

September 1, 2020

## **BREATHE EASY**

### **ELEVATOR CAB ENCLOSURE AIR FLOW EVALUATION, INFECTIOUS CONTROL TECHNIQUES AND THE EVALUATION OF AIR TREATMENT SYSTEMS – RESEARCH ARTICLE AND CONCLUSIONARY SUPPORTIVE EVIDENCE FOR STERILYFT ELEVATOR AIR STERILIZATION SYSTEM**

#### **1.1 BACKGROUND**

In late 2019 and continuing into 2020 to the present, the world's focus has been primarily on the development and continued threat of SARS-CoV-2 (Corona virus) and the illness COVID-19 created from its human infection. As with any new (or novel as it is often referred to) virus, the information on where it had originated from, how it developed into the virus we see today, and exactly how the transmission occurs from human to human, are all continuously reported, studied, revised and reiterated to what seems to be a weekly or daily change in how to battle infection. As a response, we have extensively researched the areas most pertinent, as we have understood them, to developed Sterilyft as a means to help with the elimination of SARS-CoV-2, a means to mitigate risks specifically directed to a focused high risk environment and as a permanent preventative system for the reduction of infection risks in the future with regard to viral or bacterial threat. Following is a complete dissertation on our research, development and strategically designed intent of Sterilyft, and its comparative supportive documentation as to its effectiveness and argumentative research as to the ineffectiveness of alternate methods.

#### **2.1 INFECTIOUS RISK - OVERVIEW**

We focus primarily on the main risk area of a building, as directed to by mainstream media reporting on expert opinion, the elevator. Elevators pose a unique and highly concentrated risk with respect to infection for various reasons in comparison to other areas of public access. Specifically, as the elevator is the most highly and frequently utilized space in a building, the elevator is also the most poorly ventilated.

For argument's sake, if a given building has 10 office occupancy floors with 10 offices on each floor and 10 employees for each office, the frequented occupancy rate for each office would be a factor of 10 persons per office, per day (assuming no interoffice travel) yet, the elevator's frequented occupancy rate would be 4000 persons per day assuming each tenant travels once to their office at the beginning of the day, once from their office at the end of the day and same once in and out for lunch or a break during the day. This would translate to this particular building's risk factor being 400 times more at risk in their use of the elevator, as compared to their office. Now, as 400 times the risk is substantial, translate this to a 30, 40 or 70 story building, layouts that can accept capacities of tenants in the hundreds, and factor in the added use of the elevator by inter-day traffic including visitors, additional travel during the day by tenants, deliveries, building staff use, etc. The risk is undoubtedly incalculable. With all in consideration, the unique difficulties and disadvantages to risk mitigation is further crippled for an elevator as the continual disinfection is improbable and impractical as to the continuous nature of use of the elevator by passengers, making removal from service for proper cleaning near impossible, the fact that the constant re-introduction of new possible infectious risks is added each time a passenger enters the elevator; and the fact that there is virtually little or no ventilation in an elevator to either remove contaminants or to introduce clean, fresh, contaminant free air.

### **2.1.1 INTRODUCTION OF CONTAMINANTS**

We first look at how the infectious contaminants enter and infect the elevator. In a July 9<sup>th</sup>, 2020 scientific brief by *World Health Organization (WHO) on the Transmission of SARS-CoV-2: implications for infection prevention precautions* (<https://www.wgo.int/news-room/commentaries/detail/transmission-of-sars-cov-2-implications-for-infection-prevention-precautions>), scientists examine and report on “possible modes of transmission for SARS-CoV-2, including contact, droplet, airborne, fomite, fecal-oral, blood borne, mother-to-child, and animal-to-human transmission” as an in depth focus study to determine the vehicles for transmission, as well as the categorization of risk associated with each vehicle. Their findings, and evaluations, formulate to prescribe specific key points on the specific risk factors, the preventative measure to mitigate the risks. For the purposes of our design development, we will focus on their findings of the specific vehicles of transmission in order to relate to the elevator. The brief states that transmission can occur via “*direct, indirect, or close contact with infected people through infected secretions such as saliva and respiratory secretions or their respirator droplets, which are expelled when an infected person coughs, sneezes, talks or sings.*” With this being the case, this particular risk is the reasoning behind WHO and CDC recommendations of social distancing, quarantining and the use of face coverings to help mitigate the risks. In particular to elevators though, social distancing is rarely possible, quarantining only works by voluntary means after a person is aware of infection (posing risk prior to quarantine) and face masks only work if properly fitted to the face and with proper rating.

### **2.2.2 DROPLET TRANSMISSION**

Droplet transmission (as described in the WHO brief above) as “respiratory droplets >5-10 microns in diameter” and “droplets ≤5 microns” called “droplet nuclei or aerosols.” In a May 31, 2012 *PMC published study (PMCID: PMC4676262) titled Quantity and Size Distribution of Cough-Generated Aerosol Particles Produced by Influenza Patients During and After Illness* (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4676262/>), scientific research is done on the quantity of particles per cough when multiple subjects are studied during and after illness. The significance here shows not only the volume when healthy but, a staggeringly increased volume with a comparatively respiratory illness. Study discussion shows generation of particles at a high of 516,800 particles per cough during illness, and 362,700 particles per cough when recovered. This of course is compounded higher, with a wider range of sized particles for a sneeze and conversely for talking (or singing).

### **2.2.3 AIRBORNE TRANSMISSION**

Along with the momentary risk made possible by the respiratory droplets above, the “droplet nuclei” can remain a risk long after. “Droplet nuclei (aerosols) remain infectious when suspended in air over long distances and time.” Studies mentioned in the WHO brief state “studies found SARS-CoV-2 virus RNA in air samples within aerosols for up to 3 hours in one study and 16 hours in another, reference being given to a published article in the English Journal of Medicine 2020:382:1564-7. This translates to a risk that is immediate, being generated by hundreds of thousands of possibly infectious droplets within a simple cough, followed by up to 3 to 12 hours of risk from droplet nuclei suspended within the air.

### **2.2.4 FOMITES**

Fomite transmission basically translates to “respiratory secretions or droplets” that “contaminate surfaces or object.” These range in duration of “hours to days” of infectious possibility, depending on “ambient environment” conditions and types of surfaces as to the longevity of the viral infectious risk. These settled viral carriers can either contaminate by touching the infected surface and then touching one’s mouth, nose or eyes. Further, if a draft of air disturbs the settled matter, the infectious material can again become an airborne risk until being inhaled or settle again to a surface.

#### **2.2.4 VERIFICATION OF TRANSMISSION**

With respect to the above, example was found and made of specific transmission of SARS-CoV-2 with regard to an elevator. **Centers for Disease Control and Prevention (CDC)** published a research letter (**Volume 26, Number 9 – September 2020**) titled **“Large SARS-CoV-2 Outbreak Caused by Asymptomatic Traveler, China” (ISSN:1080-6059)** to where case study and contact tracing of a specific outbreak in late 2019. The trace study shows a single person returning from a trip out of country and riding the elevator to their apartment to quarantine transitioning into infection of another tenant (patient B1.1) from another floor that rode the elevator at a different time than the initial person (patient A0). From then the spread went to family that visited the patient B1.1 and spiraled into over 70 people that had been infected by the single elevator use of the initial patient A0. Viral genome sequencing of various samples from various subjects within the 70 infected persons showed that all viral infection stemmed from patient A0.

#### **3.1 ELEVATOR VENTILATION AND CIRCULATION**

As mentioned in our introduction background, current design of an elevator cab (within the Code compliant guidelines of ASME ANSI A17 regulation), under Section 2.14.2.3 (Ventilation), no determinable circulation of air is referenced for a typical passenger elevator or any HVAC requirements stated. The only means of any ventilation consists of Natural Ventilation (2.14.2.3.1) consisting of variable dimension restricted openings below 12” from floor or above 72” from floor [Section 2.14.2.3.1(a)], small enough to reject a ball 1” in diameter if below 12” and 2” in diameter if above 72” [Section 2.14.2.3.1(b)] as well as states means to guard the openings from pass through to the shaftway. Further, natural ventilation is specified to equal a total of 3.5% of cab floor area [Section 2.14.2.3.1(e)], equally divided between the bottom and top of the enclosure. The opening area is allowed to include the opening of the space created around the car door [Section 2.14.2.3.1(f)] and the opening for a fan in the ceiling [Section 2.14.2.3.1(g)]. For typical passenger elevators however, a fan (or forced ventilation) is not required. Section 2.14.2.3.3 states that forced ventilation is only required “on observation elevators with glass walls exposed to direct sunlight” and specifies a minimum of one exchange per minute of air volume [Section 2.14.2.3.3(a)] and an auxiliary power source for one hour of emergency use [Section 2.14.2.3.3(b)]. As this concludes the Code mandated ventilation requirements on an elevator, the only proactive solutions to air handling lies within specifications for elevator work put together for an elevator contractor to adhere to with specific requirements that would be above and beyond Code requirements. Albeit, the typical ventilation scenario for an elevator would be the Code required 3.5% natural ventilation along with a mechanical exhaust fan, roof mounted, with an approximate 375 CFM rating. On occasion, or in the case of a larger elevator, the exhaust fan may be in CFM rating up to 750 to 950 CFM or, multiple 365 CFM fans used. In each case, the exhaust fan is intended to draw air from within the elevator in to the shaft, utilizing inflow of air from the natural vents or, when car doors are open, the floors the elevator serves. One overlooked issue however, is that when the elevator is in motion, the pressure of the air on the enclosure during travel (increased by speed of the respective car and by stack effect in the shaftway) creates a driven static pressure on the fan itself. As the car travels, and / or stack effect drafts on to the enclosure, the movement of that air tends to decrease, negate or in some cases reverse the flow of air being exerted by the exhaust fan. Further, with regard to stack effect, the process of opening and closing doors at each landing for ventilation can at times be counterproductive. For instance, stack effect creates a flow into and out of the shaftway into or out of the general building spaces in a shaftway. If the intent is to have air flow out of the elevator where the stack effect is flowing into the shaft, the effect would be opposite the intent. Same would be true in the opposite scenario. In addition, stack effect is by design a shaftway to landing area flow which tends to bypass the enclosure as an “air curtain” type effect is created from flow of air in between the doors of the elevator ventilating the shaftway and landing air only. Being that the cab enclosure is typically a resistive area to the flow of air (as it does not have large capacity openings to ventilate), very little air is affected, removed or added when the doors open. In addition, for either

single shaft elevators or in high speed multiple elevator shaftways, some research for purposes of smoke infiltration created by piston effect (pressures of air moving from either side of elevator to the other in the travel of the elevator), have shown fairly extensive pressures that are created by the movement of the elevator within travel that would also place extreme static pressures to the fan (***An Analysis of the Influence of Piston Effect on Elevator Smoke Control, US Department of Commerce, April 1988 and Elevator Piston Effect and the Smoke Problem, John Klote and George Tamura, Reprinted from Fire Safety Journal Vol. 11, No 2, 1986***). All cases in point, without question, there is little or no ventilation or circulation in an elevator.

#### **4.1 ELEVATOR AIR FLOW AND CIRCULATION EVALUATIONS**

To investigate the actual air circulation with respect to ventilation, we constructed a car shell enclosure complete with natural ventilation as required by Code, a typical exhaust fan and a Sterilyft sterilization unit to evaluate conditions in normal operation of fan only and operation with use of Sterilyft. Sterilyft was designed with several purposes to address elevator ventilation and circulation as well as combat airborne viral transmission by way of droplet form and / or aerosol contamination. With these designed intents in question, we conducted several case study tests utilizing an in cab mounted continual air monitor device to record data under different scenarios with focus being on ascertaining evidence of air circulation, as well as the removal of airborne particles. In addition, Sterilyft has undergone third party testing for same air quality by Underwriter's Laboratory (UL) by contract to a recipient client for unit purchased and installed in one of their buildings. The CEC in house test was performed on the same test unit (one test sequence with typical fan only , one test sequence with Sterilyft in operation), the UL test was carried out in an occupied building with one car monitored with typical fan only and one car with Sterilyft for comparison.

##### **4.1.1 CEC IN HOUSE TESTING RESULTS**

Initial air quality sampling revealed respectively, a decreased presence of particulate matter (PM) in dimension of 2.5 microns or less between the two scenarios with Sterilyft exhibiting 55.26% less contaminants than that of exhaust fan only. Further, a 33.24% reduction of VOC (Volatile Organic Compounds). Upon testing of evacuation of introduced contaminants, over 50 minutes in 3 minute intervals showed a physical removal of PM2.5 of 28% removal by fan only as compared to 95.83% removal by Sterilyft and 22.06% reduction in VOCs as compared to 76.10% by Sterilyft. Further inspection of the interval readings showed a pattern of gradual reduction of the PM2.5 content in the Sterilyft case study indicating continual reduction of the airborne particles whereas the fan only case study showed a wavering pattern, indicating the air particles were circulating in the car prior to evacuation. This indicated that the pattern of air particle removal by fan only tends to circulate within the car prior to evacuation at a much slower rate than that of Sterilyft. From this test, we were able to determine not only the superior removal and filtration of Sterilyft but, to also illustrate both the lack of circulation in a normal elevator and the proper introduction of a defined circulation by Sterilyft.

##### **4.1.2 UL AIR MONITORING TEST**

Testing as described above was conducted by Underwriter's Laboratory (UL) on Sterilyft and a typical elevator. This third party evaluation was contracted to study the air quality in two elevators, within an occupied and operating building, with one car being evaluated for typical exhaust fan set up only and one car with Sterilyft installed. By taking the average of each reading for each car over the month long test period, UL has confirmed Sterilyft to outperform the typical exhaust fan in removal of particulate matter at a rate of 95.5% less PM10 content, 95.79% less PM2.5, 97.46% less PM1 and 89.21% less PM>.3. Further, UL has confirmed VOC presence 32.7% less in the Sterilyft car in comparison to the standard fan only car. This test served as a more extensively data collected and averaged conclusion that surpassed our in house testing conclusions and exceedingly validated our initial findings of efficiency over an extended period of time. Further to this, the UL test by showing particulate matter ranging from 10 microns to less than .3 microns, expanded our understanding of the efficiency of

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Sterilyft and proved to cover a more expanded particulate material size range to cover (and surpass) the PMC published study on airborne cough material.

#### **4.2.3 EVALUATION**

From the above testing, utilizing a combination of data, given a standard cough producing an average of 439,750 particulates ranging from >5 microns to 10 microns, A typical exhaust fan would remove approximately 123,130 particles leaving 316,620 particles and Sterilyft would remove 421,412 particles leaving 18,338 particles (based on CEC evacuation testing above). Over the one month test (by average of removal of all PM data), similar would be said of the UL test results. With respect to the differences, there would be a mitigated risk factor of approximately 30% less chance of infection from an infected cough in an elevator with a typical exhaust fan as compared to an approximate 95% less chance with Sterilyft by filtration and circulation alone. This rate would then further be decreased on the Sterilyft system with respect to the germicidal effect of the UV-C irradiation.

### **5.1 SYSTEM DESIGN INCEPTION – AIR FLOW, GERMICIDAL PROPERTY**

With the research and development of Sterilyft and contemplation of the most effective source of germicidal properties to be added to the system based on efficacy, effectiveness and applicability, we researched several types of germicidal treatments. UV-C stood as the only applicable option as to the superior efficacy of inactivation of viral or bacterial matter (99.99% effectiveness), and the lack of any harmful byproducts with specially coated fused quartz glass tubes. Other disinfection / sterilization techniques were reviewed and eliminated for lack of effectiveness, problematic interaction with system or the creation of harmful byproducts to the passengers or elevator equipment; or a combination of any or all of these reasons. At present, germicidal systems currently being offered consist of similar UV-C based systems, yet lacking in the circulation properties to evacuate particulate matter properly and adds in a dangerous or undesirable result action; and ion based systems that advertise charged particle population of the air to seek and destroy viral or bacterial contaminants.

#### **5.1.1 IMPROPER CIRCULATION**

With all previously stated issues resulting from poorly designed circulation, Sterilyft serves as the ONLY system to accomplish a proper circulation of air as suggested by CDC (*Centers for Disease Control and Prevention (CDC) Corona Virus Disease 2019 (Covid-19) Covid-19 Employer Information for Office Buildings, May 27, 2020*). With consideration to the adverse effects of stack effect, shaftway movement of the elevator and the fluctuation in evacuation of particulate matter seen in CEC's air quality testing of the system, it can be indisputably noted that a proper flow of air with sufficient movement from a fan be required to properly remove particulate matter from the elevator (which is the main source of transmission of disease infection, and is the main cause of surface contamination). Systems that include poorly rated fans (some as low as 24CFMs), systems that do not direct properly placed points of exhaust and intake (such as units with only ceiling mounted ports) all will have significantly reduced performance as compared to Sterilyft. The obvious reduction in air movement will leave particulate matter in the car (such as exhaust fan only) and ceiling mounted only intake / exhaust will fail to include lower car air into the circulation. There are also systems being offered that reverse the direction of the air flow on the typical elevator fan. The intent of these systems is to force shaftway air into the car with or without an added germicidal additive. Several issues come up for this system. First and foremost, if there is ever a smoke condition in the shaft, the system would force some into the cab. This in itself should ban these systems. Additionally, the act of only forcing the air in does nothing to remove particulate matter in the elevator which leaves any untreated viral content to either remain in the car or, be pushed out onto occupied floors possibly when the car doors open at a landing. Either way, the process is not reducing risk but transferring risk to other areas of the building.

#### **5.1.2 IONS AND OZONE**

Ozone is a very effective germicidal treatment and sometimes used for this purpose. Unfortunately though, ozone is a toxic gas to humans and, the concentration of ozone required to have an effective germicidal property would be uninhabitable and dangerous. This being said, when we looked into ion

technologies (negative ion generation and positive / negative bi-polar ion generation), we researched the ozone generation, efficacy and standards on ozone levels. Per EPA (***Ozone Generators that are Sold as Air Cleaners, updated December 23, 2019***), the EPA, in collaboration with several other Federal agencies, contradicts various products and suppliers claims of air cleaning benefits of ozone and the dangers of use of ozone gas. The report goes on to iterate the dangers of ozone gas in humans “when inhaled, ozone can damage the lungs. Ozone may also worsen chronic respiratory diseases...and compromise the ability of the body to fight respiratory infection.” “Health effects may become more damaging and recovery less certain at higher levels or from longer exposures (US EPA, 1996a, 1996b)” Ozone limitations are set by Federal regulation varying from 0.05 ppm as set by FDA, 0.10 ppm in 8 hours set by OSHA, 0,10 ppm set by NIOSH and 8 hour average outdoor concentration of 0.08 ppm set by EPA. Though ozone does have germicidal properties and is used in some instances, however, these processes are done in unoccupied areas that are very well ventilated to clear the ozone before re-occupation. EPA additionally advises that “if used in concentrations that do not exceed public health standards, ozone applied to indoor air does not effectively remove viruses, bacteria, mold or other biological pollutants.” “Ozone concentration would have to be 5 – 10 times higher than public health standards allow.” Further to this letter, EPA advised of ozone being “produced indirectly by ion generators and some other electronic air cleaners” in their letter titled (***What are ionizers and other ozone generating air cleaners?, updated July 16, 2020***) being that the actual process of creating ions by high voltage charging of particles, can not be accomplished without the production of ozone (not that dissimilar to the process of welding on a microscopic scale). A study performed by BioMed Central Ltd (BMC Microbiology) was conducted to further investigate this in their research article titled (***Bacterial action of positive and negative ions in air, published April 17, 2007***) in where bacteria was subjected to positive and negative charged ions to investigate the often “often widely reported...biocidal action.” They conducted the investigative study by introducing bacteria to each produced effect by the ion generator “(i) the action of the air ions; (ii) the action of the electronic field, and (iii) the action of ozone”. BMC encapsulated their findings as “the results of the study suggest that the bacterial action attributed to negative ions by previous researchers may have been overestimated”. The study concluded that “the principle cause of cell death amongst the bacteria studied was exposure to ozone, with electroporation playing a second role (action of the electrical field of the ion generator itself)”. This in detail, verified that the use of ions does not have any germicidal effect other than to attract to the particles and cause them to settle on surfaces and not remain airborne. Though this would keep possible infectious particles away from people’s mouths, nose or eyes; the major issue would be that the virus or bacteria would still be active. Any contact with the particles, or the re-introduction of the viral carrier back into the air (in the cab or at any floor landing, would put the same exact risk in effect as it was from the initial inception of a cough, sneeze or exhale into the air. As the EPA noted, though ion generators have a germicidal effect, the effect is attributed mainly to the ozone created by the generation of the ions. Perhaps systems like this have a place in more spacious, better ventilated areas that can be unoccupied for long periods of time however, the minimally ventilated and confined space of a heavily used and occupied elevator is a recipe for disaster.

### **5.1.3 UV-C GERMICIDAL EFFICACY**

Throughout research into anti-bacterial and germicidal treatments and technologies, only UV-C was found to be an effective and permissive solution for elevators, with exception to exposure risk need be eliminated in its entirety. In our research, we have found that scientifically proven interaction between UV-C radiation and the inactivation of cellular organisms was profound, highly efficient and rapid; which made it the most fitting choice. As per studies for the last few decades, UV-C radiation has been tested on the efficacy of bacterial, protozoan, mold / mildew and viruses in a variable of ways, means and methods. The results (specific for this cause), showed that a dosage on 10 to 20 mJ/cm<sup>2</sup> of 254nm UVC radiation has been proven to inactivate SARS family viruses to a 99.9% disinfection efficiency (***International Ultraviolet Association, Covid-19 FAQ***). As COVID-19 is caused by a SARS variation (SARS-CoV-2), the speculation is that the same would apply. In calculation of dosage, many factors have

to be put in place on a speculative dosage. The strength of the UVC light (measured in Watts output), distance from the bulb to the subject material and time of exposure to the UVC source in direct line of sight. We did extensive research into formula calculation of Sterilyft based on the physical dimensions, the UVC actual output strength, and performed measured FPM calculations to determine the specific time that a particle within the air flow will be exposed in the UVC chamber. By calculation of the radiant intensity of the lamp's output to multiple locations within the UVC chamber, selection of the farthest possible distance from the UVC bulb source, calculation as to the irradiance factor of the UVC radiation at that given point and with calculation of the maximum time that the particle will be irradiated at that farthest point, we have calculated by three different formulations the actual dosage to be no less than 27.62661 mJ/cm<sup>2</sup>. As we are in process of actual laboratory 3<sup>rd</sup> party testing for efficacy, we are in expectation to assure Sterilyft will produce the same disinfection efficiency as studies have shown at 99.9% effective. For those interested in checking calculations, our recorded and utilized factors were: .8W UV Output (***Ushio specification, G4T5 Low Pressure Mercury Germicidal lamp***), 0.06364W radiance Intensity, 0.2286m furthest distance from bulb in chamber, 121.78 uW/cm<sup>2</sup> irradiance, 270fpm recorded air speed within UVC chamber between filters, .2269 seconds travel time from filter to filter (31.115cm between filters). Shorter of formula calculations used was UV Energy equals UV Lamp Output Power (W) x Exposure Time (sec). Area equals Distance from bulb (M<sup>2</sup>) x 12.57 (Pi multiplied by 4). UV Dose equals UV Energy divided by Area (in M.sec/cm<sup>2</sup> or J/cm<sup>2</sup>). This calculation (and efficiency) will most likely be multiplied by the reflectance variation as the aluminum we've selected exhibits a reflectance rate of 40-60% to the UVC irradiance (***Air-Conditioning and Refrigeration Technology Institute Report ARTI-21CR/610-40030-01 published November 2002***).

### CONCLUSION

With all of the above in account and contemplation, the development and final outcome of Sterilyft can be said to be the most specific, effective and proven system for elevator air sterilization. By way of circulatory measures, Sterilyft is the only system that utilizes a true full cab defined circulation of air to not only remove air borne particles but, to disallow particles from being allowed to settle on surfaces. For ventilation, Sterilyft has an unmatched air movement provided by an industry leading 710 CFM. On filtration purification, Sterilyft contains CDC recommended MERV 13 double filtration which is proved by third part testing for an efficiency of PM removal ranging from almost 90% of particles less than .3 microns and over 95% of particles up to 10 microns. And as for germicidal treatment, Sterilyft is designed to have an efficiency rate of disinfection of 99.9% on SARS family of virus (to be lab tested and verified for efficacy). With all in point, it can not be disputed that Sterilyft is a valid and proven, invaluable tool ; not only in the fight against SARS-CoV-2 (and COVID-19), but for ALL current and presumably future pathogens to come. Sterilyft, undeniably, should become a consideration for every elevator in the near future as the benefits of healthier living and working will serve to not only help maintain the health of the general public but also to increase the economical successes of individuals, individual companies and the cities, states and countries we all live in as the typical lost work due to illness will be greatly diminished. The return to work, school and life as we knew it in process now will undoubtedly lead to further infections, spikes in confirmed cases and further shutdowns, regulations and closings. The future of public need for the safe and healthy atmospheres to live, work and play will soon become a necessity to fill by developers, owners, managers of properties and service providers. Based on the explosion of security provisions integrated into our daily lives as a technological upgrade to existing building services, the risk mitigation industry (in the form of HVAC technologies, no contact building operations and disinfection methodology) will soon become an even more demanded expectation. The time to invest, the time to combat infection and the time to address the most risk factored space in any and all multiple level dwellings is now.



# Transmission of SARS-CoV-2: implications for infection prevention precautions

## Scientific Brief

9 July 2020

This document is an update to the scientific brief published on 29 March 2020 entitled “Modes of transmission of virus causing COVID-19: implications for infection prevention and control (IPC) precaution recommendations” and includes new scientific evidence available on transmission of SARS-CoV-2, the virus that causes COVID-19.

## Overview

This scientific brief provides an overview of the modes of transmission of SARS-CoV-2, what is known about when infected people transmit the virus, and the implications for infection prevention and control precautions within and outside health facilities. This scientific brief is not a systematic review. Rather, it reflects the consolidation of rapid reviews of publications in peer-reviewed journals and of non-peer-reviewed manuscripts on pre-print servers, undertaken by WHO and partners. Preprint findings should be interpreted with caution in the absence of peer review. This brief is also informed by several discussions via teleconferences with the WHO Health Emergencies Programme ad hoc Experts Advisory Panel for IPC Preparedness, Readiness and Response to COVID-19, the WHO ad hoc COVID-19 IPC Guidance Development Group (COVID-19 IPC GDG), and by review of external experts with relevant technical backgrounds.

The overarching aim of the global Strategic Preparedness and Response Plan for COVID-19(1) is to control COVID-19 by suppressing transmission of the virus and preventing associated illness and death. Current evidence suggests that SARS-CoV-2, the virus that causes COVID-19, is predominantly spread from person-to-person. Understanding how, when and in what types of settings SARS-CoV-2 spreads is critical to develop effective public health and infection prevention and control measures to break chains of transmission.

## Modes of transmission

This section briefly describes possible modes of transmission for SARS-CoV-2, including contact, droplet, airborne, fomite, fecal-oral, bloodborne, mother-to-child, and animal-to-human transmission. Infection with SARS-CoV-2 primarily causes respiratory illness ranging from mild disease to severe disease and death, and some people infected with the virus never develop symptoms.

## Contact and droplet transmission

Transmission of SARS-CoV-2 can occur through direct, indirect, or close contact with infected people through infected secretions such as saliva and respiratory secretions or their respiratory droplets, which are expelled when an infected person coughs, sneezes, talks or sings.(2-10) Respiratory droplets are  $>5\text{-}10\ \mu\text{m}$  in diameter whereas droplets  $\leq 5\ \mu\text{m}$  in diameter are referred to as droplet nuclei or

aerosols.(11) Respiratory droplet transmission can occur when a person is in close contact (within 1 metre) with an infected person who has respiratory symptoms (e.g. coughing or sneezing) or who is talking or singing; in these circumstances, respiratory droplets that include virus can reach the mouth, nose or eyes of a susceptible person and can result in infection. Indirect contact transmission involving contact of a susceptible host with a contaminated object or surface (fomite transmission) may also be possible (see below).

## Airborne transmission

Airborne transmission is defined as the spread of an infectious agent caused by the dissemination of droplet nuclei (aerosols) that remain infectious when suspended in air over long distances and time.(11) Airborne transmission of SARS-CoV-2 can occur during medical procedures that generate aerosols (“aerosol generating procedures”).(12) WHO, together with the scientific community, has been actively discussing and evaluating whether SARS-CoV-2 may also spread through aerosols in the absence of aerosol generating procedures, particularly in indoor settings with poor ventilation.

The physics of exhaled air and flow physics have generated hypotheses about possible mechanisms of SARS-CoV-2 transmission through aerosols.(13-16) These theories suggest that 1) a number of respiratory droplets generate microscopic aerosols (<5 µm) by evaporating, and 2) normal breathing and talking results in exhaled aerosols. Thus, a susceptible person could inhale aerosols, and could become infected if the aerosols contain the virus in sufficient quantity to cause infection within the recipient. However, the proportion of exhaled droplet nuclei or of respiratory droplets that evaporate to generate aerosols, and the infectious dose of viable SARS-CoV-2 required to cause infection in another person are not known, but it has been studied for other respiratory viruses.(17)

One experimental study quantified the amount of droplets of various sizes that remain airborne during normal speech. However, the authors acknowledge that this relies on the independent action hypothesis, which has not been validated for humans and SARS-CoV-2.(18) Another recent experimental model found that healthy individuals can produce aerosols through coughing and talking (19), and another model suggested high variability between individuals in terms of particle emission rates during speech, with increased rates correlated with increased amplitude of vocalization.(20) To date, transmission of SARS-CoV-2 by this type of aerosol route has not been demonstrated; much more research is needed given the possible implications of such route of transmission.

Experimental studies have generated aerosols of infectious samples using high-powered jet nebulizers under controlled laboratory conditions. These studies found SARS-CoV-2 virus RNA in air samples within aerosols for up to 3 hours in one study (21) and 16 hours in another, which also found viable replication-competent virus.(22) These findings were from experimentally induced aerosols that do not reflect normal human cough conditions.

Some studies conducted in health care settings where symptomatic COVID-19 patients were cared for, but where aerosol generating procedures were not performed, reported the presence of SARS-CoV-2 RNA in air samples (23-28), while other similar investigations in both health care and non-health care settings found no presence of SARS-CoV-2 RNA; no studies have found viable virus in air samples.(29-36) Within samples where SARS-CoV-2 RNA was found, the quantity of RNA detected was in extremely low numbers in large volumes of air and one study that found SARS-CoV-2 RNA in air samples reported inability to identify viable virus. (25) The detection of RNA using reverse transcription polymerase chain reaction (RT-PCR)-based assays is not necessarily indicative of replication- and infection-competent (viable) virus that could be transmissible and capable of causing infection.(37)

Recent clinical reports of health workers exposed to COVID-19 index cases, not in the presence of aerosol-generating procedures, found no nosocomial transmission when contact and droplet precautions were appropriately used, including the wearing of medical masks as a component of the personal protective equipment (PPE). (38, 39) These observations suggest that aerosol transmission did not occur in this context. Further studies are needed to determine whether it is possible to detect viable SARS-CoV-2 in air samples from settings where no procedures that generate aerosols are performed and what role aerosols might play in transmission.

Outside of medical facilities, some outbreak reports related to indoor crowded spaces (40) have suggested the possibility of aerosol transmission, combined with droplet transmission, for example, during choir practice (7), in restaurants (41) or in fitness classes.(42) In these events, short-range aerosol transmission, particularly in specific indoor locations, such as crowded and inadequately ventilated spaces over a prolonged period of time with infected persons cannot be ruled out. However, the detailed investigations of these clusters suggest that droplet and fomite transmission could also explain human-to-human transmission within these clusters. Further, the close

contact environments of these clusters may have facilitated transmission from a small number of cases to many other people (e.g., superspreading event), especially if hand hygiene was not performed and masks were not used when physical distancing was not maintained.<sup>(43)</sup>

## Fomite transmission

Respiratory secretions or droplets expelled by infected individuals can contaminate surfaces and objects, creating fomites (contaminated surfaces). Viable SARS-CoV-2 virus and/or RNA detected by RT-PCR can be found on those surfaces for periods ranging from hours to days, depending on the ambient environment (including temperature and humidity) and the type of surface, in particular at high concentration in health care facilities where COVID-19 patients were being treated.<sup>(21, 23, 24, 26, 28, 31-33, 36, 44, 45)</sup> Therefore, transmission may also occur indirectly through touching surfaces in the immediate environment or objects contaminated with virus from an infected person (e.g. stethoscope or thermometer), followed by touching the mouth, nose, or eyes.

Despite consistent evidence as to SARS-CoV-2 contamination of surfaces and the survival of the virus on certain surfaces, there are no specific reports which have directly demonstrated fomite transmission. People who come into contact with potentially infectious surfaces often also have close contact with the infectious person, making the distinction between respiratory droplet and fomite transmission difficult to discern. However, fomite transmission is considered a likely mode of transmission for SARS-CoV-2, given consistent findings about environmental contamination in the vicinity of infected cases and the fact that other coronaviruses and respiratory viruses can transmit this way.

## Other modes of transmission

SARS-CoV-2 RNA has also been detected in other biological samples, including the urine and feces of some patients.<sup>(46-50)</sup> One study found viable SARS-CoV-2 in the urine of one patient.<sup>(51)</sup> Three studies have cultured SARS-CoV-2 from stool specimens.<sup>(48, 52, 53)</sup> To date, however, there have been no published reports of transmission of SARS-CoV-2 through feces or urine.

Some studies have reported detection of SARS-CoV-2 RNA, in either plasma or serum, and the virus can replicate in blood cells. However, the role of bloodborne transmission remains uncertain; and low viral titers in plasma and serum suggest that the risk of transmission through this route may be low.<sup>(48, 54)</sup> Currently, there is no evidence for intrauterine transmission of SARS-CoV-2 from infected pregnant women to their fetuses, although data remain limited. WHO has recently published a scientific brief on breastfeeding and COVID-19.<sup>(55)</sup> This brief explains that viral RNA fragments have been found by RT-PCR testing in a few breast milk samples of mothers infected with SARS-CoV-2, but studies investigating whether the virus could be isolated, have found no viable virus. Transmission of SARS-CoV-2 from mother to child would necessitate replicative and infectious virus in breast milk being able to reach target sites in the infant and also to overcome infant defense systems. WHO recommends that mothers with suspected or confirmed COVID-19 should be encouraged to initiate or continue to breastfeed.<sup>(55)</sup>

Evidence to date shows that SARS-CoV-2 is most closely related to known betacoronaviruses in bats; the role of an intermediate host in facilitating transmission in the earliest known human cases remains unclear.<sup>(56, 57)</sup> In addition to investigations on the possible intermediate host(s) of SARS-CoV-2, there are also a number of studies underway to better understand susceptibility of SARS-CoV-2 in different animal species. Current evidence suggests that humans infected with SARS-CoV-2 can infect other mammals, including dogs<sup>(58)</sup>, cats<sup>(59)</sup>, and farmed mink.<sup>(60)</sup> However, it remains unclear if these infected mammals pose a significant risk for transmission to humans.

## When do people infected with SARS-CoV-2 infect others?

Knowing when an infected person can spread SARS-CoV-2 is just as important as how the virus spreads (described above). WHO has recently published a scientific brief outlining what is known about when a person may be able to spread, based on the severity of their illness.<sup>(61)</sup>

In brief, evidence suggests that SARS-CoV-2 RNA can be detected in people 1-3 days before their symptom onset, with the highest viral loads, as measured by RT-PCR, observed around the day of symptom onset, followed by a gradual decline over time.(47, 62-65) The duration of RT-PCR positivity generally appears to be 1-2 weeks for asymptomatic persons, and up to 3 weeks or more for patients with mild to moderate disease.(62, 65-68) In patients with severe COVID-19 disease, it can be much longer.(47)

Detection of viral RNA does not necessarily mean that a person is infectious and able to transmit the virus to another person. Studies using viral culture of patient samples to assess the presence of infectious SARS-CoV-2 are currently limited. (61) Briefly, viable virus has been isolated from an asymptomatic case,(69) from patients with mild to moderate disease up to 8-9 days after symptom onset, and for longer from severely ill patients.(61) Full details about the duration of viral shedding can be found in the WHO guidance document on “Criteria for releasing COVID-19 patients from isolation”. (61) Additional studies are needed to determine the duration of viable virus shedding among infected patients.

## **SARS-CoV-2 infected persons who have symptoms can infect others primarily through droplets and close contact**

SARS-CoV-2 transmission appears to mainly be spread via droplets and close contact with infected symptomatic cases. In an analysis of 75,465 COVID-19 cases in China, 78-85% of clusters occurred within household settings, suggesting that transmission occurs during close and prolonged contact.(6) A study of the first patients in the Republic of Korea showed that 9 of 13 secondary cases occurred among household contacts.(70) Outside of the household setting, those who had close physical contact, shared meals, or were in enclosed spaces for approximately one hour or more with symptomatic cases, such as in places of worship, gyms, or the workplace, were also at increased risk of infection.(7, 42, 71, 72) Other reports have supported this with similar findings of secondary transmission within families in other countries.(73, 74)

## **SARS-CoV-2 infected persons without symptoms can also infect others**

Early data from China suggested that people without symptoms could infect others.(6) To better understand the role of transmission from infected people without symptoms, it is important to distinguish between transmission from people who are infected who never develop symptoms(75) (asymptomatic transmission) and transmission from people who are infected but have not developed symptoms yet (pre-symptomatic transmission). This distinction is important when developing public health strategies to control transmission.

The extent of truly asymptomatic infection in the community remains unknown. The proportion of people whose infection is asymptomatic likely varies with age due to the increasing prevalence of underlying conditions in older age groups (and thus increasing risk of developing severe disease with increasing age), and studies that show that children are less likely to show clinical symptoms compared to adults.(76) Early studies from the United States (77) and China (78) reported that many cases were asymptomatic, based on the lack of symptoms at the time of testing; however, 75-100% of these people later developed symptoms. A recent systematic review estimated that the proportion of truly asymptomatic cases ranges from 6% to 41%, with a pooled estimate of 16% (12%–20%).(79) However, all studies included in this systematic review have important limitations.(79) For example, some studies did not clearly describe how they followed up with persons who were asymptomatic at the time of testing to ascertain if they ever developed symptoms, and others defined “asymptomatic” very narrowly as persons who never developed fever or respiratory symptoms, rather than as those who did not develop any symptoms at all.(76, 80) A recent study from China that clearly and appropriately defined asymptomatic infections suggests that the proportion of infected people who never developed symptoms was 23%.(81)

Multiple studies have shown that people infect others before they themselves became ill, (10, 42, 69, 82, 83) which is supported by available viral shedding data (see above). One study of transmission in Singapore reported that 6.4% of secondary cases resulted from pre-symptomatic transmission.(73) One modelling study, that inferred the date of transmission based on the estimated serial interval and incubation period, estimated that up to 44% (25-69%) of transmission may have occurred just before symptoms appeared.(62) It remains unclear why the magnitude of estimates from modelling studies differs from available empirical data.

Transmission from infected people without symptoms is difficult to study. However, information can be gathered from detailed contact tracing efforts, as well as epidemiologic investigations among cases and contacts. Information from contact tracing efforts reported to WHO by Member States, available transmission studies and a recent pre-print systematic reviews suggests that individuals without symptoms are less likely to transmit the virus than those who develop symptoms. (10, 81, 84, 85) Four individual studies from Brunei, Guangzhou China, Taiwan China and the Republic of Korea found that between 0% and 2.2% of people with asymptomatic infection infected anyone else, compared to 0.8%-15.4% of people with symptoms. (10, 72, 86, 87)

## Remaining questions related to transmission

Many unanswered questions about transmission of SARS-CoV-2 remain, and research seeking to answer those questions is ongoing and is encouraged. Current evidence suggests that SARS-CoV-2 is primarily transmitted between people via respiratory droplets and contact routes – although aerosolization in medical settings where aerosol generating procedures are used is also another possible mode of transmission - and that transmission of COVID-19 is occurring from people who are pre-symptomatic or symptomatic to others in close contact (direct physical or face-to-face contact with a probable or confirmed case within one meter and for prolonged periods of time), when not wearing appropriate PPE. Transmission can also occur from people who are infected and remain asymptomatic, but the extent to which this occurs is not fully understood and requires further research as an urgent priority. The role and extent of airborne transmission outside of health care facilities, and in particular in close settings with poor ventilation, also requires further study.

As research continues, we expect to gain a better understanding about the relative importance of different transmission routes, including through droplets, physical contact and fomites; the role of airborne transmission in the absence of aerosol generating procedures; the dose of virus required for transmission to occur, the characteristics of people and situations that facilitate superspreading events such as those observed in various closed settings, the proportion of infected people who remain asymptomatic throughout the course of their infection; the proportion of truly asymptomatic persons who transmit the virus to others; the specific factors that drive asymptomatic and pre-symptomatic transmission; and the proportion of all infections that are transmitted from asymptomatic and pre-symptomatic individuals.

## Implications for preventing transmission

Understanding how, when and in which settings infected people transmit the virus is important for developing and implementing control measures to break chains of transmission. While there is a great deal of scientific studies becoming available, all studies that investigate transmission should be interpreted bearing in mind the context and settings in which they took place, including the infection prevention interventions in place, the rigor of the methods used in the investigation and the limitations and biases of the study designs.

It is clear from available evidence and experience, that limiting close contact between infected people and others is central to breaking chains of transmission of the virus causing COVID-19. The prevention of transmission is best achieved by identifying suspect cases as quickly as possible, testing, and isolating infectious cases. (88, 89) In addition, it is critical to identify all close contacts of infected people (88) so that they can be quarantined (90) to limit onward spread and break chains of transmission. By quarantining close contacts, potential secondary cases will already be separated from others before they develop symptoms or they start shedding virus if they are infected, thus preventing the opportunity for further onward spread. The incubation period of COVID-19, which is the time between exposure to the virus and symptom onset, is on average 5-6 days, but can be as long as 14 days. (82, 91) Thus, quarantine should be in place for 14 days from the last exposure to a confirmed case. If it is not possible for a contact to quarantine in a separate living space, self-quarantine for 14 days at home is required; those in self-quarantine may require support during the use of physical distancing measures to prevent the spread of the virus.

Given that infected people without symptoms can transmit the virus, it is also prudent to encourage the use of fabric face masks in public places where there is community transmission[1] and where other prevention measures, such as physical distancing, are not possible. (12) Fabric masks, if made and worn properly, can serve as a barrier to droplets expelled from the wearer into the air and environment. (12) However, masks must be used as part of a comprehensive package of preventive measures, which includes frequent

hand hygiene, physical distancing when possible, respiratory etiquette, environmental cleaning and disinfection. Recommended precautions also include avoiding indoor crowded gatherings as much as possible, in particular when physical distancing is not feasible, and ensuring good environmental ventilation in any closed setting. (92, 93)

Within health care facilities, including long term care facilities, based on the evidence and the advice by the COVID-19 IPC GDG, WHO continues to recommend droplet and contact precautions when caring for COVID-19 patients and airborne precautions when and where aerosol generating procedures are performed. WHO also recommends standard or transmission-based precautions for other patients using an approach guided by risk assessment. (94) These recommendations are consistent with other national and international guidelines, including those developed by the European Society of Intensive Care Medicine and Society of Critical Care Medicine (95) and by the Infectious Diseases Society of America. (96)

Furthermore, in areas with COVID-19 community transmission, WHO advises that health workers and caregivers working in clinical areas should continuously wear a medical mask during all routine activities throughout the entire shift. (12) In settings where aerosol-generating procedures are performed, they should wear an N95, FFP2 or FFP3 respirator. Other countries and organizations, including the United States Centers for Diseases Control and Prevention (97) and the European Centre for Disease Prevention and Control (98) recommend airborne precautions for any situation involving the care of COVID-19 patients. However, they also consider the use of medical masks as an acceptable option in case of shortages of respirators.

WHO guidance also emphasizes the importance of administrative and engineering controls in health care settings, as well as rational and appropriate use of all PPE (99) and training for staff on these recommendations (IPC for Novel Coronavirus [COVID-19] Course. Geneva; World Health Organization 2020, available at (<https://openwho.org/courses/COVID-19-IPC-EN>)). WHO has also provided guidance on safe workplaces. (92)

## Key points of the brief

### Main findings

- Understanding how, when and in what types of settings SARS-CoV-2 spreads between people is critical to develop effective public health and infection prevention measures to break chains of transmission.
- Current evidence suggests that transmission of SARS-CoV-2 occurs primarily between people through direct, indirect, or close contact with infected people through infected secretions such as saliva and respiratory secretions, or through their respiratory droplets, which are expelled when an infected person coughs, sneezes, talks or sings.
- Airborne transmission of the virus can occur in health care settings where specific medical procedures, called aerosol generating procedures, generate very small droplets called aerosols. Some outbreak reports related to indoor crowded spaces have suggested the possibility of aerosol transmission, combined with droplet transmission, for example, during choir practice, in restaurants or in fitness classes.
- Respiratory droplets from infected individuals can also land on objects, creating fomites (contaminated surfaces). As environmental contamination has been documented by many reports, it is likely that people can also be infected by touching these surfaces and touching their eyes, nose or mouth before cleaning their hands.
- Based on what we currently know, transmission of COVID-19 is primarily occurring from people when they have symptoms, and can also occur just before they develop symptoms, when they are in close proximity to others for prolonged periods of time. While someone who never develops symptoms can also pass the virus to others, it is still not clear to what extent this occurs and more research is needed in this area.
- Urgent high-quality research is needed to elucidate the relative importance of different transmission routes; the role of airborne transmission in the absence of aerosol generating procedures; the dose of virus required for transmission to occur; the settings and risk factors for superspreading events; and the extent of asymptomatic and pre-symptomatic transmission.

## How to prevent transmission

The overarching aim of the Strategic Preparedness and Response Plan for COVID-19(1) is to control COVID-19 by suppressing transmission of the virus and preventing associated illness and death. To the best of our understanding, the virus is primarily spread through contact and respiratory droplets. Under some circumstances airborne transmission may occur (such as when aerosol

generating procedures are conducted in health care settings or potentially, in indoor crowded poorly ventilated settings elsewhere). More studies are urgently needed to investigate such instances and assess their actual significance for transmission of COVID-19.

To prevent transmission, WHO recommends a comprehensive set of measures including:

- Identify suspect cases as quickly as possible, test, and isolate all cases (infected people) in appropriate facilities;
- Identify and quarantine all close contacts of infected people and test those who develop symptoms so that they can be isolated if they are infected and require care;
- Use fabric masks in specific situations, for example, in public places where there is community transmission and where other prevention measures, such as physical distancing, are not possible;
- Use of contact and droplet precautions by health workers caring for suspected and confirmed COVID-19 patients, and use of airborne precautions when aerosol generating procedures are performed;
- Continuous use of a medical mask by health workers and caregivers working in all clinical areas, during all routine activities throughout the entire shift;
- At all times, practice frequent hand hygiene, physical distancing from others when possible, and respiratory etiquette; avoid crowded places, close-contact settings and confined and enclosed spaces with poor ventilation; wear fabric masks when in closed, overcrowded spaces to protect others; and ensure good environmental ventilation in all closed settings and appropriate environmental cleaning and disinfection.

WHO carefully monitors the emerging evidence about this critical topic and will update this scientific brief as more information becomes available.

[1] Defined by WHO as “experiencing larger outbreaks of local transmission defined through an assessment of factors including, but not limited to: large numbers of cases not linkable to transmission chains; large numbers of cases from sentinel surveillance; and/or multiple unrelated clusters in several areas of the country/territory/area” (<https://www.who.int/publications-detail/global-surveillance-for-covid-19-caused-by-human-infection-with-covid-19-virus-interim-guidance>)

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PMCID: PMC4676262

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## Quantity and Size Distribution of Cough-Generated Aerosol Particles Produced by Influenza Patients During and After Illness

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### Abstract

The question of whether influenza is transmitted to a significant degree by aerosols remains controversial, in part, because little is known about the quantity and size of potentially infectious airborne particles produced by people with influenza. In this study, the size and amount of aerosol particles produced by nine subjects during coughing were measured while they had influenza and after they had recovered, using a laser aerosol particle spectrometer with a size range of 0.35 to 10  $\mu\text{m}$ . Individuals with influenza produce a significantly greater volume of aerosol when ill compared with afterward ( $p = 0.0143$ ). When the patients had influenza, their average cough aerosol volume was 38.3 picoliters (pL) of particles per cough (SD 43.7); after patients recovered, the average volume was 26.4 pL per cough (SD 45.6). The number of particles produced per cough was also higher when subjects had influenza (average 75,400 particles/cough, SD 97,300) compared with afterward (average 52,200, SD 98,600), although the difference did not reach statistical significance ( $p = 0.1042$ ). The average number of particles expelled per cough varied widely from patient to patient, ranging from 900 to 302,200 particles/cough while subjects had influenza and 1100 to 308,600 particles/cough after recovery. When the subjects had influenza, an average of 63% of each subject's cough aerosol particle volume in the detection range was in the respirable size fraction (SD 22%), indicating that these particles could reach the alveolar region of the lungs if inhaled by another person. This enhancement in aerosol generation during illness may play an important role in influenza transmission and suggests that a better understanding of this phenomenon is needed to predict the production and dissemination of influenza-laden aerosols by people infected with this virus.

[Supplementary materials are available for this article. Go to the publisher's online edition of Journal of Occupational and Environmental Hygiene for the following free supplemental resources: a PDF file of demographic information, influenza test results, and volume and peak flow rate during each cough and a PDF file containing number and size of aerosol particles produced.]

**Keywords:** airborne particles, airborne transmission, disease transmission, human, respiratory infections

### INTRODUCTION

Influenza is a highly contagious respiratory disease that is of great concern to the public health community because of the annual burden from the illness and the possibility of a severe pandemic with high rates of morbidity and mortality. Influenza is thought to be spread by direct person-to-person transfer of infectious secretions, by indirect transfer of secretions via fomites, by large ballistic spray droplets that are produced during coughing or sneezing and splash onto mucous membranes, and by the inhalation and deposition of airborne virus-laden particles.<sup>(1)</sup> However, the relative importance of these routes of transmission is unclear and appears to depend on multiple factors, including temperature and humidity, the virus concentration in respiratory secretions, the distance from the source to the recipient, and the genotype of the virus.<sup>(2,3)</sup>

The possibility of airborne transmission in particular is hotly debated, with some studies suggesting that this pathway plays a critical role in the spread of influenza<sup>(1,4)</sup> while others concluding that it does not.<sup>(5)</sup> This controversy had a direct impact on public health policy during the 2009 novel H1N1 influenza pandemic; some health care institutes such as the U.S. Institute of Medicine recommended that health care workers in close contact with influenza patients wear respirators to prevent possible airborne transmission,<sup>(6)</sup> while other organizations such as the World Health Organization did not.<sup>(7)</sup>

Disagreement over the potential for the airborne transmission of influenza occurs in part because the quantity and size of potentially infectious aerosols produced by people with influenza have not been well characterized. Several studies have examined aerosol particle generation by people during coughing, speaking, sneezing, and breathing (reviewed by Gralton<sup>(8)</sup> and Nicas<sup>(9)</sup>). Most of these studies used healthy subjects, but a few have looked at aerosol production by individuals with respiratory viral infections, including influenza<sup>(10–12)</sup> and rhinovirus.<sup>(10,13)</sup> All of these studies have consistently found a tremendous variation in the numbers of aerosol particles expelled by individuals. Edwards et al.<sup>(14)</sup> tested 11 healthy subjects and reported that the concentration of particles in their exhaled breath varied from 1 particle/liter to over 10,000 particles/liter. Fabian et al.<sup>(11)</sup> tested 10 patients with influenza and found that the concentration of particles exhaled by these subjects ranged from 67 to 8500 particles/liter of air; similar results were later reported for patients with rhinovirus infections.<sup>(13)</sup> Almstrand et al.<sup>(15)</sup> also found that the exhaled particle concentrations varied considerably from subject to subject and with the depth of breathing, but that the exhaled concentrations for each subject were fairly consistent during repeated testing for each respiratory maneuver studied.

Although these papers and others have added considerably to the body of knowledge about respiratory aerosol production, one important question that remains unanswered is how respiratory infections affect aerosol particle production by patients. Because respiratory infections generally increase airway mucus production, it is typically assumed that aerosol production also increases, but the actual amount of any change is unknown, and it is also unclear whether the particle size or distribution of the aerosol is shifted. The studies cited above that looked at particle production in individuals with respiratory infections also included healthy individuals in some cases, but because of the large interpersonal variability in aerosol production, it is impossible to know if any differences observed are due to illness or other factors.

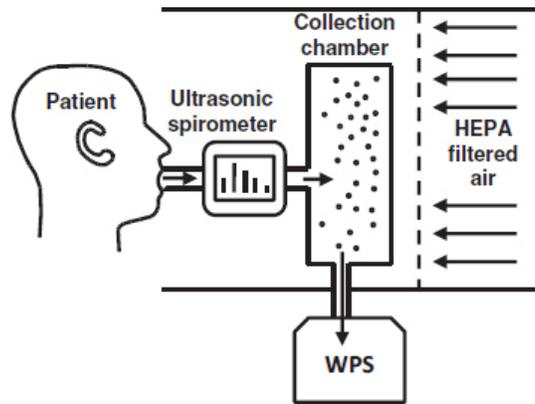
This study measured aerosol particle production during coughing by patients while they had an active influenza infection, and then measured cough aerosol production by these same patients after they had recovered. By performing the first direct comparison of respiratory aerosol production during and after illness, these results show more clearly how influenza affects aerosol generation. A better understanding of the effects of influenza on aerosol production will help with efforts to study the potential for the airborne transmission of this illness and to devise interventions to reduce its spread.

## METHODS

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### Equipment

The cough particle measurement system used in these experiments included a HEPA-filtered enclosure (Model 100-Plus; Enviroco Corporation, Sanford, N.C.), an ultrasonic spirometer (EasyOne; ndd Medical Technologies, Andover, Mass.), and a 20-L stainless steel box that served as a collection chamber for the cough aerosols (Figure 1). The cough aerosol collection chamber was fitted with an inlet port for the spirometer and two outlets. A Wide-Range Particle Spectrometer (WPS) (Model M1000XP; MSP Corporation, Shoreview, Minn.) was connected to the chamber to analyze the cough aerosols. The WPS includes a laser particle spectrometer (LPS) to measure larger aerosol particles and a differential mobility analyzer-condensation particle counter (DMA-CPC) to measure smaller particles. According to the manufacturer, the LPS detection range is 0 to 500,000 particles/liter, while the DMA-CPC detection range is 20,000 to 1010 particles/liter. Most of the cough particle concentrations were below the detection limit of the DMA-CPC, and thus, only the LPS data are reported here. A 32-L/min air pump (Model 1532; Gast Manufacturing, Benton Harbor, Mich.) was also connected to the chamber to remove particles from it between tests.



**FIGURE 1.**

Cough aerosol particle measurement system.

### Test Procedure

All procedures involving human subjects were reviewed and approved by the National Institute for Occupational Safety and Health (NIOSH) and West Virginia University (WVU) Institutional Review Boards. Written informed consent was obtained from all study participants.

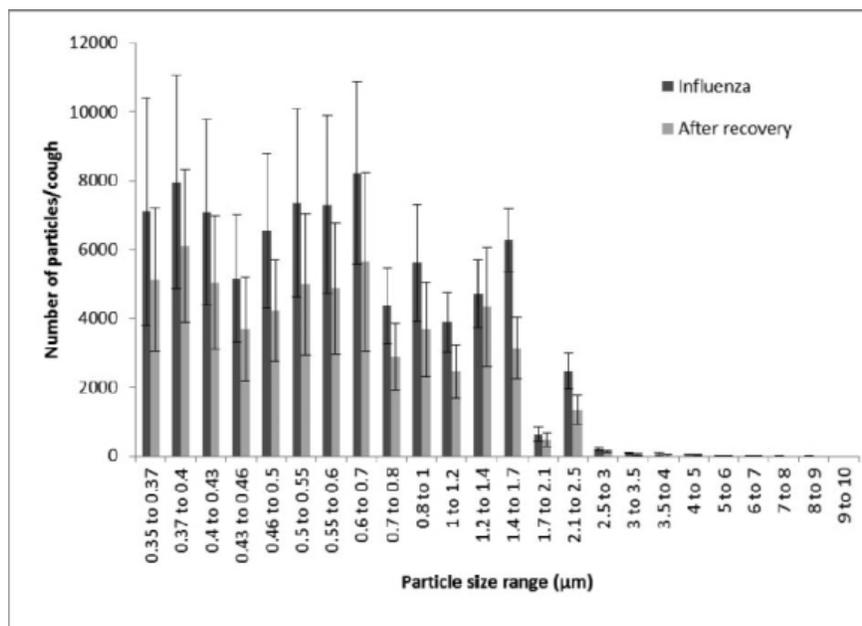
During February and March 2009, patients presenting to WELLWVU Student Health with influenza-like symptoms were recruited. To be included in the study, subjects were required to be male or non-pregnant female, age 18 to 35, lifetime non-smoker, have influenza-like symptoms including fever for 3 days or less, in otherwise good health, and must not have been vaccinated against influenza within the last 6 months. Subjects were asked a few questions about their general health, including current respiratory symptoms or illnesses. Their oral temperature was taken and two nasal swabs were collected, with the first one used for a standard clinical rapid influenza test (QuickVue Influenza test, Quidel Corp., San Diego, Calif.). If the rapid test was negative, the second swab was sent to a contract laboratory for analysis by polymerase chain reaction (PCR). The study technician then gave the participant specific instructions for the cough procedure.

For each test, the participant was seated directly in front of the HEPA-filtered air cabinet and asked to breathe normally for 5 min to remove background aerosols from their respiratory tract. During this time, the air pump was used to remove background aerosols from the collection chamber. After 5 min the air pump was turned off, and the subject was asked to exhale completely, inhale as much as possible, seal their lips around the spirometer mouthpiece, and cough. The subjects were asked to cough forcefully using as much of the air in their lungs as possible and to remove their lips from the mouthpiece at the end of the cough. After coughing, the subject resumed breathing HEPA-filtered air while the aerosol was collected and analyzed for 5 min. After analysis, the chamber was evacuated for 2 min using the air pump, and the subject was asked to repeat the cough procedure two more times for a total of three coughs. After each participant was finished, the spirometer mouthpiece was changed, and the HEPA cabinet and equipment were cleaned with disinfectant.

After completing the initial cough procedure, participants were scheduled for a return visit approximately 2 weeks later. On the return visit, the participants were asked if they had any influenza-like symptoms. If the person was no longer exhibiting symptoms, they were again asked to perform three coughs using the same procedure as above.

### Calculations

The LPS provided aerosol particle counts in 24 size bins with optical diameters from 0.35 to 10  $\mu\text{m}$  (Figure 2). Particle size and count data were collected continuously during each trial. The data were adjusted for the fraction of the sample flow stream that was counted by the LPS (0.7), the sample count time vs. the time for the total measurement cycle (30 vs. 51 sec), and the fraction of the air in the collection chamber that was sampled (5 of 20 L) to get the total number of aerosol particles and calculate the average number of particles/ cough. Because the data distributions are somewhat skewed, the average and the median of each data set are reported, along with the standard deviation, the geometric mean, and the geometric standard deviation (GSD). For the particle size distribution parameter calculations (count median diameter, volume median diameter, and GSD), the particle counts for each size bin were summed for all three coughs, and the parameters were calculated based on the summed counts. For each subject, the average results from all three coughs while ill were compared with the average results from all three coughs after recovery using a paired two-tailed t-test. Results were considered significant if  $p \leq 0.05$ .



**FIGURE 2.**

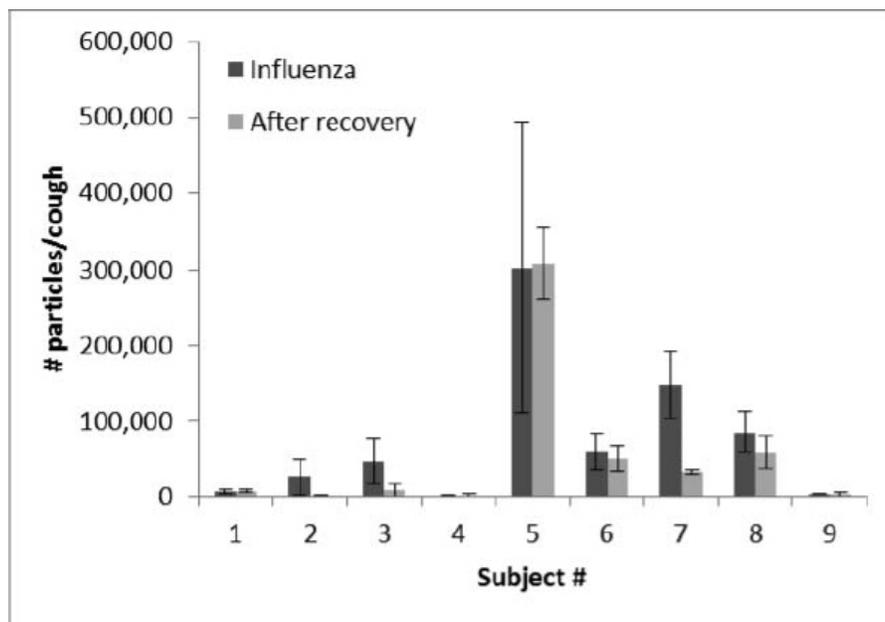
Number of particles per cough for different particle sizes for Subject 8. The number of particles detected per cough is shown while the subject had influenza and after recovery. Particle sizes are optical diameters. Each bar shows the average of three coughs. Error bars show the standard deviation.

The total volume of the aerosol particles in each size bin was estimated by assuming that the particles were spherical and that the physical diameter was approximately equal to the optical diameter. The volume of each aerosol particle was then computed, and the number of particles in each size bin was multiplied by the volume of each particle to get the total aerosol particle volume for each size bin.

The ACGIH® defines respirable aerosol particles as those particles small enough to reach the alveolar region of the lungs. The aerodynamic cutoff diameter for respirable particles is 4 µm.<sup>(16)</sup> To estimate the fraction of the cough aerosol that was in the respirable size range, the particles were assumed to have a density of 1.704 g/cm<sup>3</sup> based on the calculation by Nicas et al.<sup>(9)</sup> The particle optical diameters were thus multiplied by  $\sqrt{1.704}$  to get the aerodynamic diameter.<sup>(17)</sup> The aerosol particle volume in each size bin was then multiplied by the corresponding respirable fraction for that aerodynamic diameter as calculated using the ACGIH's formula.<sup>(16)</sup>

## RESULTS

Twenty-three subjects were recruited for this study. Of these, nine subjects (six male, three female, ages 18 to 22) were confirmed to have influenza on their first visit and returned for a second test session after their symptoms had resolved (average time from first visit to second 13.8 days, SD 1.3). A typical plot of the number of aerosol particles detected per cough in each size bin is shown in [Figure 2](#). The number of particles expelled per cough while the subjects had influenza varied considerably, with an average of 75,400 particles/cough, a median of 46,400, and a standard deviation (SD) of 97,300 ([Figure 3](#), [Table 1](#)). After the patients had recovered, their average and median particles per cough were lower (average 52,200; median 8300; SD 98,600), although the difference did not reach statistical significance ( $p = 0.1042$ ).



**FIGURE 3.**

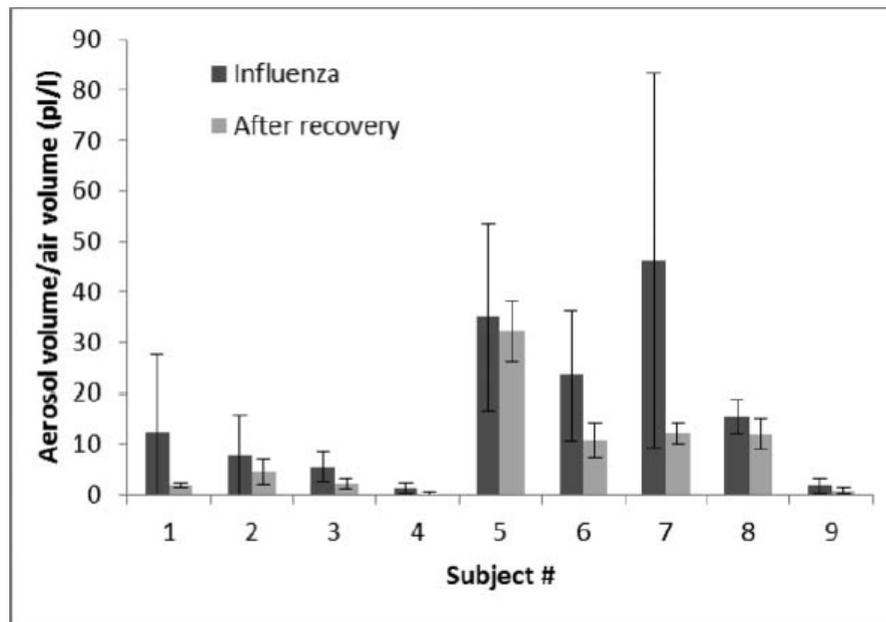
Number of particles per cough during influenza and after recovery. For each subject, the total number of aerosol particles per cough from 0.35 to 10  $\mu\text{m}$  in optical diameter is shown while they had influenza and after they had recovered. Each bar shows the average of three coughs. Error bars show the standard deviation.

**TABLE I.**

**Statistical Analysis of Cough Aerosol Particle Data from Nine Subjects During Influenza and After Recovery**

		Average	Median	SD	GM	GSD
Number of particles per cough	While ill	75,400	46,400	97,300	26,600	6.50
	After recovery	52,200	8300	98,600	12,600	6.63
Number of particles per liter of air coughed	While ill	29,600	13,700	34,200	12,100	5.47
	After recovery	16,800	3800	22,200	5800	5.89
Volume of particles per cough (pL)	While ill	38.3	20.9	43.7	20.2	3.90
	After recovery	26.4	13.5	45.6	8.4	5.74
Volume of particles per liter of air coughed (pL)	While ill	14.9	12.3	12.7	9.2	3.32
	After recovery	8.5	4.5	10.1	3.9	4.56

The volume of the aerosol particles (that is, the total physical volume of the particles themselves, not the air volume of the coughs) also varied from patient to patient. When the patients had influenza, their average aerosol volume was 38.3 pL/cough; after patients recovered, the average volume was 26.4 pL/cough. In this case, the difference in aerosol volume during sickness and after recovery was statistically significant ( $p = 0.0143$ ). The volume of aerosol particles per liter of air coughed changed in a similar manner; the average cough aerosol volume per liter of air was 14.9 pL/l when ill and 8.5 pL/l after recovery (Figure 4), and this difference was also significant ( $p = 0.0215$ ).



**FIGURE 4.**

Volume of aerosol particles per liter of air coughed during influenza and after recovery. Total volume of aerosol particles expelled in picoliters per liter of air coughed (pL/l) is shown for each subject with influenza and after they had recovered. Each bar shows the average of three coughs. Error bars show the standard deviation.

The air volume of each cough did not change significantly during and after illness ( $p = 0.7201$ ); the average cough air volume was 2.48 L when the subjects had influenza (SD 1.09) and 2.33 L after recovery (SD 1.00). The peak airflow during coughing increased somewhat after recovery; the peak flow was 5.33 L/second with influenza (SD 1.36) and 5.86 L/sec after recovery (SD 1.51). However, the difference did not reach statistical significance ( $p = 0.1018$ ).

The count median diameter (CMD) of the cough aerosol particles was fairly similar for all subjects and did not vary significantly during and after illness ( $p = 0.9340$ ). When the subjects had influenza, the CMDs of all the cough aerosols were between 0.57 and 0.71  $\mu\text{m}$  (average 0.63  $\mu\text{m}$ , SD 0.05), and the geometric standard deviations (GSD) were between 1.54 and 1.83. After the subjects had recovered, the cough aerosol CMDs ranged from 0.57 to 0.89  $\mu\text{m}$  (average 0.63  $\mu\text{m}$ , SD 0.100), and the GSDs were between 1.53 and 2.28. Similar results were seen for the volume median diameters (VMD); the average cough aerosol VMD while ill was 2.44  $\mu\text{m}$  (SD 1.17) and was 2.24  $\mu\text{m}$  (SD 1.50) after recovery. The GSDs ranged from 1.66 to 2.31 while ill and 1.54 to 2.21 after recovery. The difference in VMD while ill and after recovery also was not statistically significant ( $p = 0.7780$ ).

The ambient temperature during testing was 21°C (SD 1) and the relative humidity was 28% (SD 8%). The experimental data for all subjects are presented as an online supplement.

## DISCUSSION

The ability of influenza to spread by airborne transmission is a critical issue for those in the public health community who must plan for a possible pandemic. However, many questions remain about the production of potentially infectious aerosols by people with influenza. Our study shows that people produce aerosols with a greater volume of particles when they are sick with influenza compared with after they have recovered. Our results also show that the increase in particle volume occurs across a broad range of particle sizes, and that the overall CMD and VMD of the cough particle size distribution do not change significantly after recovery from the illness.

The number of cough aerosol particles expelled by subjects in our study varied greatly from patient to patient. The number of particles generated ranged from a low of 400 particles/cough to a high of 516,800 particles/cough while subjects had an influenza infection, and 300 to 362,700 particles/cough after recovery. These results are consistent with those reported by others; virtually all studies of human aerosol particle generation that have compared subjects have found tremendous person-to-person variation.<sup>(8–13)</sup> This phenomenon may lead to a “superspreader” effect; that is, if some people produce much greater quantities of infectious aerosols, they may be much more likely to spread influenza to others.<sup>(9,18)</sup>

The cough aerosol particle volume also varied greatly from patient to patient; the average aerosol volumes ranged from 2.4 to 144 pL/cough when ill and 0.5 to 145 pL/cough after recovery. Interestingly, the ratio of cough aerosol volume when sick to the volume after recovery was more consistent; this ratio was always between 0.99 and 5.23 and had an average of 2.77 (SD 1.55). Thus, even

though the absolute volume of aerosol coughed out by the patients varied greatly, the change in cough aerosol volume due to influenza for a given patient was more predictable. This was also true for the aerosol volume per liter of cough; in this case, the ratio ranged from 1.08 to 6.95, with an average of 2.79 (SD 1.87).

The respirable fraction of influenza-laden aerosols is of particular concern because these particles are capable of reaching the alveolar region of the lung during inhalation; human challenge studies have suggested that the infectious dose required for influenza to develop is considerably lower for particles depositing deeply into the lungs compared with particles depositing in the nasal region.<sup>(19)</sup> For our subjects, an average of 63% of the cough aerosol particle volume that was detected was in the respirable particle fraction while the subject had influenza (SD 22%). Cough aerosols have a much broader size range<sup>(8,9)</sup> than was covered by our instrument (0.35 to 10  $\mu\text{m}$ ), and thus our data do not mean that 63% of the entire cough aerosol was in the respirable fraction.

However, our results do show that a substantial volume of cough aerosol particles are produced that are in the respirable fraction, and thus potentially capable of reaching the alveolar region of the lungs. It is also interesting to compare this result with other reports of the sizes of influenza-laden airborne particles; a study of cough aerosols collected from influenza patients found that 65% of the influenza virus RNA was contained in particles in the respirable size fraction,<sup>(12)</sup> and two previous studies of airborne particles in a hospital emergency department<sup>(20)</sup> and an urgent care clinic<sup>(21)</sup> found that 53% and 42% of the influenza virus RNA was in particles in the respirable size fraction. Taken together, these studies all suggest that a substantial portion of the airborne particles containing influenza that are expelled by patients are in the respirable size range and support the hypothesis that influenza could in fact be transmitted by the airborne route.

The total volume of the expelled particles recorded during our experiments was considerably lower than the amounts that have been reported by other researchers.<sup>(22–24)</sup> The primary reason for this is likely because these authors included much larger particles in their measurements (up to 2 mm for one study).<sup>(23)</sup> Since a 2-mm particle has a volume that is  $10^9$  greater than a 2- $\mu\text{m}$  particle, a small number of large particles can have a tremendous impact on the measurement of the total volume expelled. These larger drops fall very quickly (in fact, they were usually collected onto nearby settling plates during the experiments), while smaller droplets are capable of remaining airborne for an extended period. Thus, these results also illustrate the critical role of particle size in airborne disease transmission. At close range, larger drops can be important because of their greater volume and subsequent pathogen content, but as one moves farther away from an infected person, smaller particles may gradually become more important because they are able to stay airborne longer, reach more people, and are more easily inhaled.

Finally, some of the limitations of our study must be noted. The participants were young, ambulatory, otherwise healthy adults, and thus their results may not be representative of a broader and more diverse population. The number of subjects was small, which may be important given the large person-to-person variation seen in aerosol production. Our subjects were all symptomatic at the time of initial testing, but they were likely at different stages of their illness and some were more ill than others; both of these factors may influence aerosol production. Aerosol particles toward the upper limit of the WPS (10  $\mu\text{m}$ ) were more likely than small ones to deposit by impaction or settling before detection and thus may have been undercounted in our cough aerosol measurement system. Finally, it is not known how much influenza virus was contained in the airborne particles, how the influenza virus content varies with particle size, or how the amount of virus in each particle changes over the course of the illness.

## CONCLUSION

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People with influenza release potentially infectious aerosol particles when they cough, sneeze, speak, and breathe. However, many questions remain about the likelihood of the transmission of influenza from person to person by the airborne route. Our study shows for the first time that individuals with influenza cough out a greater volume of aerosol particles than they do when they are healthy. Further, many of these particles are in the respirable size fraction and thus can be easily inhaled and drawn down into the deepest parts of the lungs where they may be more likely to cause an infection. This enhancement in aerosol generation may play an important role in influenza transmission and suggests that a better understanding of this phenomenon is needed to predict the production and dissemination of influenza-laden aerosols by people infected with this virus.

## Supplementary Material

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**UOEH\_A\_684582\_SM6450.pdf:**

[Click here to view.](#)<sup>(17K, pdf)</sup>

**UOEH\_A\_684582\_SM6452.pdf:**

[Click here to view.](#)<sup>(36K, pdf)</sup>

## Footnotes

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The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health.

## ACKNOWLEDGMENTS

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The authors would like to thank the study participants for their willingness to assist with this project, and the staff of WELLWVU Student Health for their cheerful help and cooperation. We would also like to thank Michael Commodore of NIOSH for his assistance with assembling the cough measurement system and conducting the experiments, and Michael Andrew of NIOSH for his consultations with us about the statistical analysis.

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Volume 26, Number 9—September 2020

*Research Letter*

## Large SARS-CoV-2 Outbreak Caused by Asymptomatic Traveler, China

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## Abstract

An asymptomatic person infected with severe acute respiratory syndrome coronavirus 2 returned to Heilongjiang Province, China, after international travel. The traveler's neighbor became infected and generated a cluster of  $\geq 71$  cases, including cases in 2 hospitals. Genome sequences of the virus were distinct from viral genomes previously circulating in China.

Coronavirus disease (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has spread rapidly around the world since the first cases were reported in late 2019 (1,2). Prior to April 9, 2020, Heilongjiang Province, China, had not reported a new COVID-19 diagnosis since March 11, 2020. On April 9, SARS-CoV-2 was diagnosed in 4 patients. By April 22,  $\geq 71$  persons had been infected. The likely origin of this cluster is an imported case from an asymptomatic traveler.

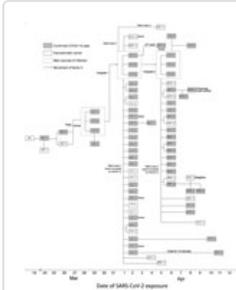


Figure. Timeline of exposure and connections between cases of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) among persons in Heilongjiang Province, China. A0 returned from the United States on March 19, tested...

We collected and analyzed epidemiologic data published on the website of the Health Commission of Heilongjiang Province for April 9–23, 2020 (3). We defined confirmed COVID-19 cases as persons who tested positive for SARS-CoV-2 and had clinical symptoms. We defined asymptomatic carriers as persons without clinical symptoms who tested positive for SARS-CoV-2. We refer to case-patients by a letter for each family (A–Z, AA–ZZ), then by the assumed transmission generation (1–2), and finally in sequential order of exposure to SARS-CoV-2-positive persons in generations 1–3 (Figure) (4).

On March 19, 2020, case-patient A0 returned to Heilongjiang Province from the United States; she was asked to quarantine at home. She lived alone during her stay in Heilongjiang Province. She had negative SARS-CoV-2 nucleic acid and serum antibody tests on March 31 and April 3.

Patient B1.1 was the downstairs neighbor of case-patient A0. They used the same elevator in the building but not at the same time and did not have close contact otherwise. On March 26, B1.1's mother, B2.2, and her mother's boyfriend, B2.3, visited and stayed in B1.1's home all night. On March 29, B2.2 and B2.3 attended a party with patient C1.1 and his sons, C1.2 and C1.3.

On April 2, C1.1 suffered a stroke and was admitted to hospital 1. His sons, C1.2 and C1.3, cared for him in ward area 1 of the hospital. Patient C1.1 shared the same clinical team and items, such as a microwave, with other patients in the ward. On April 6, patient C1.1 was transferred to hospital 2 because of fever; C1.2 and C1.3 accompanied him.

On April 7, patient B2.3 first noted symptoms of COVID-19. He tested positive for SARS-CoV-2 on April 9, the first confirmed case in this cluster. His close contacts, B1.1, B2.1, B2.2, and C1.1, subsequently tested positive for SARS-CoV-2 on April 9 or 10. Patient C1.1 was quarantined in hospital 2 when he tested positive on April 9. The epidemiologic investigation showed that none of these 5 persons had a history of travel or residence in affected areas with sustained transmission of SARS-CoV-2 during the 14 days before diagnosis, suggesting that SARS-CoV-2 came from contact with other persons.

During C1.1's admission at hospital 1, a total of 28 other persons, D1.1–BB1.1, were infected with SARS-CoV-2 in ward area 1. Because all patients in the ward could ambulate, 4 persons, CC1.1, DD1.1, EE1.1, and FF1.1, were infected in other wards and in the computed tomography room of hospital 1. Among hospital 1 staff, 5 nurses and 1 doctor were infected. In hospital 2, another 20 persons, GG1.1–VV1.1, were infected in the ward where C1.1 stayed ([Figure](#)).

On April 9, investigators also learned that A0, B1.1's neighbor, had returned on March 19 from the United States, where COVID-19 cases had been detected. Investigators performed SARS-CoV-2 serum antibody tests on A0 on April 10 and 11. SARS-CoV-2 serum IgM was negative but IgG was positive, indicating that A0 was previously infected with SARS-CoV-2 ([5,6](#)). Therefore, we believe A0 was an asymptomatic carrier ([7,8](#)) and that B1.1 was infected by contact with surfaces in the elevator in the building where they both lived ([9](#)). Other residents in A0's building tested negative for SARS-CoV-2 nucleic acids and serum antibodies.

On April 15, the Chinese Center for Disease Control and Prevention sequenced the entire genomes of 21 samples from the cluster. Viral genomes were identical in 18 cases and 3 other cases had a difference of 1–2 nucleotides, indicating that SARS-CoV-2 came from the same point of origin. The viral genome sequences from the cluster were distinct from the viral genomes previously circulating in China, indicating the virus originated abroad ([10](#)) and suggesting case A0 was the origin of infection for this cluster.

All persons in this cluster, including those who lived in the same community and had close contact with SARS-CoV-2-positive patients or visited the 2 hospitals during April 2–15, were tested for SARS-CoV-2 nucleic acids and serum antibodies. As of April 22, 2020, A0 remained asymptomatic, and a total of 71 SARS-CoV-2-positive cases had been identified in the cluster.

Our results illustrate how a single asymptomatic SARS-CoV-2 infection could result in widespread community transmission. This report also highlights the resources required for case investigation and challenges associated with containment of SARS-CoV-2. Continued measures to protect, screen, and isolate infected persons are essential to mitigating and containing the COVID-19 pandemic.

Mr. Liu is a PhD candidate in Huazhong University of Science and Technology. He also conducts research in Hubei University of Medicine. His primary research interest is neurodegenerative diseases.

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## Acknowledgments

We thank the patients described in this report, the health care personnel who cared for them, the staff members of Health Commission of Heilongjiang Province, and the staff members of Heilongjiang Provincial Center for Disease Control and Prevention.

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## Figure

**Figure.** Timeline of exposure and connections between cases of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) among persons in Heilongjiang Province, China. A0 returned from the United States on March 19,...

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*Suggested citation for this article:* Liu J, Huang J, Xiang D. Large SARS-CoV-2 outbreak caused by asymptomatic traveler, China. *Emerg Infect Dis.* 2020 Sep [date cited]. <https://doi.org/10.3201/eid2609.201798>

DOI: 10.3201/eid2609.201798

Original Publication Date: June 30, 2020

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NBSIR 88-3751

**AN ANALYSIS OF THE INFLUENCE OF  
PISTON EFFECT ON ELEVATOR SMOKE  
CONTROL**

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April 1988

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**NBSIR 88-3751**

# **An Analysis of the Influence of Piston Effect on Elevator Smoke Control**

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# AN ANALYSIS OF THE INFLUENCE OF PISTON EFFECT ON ELEVATOR SMOKE CONTROL

John H. Klote

## Abstract

This paper is part of a joint project between the United States and Canada to evaluate the feasibility of using elevators for the evacuation of the handicapped during a fire. The transient pressures produced when an elevator car moves in a shaft are a potential problem for elevator smoke control. Such piston effect can pull smoke into a normally pressurized elevator lobby. This paper presents an analysis of an elevator smoke control system emphasizing the influence of piston effect on system performance. For most elevators the problem can be overcome by designs that prevent smoke from being pulled into lobbies, and equations for the amount of pressurization air to accomplish this are developed for two arrangements of supply air outlets. Where this approach is not feasible, the methods of analysis presented in this paper can be used to determine smoke infiltration for a hazard analysis.

Key words: elevators (lifts), hazard analysis, piston effect, pressurization, smoke, smoke control.

## 1. INTRODUCTION

The problem of fire evacuation of the handicapped has become a topic of concern within the fire protection community. One solution would be the use of elevators. Logistics of evacuation, reliability of electrical power, elevator door jamming, and fire and smoke protection are long-standing obstacles to the use of elevators for fire evacuation. All of these obstacles except smoke protection can be addressed by existing technology as discussed by Klote [1].

The National Bureau of Standards (NBS) in the United States and the National Research Council of Canada (NRCC) are engaged in a joint project to develop smoke control technology for elevators. The initial report [2] of this project was a concept study evaluating several elevator smoke control systems by computer analysis using the NBS program for analysis of smoke control systems[3]. The transient pressures due to 'piston effect' when an elevator car moves is a concern of building designers relative to elevator smoke control. The second report [4] of this project developed an analysis of the pressures due to piston effect in a building without smoke control and evaluates piston effect in light of that analysis. This paper presents an analysis of piston effect incorporating elevator smoke control, and addresses the problem based on this analysis.

## 2. SMOKE CONTROL SYSTEM

The term 'smoke control' is used in this report to mean the limiting of smoke movement by pressurization produced by mechanical fans. This meaning has attained some level of acceptance in North America. Ideally, an elevator smoke control system **should** protect the elevator shaft and the elevator lobbies such that smoke contamination in these areas does not present a hazard.

Most elevator doors have large gaps around them [5]. Such large leakage areas around the doors result in lobby and shaft pressures that are nearly equal under most conditions. Thus if pressurization air is supplied to the elevator shaft, the lobbies will be pressurized indirectly to almost the same pressure as the shaft. A concern with such systems is that a few open doors might result in significant loss of pressurization. The first paper [2] of this project demonstrates that this problem can be overcome by use of a system with feedback control. **The** flow rate of air into the **shaft** is controlled **by** a differential pressure sensor **to** maintain a constant pressure difference across **the** elevator lobby **door** on the fire floor. One method of varying the flow rate is a fan bypass system. It may be possible to develop **other systems** that can also **solve** the pressure loss problem. Because the elevator smoke control

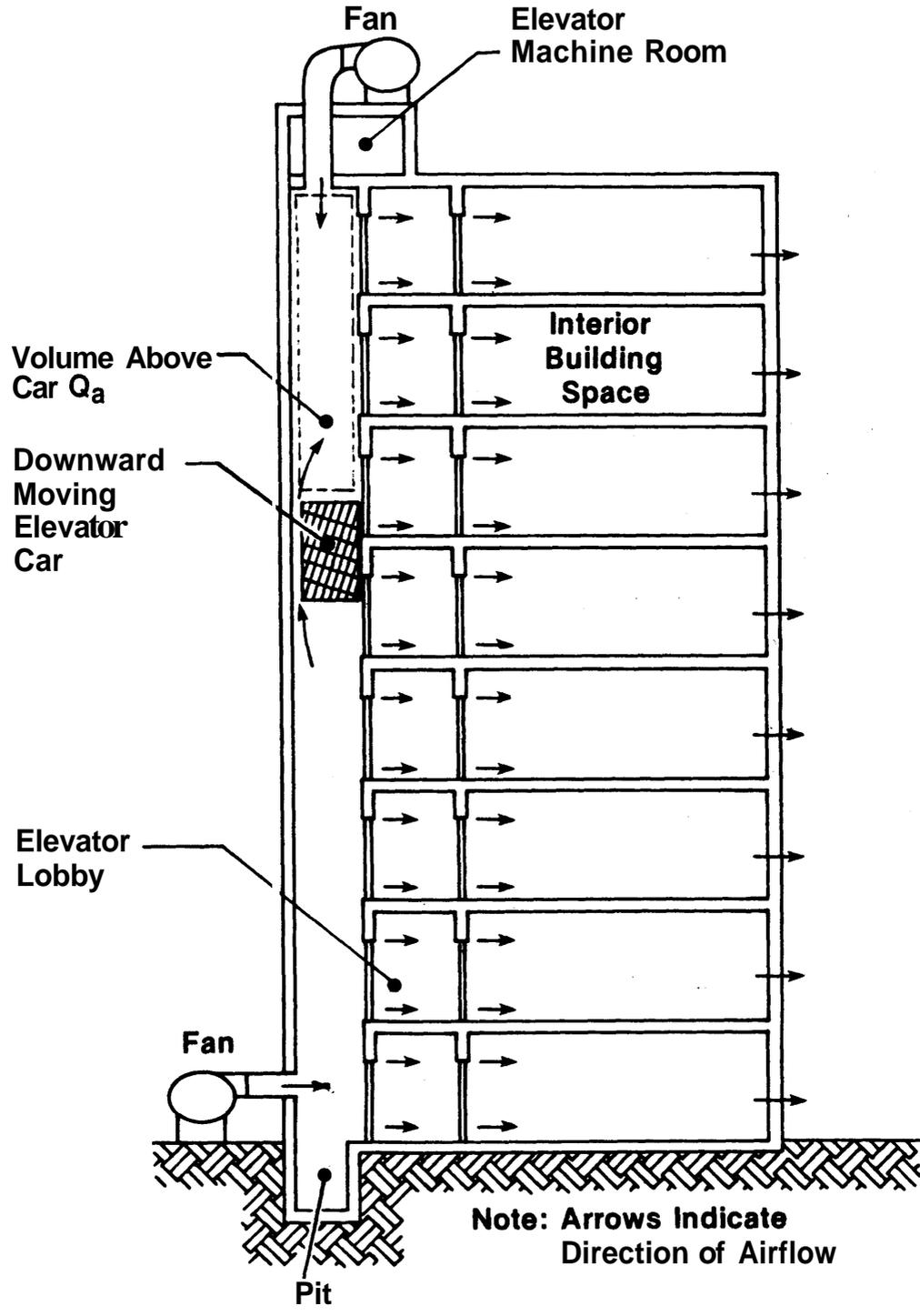


Figure 1. Airflow due to shaft pressurization and car motion

system discussed above can deal with this problem, it is the basis of the analysis and discussions that follow.

### 3. ANALYSIS

The direction of airflow illustrated in figure 1 is that which would result from shaft pressurization. When an elevator car moves downward, piston effect tends to increase the pressure below the car and to reduce the pressure above the car. In extreme cases the reduction of pressure could overcome a shaft pressurization system and result in smoke infiltration of the elevator lobby or shaft. For the sake of simplicity, buoyancy, wind, stack effect, and the heating and ventilating system have been omitted from this analysis.' Omitting stack effect is equivalent to stipulating that the building air temperature and the outside air temperature are equal. Because these temperatures are the same, the gravity effects on air density and pressure are negligible. For this analysis elevator car motion is limited to a single car moving in a single car shaft or in a multiple car shaft. The analysis is for a downward-moving elevator car (as illustrated in figure 1), however the problem of an upward-moving one is same mathematically. Thus the equations developed can be extended for an upward-moving car by reversing subscripts a (above the car) and b (below the car).

#### 3.1 Equations for Conservation of Mass

The law of conservation of mass can be written for the volume,  $Q_a$ , above the car

$$\left[ \begin{array}{l} \text{Net mass flow} \\ \text{into volume } Q_a \end{array} \right] = \left[ \begin{array}{l} \text{Rate of mass change} \\ \text{within volume } Q_a \end{array} \right] \quad (1)$$

$$\dot{m}_{p_a} + \dot{m}_{b_a} - \dot{m}_{a_o} = \frac{d}{dt} (\rho Q_a)$$

where

- $\dot{m}_{pa}$  = mass flow rate of pressurization air to the shaft space above the car
- $\dot{m}_{ba}$  = mass flow rate from below the elevator car,  $Q_b$ , to volume,  $Q_a$
- $\dot{m}_{ao}$  = mass flow rate from volume,  $Q_a$ , to the outside
- $\rho$  = air density within the shaft.

For a downward moving car velocity,  $V$ , and a cross-sectional area of the shaft,  $A_s$ , the derivative of the volume,  $Q_a$ , can be expressed as

$$\frac{d Q_a}{dt} = A_s V \quad (2)$$

The air density is essentially constant within the shaft. Therefore, Substituting equation (2) into equation (1) yields

$$\dot{m}_{pa} + \dot{m}_{ba} - \dot{m}_{ao} = \rho A_s V \quad (1a)$$

The conservation of mass equation for the entire shaft is

$$\dot{m}_{pa} + \dot{m}_{pb} - \dot{m}_{ao} - \dot{m}_{bo} = 0 \quad (3)$$

Where  $\dot{m}_{pb}$  is the mass flow rate of pressurization air to the shaft below the car.

### 3.2 Equations for Mass Flow

To expedite the analysis, the flow areas are chosen such that they are the same for each floor of the building and that the only vertical airflow in the building is within the elevator shaft. The flow from  $Q_a$  to the outside is

$$\dot{m}_{ao} = N_a C A_a S_a \sqrt{2\rho |P_a|} \quad (4)$$

where

$N_a$  = number of floors above the car

$C$  = flow coefficient

$A_e$  = effective flow area per floor between the shaft and the outside

$P_a$  = pressure of the air in  $Q_a$  relative to the outside

$S_a$  = the sign of  $P_a$ .

The absolute value signs and  $S_a$  are included in equation (4) to allow for pressurization failure above the downward moving car. The outside pressure is not explicitly incorporated in equation (4), because  $P_a$  is a gage pressure which is the difference between the absolute pressure and the atmospheric pressure outside the building. The effective flow area,  $A_e$ , is the area that results in the same flow as the system of flow areas from the building to the outside when  $A_e$  is subjected to the same pressure difference as the system. The system of flow areas can consist of areas in parallel with one another, in series, or a combination of both parallel and series. The ASHRAE smoke control manual [6] presents a detailed discussion of effective flow areas, and an example evaluation of  $A_e$  for a system of flow paths is presented later in this paper.

The flow rate from  $Q_b$  to the outside is

$$\dot{m}_{b_o} = N_b C A_e \sqrt{2\rho P_b} \quad (5)$$

where

$N_b$  = number of floors below the car

$P_b$  = pressure of the air in  $Q_b$  relative to the outside.

If the car were standing still,  $\dot{m}_{b_o}$  would be positive. A downward-moving car only increases the positive pressurization below the car. Thus the analysis only accounts for positive pressurization of the shaft below the car as can be observed from equation (5). Neglecting hydrostatic pressure

difference, the mass flow rate from below the car to above it can be expressed as

$$\dot{m}_{b_a} = C_c A_f S_{b_a} \sqrt{2\rho |P_b - P_a|} \quad (6)$$

where

- $A_f$  = free flow area in the shaft around the car
- $C_c$  = flow coefficient for flow around the car
- $S_{b_a}$  = sign of  $(P_b - P_a)$ .

Equation (6) includes the absolute value signs and  $S_{b_a}$  to allow for flow from above the car to below it. This can occur when the pressurization air,  $\dot{m}_{p_a}$ , above the car is very large. Tests were conducted to evaluate  $C_c$  on a twelve-story elevator shaft at the NBS administration building [4]. For one car traveling in a two car shaft, the flow coefficient was .94, and for two cars traveling side-by-side together the flow coefficient was .83. The case of the two cars moving together was measured to obtain an approximation of a car moving in a single car shaft.

### 3.3 Equations for $P_a$ and $P_b$

Substituting equations (4) and (5) into equation (3), and solving for  $P_b$  yields

$$P_b = (c - b \sqrt{|P_a|})^2 \quad (7)$$

where

$$c = (\dot{m}_{p_a} + \dot{m}_{p_b}) / K_{b_o}$$

$$b = S_a N_a / N_b$$

$$K_{b_o} = N_b C A_o \sqrt{2\rho}$$

Combining equations (1a), (4), (5), (6) and (7) yields

$$\dot{m}_{p_a} + K_{b_a} S_{b_a} \sqrt{\left| \left[ c - b \sqrt{|P_a|} \right]^2 - P_a \right|} - K_{a_o} S_a \sqrt{|P_a|} - \rho A_s V = 0 \quad (8)$$

where

$$K_{b_a} = A_f C_c \sqrt{2 \rho}$$

$$K_{a_o} = N_a C A_e \sqrt{2 \rho}$$

As might be expected, for  $\dot{m}_{p_a} = \dot{m}_{p_b} = 0$ , equation (8) reduces to equation (7) from the earlier paper [3] on piston effect without shaft pressurization (note that the sign convention for  $P_a$  is opposite in the two papers).

#### 4. MOTION OF CAR

For this paper three phases of elevator car motion are considered: constant acceleration, transitional and constant velocity motion. A car starting from rest accelerates at a constant rate,  $a$ , until the transitional velocity,  $V_t$ , is reached. The time,  $t_t$ , to reach this velocity is

$$t_t = \frac{V_t}{a} \quad (9)$$

The distance,  $X_t$ , the car travels in this time is

$$X_t = \frac{V_t^2}{2 a} \quad (10)$$

During the transitional phase the acceleration decreases until full operational velocity,  $V_p$ , is reached. Strakosch [7] uses the following approximate relations for this phase:

$$t_p = \frac{V_p^2 - V_t^2}{2 a V_t} \quad (11)$$

and

$$X_p = \frac{1}{3 a} \left[ \frac{V_p^3}{V_t} - V_t^2 \right] + X_t \quad (12)$$

where  $X_p$  is the distance the car travels before it reaches full operational velocity at time  $t_p$  from the start of motion. These equations leave the motion between  $X_t$  and  $X_p$  undefined. Motion in this region is not necessary for evaluation of piston effect in the context of this paper as is demonstrated by the example in the following section.

## 5. PRESSURE DIFFERENCE ACROSS LOBBY DOORS

For fire evacuation by elevators, the pressure difference,  $\Delta P_{11}$ , across the elevator lobby doors is of major importance. If  $\Delta P_{11}$  is positive, the resulting airflow from the lobby to the building will act to prevent smoke infiltration of the lobby. This pressure difference can be evaluated by examination of the effective flow area. For the system of three series flow paths from the shaft to the outside illustrated in Figure 1, the effective flow area per floor is

$$A_e = \left| \frac{1}{A_{1s}^2} + \frac{1}{A_{11}^2} + \frac{1}{A_{o1}^2} \right|^{-1/2} \quad (13)$$

where

$A_{1s}$  = leakage area between the lobby and the shaft

$A_{i1}$  = leakage area between the building and the lobby

$A_{o1}$  = leakage area between the outside and the building.

For paths in series the pressure difference across one path equals the pressure difference across the system times the square of the ratio of the effective area of the system to the flow area of the path in question. Thus for flows above the elevator car,  $\Delta P_{i1}$  can be expressed as

$$\Delta P_{i1} = P_a (A_e / A_{i1})^2 \quad (14)$$

This equation is general in that it applies to any system of flow paths not just those shown in figure 1, provided that  $A_e$  is evaluated for that particular system. This analysis does not include the effects of other shafts such as stairwells and dumbwaiters. Provided that the leakage of these other shafts is relatively small compared to  $A_{o1}$ , equation (13) is appropriate for evaluation of  $A_e$  for buildings with open floor plans. The configuration of figure 1 was selected because many buildings are constructed with open floor plans and because evaluation of this system may provide some understanding of more complicated systems with interior partitions. The complicated flow path systems probably require case by case evaluation which can be done by using the effective area techniques presented in the ASHRAE smoke control manual [6].

A computer program was developed which solved equation (8) for  $P_a$  by the **method** of bisection [8] using the car velocities and displacements of equations (9) through (12). Equations (13) and (14) were used to obtain  $\Delta P_{i1}$ . In the preceding analysis, the number of floors,  $N_e$ , above the car might be **thought** of as an integer, however, a real number value for  $N_e$  was used for the computer program to allow calculations when the car is not located exactly at a floor. This occurs at the points where transitional acceleration begins and ends. The real number approach assumes that at each floor the leakage area,  $A_e$ , is uniformly distributed over the floor height. Even though it is obvious

that leakage areas in buildings are not uniform, it is believed that the errors due to this assumption are insignificant.

Table 1. Flow Areas of Eleven Story Elevator Shaft for Example Piston Effect Analysis

	m <sup>2</sup>	ft <sup>2</sup>
For Single Car Shaft		
A <sub>l</sub> , area between lobby and shaft	0.167	1.80
A <sub>b</sub> , area between building and lobby	0.0390	0.42
A <sub>oi</sub> , area between outside and building	0.0502	0.54
A <sub>s</sub> , cross-sectional area of shaft	5.61	60.4
A <sub>f</sub> , free flow area around car	1.80	19.4
For Double Car Shaft		
A <sub>l</sub> , area between lobby and shaft	0.0836	0.90
A <sub>b</sub> , area between building and lobby	0.0390	0.42
A <sub>oi</sub> , area between outside and building	0.0502	0.54
A <sub>s</sub> , cross-sectional area of shaft	11.22	120.8
A <sub>f</sub> , free flow area around car	7.41	79.8

Note: For the single car shaft a value of  $C_o = 0.83$  was used, and for the double car shaft a value of  $C_o = 0.94$  was used. The flow coefficient was  $C = 0.65$ . Pressurization air was  $\dot{m}_{pa} = 0$  and  $\dot{m}_{pb} = 2.160$  kg/s (3810 standard cfm at 68 °F and one atmosphere). Car acceleration was 1.22 m/s<sup>2</sup> (4 ft/sec<sup>2</sup>), and  $V_t$  at 60 % of  $V_p$ .

Figure 2. shows computer calculated values of  $AP_{f,}$  for a single and a double car shaft for two values of  $V_t$ . The flow areas for these examples are listed in table 1. These flow areas are based on the measured values of  $A_l$  and  $A_b$  from tests of the NBS administration building [4] and average leakage values from Appendix C of the ASHRAE smoke control manual for a building with a floor size of 14.0 m x 67.7 m (46.0 ft x 222 ft) and 3.099 m (10.17 ft) between floors. Pressurization air was supplied below the car and at a rate such that  $\Delta P_{i1}$  was 25 Pa (0.10 in H<sub>2</sub>O) when the cars were still.

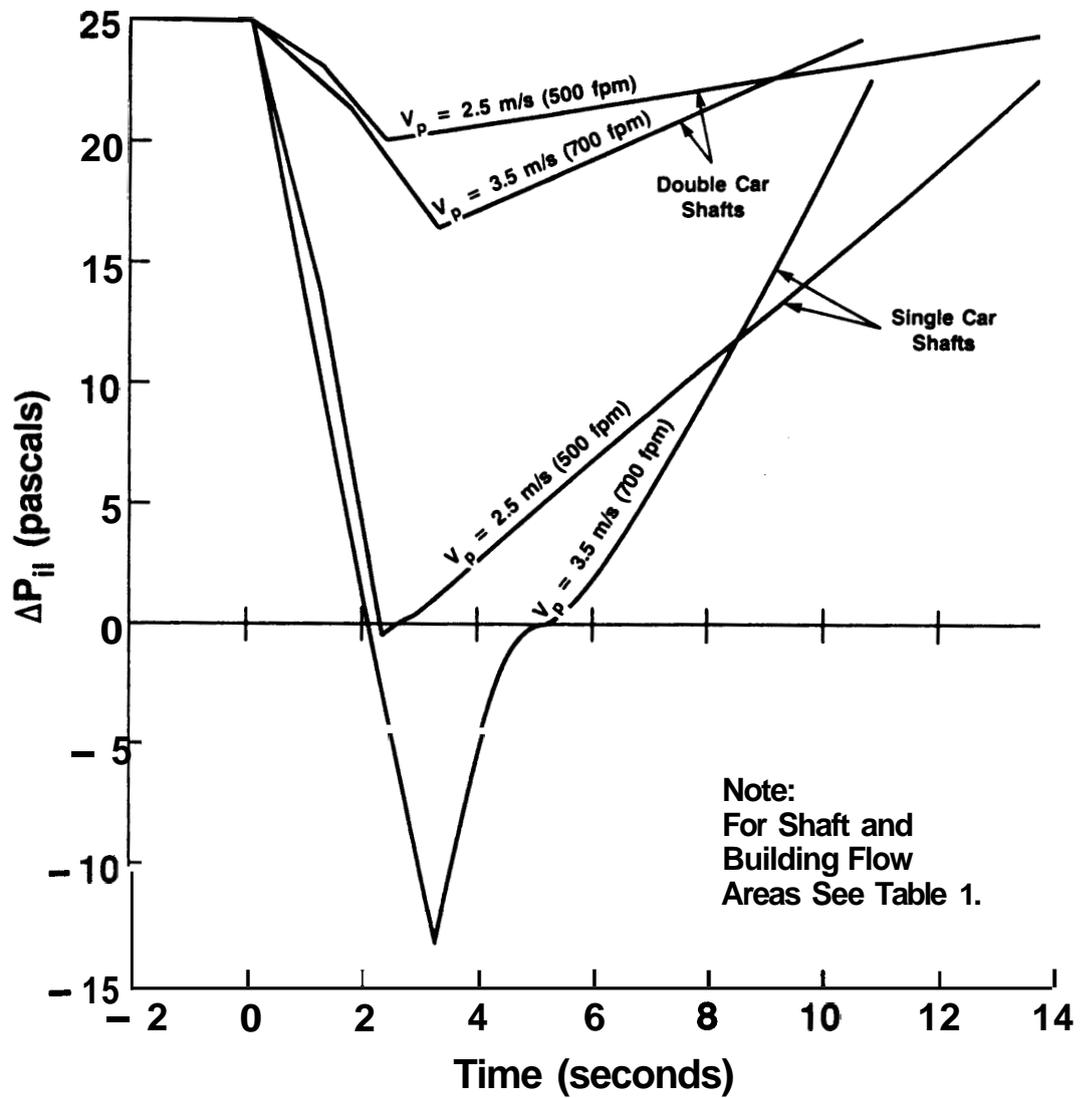


Figure 2. Calculated pressure differences across elevator lobby doors due to piston effect of a single car moving in an eleven story shaft

As expected figure 2 illustrates that piston effect is more pronounced for greater car velocities and for single car shafts. For the double car shaft of this example, piston effect does not adversely effect lobby pressurization. This is because the free flow area,  $A$ , around the car is so large that there is little resistance to air flow around the car. Because  $A$  is large for all multiple car shafts, piston effect in these shafts will probably not have an adverse effect on lobby pressurization except at high car velocities.

For the example single car shaft at  $V_p = 3.5$  m/s (700 fpm), piston effect caused a flow from the building to the lobby for a duration of about 3 seconds. In a fire situation this could result in smoke being pulled into the lobby. The severity of such a situation would depend on the speed of the car, the size of the lobby, the toxicity of the smoke and the number of times the car passes the floor. Modern elevators operate at speeds up to 10 m/s (2000 fpm). At these high speeds piston effect is much more significant. Fortunately such high speeds are not common for single car shafts.

## 6. HAZARD ANALYSIS

One approach to the piston effect problem is an analysis determining the quantity of toxic gases pulled into an elevator lobby by piston action and determining the resulting hazard to life. The mass,  $m$ , of smoke laden air pulled into the elevator lobby during the decent of a car is

$$m = \int_{t_1}^{t_2} \dot{m}_{a.o} dt$$

where the smoke and air mixture is pulled into the lobby during the time interval from  $t_1$  to  $t_2$ . The mass flow rate can be obtained from equation (4) using the pressures as defined by equation (8). The mass of gases pulled into a lobby due to an upward moving car can be determined by a similar approach.

An evaluation of the resulting hazard must include considerations of mixing of air and smoke within the lobby, an estimate of the number of times a car passes the floor of concern and an analysis of the effects of the toxic gases on people waiting in the lobby. Such an analysis is beyond the scope of this paper. An alternate approach is to design elevator systems such that piston effect does not cause any smoke infiltration of elevator lobbies. This approach is developed in the following section.

## 7. CRITICAL PRESSURIZATION RATE

For an unpressurized shaft Klote and Tamura [4] developed an expression for the limit of the extent of piston effect, and this limit was obtained for the conditions of the elevator car being at the top ( $N_c = 0$ ) of the shaft and a car velocity of  $V_p$ . Similarly, a limit or minimum value of  $P_a$  can be obtained for a pressurized shaft using the same conditions. Observation of figure 2 reveals that once the car reaches  $V_p$ ,  $\Delta P_{11}$  increases with time or with distance from the top of the shaft. If the car were traveling at  $V_p$  over all its descent,  $\Delta P_{11}$  would increase with distance traveled from the top of the shaft. Obviously for the above conditions, equation (8) yields a value of  $P_a$  which is less than that resulting from realistic car motion starting at rest. For a pressurized shaft, setting  $N_c$  to zero results in equation (4) becoming  $m_{a0} = 0$  and by definition  $b = 0$ . Equation (8) becomes

$$(P_a)_{\min} = \frac{1}{2 \rho} \left[ \left( \frac{\dot{m}_{pa} + \dot{m}_{pb}}{N_{tot} C A_e} \right)^2 - \left( \frac{\rho A_s V_p - \dot{m}_{pa}}{A_f C_c} \right)^2 \right] \quad (15)$$

where

$(P_a)_{\min}$  = minimum level of pressurization above a downward moving elevator car  
 $N_{tot}$  = total number of **floors**.

A negative value of  $(P_a)_{\min}$  indicates a failure of pressurization due to piston effect. The velocity  $V_p$  was used in equation (15) because this is the

maximum speed at which the car can travel making the resulting pressure a limit below which the pressure  $P_a$  would not fall.

A value of  $(P_a)_{\min} = 0$  means that elevator pressurization remains positive throughout the car's descent. The total mass flow rate to achieve this condition is referred to as the critical pressurization rate,  $\dot{m}_{crit}$ . A common situation is to supply all of the air at one point. If all the air is supplied below the car ( $\dot{m}_{pa} = 0$ ), the critical mass flow rate is

$$\dot{m}_{crit} = \rho A_s V_p \frac{N_{tot} A_e C}{A_f C_c} \quad (16)$$

If pressurization air is supplied at this rate or greater to the space below a downward moving elevator car, piston effect will not result in loss of shaft pressurization. It is obvious that equation (16) also applies to an upward-moving car with all the air supplied above the car. Thus it can be stated in general that equation (16) defines the critical mass flow rate for a shaft with an air pressurization inlet at only one location.

For pressurization air supplied evenly at the top and bottom of the shaft ( $\dot{m}_{pa} = \dot{m}_{pb} = \dot{m}_{crit}/2$ ), the critical mass flow rate is

$$\dot{m}_{crit} = \frac{2 \rho A_s V_p}{1 + \frac{2 A_f C_c}{N_{tot} A_e C}} \quad (17)$$

If at least half this amount of pressurization air is supplied above the car and an equal amount below it, piston effect will not result in loss of shaft pressurization. Equations (16) and (17) can be used to check during smoke control design to assure that piston effect does not result in loss of shaft pressurization. Table 2 lists critical mass flow rates calculated from these equations for shafts of the previous example. An elevator smoke control system may need a much greater supply rate of pressurization air in order to

produce the pressure differences desired for smoke control as discussed in previous reports [1,2]. It can be observed from table 2 that  $\dot{m}_{crit}$  is larger when air is supplied at only one location as opposed to being supplied both at the top and bottom of the shaft. Obviously, injecting air into the shaft above the car reduces piston effect for a downward moving car. It can also be observed from table 2 that a double car shaft has a much lower  $\dot{m}_{crit}$  than a similar single car shaft. This supports the belief that generally piston effect would not be a problem for multiple car shafts.

Table 2. Critical mass flow rates calculated from equations (16) and (17) for example shafts

	Single Car Shafts		<b>Double</b> Car Shafts	
	kg/s	Standard cfm	<b>kg/s</b>	Standard cfm
FOR $V_p = 2.5$ m/s (500 ft/min)				
Pressurization air supplied at one point	<b>2.44</b>	4310	1.00	1770
Pressurization air evenly divided between top and bottom of shaft	<b>2.27</b>	4010	<b>0.96</b>	1700
FOR $V_p = 3.5$ m/s (700 ft/min)				
Pressurization air supplied at one point	<b>3.42</b>	<b>6040</b>	1.40	<b>2470</b>
Pressurization air evenly divided between top and bottom of shaft	<b>3.18</b>	<b>5620</b>	<b>1.35</b>	<b>2380</b>

Note: For areas and flow coefficients see table 1. Standard cfm is at 68 °F and one atmosphere.

## 8. FUTURE EFFORT

Elevator smoke control tests are being conducted by the NRCC at the Fire Research Tower near Ottawa to evaluate various system concepts under full scale fire conditions. Tests will be conducted by the NRCC on an existing building with a pressurized elevator shaft in an attempt to verify the analysis presented in this paper. The final effort planned for this project will be for NRCC and NBS to jointly develop practical engineering design information for elevator smoke control based on this research.

## 9. CONCLUSIONS

1. For most elevators, especially those in multiple car shafts, it is feasible to deal with the piston effect problem by designing **so** as to prevent smoke from being pulled into the elevator lobby by piston effect.
2. For an elevator shaft with only one pressurization air inlet, piston effect will not result in loss of shaft pressurization provided the mass flow rate of pressurization air is at least as great as the critical mass flow rate determined from equation (16).
3. For an elevator shaft with equal amounts of pressurization air supplied evenly at the top and bottom of the shaft, piston effect will not result in loss of shaft pressurization provided the mass flow rate of pressurization air is at least as great as the critical mass flow rate determined from equation (17).
4. For single car shafts with high velocities or multiple car shafts with very high velocities the approach of 1 above may not be feasible. For such cases a hazard analysis may provide useful information, and the methods of analysis presented in this **paper** can be used for the fluid flow portion of such a hazard analysis.

## 10. NOMENCLATURE

A	area
a	acceleration
b	$S_a N_a / N_b$
C	flow coefficient
c	$(\dot{m}_{pa} + \dot{m}_{pb}) / K_{bo}$
K	coefficient
m	mass
m	mass flow rate
N	number of floors
P	pressure
Q	volume
t	time
V	elevator car velocity
X	distance of car travel
$\rho$	density
$\Delta P$	pressure difference

### Subscripts

a	above elevator car
b	below elevator car
c	elevator car
crit	critical
e	effective
f	free flow around
i	building
l	lobby
min	minimum
o	outside
s	shaft
p	full operational
t	transitional
tot	total

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<b>U.S. DEPT. OF COMM.</b> <b>BIBLIOGRAPHIC DATA SHEET</b> (See instructions)	<b>1. PUBLICATION OR REPORT NO.</b> <b>NBSIR-88/3751</b>	<b>2. Performing Organ. Report No.</b>	<b>3. Publication Date</b> <b>April 1988</b>
<b>5. AUTHOR(S)</b> John H, Klote			
<b>6. PERFORMING ORGANIZATION</b> (If joint or other than <b>NBS</b> , see instructions) <b>NATIONAL BUREAU OF STANDARDS</b> <b>DEPARTMENT OF COMMERCE</b> <del>WASHINGTON, DC 20334</del> Gaithersburg, MD 20899		<b>7. Contract/Grant No.</b>	<b>8. Type of Report &amp; Period Covered</b>
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<b>10. SUPPLEMENTARY NOTES</b>  <input type="checkbox"/> Document describes a computer program; SF-185, <b>FIPS Software Summary, is attached.</b>			
<b>11. ABSTRACT</b> (A 200-word or less factual summary of most significant <i>information</i> . If document includes a significant bibliography or literature survey, mention it here)			
<b>12. KEY WORDS</b> (Six to twelve entries; alphabetical order; capitalize only proper names; and separate key words by semicolons) elevators (lifts); hazard analysis; piston effect; pressurization; smoke; smoke control			
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## **AIR QUALITY TEST REPORT AND COMPARISON** **(STERILYFT vs STANDARD PASSENGER ELEVATOR FAN)**

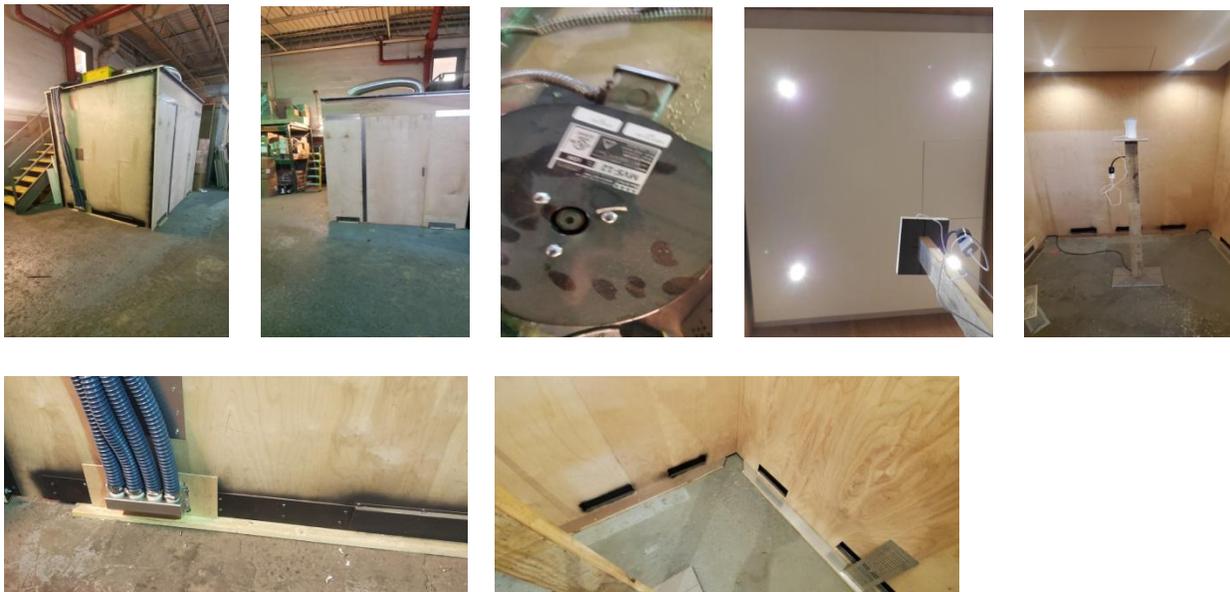
This test, conducted by CEC on 7/8/2020 was conducted to test, record and evaluate the performance of Sterilyft as compared to a standard passenger elevator exhaust fan in the removal of Total Volatile Organic Compounds (TVOC) and Particulate Matter 2.5 microns and less (PM2.5).

### **TESTING EQUIPMENT:**

A mock elevator cab enclosure was constructed at CEC facility, 540 Manida Street, Bronx, NY in dimension to match a standard 3500 Lbs capacity interior volume including a standard drop ceiling (mounted 6" below canopy) and including standard escape hatch, exhaust fan cut out with Man D Tec standard MVS-12 exhaust fan, standard natural base ventilation and SoloBeam LED downlights. Cab doors were replaced by hinged wood door for sealing cab off from shop facility.

Sterilyft unit installed is standard Sterilyft base model air sterilizer with 1 ½" diameter exhaust duct and 6" intake duct with standard canopy start collar and base register installed at opposite corners of cab.

Sampling device is Uho Indoor Air Quality – 9 in 1 Smart Air Monitor which was mounted at center of elevator cab floor and at 5' above floor grade.



Simulated "sneeze" as produced by atomized vegetable glycerol with polyglycerin and artificial additives to produce vaporized particles to fill cab enclosure. VOC content consisted of vaporized ingredients listed above with expelled human exhaled air content. Particulate size of injected media is approximately .03 to 1 micron.

**TESTING ENVIRONMENT:**

Test cab is located in elevator cab manufacturing facility. Ambient average temperature 82.5 deg F, 64% relative humidity. VOCs include cleaning fluids, adhesives, paint fumes, wood dust, common dust, welding fumes and minor intermediate vehicle emissions as well as approximately 50 human occupants. Test cab is closed off from outside shop by wood door however, still open to outside air by way of natural ventilation at base. Test conducted midday during prime occupancy and activity in facility.

**TEST 1 PROCEDURE:**

Initial Air Sampling test is performed by recording of air monitor readings from with the closed cab enclosure, readings in intervals of 10 minutes for a period of 40 minutes. Test is conducted first on enclosure with only standard exhaust fan in use, second test is with Sterilyft unit in use.

**TEST 1.1 (Air Quality sampling, fan only)**

		EXHAUST FAN ONLY				
MEASUREMENT	UNITS	2:30	2:40	2:50	3:00	3:10
TEMPERATURE	DEG F	82.2	82	81.9	81.9	81.7
AIR PRESSURE	hPa	1019.1	1019.1	1019	1019.1	1019.1
TVOC	ppb	310	260	270	160	65
CO	ppm	0	0	0	0	0
OZONE	ppb	15.6	15.2	15.2	16.1	16.7
HUMIDITY	REL %	60.5	61	60.8	61.2	61.6
CO2	ppm	490	497	449	429	415
PM2.5	ug/m3	3	8	18	5	4
NO2	ppb	140.8	136.1	136.5	150.2	161.6

**TEST 1.2 (Air Quality sampling, Sterilyft in use)**

		STERILYFT				
MEASUREMENT	UNITS	3:20	3:30	3:40	3:50	4:00
TEMPERATURE	DEG F	81.9	82	81.9	82.2	82.2
AIR PRESSURE	hPa	1018.9	1018.8	1018.8	1018.5	1018.6
TVOC	ppb	128	107	122	209	145
CO	ppm	0	0	0	0	0
OZONE	ppb	16.5	16.6	16.8	15.6	16.6
HUMIDITY	REL %	61.6	61.3	61.3	60.6	61.4
CO2	ppm	427	422	420	435	425
PM2.5	ug/m3	1	1	9	1	5
NO2	ppb	157.1	157.5	162.4	141.8	157.3

**TEST 1 ANALYSIS**

It can be seen by averaging of data that Sterilyft shows a substantial improvement in air quality readings of decreased TVOC content (33.24% reduction) as well as PM2.5 content (55.26% reduction than in use of fan alone). This is substantial as the lowering of both VOCs and PMs helps reduce respiratory inhalation as well as the existence of possibly harmful particulates such as viruses, bacteria and germs.

AIR SAMPLING - 40 MINUTE - 10 MIN INTERVALS				
AVERAGE				
MEASUREMENT	UNITS	FAN	STERILYFT	% CHANGE
TEMPERATURE	DEG F	81.94	82.04	0.12%
AIR PRESSURE	hPa	1019.08	1018.72	-0.04%
<b>TVOC</b>	<b>ppb</b>	<b>213</b>	<b>142.2</b>	<b>-33.24%</b>
CO	ppm	0	0	0.00%
OZONE	ppb	15.76	16.42	4.19%
HUMIDITY	REL %	61.02	61.24	0.36%
CO2	ppm	456	425.8	-6.62%
<b>PM2.5</b>	<b>ug/m3</b>	<b>7.6</b>	<b>3.4</b>	<b>-55.26%</b>
NO2	ppb	145.04	155.22	7.02%

### TEST 2 PROCEDURE:

VOC / PM2.5 Introduction and Evacuation test is performed by recording of air monitor readings from with the closed cab enclosure, readings in intervals of 3 minutes for a period of 21 minutes and final reading at 50 minutes after test initiation. Test is conducted first on enclosure with only standard exhaust fan in use, second test is with Sterilyft unit in use. Introduction of VOC / PM2.5 (simulated sneeze) is introduced at 2 minutes into test and recorded at first 3 minute interval.

### TEST 2.1 (VOC/PM 2.5 Handling, fan only)

VOC / PM2.5 INTRODUCTION AND EVACATION TEST - FAN ONLY										
FAN ONLY		10:40	10:43	10:46	10:49	10:52	10:55	10:58	11:01	11:30
MEASUREMENT	UNITS	START	INTRO							
TEMPERATURE	DEG F	82.4	82.2	82.4	82.4	82.4	82.2	82.2	82.4	82.4
AIR PRESSURE	hPa	1016.5	1016.5	1016.5	1016.5	1016.5	1016.4	1016.4	1016.5	1016.4
<b>TVOC</b>	<b>ppb</b>	<b>331</b>	<b>408</b>	<b>398</b>	<b>372</b>	<b>358</b>	<b>369</b>	<b>382</b>	<b>363</b>	<b>318</b>
CO	ppm	0	0.9	0.9	1.2	0	0	0.9	0	0
OZONE	ppb	9.3	9.2	9.3	9.3	9.4	9.3	9.3	9.4	14.7
HUMIDITY	REL %	64	64.3	64.1	64	63.9	64.3	64.3	64.1	64.2
CO2	ppm	491	503	500	500	495	482	490	496	474
<b>PM2.5</b>	<b>ug/m3</b>	<b>6</b>	<b>25</b>	<b>12</b>	<b>18</b>	<b>17</b>	<b>19</b>	<b>10</b>	<b>22</b>	<b>18</b>
NO2	ppb	37.5	35.7	37	37.6	38.2	37.8	37.4	38.5	125

### TEST 2.2 (VOC/PM 2.5 Handling, Sterilyft)

VOC / PM2.5 INTRODUCTION AND EVACATION TEST - STERILYFT										
STERILYFT		11:30	11:33	11:36	11:39	11:42	11:45	11:48	11:51	12:20
MEASUREMENT	UNITS	START	INTRO							
TEMPERATURE	DEG F	82.4	82.4	82.4	82.6	82.6	82.6	82.6	82.6	82.9
AIR PRESSURE	hPa	1016.5	1016.2	1016.3	1016.3	1016.3	1016.3	1016.2	1016.3	1015.8
<b>TVOC</b>	<b>ppb</b>	<b>315</b>	<b>385</b>	<b>152</b>	<b>148</b>	<b>145</b>	<b>137</b>	<b>135</b>	<b>127</b>	<b>92</b>
CO	ppm	0	0.5	0	0	0	0	0	0	0
OZONE	ppb	14.8	14.7	14.8	14.9	14.9	15.1	14.9	15	15.3
HUMIDITY	REL %	64.3	64.2	64	64	63.7	63.8	63.8	63.8	63.5
CO2	ppm	485	474	482	485	484	476	475	473	463
<b>PM2.5</b>	<b>ug/m3</b>	<b>8</b>	<b>24</b>	<b>9</b>	<b>10</b>	<b>5</b>	<b>8</b>	<b>3</b>	<b>5</b>	<b>1</b>
NO2	ppb	120	125	125.5	127.6	126.8	130.7	127	128.7	131.5

TEST 2 ANALYSES

It can be seen by comparison of test data that use of fan only exhibits a minimal result of removal of both TVOC and PM2.5 by showing of a circulation of air contaminants which remain in the enclosure. This shows an inability of a standard fan to effectively lift and remove airborne particles and contaminants from within the volume of air below. This is due to lesser CFM draw coupled by lack of introduction of forced air to push up the air volume. By use of higher air movement (710CFM) as well as looped circulation creating a distinct air flow, **Sterilyft's efficiency is by far more effective.** Variable from introduction of contaminant to enclosure to completion for fan is a reduction in VOC of 22.06% whereas Sterilyft is 76.10%, netting an increased evacuation efficiency of 54.06%. Variable from introduction of contaminant to enclosure to completion for fan is a reduction in PM 2.5 content of 28% whereas Sterilyft is 95.83%, netting an increased evacuation efficiency of 67.83%. Furthermore, variable from start of test to completion for fan is a reduction in VOC of 3.93% whereas Sterilyft is 70.79%, netting an increased evacuation efficiency of 66.86%. Variable from start of test to completion for fan is a INCREASE in PM 2.5 content of 200% whereas Sterilyft is reduced 87.5%, netting an increased evacuation efficiency of 287.5%. The data leads to a clear conclusion that the **Sterilyft system shows a substantially improved rate of circulation of the air within an elevator as well as in the removal of elevator cab enclosure contaminants within the air.** This coupled by UV-C irradiation should prove to effectively remove all or most of the contaminants and de-activate any remainder prior to providing clean air to the passengers within the cab.

VOC / PM2.5 EVALUATION FAN ONLY					VOC / PM2.5 EVALUATION FAN ONLY				
START	INTRO	COMPL	VAR S-C	VAR I-C	START	INTRO	COMPL	VAR S-C	VAR I-C
82.4	82.2	82.4	0.00%	0.24%	82.4	82.4	82.9	0.61%	0.61%
1016.5	1016.5	1016.4	-0.01%	-0.01%	1016.5	1016.2	1015.8	-0.07%	-0.04%
331	408	318	-3.93%	-22.06%	315	385	92	70.79%	-76.10%
0	0.9	0	0.00%	100.00%	0	0.5	0	0.00%	100.00%
9.3	9.2	14.7	58.06%	59.78%	14.8	14.7	15.3	3.38%	4.08%
64	64.3	64.2	0.31%	-0.16%	64.3	64.2	63.5	-1.24%	-1.09%
491	503	474	-3.46%	-5.77%	485	474	463	-4.54%	-2.32%
6	25	18	200.00%	-28.00%	8	24	1	87.50%	-95.83%
37.5	35.7	125	233.33%	250.14%	120	125	131.5	9.58%	5.20%

CONCLUSION

Sterilyft is an obvious solution to circulating and sterilizing the air from within an elevator enclosure.

## **UL Remote Air Quality Monitoring**

CEC is pleased to receive copy of UL testing of the in cab air quality of Sterilyft in comparison side by side to a standard exhaust fan ventilated elevator during a one month trial period utilizing two actively used passenger elevators in an occupied building.

CEC is very thankful for the cooperation and coordination of this test in conjunction with an installation performed by ELCON Enterprises, Inc in Virginia.

For the test results below, Car 12 represents passenger elevator with Sterilyft and car 13 represents passenger elevator with only typical type exhaust fan.

Following is our takeaway of the attached UL results:

### **PARTICULATE MATTER (PM10) – Particles less than 10 microns**

UL REL = 50 ug/m<sup>3</sup> [World Health Organization (WHO) respirable particles (24-Hour mean)]

Car 13 = 13.1 ug/m<sup>3</sup> avg

Car 12 = 0.59 ug/m<sup>3</sup> avg

Sterilyft represents 95.5% less contamination than exhaust fan only. It should be noted that viral carriers (sneeze, cough or expelled particulate) falls within about 75% approximately 10 microns and less than 25% 1-5 microns.

### **PARTICULATE MATTER (PM2.5) – Particles less than .5 microns**

UL REL = 35 ug/m<sup>3</sup> [EPA (NAAQS) (24-Hour mean)]

Car 13 = 12.6 ug/m<sup>3</sup> avg

Car 12 = 0.53 ug/m<sup>3</sup> avg

Sterilyft represents 95.79% less contamination than exhaust fan only. It should be noted that viral carriers (sneeze, cough or expelled particulate) falls within about 75% approximately 10 microns and less than 25% 1-5 microns.

### **PARTICULATE MATTER (PM1) – Particles less than 1 micron**

Car 13 = 9.86 ug/m<sup>3</sup> avg

Car 12 = 0.25 ug/m<sup>3</sup> avg

Sterilyft represents 97.46% less contamination than exhaust fan only. It should be noted that bacteria measures approximately .4 microns and virus range from .02 to .25 microns. Corona virus in particular is approximately .125 microns

### **PARTICULATE MATTER (PM>0.3) – Particles less than .3 microns**

Car 13 = 2197 particles avg  
Car 12 = 237 particles avg

Sterilyft represents 89.21% less contamination than exhaust fan only. It should be noted that bacteria measures approximately .4 microns and virus range from .02 to .25 microns. Corona virus in particular is approximately .125 microns

### **VOLITILE ORGANIC COMPOUNDS (VOC)**

UL REL = 500 ug/m3 (300 ppb) [US Green Building Council]

Car 13 = 422 ug/m3 avg  
Car 12 = 284 ug/m3 avg

Sterilyft represents 32.7% less contamination than exhaust fan only. As car 13 is at the threshold for Green Building standards, car 12 is at slightly over half of the standard.

### **SIGNIFICANCE**

As we are all reminded by CDC, WHO and NIH, the spread of Corona virus (as well as many other infectious contagion, is transmitted via viral carriers be it sneezing, coughing or other expelled droplet particles from human to human. This being said, it is abundantly clear that by the reductions of these particles in excess of 95% as in the case of a typical elevator will substantially reduce the exposure of passengers in the elevator to these transmission vehicles. In addition, as to free floating bacterial or viral pathogens, the further reduction of particles in the sub micron area clearly shows further risk mitigation of bacterial or viral transmission by the reasonable result in reduction of particles less than 0.3 microns by over 89%.

Further efficacy testing will be conducted on bacterial and viral inactivation however, with UV-C 254nm irradiation trials widely tested and accepted, the added result of the efficacies in the area of 99.97% success is substantial be it as the carriers of infectious particulates and infectious particulates themselves are removed from the passenger cab enclosure in excess of 89-97% as determined above.

ATTACHED: UL remote air quality monitoring test results dated 8/20/20, data collection from 7/20/20 through 8/20/20. Building and building owner name has been redacted for privacy.



UL  
 3251 Old Lee Hwy #100  
 Fairfax, VA 22030  
 o: (703) 323-4400  
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August 20, 2020

[Redacted]

Dear [Redacted]

**Remote Air Quality Monitoring**

As requested by [Redacted], UL is conducting screening level air quality monitoring in the above listed address in McLean, Virginia. Remote air quality monitoring units were installed July 20, 2020 through August 20, 2020. This interim letter style report records our measurements, conclusions and recommendations.

**Screening Locations**

Monitoring was conducted in the following areas:

- Elevator 12 Cab
- Elevator 13 Cab (control)

**Description**

Monitoring in Elevator 12 and 13 Cab was conducted as part of a comparative study performed over a month to determine the effectiveness of an air purifier system installed within the cab. The CEC cab sanitizing unit was installed in Elevator 12 Cab. The Elevator 13 Cab served as the control location with no system installed for comparison. The CEC system is equipped with a dual pleated HEPA filter, UVC germicidal lamp, and 4.5-watt UVC germicidal fluorescent lamp.

**Monitoring Hardware Specifications**

The Senseware IAQ Indoor Air Package Modules remote monitors were deployed with the following sensors: Temperature, Particulate Matter less than 2.5 microns (PM<sub>2.5</sub>), Particulate Matter less than 10 microns (PM<sub>10</sub>), Carbon Monoxide (CO), Carbon Dioxide (Calculated Equivalency), Volatiles Organic Compounds (VOCs), Nitrogen Dioxide (NO<sub>2</sub>) and Noise.

Sensor	Resolution	Measurement Range	Accuracy
Nitrogen dioxide (NO <sub>2</sub> )	20 ppb	0 to 5 ppm	+/-15%
Particulate Matter ≤2.5 microns (PM <sub>2.5</sub> )	1 µg/m <sup>3</sup>	0-500 µg/m <sup>3</sup>	N/A
Particulate Matter ≤10 microns (PM <sub>10</sub> )	1 µg/m <sup>3</sup>	0-500 µg/m <sup>3</sup>	N/A
Volatile Organic Compounds (VOCs)	1 ppb (0-2008) 6 ppb (2008-11110)	0 to 60000 ppb	N/A

	32 ppb (11110 -60000)		
Temperature (degrees Fahrenheit [°F])	0.1 °F	-40 to 257 °F	0.5 °F

### Recommended Standards

In this report, UL has included various standards, threshold limit values, time weighted averages, or other recommended acceptable standards for various indoor air pollutants based on the findings and publications of several U.S. government agencies, independent industrial hygiene organizations, and other bodies. Furthermore, based on our professional opinion, we have selected the most appropriate guidelines in interpreting the data gathered during this screening. If further information is required in appreciating the guidelines used by UL, please feel free to request such information.

UL has selected the standards listed in Table below to be used as comparative levels for the air quality screening. The standards below reflect the most stringent standard available where more than one exists. It should be noted that standards from the ACGIH reflect personal exposure levels which cannot be derived from the screening data in the tables below.

Table Key	Name	Recommended Exposure Limit (REL)	Regulatory Agency
CO	Carbon Dioxide	9 ppm	ASHRAE Std 62.1-2016 Selected EPA NAAQS
CO <sub>2</sub> Equivalency	Carbon Dioxide	Outdoors plus 700	ASHRAE Std 62.1-2016
NO <sub>2</sub>	Nitrogen dioxide	200 ppb	ACGIH TLV (8-hour TWA)
PM <sub>2.5</sub>	Particulate Matter 2.5 microns	35 µg/m <sup>3</sup> (24 hours)	EPA (NAAQS) (24-hr mean)
PM <sub>10</sub>	Particulate Matter 10 microns	50 µg/m <sup>3</sup>	World Health Organization (WHO) (PM <sub>10</sub> ) respirable particles (24-hr mean)
VOC	Volatile Organic Compounds	500 ug/m <sup>3</sup> (300 ppb)	US Green Building Council *See additional information regarding this standard in Attachment B
Temperature	Temperature	Summer: 73-79°F Winter: 68-75°F	ASHRAE Std 62.1-2016
Humidity	Humidity	Summer: 30-65% Winter: 20-65%	ASHRAE Std 62.1-2016

## Results and Conclusions

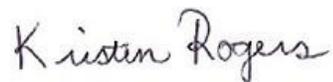
UL conducted continuous air monitoring from July 20, 2020 through August 20, 2020 in the Elevator 12 Cab and Elevator 13 Cab.

Please note the following findings:

- The data collected indicate that the indoor air quality in the monitored locations are within the selected recommended regulatory air quality standards. Measurements were collected for dusts (PM<sub>10</sub> and PM<sub>2.5</sub>), VOCs, and carbon dioxide. While fluctuations were observed, no sustained exceedances in contaminant levels were identified. VOC levels were briefly elevated likely due to cleaning and sanitizing products used during the scheduled times of janitorial operations in the building.
- Temperature and relative humidity were within the ASHRAE thermal comfort envelope during normal office hours.

See details in graphical form in Attachment C for each constituent.

For UL,



Kristen Rogers  
Senior Client Services Specialist  
Environment and Sustainability  
UL

Attachments:

- Glossary of Technical Terms
- Attachment A. Senseware Module Technical Information
- Attachment B. Description of Recommended Volatile Organic Compound (VOC) Concentrations
- Attachment C. Monitoring Data Results Graphs

<b>Glossary of Technical Terms</b>	
<b>ASHRAE Std 62.1-2016</b>	American Society of Heating, Refrigerating and Air Conditioning Engineers standard – Ventilation for Acceptable Indoor Air Quality. Standard selected from Table C-1 of the standard, which is based upon the EPA NAAQS for Carbon Monoxide (not to be exceeded more than once per year).
<b>ACGIH TLV (8-hour TWA)</b>	American Conference of Industrial Hygienist – Threshold Limit Value, Time-Weighted Average – average exposure limit based upon a 8 hour work day
<b>EPA (NAAQS) (24-hr mean)</b>	Environmental Protection Agency, National Ambient Air Quality Standard, 24 hour averaging time.
<b>Carbon dioxide (CO<sub>2</sub>)</b>	A byproduct of respiration and normal constituent of the atmosphere. Measurement of carbon dioxide can provide an indication of ventilation rates in a building and/or a threshold of comfort
<b>Carbon monoxide (CO)</b>	A toxic byproduct of fuel combustion. While odorless, carbon monoxide gas is often accompanied by other odorous combustion products (aldehydes, oxides of nitrogen, etc.)
<b>Counts per cubic meter of air (count/m<sup>3</sup>)</b>	A calculated unit of measurement for quantifying airborne mold spores per unit volume of air
<b>Formaldehyde (HCHO)</b>	A pungent, organic compound associated with certain new furnishings, glues, pressed woods, vehicle exhaust and tobacco smoke. Reacts in the atmosphere to become a component of smog
<b>Hydrogen Sulfide (H<sub>2</sub>S)</b>	Or sewer gas. A toxic and flammable gas associated with rotten egg odor from the bacterial breakdown or organic matter. Detectable by the human sense of smell at extremely low concentration.
<b>Micrograms per cubic meter (µg/m<sup>3</sup>)</b>	A unit of concentration common to particles and gases which describes the weight or mass of the contaminant per unit volume of air. A microgram is 1/1,000,000th of a gram.
<b>Micrometer (µm)</b>	A common unit of measurement for microscopic particles. Unit of measure that is 1/1,000,000th the length of a meter
<b>Nitrogen Dioxide (NO<sub>2</sub>)</b>	A toxic and pungent gas common to internal combustion engines and power plants. Reaction products include ozone.
<b>Relative humidity</b>	The amount of water vapor that exists in a gaseous mixture of air and water relative to temperature. Measured in %.
<b>Respirable Suspended Particulate</b>	A classification of dust which describes a particle size range averaging less than 10 micrometers (µm) in diameter. Excessive particles in the respirable range are more likely to be implicated in respiratory distress
<b>Sulfur Dioxide (SO<sub>2</sub>)</b>	A toxic gas associated common to coal burning, power plants, and the incomplete combustion of lower grade fuels.
<b>Temperature</b>	A physical property of air describing heat or cold measured in degrees Fahrenheit
<b>Total Volatile Organic Compounds (TVOCs)</b>	An aggregate measure of volatile organic compounds in air expressed in ppm or µg/m <sup>3</sup> .
<b>Ultrafine Particles</b>	A classification of airborne particles with diameters in the range of 0.02 – 1.0 micrometers characterized by their ability to reach the gas exchange regions of the lung; under considerable investigation as a trigger for respiratory distress
<b>Volatile Organic Compounds</b>	Classes of organic chemical compounds (containing carbon) with high enough vapor pressures to exist as gases under normal temperature and pressure conditions. Odors common to fuels, paints, new furnishings, etc.



## ATTACHMENT A

### Senseware Module Technical Information

IAQ Sensor Package - T/RH, VOC, PM, CO<sub>2</sub>, and more, all in one place

Written by Nathan Sacks:

Senseware's Indoor Air Quality (IAQ) Sensor Package makes it easy to monitor a variety of environmental conditions. The sensor can provide the following sensor data depending on your ordered configuration: Temperature, Relative Humidity, Total Volatile Organic Compounds (TVOC), Particulate Matter (PM<sub>1.0</sub>, PM<sub>2.5</sub>, PM<sub>10</sub>, and total number of counted particle sizes), CO<sub>2</sub>, and additional gases.



#### Installation:

Plug the IAQ Sensor Package into a wall outlet using the provided wall power adapter. The CO<sub>2</sub> and VOC sensors automatically calibrate and will reach full operational accuracy within 24-48 hours, although readings will be available before this time.

**ATTACHMENT B****Description of Recommended Volatile Organic Compound (VOC) Concentrations**Volatile Organic Compounds (VOCs) – Background Information

Organic chemicals that may be emitted as gases or “volatilize” at room temperatures are described as volatile organic compounds. These compounds are released by “off-gassing” from diverse sources, such as occupants, pests, manufactured building products, furnishings, paints, cleaners, etc. In commercial buildings, it is not uncommon to find VOC levels two to ten times higher than outdoor levels since there are multiple sources in buildings. However, they are normally present in very dilute concentrations in the air, usually measurable in parts per billion (ppb) or micrograms per cubic meter of air ( $\mu\text{g}/\text{m}^3$ ). In assessing these compounds there are three factors of concern: odors, irritation and potential health effects to toxic effects. No federal agency has set permissible exposure levels for mixtures of these compounds in the indoor air. The Molhave Dose Response scale is generally accepted as a guideline for interpreting TVOC exposure in commercial environments. Molhave developed a classification of four grades of TVOC concentrations and Healthy Buildings has adopted that classification with the slight modification that extends the “comfort range” for TVOCs from undetectable to  $500 \mu\text{g}/\text{m}^3$  (300 ppb). This reflects the standard adopted by the US Green Building Council in their Leadership in Energy and Environmental Design (LEED™) rating system for “Green,” or “environmentally friendly” buildings. This was in turn based on the standard adopted by the State of Washington and is still the most stringent standard for TVOCs in the United States.

<b>Tentative Dose Response to TVOCs</b>			
<b>Grade</b>	<b>Airborne TVOC* concentration (<math>\mu\text{g}/\text{m}^3</math>)</b>	<b>Symptoms</b>	<b>Effects</b>
A	<500 (<300 ppb)	No irritation or discomfort expected	The comfort range
B	500-3,000 (300 – 1,800 ppb)	Irritation and discomfort possible if other exposures interact	The multi-factorial exposure range
C	3,000-25,000 (1,800 – 15,000 ppb)	Exposure effect and probable headache possible if other exposures interact	The discomfort range
D	>25,000 (25,000 ppb)	Additional neurotoxic effects other than headache may occur	The toxic range

\*Via photoionization measurement, equivalent to isobutylene.



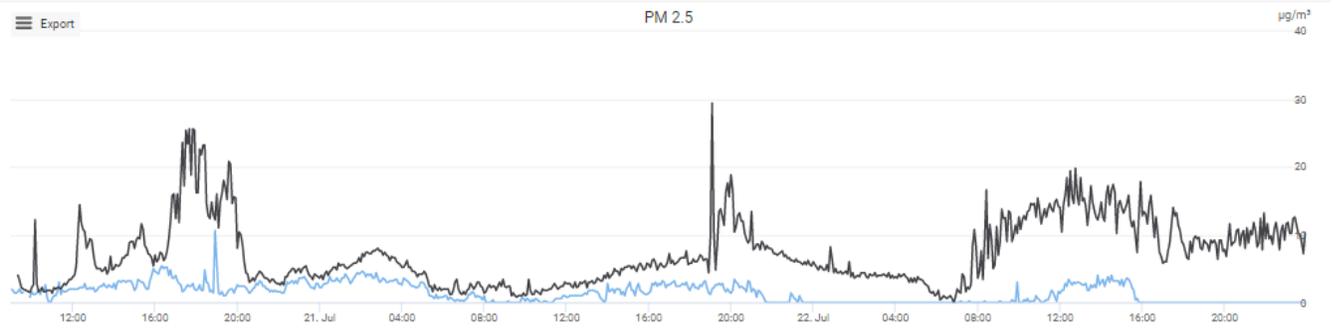
## ATTACHMENT C

### Monitoring Data Results Graphs

Blue: Elevator 12

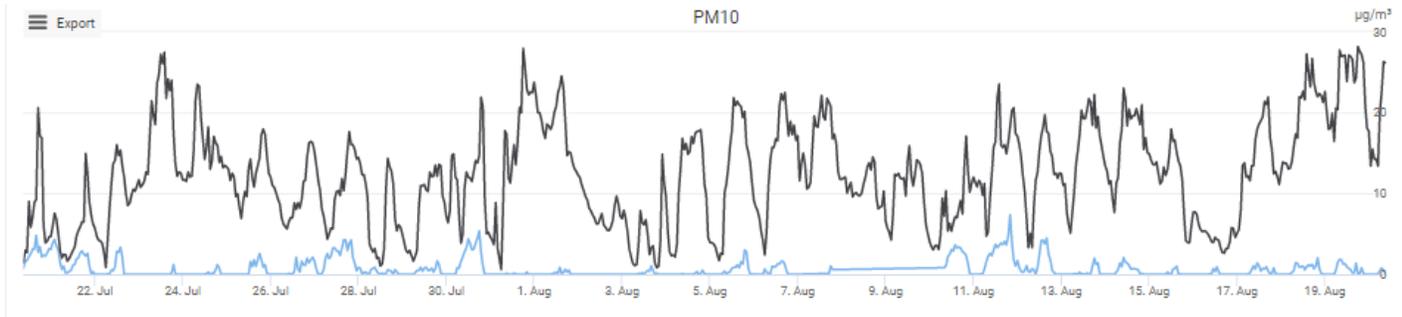
Black = Elevator 13

#### Particulate Matter (PM<sub>2.5</sub>)



PM<sub>2.5</sub> levels were noted to average higher in Elevator 13. All peaks fell below 30 µg/m<sup>3</sup>.

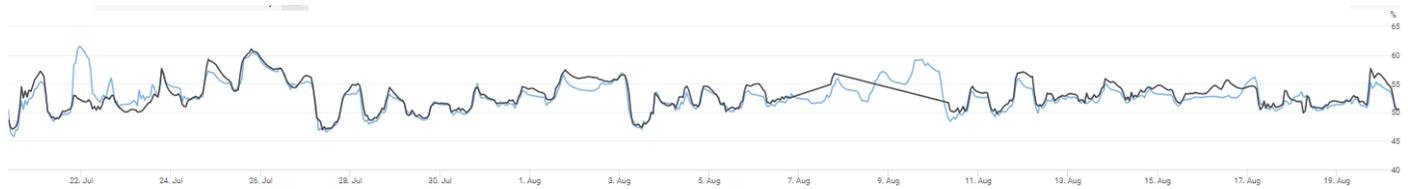
#### Particulate Matter (PM<sub>10</sub>)



PM<sub>10</sub> levels were noted higher in Elevator 13. All levels averaged below our recommended limit of 50 µg/m<sup>3</sup>.

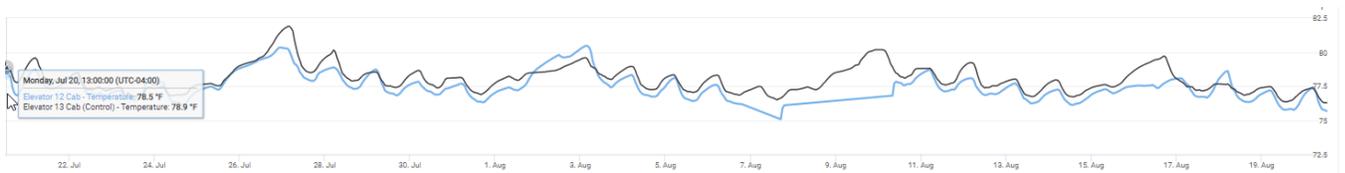


### Humidity



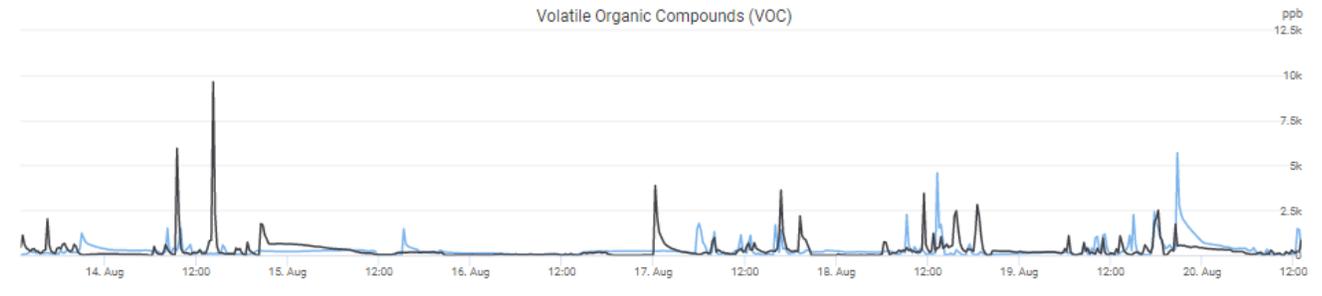
Humidity was noted to be tightly controlled and well within seasonal norms in both sample areas.

### Temperature



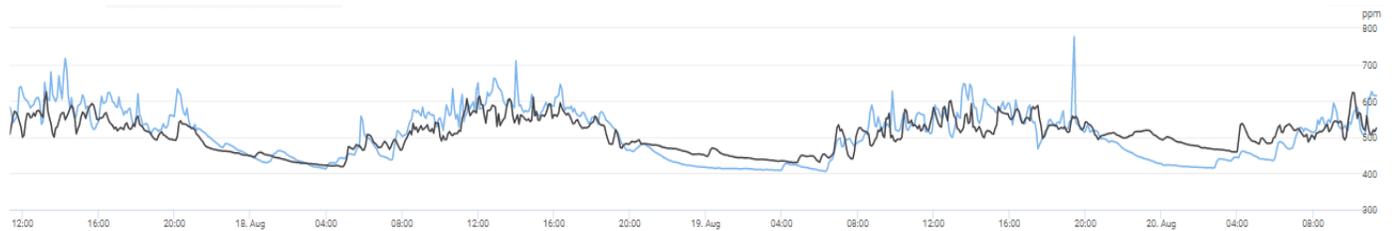
Temperatures were noted to be within seasonal norms during occupied periods.

### Volatile Organic Compounds



VOC levels averaged 284  $\mu\text{g}/\text{m}^3$  in Elevator 12 and 422  $\mu\text{g}/\text{m}^3$  in Elevator 13 with several peaks that coincided with janitorial operations in the Elevator Lobby.

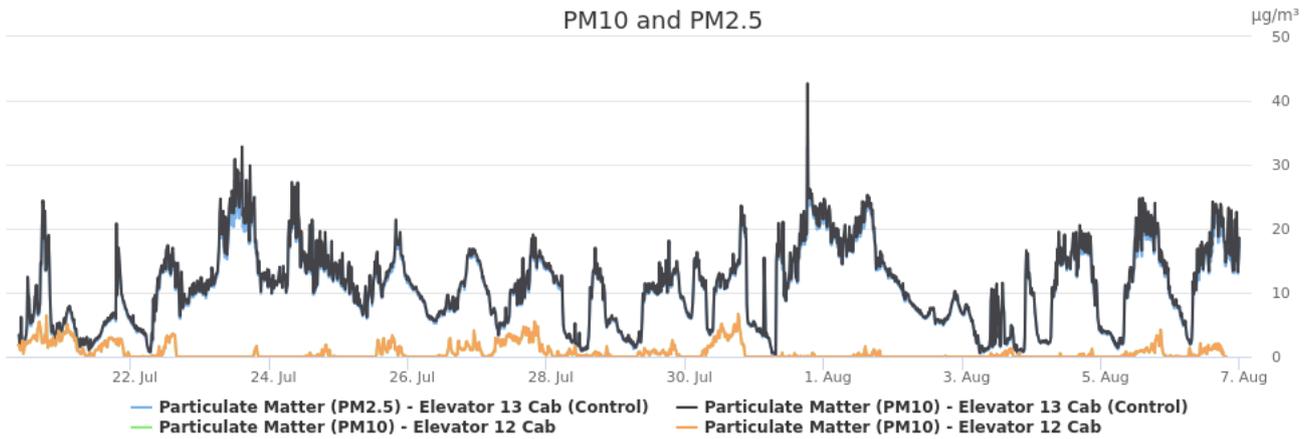
### Carbon Dioxide



Carbon Dioxide levels were well below our recommended limit of outdoors plus 700 ppm.

2007057DC

Elevator 12 is equipped with a CEC Cab Sanitizing Unit System



Particulate Matter (PM10) - Elevator 13 Cab (Control)

**13.1 µg/m<sup>3</sup>**

Average

Particulate Matter (PM10) - Elevator 12 Cab

**0.59 µg/m<sup>3</sup>**

Average

Particulate Matter (PM2.5) - Elevator 13 Cab (Control)

**12.6 µg/m<sup>3</sup>**

Average

Particulate Matter (PM2.5) - Elevator 12 Cab

**0.53 µg/m<sup>3</sup>**

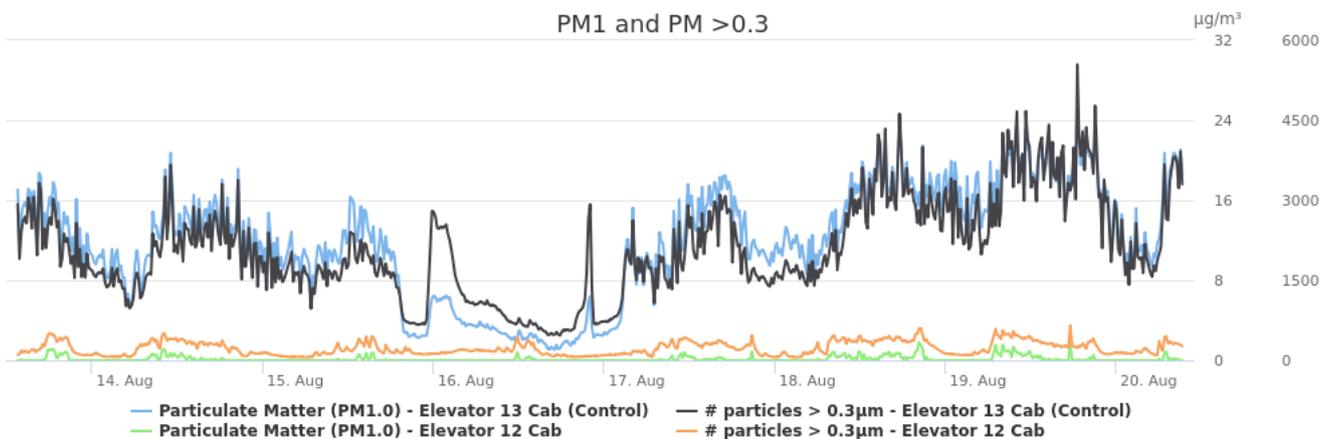
Average

Time	Particulate Matter (PM10) - Elevator 13 Cab (Control) µg/m <sup>3</sup>	Particulate Matter (PM10) - Elevator 12 Cab µg/m <sup>3</sup>
07/20/20 12:00 AM	8.41	2.67
07/21/20 12:00 AM	5.49	1.80
07/22/20 12:00 AM	9.07	0.55
07/23/20 12:00 AM	18.2	0.06
07/24/20 12:00 AM	15.4	0.14
07/25/20 12:00 AM	12.3	0.58
07/26/20 12:00 AM	9.88	0.63
07/27/20 12:00 AM	10.9	2.22
07/28/20 12:00 AM	7.60	0.33
07/29/20 12:00 AM	8.20	0.42

Time	Particulate Matter (PM10) - Elevator 13 Cab (Control) $\mu\text{g}/\text{m}^3$	Particulate Matter (PM10) - Elevator 12 Cab $\mu\text{g}/\text{m}^3$
07/30/20 12:00 AM	11.1	1.81
07/31/20 12:00 AM	14.1	0.03
08/01/20 12:00 AM	18.8	0.14
08/02/20 12:00 AM	7.98	0.00
08/03/20 12:00 AM	4.78	0.14
08/04/20 12:00 AM	11.5	0.06
08/05/20 12:00 AM	12.2	0.63
08/06/20 12:00 AM	13.9	0.59
08/07/20 12:00 AM	16.5	0.59
08/08/20 12:00 AM	11.0	
08/09/20 12:00 AM	10.5	
08/10/20 12:00 AM	7.48	2.27
08/11/20 12:00 AM	14.3	2.39
08/12/20 12:00 AM	12.7	1.33
08/13/20 12:00 AM	15.2	0.28
08/14/20 12:00 AM	16.3	0.54
08/15/20 12:00 AM	12.1	0.12
08/16/20 12:00 AM	4.85	0.10
08/17/20 12:00 AM	14.4	0.39
08/18/20 12:00 AM	19.5	0.67
08/19/20 12:00 AM	23.6	0.56
08/20/20 12:00 AM	15.7	0.09

Time	Particulate Matter (PM2.5) - Elevator 13 Cab (Control) $\mu\text{g}/\text{m}^3$	Particulate Matter (PM2.5) - Elevator 12 Cab $\mu\text{g}/\text{m}^3$
07/20/20 12:00 AM	8.09	2.45
07/21/20 12:00 AM	5.19	1.65
07/22/20 12:00 AM	8.78	0.49
07/23/20 12:00 AM	17.2	0.05
07/24/20 12:00 AM	15.0	0.11
07/25/20 12:00 AM	12.0	0.52
07/26/20 12:00 AM	9.55	0.58
07/27/20 12:00 AM	10.6	2.09
07/28/20 12:00 AM	7.34	0.27
07/29/20 12:00 AM	7.97	0.38
07/30/20 12:00 AM	10.8	1.72

Time	Particulate Matter (PM2.5) - Elevator 13 Cab (Control) $\mu\text{g}/\text{m}^3$	Particulate Matter (PM2.5) - Elevator 12 Cab $\mu\text{g}/\text{m}^3$
07/31/20 12:00 AM	13.5	0.02
08/01/20 12:00 AM	18.2	0.12
08/02/20 12:00 AM	7.76	0.00
08/03/20 12:00 AM	4.59	0.11
08/04/20 12:00 AM	11.2	0.05
08/05/20 12:00 AM	11.7	0.56
08/06/20 12:00 AM	13.2	0.51
08/07/20 12:00 AM	15.7	0.51
08/08/20 12:00 AM	10.8	
08/09/20 12:00 AM	10.1	
08/10/20 12:00 AM	7.13	2.12
08/11/20 12:00 AM	13.8	2.21
08/12/20 12:00 AM	12.4	1.18
08/13/20 12:00 AM	14.5	0.23
08/14/20 12:00 AM	15.5	0.45
08/15/20 12:00 AM	11.8	0.11
08/16/20 12:00 AM	4.63	0.08
08/17/20 12:00 AM	13.9	0.31
08/18/20 12:00 AM	18.7	0.60
08/19/20 12:00 AM	22.2	0.49
08/20/20 12:00 AM	15.0	0.04



# particles > 0.3 $\mu\text{m}$  - Elevator 13 Cab (Control)

**2197**

Average

# particles > 0.3 $\mu\text{m}$  - Elevator 12 Cab

**237**

Average

Particulate Matter (PM1.0) - Elevator 13 Cab (Control)

**9.86  $\mu\text{g}/\text{m}^3$**

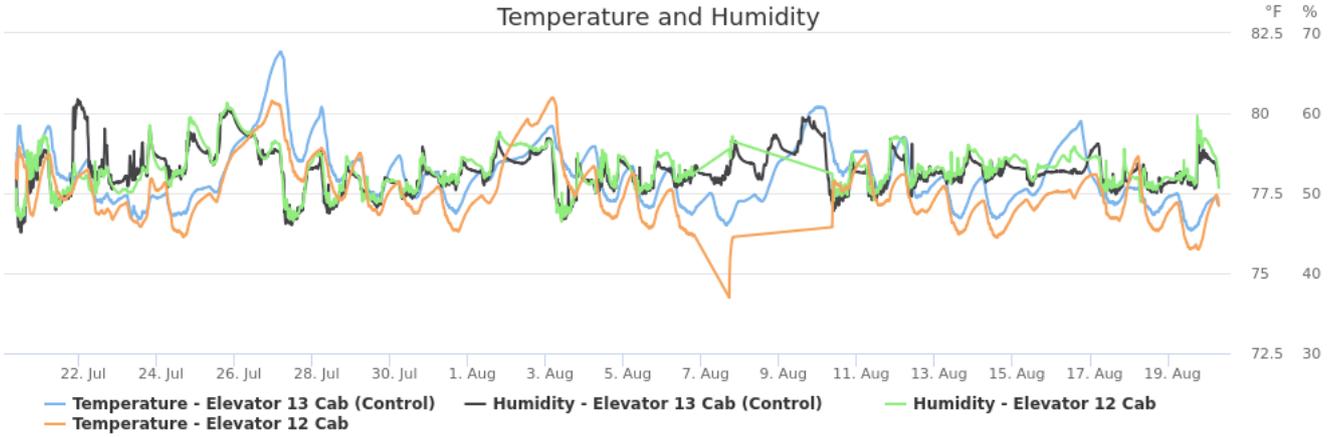
Average

Particulate Matter (PM1.0) - Elevator 12 Cab

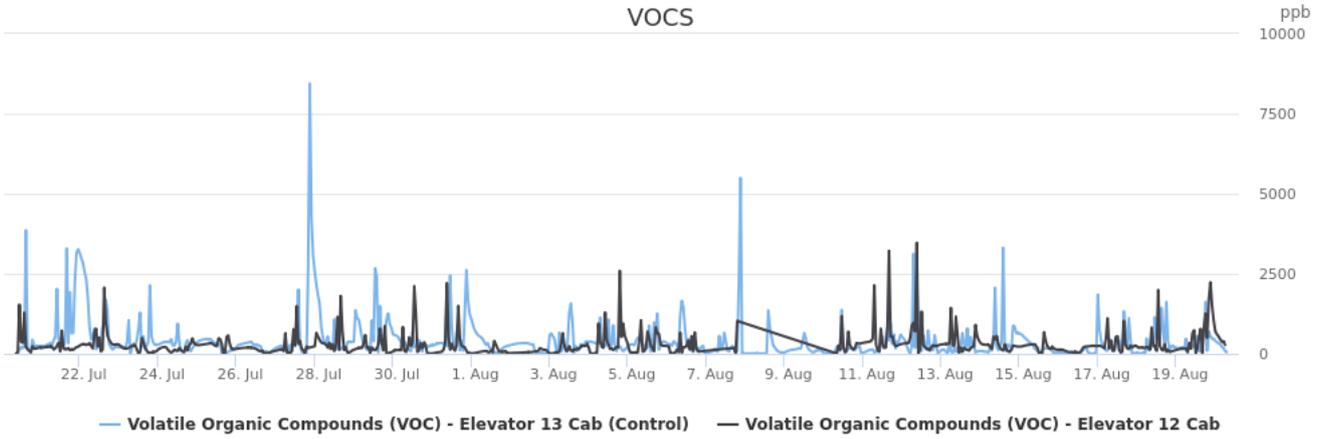
**0.25  $\mu\text{g}/\text{m}^3$**

Average

### Temperature and Humidity



### VOCS





## Coronavirus Disease 2019 (COVID-19)

# COVID-19 Employer Information for Office Buildings

Workers in office buildings may be at risk for exposure to the virus that causes coronavirus disease 2019 (COVID-19). Office building employers, building owners and managers, and building operations specialists can take steps to create a safe and healthy workplace and protect workers and clients.

## Create a COVID-19 workplace health and safety plan.

- Start by reviewing the [CDC Interim Guidance for Businesses and Employers](#). This will provide guidelines and recommendations that all employers can use to protect their workers and clients.

## Before resuming business operations, check the building to see if it's ready for occupancy.

- **Ensure that ventilation systems in your facility operate properly. For building heating, ventilation, and air conditioning (HVAC systems) that have been shut down or on setback, review new construction start-up guidance provided in ASHRAE Standard 180-2018, Standard Practice for the Inspection and Maintenance of Commercial Building HVAC Systems**  .
- Increase circulation of outdoor air as much as possible by opening windows and doors, using fans, and other methods. Do not open windows and doors if doing so poses a safety or health risk for current or subsequent occupants, including children (e.g., allowing outdoor environmental contaminants including carbon monoxide, molds, or pollens into the building).
- Evaluate the building and its mechanical and life safety systems to determine if the building is ready for occupancy. Check for hazards associated with prolonged facility shutdown such as [mold growth](#) , [rodents or pests](#)  , or [issues with stagnant water systems](#), and take appropriate remedial actions.

## Identify where and how workers might be exposed to COVID-19 at work.

Employers are responsible for providing a [safe and healthy workplace](#) .

- Conduct a thorough [hazard assessment](#)  of the workplace to identify potential workplace hazards that could increase risks for COVID-19 transmission.
- **Identify work and common areas where employees could have close contact (within 6 feet) with others — for example meeting rooms, break rooms, the cafeteria, locker rooms, check-in areas, waiting areas, and routes of entry and exit.**
- Include all employees in the workplace in communication plans — for example management, staff, utility employees, relief employees, janitorial staff, maintenance staff, and supervisory staff.
- If contractors are employed in the workplace, develop plans to communicate with the contracting company regarding modifications to work processes and requirements for the contractors to prevent transmission of COVID-19.

Develop hazard controls using [the hierarchy of controls](#) to reduce transmission among workers. Include a combination of controls noted below.

### Engineering controls: Isolate workers from the hazard

- [Modify or adjust seats, furniture, and workstations](#)   to maintain social distancing of 6 feet between employees.
  - Install transparent shields or other physical barriers where possible to separate employees and visitors where social distancing is not an option.
  - Arrange reception or other communal seating area chairs by turning, draping (covering chair with tape or fabric so seats cannot be used), spacing, or removing chairs to maintain social distancing.
- Use methods to physically separate employees in all areas of the facilities including work areas and other areas such as meeting rooms, break rooms, parking lots, entrance and exit areas, and locker rooms.
  - Use signs, tape marks, or other visual cues such as decals or colored tape on the floor, placed 6 feet apart, to indicate where to stand when physical barriers are not possible.
  - Replace high-touch communal items, such as coffee pots, water coolers, and bulk snacks, with alternatives such as pre-packaged, single-serving items.
- **Take steps to improve ventilation in the building:**
  - Increase the percentage of outdoor air (e.g., using economizer modes of HVAC operations) potentially as high as 100% (first verify compatibility with HVAC system capabilities for both temperature and humidity control as well as compatibility with outdoor/indoor air quality considerations).
  - **Increase total airflow supply to occupied spaces, if possible.**
  - Disable demand-control ventilation (DCV) controls that reduce air supply based on temperature or occupancy.
  - Consider using natural ventilation (i.e., opening windows if possible and safe to do so) to increase outdoor air dilution of indoor air when environmental conditions and building requirements allow.
  - **Improve central air filtration:**
    - **Increase air filtration**  to as high as possible (MERV 13 or 14) without significantly diminishing design airflow.
    - Inspect filter housing and racks to ensure appropriate filter fit and check for ways to minimize filter bypass
  - **Consider running the building ventilation system even during unoccupied times to maximize dilution ventilation.**
  - [Generate clean-to-less-clean air movement](#)   **by re-evaluating the positioning of supply and exhaust air diffusers and/or dampers and adjusting zone supply and exhaust flow rates to establish measurable pressure differentials. Have staff work in areas served by “clean” ventilation zones that do not include higher-risk areas such as visitor reception or exercise facilities (if open).**
- Consider using portable high-efficiency particulate air (HEPA) fan/filtration systems to help [enhance air cleaning](#)   (especially in higher risk areas).
- Ensure exhaust fans in restroom facilities are functional and operating at full capacity when the building is occupied.
- **Consider using ultraviolet germicidal irradiation (UVGI)**   **as a supplement to help inactivate the virus.**

## Administrative controls: Change the way people work

- Actively encourage employees who have [symptoms](#) of COVID-19 or who have a sick family member at home with COVID-19 to notify their supervisor and stay home.
  - Employees who appear to have [symptoms](#) upon arrival at work or who become sick during the day should immediately be separated from others, provided a face mask if they are not using one, and sent home with instructions and guidance on how to follow-up with their health care professional.
  - Sick employees should follow [CDC-recommended steps](#). Employees should not return to work until the criteria to [discontinue home isolation](#) are met, in consultation with their healthcare provider.
  - Perform enhanced [cleaning and disinfection](#) after anyone suspected or confirmed to have COVID-19 has been in the workplace.
- Consider conducting daily in-person or virtual health checks (e.g., symptoms and/or temperature screening) of employees before they enter the work site.
  - See [CDC's COVID-19 General Business FAQs](#) for guidance on how to safely conduct employee screening.
  - Develop and implement a policy to prevent employees from congregating in groups while waiting for screening, and maintain a 6-foot separation between employees.
- Stagger shifts, start times, and break times as feasible to reduce the density of employees in common areas such as screening areas, break rooms, and locker rooms.
- Consider posting signs in parking areas and entrances that ask guests and visitors to phone from their cars to inform the administration or security when they reach the facility.
  - Provide directions for visitors to enter the building at staggered times.
- Consider posting signs in parking areas and entrances that ask guests and visitors to wear cloth face coverings if possible, to not enter the building if they are sick, and to stay 6 feet away from employees, if possible.
- [Clean and disinfect](#) high-touch surfaces
  - Follow the [Guidance for Cleaning and Disinfecting](#) to develop, follow, and maintain a plan to perform regular cleanings to reduce the risk of people's exposure to the virus that causes COVID-19 on surfaces.
  - Routinely clean all frequently touched surfaces in the workplace, such as workstations, keyboards, telephones, handrails, printer/copiers, drinking fountains, and doorknobs.
    - If hard surfaces are visibly soiled (dirty), clean them using a detergent or soap and water before you disinfect them.
    - For disinfection, most common, EPA-registered, household disinfectants should be effective as well as [diluted household bleach solutions](#) or alcohol solutions with at least 70% alcohol. A list of [products that are EPA-approved for use against the virus that causes COVID-19](#) [↗](#) is available on the EPA website. Follow the manufacturer's instructions for all cleaning and disinfection products (e.g., concentration, application method, and contact time).
  - Provide employees with disposable wipes and other cleaning materials so that they can properly wipe down frequently touched surfaces before each use.
- Provide employees adequate time to wash their hands and access to soap, clean water, and single use paper towels.
  - Remind employees to [wash their hands](#) often with soap and water for at least 20 seconds. If soap and water are not available, they should use hand sanitizer with at least 60% alcohol.
- Establish policies and practices for social distancing:
  - Remind employees that people may be able to spread COVID-19 even if they do not show symptoms. **Consider all close interactions (within 6 feet) with employees, clients, and others as a potential source of exposure.**

- Prohibit handshaking, hugs, and fist bumps.
- **Limit use and occupancy of elevators to maintain social distancing of at least 6 feet.**
- Encourage the use of outdoor seating areas and social distancing for any small group activities such as lunches, breaks, and meetings.
- For employees who commute to work using public transportation or ride sharing, consider offering the following support:
  - If feasible, offer employees incentives to use forms of transportation that minimize close contact with others (e.g., biking, walking, driving or riding by car either alone or with household members).
  - Ask employees to follow the CDC guidance on how to [protect yourself when using transportation](#).
  - Allow employees to shift their hours so they can commute during less busy times.
  - Ask employees to [wash their hands](#) as soon as possible after their trip.
- [Post signs and reminders](#) at entrances and in strategic places providing instruction on [hand hygiene](#), [COVID-19 symptoms](#), and [cough and sneeze etiquette](#). This should include signs for non-English speakers, as needed.
- Use no-touch waste receptacles when possible.
- Remind employees to avoid touching their eyes, nose, and mouth.
- Employees should wear [a cloth face covering](#) to cover their nose and mouth in all areas of the business.
- CDC recommends wearing a cloth face covering as a measure to contain the wearer's respiratory droplets and help protect their co-workers and members of the general public. Employees should not wear cloth face coverings at work if they have trouble breathing, any inability to tolerate wearing it, or if they are unable to remove it without assistance.
  - Cloth face coverings are not considered personal protective equipment. They may prevent workers, including those who don't know they have the virus, from spreading it to others but may not protect the wearers from exposure to the virus that causes COVID-19.
  - Remind employees and clients that CDC recommends wearing cloth face coverings in public settings where other social distancing measures are difficult to maintain, **especially** in areas of significant community-based transmission. Wearing a cloth face covering, however, does not replace the need to practice social distancing.

## Educate employees and supervisors about steps they can take to protect themselves at work.

- Communication and training should be easy to understand, be in preferred languages spoken or read by the employees, and include accurate and timely information. Topics should include signs and symptoms of infection, staying home when ill, social distancing, personal protective equipment, hand hygiene practices, and identifying and minimizing potential routes of transmission at work, at home, and in the community. Other topics may be considered based on local context and need.
- CDC has free, simple [posters available](#) to download and print, some of which are translated into different languages.
- Provide information and training on what actions employees should take when they are not feeling well (e.g., workplace leave policies, local and state health department information).

## Take actions to maintain a healthy work environment for your employees and clients.

Read the [CDC Interim Guidance for Businesses and Employers](#) to learn about more recommendations for creating new sick leave policies, cleaning, and employee communication policies to help protect your workers and clients.

# Where can I get more information?

You, as the employer, are responsible for responding to COVID-19 concerns and informing employees of the hazards in your workplace. You can use these additional sources for more information on reducing the risk of exposures to COVID-19 at work:

- [CDC Interim Guidance for Businesses and Employers \(COVID-19\)](#)
- [CDC General Business Frequently Asked Questions](#)
- [NIOSH COVID-19 Workplace Safety and Health Topic](#)
- [CDC COVID-19](#)
- [OSHA COVID-19](#) 
- [OSHA Guidelines on Preparing Workplaces for COVID](#)  
- [AIHA Reopening: Guidance for General Office Settings](#)  
- [Building Owners & Managers Association International \(BOMA\): Getting Back to Work: Preparing Buildings for Re-Entry Amid COVID-19](#)  
- CDCINFO: 1-800-CDC-INFO (1-800-232-4636) | TTY: 1-888-232-6348 | website: [cdc.gov/info](https://www.cdc.gov/info)

Page last reviewed: May 27, 2020

Content source: [National Center for Immunization and Respiratory Diseases \(NCIRD\)](#), Division of Viral Diseases

An official website of the United States government.

[Close](#)

We've made some changes to [EPA.gov](#). If the information you are looking for is not here, you may be able to find it on the [EPA Web Archive](#) or the [January 19, 2017 Web Snapshot](#).



## Ozone Generators that are Sold as Air Cleaners

### Additional IAQ Resources

- [Care for Your Air: A Guide to Indoor Air Quality](#)
- [The Inside Story: A Guide to Indoor Air Quality](#)
- [Indoor Air Pollution: An Introduction for Health Professionals](#)
- [Residential Air Cleaners \(Third Edition\) A Summary of Available Information](#)

#### On this page:

- [Introduction and Purpose](#)
- [What is ozone?](#)
- [How is ozone harmful?](#)
  - [Ozone Health Effects and Standards](#)
- [Is there such a thing as "good ozone," and "bad ozone"?](#)
- [Are ozone generators effective in controlling indoor air pollution?](#)
- [If I follow manufacturers' directions, can I be harmed?](#)
- [Why is it difficult to control ozone exposure with an ozone generator?](#)
- [Can ozone be used in unoccupied spaces?](#)
- [What other methods can be used to control indoor air pollution?](#)
- [Conclusions](#)
- [Recommendation](#)
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- [Bibliography](#)

There is a large body of written material on ozone and the use of ozone indoors. However, much of this material makes claims or draws conclusions without substantiation and sound science. In developing *Ozone Generators that are Sold as Air Cleaners*, the EPA reviewed a wide assortment of this literature, including information provided by a leading manufacturer of ozone generating devices. In keeping with EPA's policy of insuring that the information it provides is based on sound science, only peer reviewed, scientifically supported findings and conclusions were relied upon in developing this document.

Several brands of ozone generators have EPA establishment number on their packaging. This number helps EPA identify the specific facility that produces the product. **The display of this number does not imply EPA endorsement or suggest in any way that EPA has found the product to be either safe or effective.**

**Please Note:** EPA does not certify air cleaning devices. The Agency does not recommend air cleaning devices or manufacturers. If you need information on specific devices or manufacturers, one resource you can consult is the [Association of Home Appliance Manufacturers \(AHAM\) EXIT](#), (202) 872-5955. AHAM conducts four certification programs for each category - room air cleaners, room air conditioners, dehumidifiers and refrigerator/freezers. The air cleaner certification program is known as AC-1.

## Introduction and Purpose

Ozone generators that are sold as air cleaners intentionally produce the gas ozone. Often the vendors of ozone generators make statements and distribute material that lead the public to believe that these devices are always safe and effective in controlling indoor air pollution. For almost a century, health professionals have refuted these claims (Sawyer, et. al 1913; Salls, 1927; Boeniger, 1995; American Lung Association, 1997; Al-Ahmady, 1997). The purpose of this document is to provide accurate information regarding the use of ozone-generating devices in indoor occupied spaces. This information is based on the most credible scientific evidence currently available.

Some vendors suggest that these devices have been approved by the federal government for use in occupied spaces. To the contrary, **NO** agency of the federal government has approved these devices for use in occupied spaces. Because of these claims, and because ozone can cause health problems at high concentrations, several federal government agencies have worked in consultation with the U.S. Environmental Protection Agency to produce this public information document.

## What is Ozone?

Ozone is a molecule composed of three atoms of oxygen. Two atoms of oxygen form the basic oxygen molecule--the oxygen we breathe that is essential to life. The third oxygen atom can detach from the ozone molecule, and re-attach to molecules of other substances, thereby altering their chemical composition. It is this ability to react with other substances that forms the basis of manufacturers' claims.

## How is Ozone Harmful?

The same chemical properties that allow high concentrations of ozone to react with organic material outside the body give it the ability to react with similar organic material that makes up the body, and potentially cause harmful health consequences. When inhaled, ozone can damage the lungs. Relatively low amounts can cause chest pain, coughing, shortness of breath and throat irritation. Ozone may also worsen chronic respiratory diseases such as asthma and compromise the ability of the body to fight respiratory infections. People vary widely in their susceptibility to ozone. Healthy people, as well as those with respiratory difficulty, can experience breathing problems when exposed to ozone. Exercise during exposure to ozone causes a greater amount of ozone to be inhaled, and increases the risk of harmful respiratory effects. Recovery from the harmful effects can occur following short-term exposure to low levels of ozone, but health effects may become more damaging and recovery less certain at higher levels or from longer exposures (US EPA, 1996a, 1996b).

- [Ozone and Your Health \(PDF\)](#) (2 pp, 2.5 MB, [About PDF](#))

Manufacturers and vendors of ozone devices often use misleading terms to describe ozone. Terms such as "energized oxygen" or "pure air" suggest that ozone is a healthy kind of oxygen. Ozone is a toxic gas with vastly different chemical and toxicological properties from oxygen. Several federal agencies have established health standards or recommendations to limit human exposure to ozone. These exposure limits are summarized in Table 1.

**Table 1. Ozone Heath Effects and Standards**

Health Effects	Risk Factors	Health Standards*
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<p><b>Potential risk of experiencing:</b></p> <p>Decreases in lung function</p> <p>Aggravation of asthma</p> <p>Throat irritation and cough</p> <p>Chest pain and shortness of breath</p> <p>Inflammation of lung tissue</p> <p>Higher susceptibility to respiratory infection</p>	<p><b>Factors expected to increase risk and severity of health effects are:</b></p> <p>Increase in ozone air concentration</p> <p>Greater duration of exposure for some health effects</p> <p>Activities that raise the breathing rate (e.g., exercise)</p> <p>Certain pre-existing lung diseases (e.g., asthma)</p>	<p>The <b>Food and Drug Administration (FDA)</b> requires ozone output of indoor medical devices to be no more than 0.05 ppm.</p> <p>The <b>Occupational Safety and Health Administration (OSHA)</b> requires that workers not be exposed to an average concentration of more than 0.10 ppm for 8 hours.</p> <p>The <b>National Institute of Occupational Safety and Health (NIOSH)</b> recommends an upper limit of 0.10 ppm, not to be exceeded at any time.</p> <p>EPA's National Ambient Air Quality Standard for ozone is a maximum 8 hour average outdoor concentration of 0.08 ppm</p> <ul style="list-style-type: none"> <li>• See - <a href="#">the Clean Air Act</a></li> </ul>
<p>(* ppm = parts per million)</p>		

## Is There Such a Thing as "Good Ozone" and "Bad Ozone"?

The phrase "good up high - bad nearby" has been used by the U.S. Environmental Protection Agency (EPA) to make the distinction between ozone in the upper and lower atmosphere. Ozone in the upper atmosphere--referred to as "stratospheric ozone"--helps filter out damaging ultraviolet radiation from the sun. Though ozone in the stratosphere is protective, ozone in the atmosphere - which is the air we breathe - can be harmful to the respiratory system. Harmful levels of ozone can be produced by the interaction of sunlight with certain chemicals emitted to the environment (e.g., automobile emissions and chemical emissions of industrial plants). These harmful concentrations of ozone in the atmosphere are often accompanied by high concentrations of other pollutants, including nitrogen dioxide, fine particles and hydrocarbons. *Whether pure or mixed with other chemicals, ozone can be harmful to health.*

- "[Good up High - Bad Nearby](#)"
  - You can order the Office of Air Quality Planning and Standard's "**Good Up High Bad Nearby**", EPA publication number **EPA-451/K-03-001**, June 2003
- and
- [Ozone and Your Health \(PDF\)](#)  
(2 pp, 2.5 MB, [About PDF](#)) EPA publication number **EPA-452/F-99-003**, September 1999

## Are Ozone Generators Effective in Controlling Indoor Air Pollution?

*Available scientific evidence shows that at concentrations that do not exceed public health standards, ozone has little potential to remove indoor air contaminants.*

Some manufacturers or vendors suggest that ozone will render almost every chemical contaminant harmless by producing a chemical reaction whose only by-products are carbon dioxide, oxygen and water. This is misleading.

- First, a review of scientific research shows that, for many of the chemicals commonly found in indoor environments, the reaction process with ozone may take months or years (Boeniger, 1995). For all practical purposes, ozone does not react at all with such chemicals. And contrary to specific claims by some vendors, ozone generators are not effective in removing carbon monoxide (Salls, 1927; Shaughnessy et al., 1994) or formaldehyde (Esswein and Boeniger, 1994).
- Second, for many of the chemicals with which ozone does readily react, the reaction can form a variety of harmful or irritating by-products (Weschler et al., 1992a, 1992b, 1996; Zhang and Liroy, 1994). For example, in a laboratory experiment that mixed ozone with chemicals from new carpet, ozone reduced many of these chemicals, including those which can

produce new carpet odor. However, in the process, the reaction produced a variety of aldehydes, and the total concentration of organic chemicals in the air increased rather than decreased after the introduction of ozone (Weschler, et. al., 1992b). In addition to aldehydes, ozone may also increase indoor concentrations of formic acid (Zhang and Liroy, 1994), both of which can irritate the lungs if produced in sufficient amounts. Some of the potential by-products produced by ozone's reactions with other chemicals are themselves very reactive and capable of producing irritating and corrosive by-products (Weschler and Shields, 1996, 1997a, 1997b). Given the complexity of the chemical reactions that occur, additional research is needed to more completely understand the complex interactions of indoor chemicals in the presence of ozone.

- Third, ozone does not remove particles (e.g., dust and pollen) from the air, including the particles that cause most allergies. However, some ozone generators are manufactured with an "ion generator" or "ionizer" in the same unit. An ionizer is a device that disperses negatively (and/or positively) charged ions into the air. These ions attach to particles in the air giving them a negative (or positive) charge so that the particles may attach to nearby surfaces such as walls or furniture, or attach to one another and settle out of the air. In recent experiments, ionizers were found to be less effective in removing particles of dust, tobacco smoke, pollen or fungal spores than either high efficiency particle filters or electrostatic precipitators. (Shaughnessy et al., 1994; Pierce, et al., 1996). However, it is apparent from other experiments that the effectiveness of particle air cleaners, including electrostatic precipitators, ion generators, or pleated filters varies widely (U.S. EPA, 1995).

***There is evidence to show that at concentrations that do not exceed public health standards, ozone is not effective at removing many odor-causing chemicals.***

- In an experiment designed to produce formaldehyde concentrations representative of an embalming studio, where formaldehyde is the main odor producer, ozone showed no effect in reducing formaldehyde concentration (Esswein and Boeniger, 1994). Other experiments suggest that body odor may be masked by the smell of ozone but is not removed by ozone (Witheridge and Yaglou, 1939). Ozone is not considered useful for odor removal in building ventilation systems (ASHRAE, 1989).
- While there are few scientific studies to support the claim that ozone effectively removes odors, it is plausible that some odorous chemicals will react with ozone. For example, in some experiments, ozone appeared to react readily with certain chemicals, including some chemicals that contribute to the smell of new carpet (Weschler, 1992b; Zhang and Liroy, 1994). Ozone is also believed to react with acrolein, one of the many odorous and irritating chemicals found in secondhand tobacco smoke (US EPA, 1995).

***If used at concentrations that do not exceed public health standards, ozone applied to indoor air does not effectively remove viruses, bacteria, mold, or other biological pollutants.***

- Some data suggest that low levels of ozone may reduce airborne concentrations and inhibit the growth of some biological organisms while ozone is present, but ozone concentrations would have to be 5 - 10 times higher than public health standards allow before the ozone could decontaminate the air sufficiently to prevent survival and regeneration of the organisms once the ozone is removed (Dyas, et al., 1983; Foarde et al., 1997).
- Even at high concentrations, ozone may have no effect on biological contaminants embedded in porous material such as duct lining or ceiling tiles (Foarde et al, 1997). In other words, ozone produced by ozone generators may inhibit the growth of some biological agents while it is present, but it is unlikely to fully decontaminate the air unless concentrations are high enough to be a health concern if people are present. Even with high levels of ozone, contaminants embedded in porous material may not be affected at all.

## **If I Follow Manufacturers' Directions, Can I be Harmed?**

***Results of some controlled studies show that concentrations of ozone considerably higher than these standards are possible even when a user follows the manufacturer's operating instructions.***

There are many brands and models of ozone generators on the market. They vary in the amount of ozone they can produce. In many circumstances, the use of an ozone generator may not result in ozone concentrations that exceed public health standards. But many factors affect the indoor concentration of ozone so that under some conditions ozone concentrations may exceed public health standards.

- In one study (Shaughnessy and Oatman, 1991), a large ozone generator recommended by the manufacturer for spaces "up to 3,000 square feet," was placed in a 350 square foot room and run at a high setting. The ozone in the room quickly reached concentrations that were exceptionally high--0.50 to 0.80 ppm which is 5-10 times higher than public health limits (see [Table 1.](#))
- In an EPA study, several different devices were placed in a home environment, in various rooms, with doors alternately opened and closed, and with the central ventilation system fan alternately turned on and off. The results showed that some ozone generators, when run at a high setting with interior doors closed, would frequently produce concentrations of 0.20 - 0.30 ppm. A powerful unit set on high with the interior doors opened achieved values of 0.12 to 0.20 ppm in adjacent rooms.

When units were not run on high, and interior doors were open, concentrations generally did not exceed public health standards (US EPA, 1995).

- The concentrations reported above were adjusted to exclude that portion of the ozone concentration brought in from the outdoors. Indoor concentrations of ozone brought in from outside are typically 0.01- 0.02 ppm, but could be as high as 0.03 - 0.05 ppm (Hayes, 1991; U.S. EPA, 1996b; Weschler et al., 1989, 1996; Zhang and Liou, 1994). *If the outdoor portion of ozone were included in the indoor concentrations reported above, the concentrations inside would have been correspondingly higher, increasing the risk of excessive ozone exposure.*
- None of the studies reported above involved the simultaneous use of more than one device. The simultaneous use of multiple devices increases the total ozone output and therefore greatly increases the risk of excessive ozone exposure.

## Why is it Difficult to Control Ozone Exposure with an Ozone Generator?

The actual concentration of ozone produced by an ozone generator depends on many factors. Concentrations will be higher if a more powerful device or more than one device is used, if a device is placed in a small space rather than a large space, if interior doors are closed rather than open and, if the room has fewer rather than more materials and furnishings that adsorb or react with ozone and, provided that outdoor concentrations of ozone are low, if there is less rather than more outdoor air ventilation.

The proximity of a person to the ozone generating device can also affect one's exposure. The concentration is highest at the point where the ozone exits from the device, and generally decreases as one moves further away.

Manufacturers and vendors advise users to size the device properly to the space or spaces in which it is used. Unfortunately, some manufacturers' recommendations about appropriate sizes for particular spaces have not been sufficiently precise to guarantee that ozone concentrations will not exceed public health limits. Further, some literature distributed by vendors suggests that users err on the side of operating a more powerful machine than would normally be appropriate for the intended space, the rationale being that the user may move in the future, or may want to use the machine in a larger space later on. Using a more powerful machine increases the risk of excessive ozone exposure.

Ozone generators typically provide a control setting by which the ozone output can be adjusted. The ozone output of these devices is usually **not** proportional to the control setting. That is, a setting at medium does not necessarily generate an ozone level that is halfway between the levels at low and high. The relationship between the control setting and the output varies considerably among devices, although most appear to elevate the ozone output much more than one would expect as the control setting is increased from low to high. In experiments to date, the high setting in some devices generated 10 times the level obtained at the medium setting (US EPA, 1995). Manufacturer's instructions on some devices link the control setting to room size and thus indicate what setting is appropriate for different room sizes. However, room size is only one factor affecting ozone levels in the room.

In addition to adjusting the control setting to the size of the room, users have sometimes been advised to lower the ozone setting if they can smell the ozone. Unfortunately, the ability to detect ozone by smell varies considerably from person to person, and one's ability to smell ozone rapidly deteriorates in the presence of ozone. While the smell of ozone may indicate that the concentration is too high, lack of odor does not guarantee that levels are safe.

At least one manufacturer is offering units with an ozone sensor that turns the ozone generator on and off with the intent of maintaining ozone concentrations in the space below health standards. EPA is currently evaluating the effectiveness and reliability of these sensors, and plans to conduct further research to improve society's understanding of ozone chemistry indoors. EPA will report its findings as the results of this research become available.

## Can Ozone be Used in Unoccupied Spaces?

Ozone has been extensively used for water purification, but ozone chemistry in water is not the same as ozone chemistry in air. High concentrations of ozone in air, **when people are not present**, are sometimes used to help decontaminate an unoccupied space from certain chemical or biological contaminants or odors (e.g., fire restoration). However, little is known about the chemical by-products left behind by these processes (Dunston and Spivak, 1997). While high concentrations of ozone in air may sometimes be appropriate in these circumstances, *conditions should be sufficiently controlled to insure that no person or pet becomes exposed*. Ozone can adversely affect indoor plants, and damage materials such as rubber, electrical wire coatings and fabrics and art work containing susceptible dyes and pigments (U.S. EPA, 1996a).

## What Other Methods Can Be Used to Control Indoor Air Pollution?

The three most common approaches to reducing indoor air pollution, in order of effectiveness, are:

1. **Source Control:** Eliminate or control the sources of pollution;
2. **Ventilation:** Dilute and exhaust pollutants through outdoor air ventilation and
3. **Air Cleaning:** Remove pollutants through proven air cleaning methods.

Of the three, the first approach — **source control** — is the most effective. This involves minimizing the use of products and materials that cause indoor pollution, employing good hygiene practices to minimize biological contaminants (including the control of humidity and moisture, and occasional cleaning and disinfection of wet or moist surfaces), and using good housekeeping practices to control particles.

The second approach — **outdoor air ventilation** — is also effective and commonly employed. Ventilation methods include installing an exhaust fan close to the source of contaminants, increasing outdoor air flows in mechanical ventilation systems, and opening windows, especially when pollutant sources are in use.

The third approach — **air cleaning** — is not generally regarded as sufficient in itself, but is sometimes used to supplement source control and ventilation. Air filters, electronic particle air cleaners and ionizers are often used to remove airborne particles, and gas adsorbing material is sometimes used to remove gaseous contaminants when source control and ventilation are inadequate.

See Additional Resources section below for more detailed information about these methods.

## Conclusions

*Whether in its pure form or mixed with other chemicals, ozone can be harmful to health.*

When inhaled, ozone can damage the lungs. Relatively low amounts of ozone can cause chest pain, coughing, shortness of breath and, throat irritation. It may also worsen chronic respiratory diseases such as asthma as well as compromise the ability of the body to fight respiratory infections.

*Some studies show that ozone concentrations produced by ozone generators can exceed health standards even when one follows manufacturer's instructions.*

Many factors affect ozone concentrations including the amount of ozone produced by the machine(s), the size of the indoor space, the amount of material in the room with which ozone reacts, the outdoor ozone concentration, and the amount of ventilation. These factors make it difficult to control the ozone concentration in all circumstances.

*Available scientific evidence shows that, at concentrations that do not exceed public health standards, ozone is generally ineffective in controlling indoor air pollution.*

The concentration of ozone would have to greatly exceed health standards to be effective in removing most indoor air contaminants. In the process of reacting with chemicals indoors, ozone can produce other chemicals that themselves can be irritating and corrosive.

## Recommendation

*The public is advised to use proven methods of controlling indoor air pollution.* These methods include eliminating or controlling pollutant sources, increasing outdoor air ventilation and using proven methods of air cleaning.

## Additional Resources

See Indoor Air Quality Publications

### Publications

- [The Inside Story: A Guide to Indoor Air](#), EPA Document Number EPA 402-K-93-007. U.S. EPA, U.S. CPSC. April 1995.
- [Guide to Air Cleaners in the Home \(Second Edition\)](#) This brochure replaces "Residential Air Cleaners - Indoor Air Facts No. 7, EPA 20A-4001, February 1990.", EPA 402-F-08-004, May 2008
- [Residential Air Cleaners \(Third Edition\) A Summary of Available Information](#)
- [Indoor Air Pollution: An Introduction for Health Professionals](#), EPA Document Number EPA 402-R-94-007. American Lung Association, EPA, CPSC, American Medical Association.

## Information Sources

California Department of Health Services, Indoor Air Quality Program, DHS-IAQ Program Assistance Line: (510) 620-2874, Fax: (510) 620-2825

[Federal Trade Commission](#), Consumer Response Center, (202) 326-3128.

[U.S. Consumer Product Safety Commission](#), or call Consumer Hotline, English/Spanish: (800) 638-2772, Hearing/Speech Impaired: (800) 6388270.

The Association of Home Appliance Manufacturers (AHAM) has developed an American National Standards Institute (ANSI)-approved standard for portable air cleaners (ANSI/AHAM Standard AC-1-1988). This standard may be useful in estimating the effectiveness of portable air cleaners. Under this standard, room air cleaner effectiveness is rated by a clean air delivery rate (CADR) for each of three particle types in indoor air: tobacco smoke, dust and pollen. Only a limited number of air cleaners had been certified under this program when this document was written.

A listing of AHAM-certified room air cleaners and their CADRs can be obtained from [Aham Verifide](#) **EXIT**

[Association of Home Appliance Manufacturers \(AHAM\)](#) **EXIT**  
(202) 872-5955

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LAST UPDATED ON DECEMBER 23, 2019

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## What are ionizers and other ozone generating air cleaners?

From: [Indoor Air Pollution: An Introduction for Health Professionals](#)

Ion generators act by charging the particles in a room so that they are attracted to walls, floors, tabletops, draperies, occupants, etc. Abrasion can result in these particles being resuspended into the air. In some cases these devices contain a collector to attract the charged particles back to the unit. While ion generators may remove small particles (e.g., those in tobacco smoke) from the indoor air, they do not remove gases or odors, and may be relatively ineffective in removing large particles such as pollen and house dust allergens. Although some have suggested that these devices provide a benefit by rectifying a hypothesized ion imbalance, no controlled studies have confirmed this effect.

Ozone, a lung irritant, is produced indirectly by ion generators and some other electronic air cleaners and directly by ozone generators. While indirect ozone production is of concern, there is even greater concern with the direct, and purposeful introduction of a lung irritant into indoor air. There is no difference, despite some marketers' claims, between ozone in smog outdoors and ozone produced by these devices. Under certain use conditions ion generators and other ozone generating air cleaners (see [www.epa.gov/indoor-air-quality-iaq/ozone-generators-are-sold-air-cleaners](http://www.epa.gov/indoor-air-quality-iaq/ozone-generators-are-sold-air-cleaners)) can produce levels of this lung irritant significantly above levels thought harmful to human health. A small percentage of air cleaners that claim a health benefit may be regulated by FDA as a medical device. The Food and Drug Administration has set a limit of 0.05 parts per million of ozone for medical devices. Although ozone can be used in reducing odors and pollutants in unoccupied spaces (such as removing smoke odors from homes involved in fires) the levels needed to achieve this are above those generally thought to be safe for humans.

See also:

- [The Inside Story: A Guide to Indoor Air Quality](#) EPA 402-K-93-007, April 1995
- [Indoor Air Pollution: An Introduction for Health Professionals](#) EPA 402-R-94-007, 1994
- [Residential Air Cleaning Devices: A Summary of Available Information](#) EPA 400/1-90-002, February 1990
- [Guide to Air Cleaners in the Home](#) EPA 402-F-07-018, October 2007
- [Ozone Generators That are Sold as Air Cleaners](#) (only available via the web site)

LAST UPDATED ON JULY 16, 2020

Research article

Open Access

## Bactericidal action of positive and negative ions in air

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Published: 17 April 2007

Received: 19 December 2006

BMC Microbiology 2007, 7:32 doi:10.1186/1471-2180-7-32

Accepted: 17 April 2007

This article is available from: <http://www.biomedcentral.com/1471-2180/7/32>

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### Abstract

**Background:** In recent years there has been renewed interest in the use of air ionisers to control of the spread of airborne infection. One characteristic of air ions which has been widely reported is their apparent biocidal action. However, whilst the body of evidence suggests a biocidal effect in the presence of air ions the physical and biological mechanisms involved remain unclear. In particular, it is not clear which of several possible mechanisms of electrical origin (i.e. the action of the ions, the production of ozone, or the action of the electric field) are responsible for cell death. A study was therefore undertaken to clarify this issue and to determine the physical mechanisms associated with microbial cell death.

**Results:** In the study seven bacterial species (*Staphylococcus aureus*, *Mycobacterium parafortuitum*, *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, *Burkholderia cenocepacia*, *Bacillus subtilis* and *Serratia marcescens*) were exposed to both positive and negative ions in the presence of air. In order to distinguish between effects arising from: (i) the action of the air ions; (ii) the action of the electric field, and (iii) the action of ozone, two interventions were made. The first intervention involved placing a thin mica sheet between the ionisation source and the bacteria, directly over the agar plates. This intervention, while leaving the electric field unaltered, prevented the air ions from reaching the microbial samples. In addition, the mica plate prevented ozone produced from reaching the bacteria. The second intervention involved placing an earthed wire mesh directly above the agar plates. This prevented both the electric field and the air ions from impacting on the bacteria, while allowing any ozone present to reach the agar plate. With the exception of *Mycobacterium parafortuitum*, the principal cause of cell death amongst the bacteria studied was exposure to ozone, with electroporation playing a secondary role. However in the case of *Mycobacterium parafortuitum*, electroporation resulting from exposure to the electric field appears to have been the principal cause of cell inactivation.

**Conclusion:** The results of the study suggest that the bactericidal action attributed to negative air ions by previous researchers may have been overestimated.

## Background

In recent years there has been renewed interest in the use of air ionisers to control the spread of airborne infection. In particular, results from a clinical trial in an intensive care unit suggest that negative air ions may have the potential to control some hospital acquired infections (HAIs) [1]. One characteristic of 'air' ions which has been widely reported is their apparent biocidal action. Over many years, various researchers have reported that 'air' ions inhibit the growth of *Penicillium notatum* [2,3], *Neurospora crassa* [3], *Serratia marcescens* [4], "*Staphylococcus albus*" [5], *Candida albicans* [6], *Escherichia coli* [7], *Pseudomonas veronii* [8], *Aspergillus versicolor* [9], *Enterococcus malodoratus* [9], *Staphylococcus chromogenes* [9] and *Sarcina flava* [9]. However, while this body of work collectively suggests a biocidal effect in the presence of ions, in both air [2-7,9] and nitrogen [7,8], the physical and biological mechanisms involved remain unclear. In particular, it is not clear which of several possible mechanisms of electrical origin (i.e. the action of the ions, the production of ozone, or the action of the electric field) are responsible for cell death. A study was therefore undertaken to clarify this issue and to determine the physical mechanisms associated with microbial cell death. In this study seven bacterial species were exposed to both positive and negative ions in the presence of air for various durations and the bactericidal effects recorded.

## Results

In the study bacteria in pure culture on agar were exposed to air ions in a test rig, as shown in Figure 1. Experimentation was undertaken on sessile cultures of *Staphylococcus aureus* (NCTC 10399/ATCC 13709), *Mycobacterium parafortuitum* (NCTC 10410/ATCC 19687), *Pseudomonas aeruginosa* (NCIMB 10848), *Acinetobacter baumannii* (NCTC 12156/ATCC 19606), *Burkholderia cenocepacia* (NCTC 10744/ATCC25609), *Bacillus subtilis* (NCTC 10106/ATCC33234) and *Serratia marcescens* (NCTC 1377/ATCC 274). Cultures were exposed to positive air ions and negative air ions in separate experiments. In order to ensure uniform ionic exposure the agar plates were earthed and placed directly below a 7-pin electrode set, with the surface of the agar being 25 mm below the electrode tips. A high voltage generator (Brandenburg Alpha III, Brandenburg, UK) was used to apply a DC potential between the pin electrodes and the earthed agar plate. During experimentation an ammeter located on the earth conductor measured the current associated with the ions impinging on the agar plate.

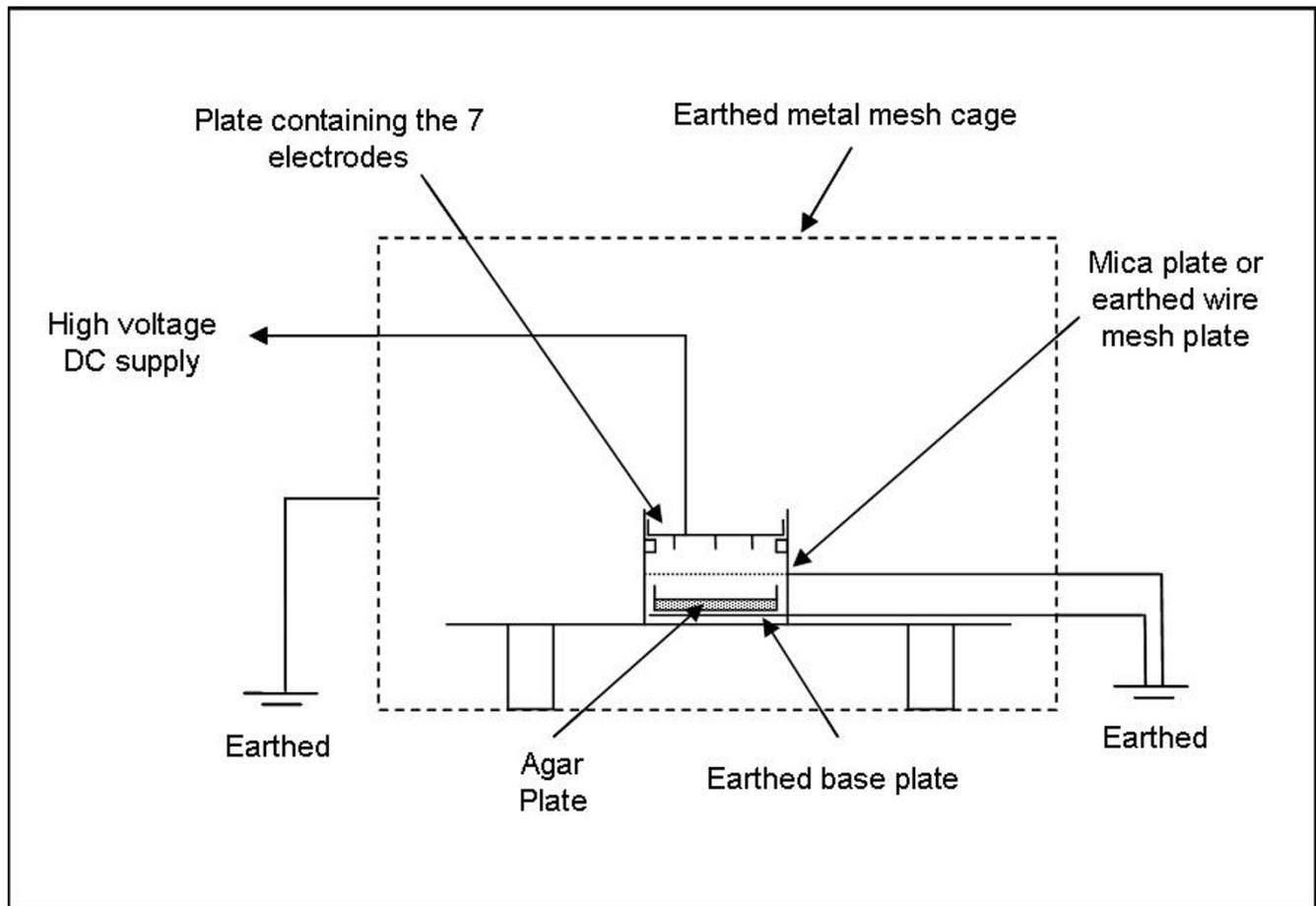
In order to distinguish between effects arising from: (i) the action of the air ions; (ii) the action of the electric field, and (iii) the action of any ozone produced as a by-product of the corona discharge, two interventions were made to the experimental procedure. The first intervention

involved placing a thin mica sheet between the 7-pin electrode set and the agar plate, which while leaving the electric field largely unaltered, prevented the air ions from reaching the bacteria. It should be noted that with free charges, the mica plate may have altered the space charge generated by the corona and thus may have modified the field distribution slightly. In addition, the mica plate prevented ozone generated by the electrical discharge from reaching the bacteria. The second intervention involved placing an earthed wire mesh 20 mm below the electrode set, and above the agar plates. This had the effect of greatly reducing (i.e. to negligible levels) the electric field strength and the number of air ions to which the bacteria were exposed. However, ozone generated by the discharge could still reach the agar plate.

All the ion exposure experiments were undertaken under ambient room conditions. All seven bacterial species were exposed to negative air ions (with an electrode potential of -10 kV) for periods of 5, 10 and 15 minutes, with five replicates taken on each occasion. The process was then repeated for samples exposed to positive ions, with an electrode potential of +10 kV. The negative ion exposure experiments were repeated twice more, first with a mica sheet placed above the agar plate and then with an earthed wire mesh located as described above. During experimentation the earth current and moisture loss from the agar plates were recorded. Ozone levels were also monitored using a portable detector (Model A-21ZX, Eco Sensors, USA).

The results of the negative air ion experiment (i.e. without any interventions) are presented in Figure 2, which shows the survival fraction versus exposure time for each bacterial species. From these it can be seen that in every case exposure to negative air ions was associated with a marked reduction in colony count. Statistical analysis using a T-test (two-tailed with equal variance) reveals that for all bacteria species this reduction was significant ( $p < 0.01$ ). Interestingly, little difference was observed between the behaviour of the Gram-positive and Gram-negative species.

The results of the positive air ion experiment are presented in Figure 3. In marked contrast to the negative ion results, it can be seen that exposure to positive ions produced a dissimilar effect in the various test bacteria. A substantial reduction was observed for *Mycobacterium parafortuitum* (96.0% after 15 minutes exposure) ( $p < 0.001$ ) and *Bacillus subtilis* (70.8% after 15 minutes) ( $p < 0.001$ ), with a much smaller reduction for *Pseudomonas aeruginosa* (31.4% after 15 minutes) ( $p < 0.001$ ), *Acinetobacter baumannii* (31.2% after 15 minutes) ( $p < 0.001$ ), *Burkholderia cenocepacia* (32.1% after 15 minutes) ( $p = 0.059$ ) and *Serratia marcescens* (24.2% after 15 minutes) ( $p = 0.002$ ).



**Figure 1**  
Diagram of the test rig.

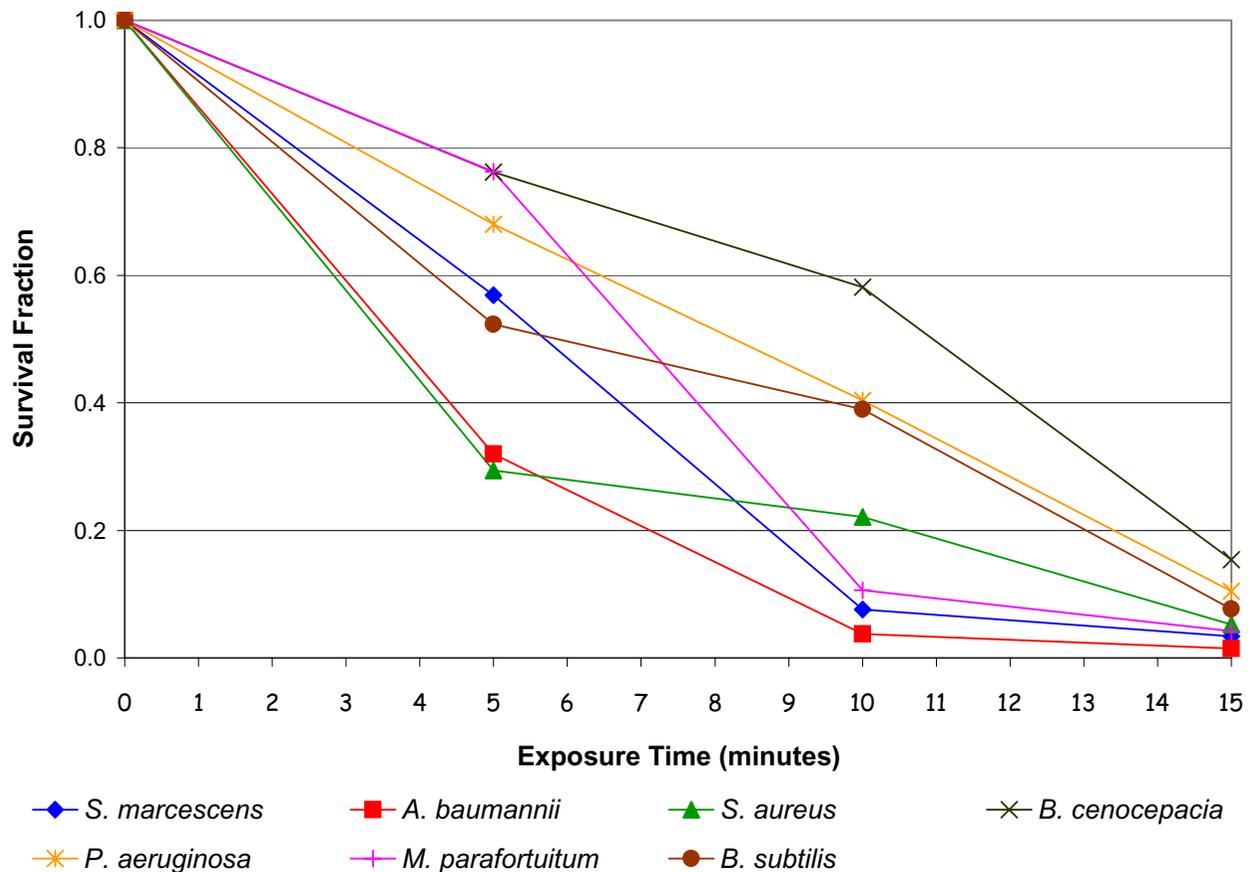
However, no bactericidal effect was observed for *Staphylococcus aureus* ( $p = 0.345$ ). With the exception of *Mycobacterium parafortuitum* the bactericidal effect produced when the bacteria were exposed to positive ions was much less than that achieved by the negative ions.

The results of the mica plate experiment, revealing the effect of the electric field alone, are presented in Figure 4. Comparison of these results with those in Figure 2 reveals that the intervention of the mica plate had a marked effect on the bactericidal action of the negative ions. This suggests that for most of the bacteria species tested, the bactericidal action observed in Figure 2 was not primarily due to the action of the electric field. However, from Figure 4 it can be seen that for one species in particular, *Mycobacterium parafortuitum*, the action of the electric field alone appears to have resulted in a strong bactericidal effect, with a 94.9% reduction ( $p < 0.001$ ) occurring after 15 minutes exposure. Lesser reductions were also observed for *Burkholderia cenocepacia* (48.7% after 15 minutes) ( $p < 0.001$ ), *Acinetobacter baumannii* (44.0% after 15 minutes)

( $p < 0.001$ ), *Staphylococcus aureus* (32.3% after 15 minutes) ( $p = 0.005$ ), with only marginal reductions occurring for *Bacillus subtilis* (14.9% after 15 minutes) ( $p = 0.516$ ) and *Pseudomonas aeruginosa* (9.3% after 15 minutes) ( $p = 0.192$ ).

The results of the wire mesh experiment, in which the action of the electric field and the air ions were reduced to negligible levels, are presented in Figure 5. All the bacterial species exhibited marked reductions ( $p < 0.01$ ) in colony count during the exposure period. This indicates that all the bacteria were susceptible to ozone damage.

The mean currents (together with the maximum and minimum values) recorded during the various experiments are presented in Table 1. These values for the positive and negative ion experiments (i.e. columns 2 and 3) indicate that although the potential of the electrode was constant for the each experiment, there was some variation in the current produced. This phenomenon was probably due to variations in the conductivity of the agar, and day-to-day



**Figure 2**  
Reduction in viability of bacteria following negative energisation of the electrodes. The survival fractions shown are the mean of 5 replicates.

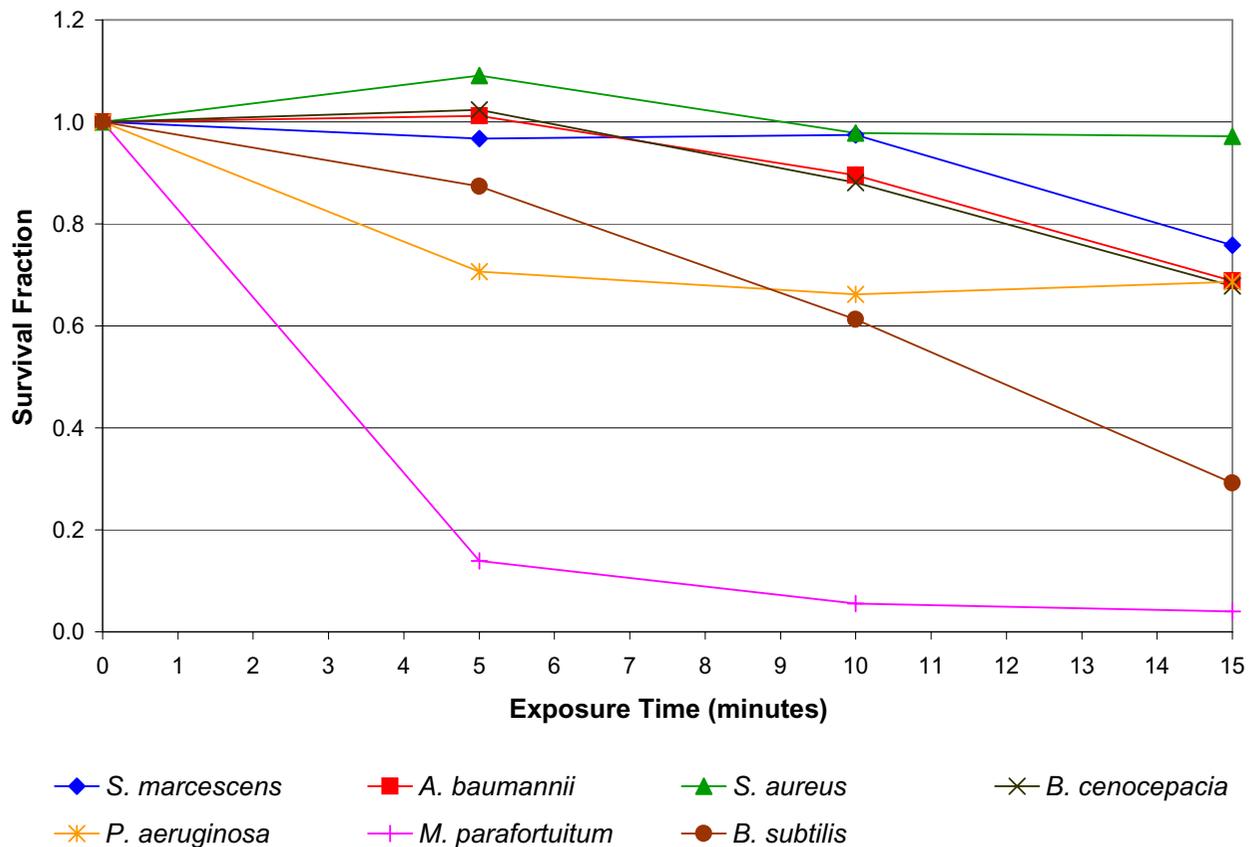
variations in temperature, humidity and air movement in the laboratory. With regard to the mica plate experiment, it can be seen that the current flow was negligible, indicating the lack of any ion flow to the agar plate. Similarly, the earthed wire mesh prevented any current flowing to the agar plate.

During production of negative ions the ozone concentration in the vicinity of the agar plate was found to be 2.3 ppm. However, during positive ion production this value was found to be considerably lower, about 0.8 ppm.

## Discussion

Although a number researchers have noted the phenomenon of microbial death 'when exposed to air ions', to the authors' best knowledge, none of the previous studies have made a rigorous attempt to separate out the various effects of electrical origin that may be responsible for cell death. For example, Kellogg *et al.* [5] and Digel *et al.* [9]

made no attempt to distinguish effects arising from ozone or electric fields, from those caused by air ions themselves. By contrast Shargawi *et al.* [6] and Noyce and Hughes [7,8] did attempt to eliminate the effects of ozone, by performing a series of negative 'air' ion experiments in a pure nitrogen atmosphere. However this approach is inherently flawed, because although negative air ions readily form in air, they cannot form in pure nitrogen, and the mechanism is therefore believed to be electronic. One feature of negative coronas is that they can only be sustained in fluids which contain electronegative molecules, such as O<sub>2</sub>, H<sub>2</sub>O and CO<sub>2</sub>, since these gases have molecules which readily scavenge free electrons. Without electronegative molecules to capture free electrons, small negative cluster ions cannot form, with the result that a simple flow of electrons will occur in an ionized gas between the two electrodes and an arc will develop. Therefore any direct comparison between the action of negative ions in air and in nitrogen, such as that performed by Shargawi *et al.* [6]



**Figure 3**

Reduction in viability of bacteria following positive energisation of the electrodes. The survival fractions shown are the mean of 5 replicates.

is somewhat erroneous, since both ozone and negative ions will form in air but not in nitrogen. Furthermore, irrespective of the nature of the atmosphere used, an electric field will always be present and this may have an impact on microbial viability. Given this, it is difficult to draw any firm conclusions about possible bactericidal mechanisms from much of the previous work.

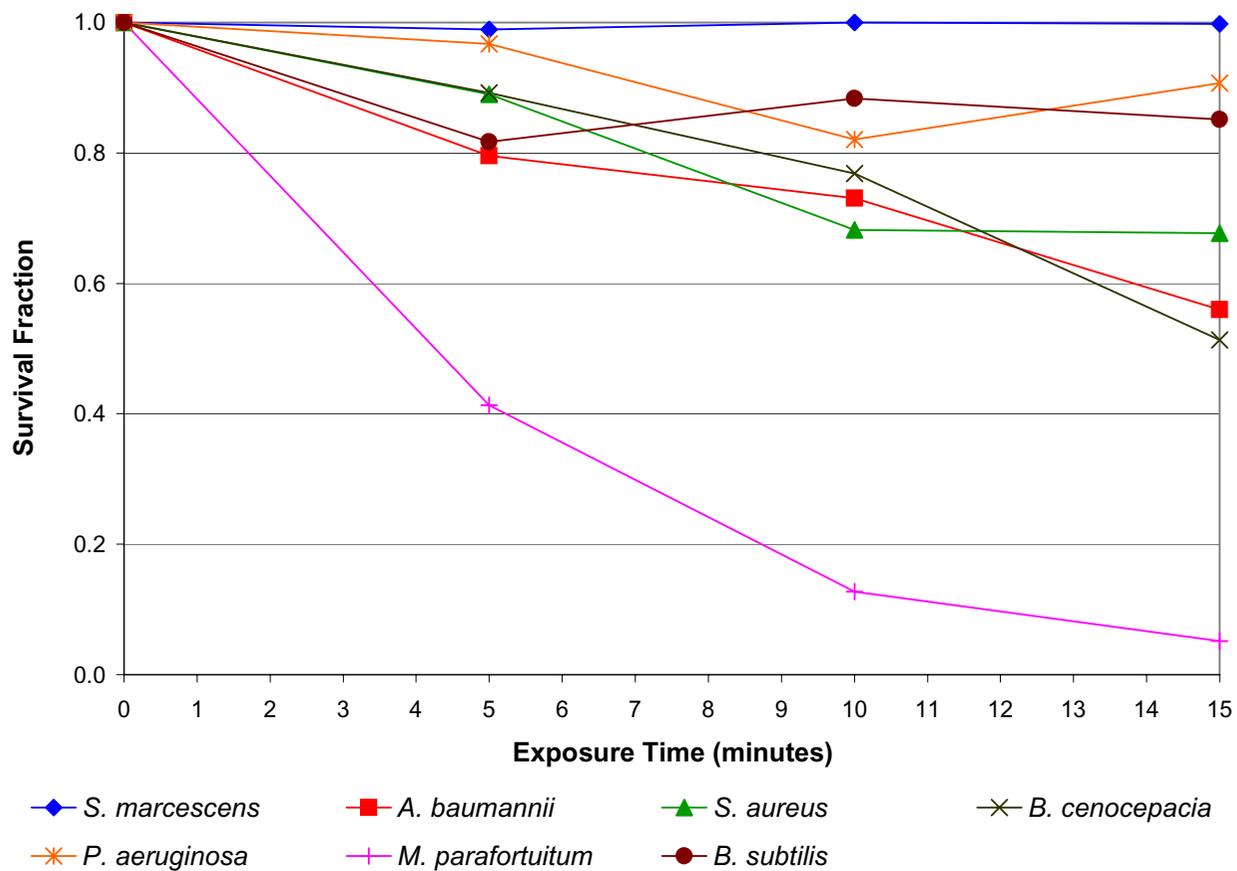
Experiments such as described in this paper and those of previous researchers can produce microbial death by three, quite distinct, electrical phenomena:

- **Electrodynamics** – Ions, electrons and other ionising radiations
- **Electrostatics** – Electric charge/Electric field
- **Electro-chemical effects** – Ozone production

The experiments described in this paper have been designed to distinguish, for the first time, between these different effects. A mica plate was used to isolate the

microorganisms from the ions and any ozone produced by the electric discharge. Similarly, an earthed wire mesh was used to prevent exposure of the microorganisms to both the ions and the electric field, allowing only the ozone to pass. In this way, it was possible to quantify with some accuracy the relative proportions of microbial mortality attributable to the different bactericidal mechanisms.

From the data presented in figures 2, 4 and 5 a clear consistent picture emerges. When exposed to 'negative ions' the principal bactericidal mechanism affecting most of the test species appears to be oxidation damage arising from exposure to ozone. This is clearly evident from Figure 5 which shows the reduction in the microbial population primarily due to the action of ozone. This finding echoes that of Shargawi *et al.* [6], who when working with *Candida albicans* found a strong correlation between cell death and the level of ozone present. The results achieved with the mica plate in place reinforce the opinion that ozone played an important role in the inactivation of most of the species tested. The results in Figure 4 indicate that for



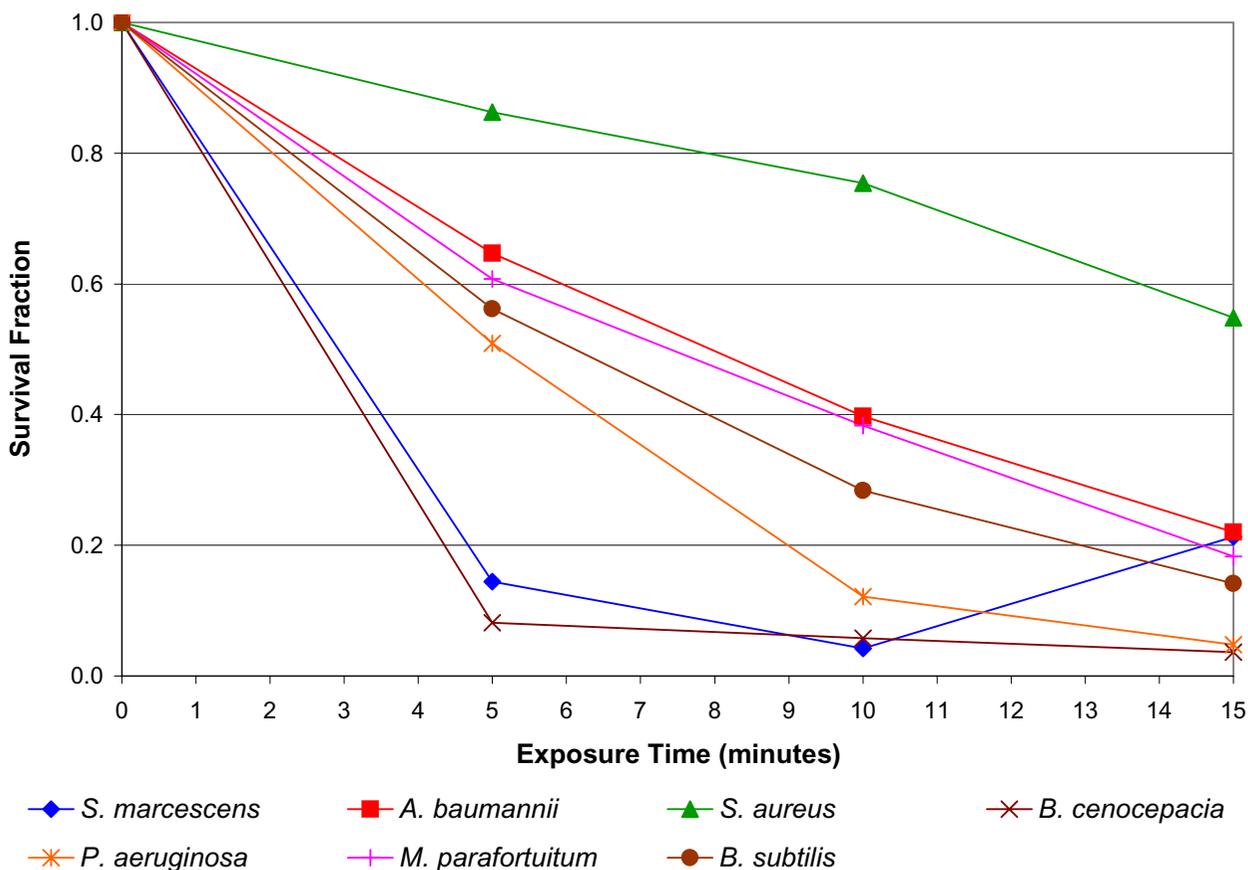
**Figure 4**

Reduction in viability of bacteria following negative energisation of the electrodes, with the intervention of a mica plate. The survival fractions shown are the mean of 5 replicates.

most of the species tested the negative ions and the electric field played only a minor role in cell death compared with the action of the ozone. Further evidence supporting this comes from the positive ion data presented in Figure 3. The mechanism by which positive ions are produced generates much less ozone than does its negative counterpart. This is because of the greater number of free electrons associated with negative coronas and the fact that the reactions which produce ozone are relatively low-energy. Consequently, when exposed to positive ions the test bacteria were also exposed to reduced levels of ozone compared with levels associated with negative air ionization, with the result that for most of the test species only a modest bactericidal action was observed. Interestingly, from the results in figures 2 and 5 it appears that some microorganisms (i.e. *Serratia marcescens* and *Acinetobacter baumannii*) are more susceptible to ozone alone compared with the combined action of ozone, negative ions and the

electric field. However, the reasons for this phenomenon are unclear.

The behaviour of *Mycobacterium parafortuitum* is of particular interest. For this bacterium in the absence of ozone or negative ions (see Figure 4), a substantial reduction (i.e. 94.9% at 15 minutes) was achieved solely through the intervention of the electric field. This suggests that unlike the other bacteria, with this species the principal inactivation mechanism is electroporation. Corroboration of this comes from Figure 5 which shows that for this bacterium, the kill achieved through the action of ozone alone was actually less than that achieved by the electric field alone. Further evidence suggesting that ozone played only a relatively minor role in the inactivation of *Mycobacterium parafortuitum* comes from Figure 3, where it can be seen that under conditions of reduced ozone a large kill was still achieved. Of the bacteria tested this behavior appears



**Figure 5**  
Reduction in viability of bacteria following negative energisation of the electrodes, with the intervention of a wire mesh. The survival fractions shown are the mean of 5 replicates.

**Table 1: Mean currents recorded during experiments, with range indicated in brackets.**

Bacterium	Average Current (range) (µA)			
	Negative ions	Positive ions	Negative ions with mica plate	Negative ions with wire mesh
<i>Serratia marcescens</i>	76.0 (65 – 95)	42.3 (30 – 50)	0.000 (0 – 0.005)	0.001 (0 – 0.01)
<i>Acinetobacter baumannii</i>	102.0 (40 – 150)	35.3 (30 – 45)	0.003 (0 – 0.005)	0.000 (0 – 0)
<i>Staphylococcus aureus</i>	65.00 (30 – 90)	36.0 (25 – 45)	0.003 (0 – 0.05)	0.000 (0 – 0)
<i>Burkholderia cenocepacia</i>	109.0 (95 – 120)	32.0 (30 – 35)	0.022 (0 – 0.2)	0.000 (0 – 0)
<i>Pseudomonas aeruginosa</i>	97.0 (80 – 105)	36.3 (30 – 40)	0.003 (0 – 0.025)	0.000 (0 – 0)
<i>Mycobacterium parafortuitum</i>	113.0 (95 – 130)	36.3 (25 – 45)	0.009 (0 – 0.05)	0.000 (0 – 0)
<i>Bacillus subtilis</i>	114.3 (90 – 135)	27 (20 – 40)	0.002 (0 – 0.025)	0.000 (0 – 0)

unique to *Mycobacterium parafortuitum*. By comparison, the electric field appears to have played only a minor role in the inactivation of the other bacteria.

Collectively the data in figures 2, 4 and 5 suggests that for most of the bacterial species tested, negative air ions play only a relatively minor role in the bactericidal process. Microbial inactivation appears to occur mainly through a combination of ozone damage and damage caused by the electric field, with very little contribution from the negative ions themselves. Although not conclusive, Figure 3 suggests that the positive ions also have only a very limited affect on the bacteria studied. Indeed, the similarity between figures 3 and 4 is striking. Thus, a very interesting picture emerges. It would appear that some bactericidal action results from all the electrical phenomena tested. The disinfectant properties of ozone are well known and this mechanism seems to be responsible for the majority of the kills. However, it also appears that both the electric field and air ions have a contributory role.

Although it might be argued that the magnitude of the applied field is not high in relative terms, the complete dynamics of the time development of the electric field in a spherical dielectric shell representing the cellular membrane can be obtained using an analytical solution of the Ohmic conduction problem. Indeed, it has been found that the field in the membrane can reach a maximum value two orders of magnitude higher than the original Laplacian electrical field [10]. The plasma membrane of a cell serves the vital function of partitioning the contents of the cytoplasm from the external environment. These membranes are largely composed of amphiphilic lipids which self-assemble into highly insulating structures and thus present a large energy barrier to trans-membrane ionic transport. However, the lipid matrix can be disrupted by a strong external electric field leading to an increase in trans-membrane conductivity and diffusive permeability. These effects are the result of formation of aqueous pores in the membrane, which alter the electrical potential across the membrane and may ultimately lead to cell lysis.

Electroporation is a mechanical method used to introduce polar molecules into a host cell through the cell membrane. In this procedure, a large electric pulse temporarily disturbs the phospholipid bilayer, allowing molecules like DNA to pass into the cell. Electroporation is also the basic mechanism of tissue injury in high-voltage electric shock. If the strength of electrical field and duration of exposure to it are properly chosen, the pores formed by the electrical pulse reseal after a short period of time, during which the extracellular compounds have a chance to get inside the cell. However, excessive exposure of live cells to electrical fields can also cause apoptosis. Indeed,

such harsh treatments have been used for killing tumour cells.

Care has been taken in the experimental protocols described in this paper to distinguish between the potential biocidal effects of the ions and the possibility of cell death induced by the electric field alone. Onset of cell lysis generally takes place at electric field strengths of around 100 V/mm or equivalently 100 kV/m. The ionising electrodes in our experiments are located 25 mm from the agar surface. At a potential of 10 kV this results in an 'estimated' field strength of 400 kV/m, which suggests that cell death due to electroporation may indeed have occurred. (It should be noted that because of the large field gradient at the electrode tip, this simple computation over-estimates the field strength at the agar surface. However, for the purposes of this study this estimate is a useful enough indicator of the field strength at the agar surface.) To quantify the effect of the electric field, a mica barrier was inserted between the ion source and the agar surface. The mica stopped the ions and ozone but had little effect on the electric field. In this way it was possible to measure the background effect of the electric field in isolation. From Figure 4 it can be seen that in addition to the dramatic effect on *Mycobacterium parafortuitum*, the electric field alone appears to have resulted in a modest kill in most of the other microbial species tested.

Collectively, the findings of the study indicate that negative air ions have little bactericidal effect on the species studied, despite being generated in close proximity to the microorganisms. This confirms the observations of Kerr *et al.* [1], who in a study on an intensive care unit (ICU), found the action of negative air ions to be associated with; (a) increased environmental isolates of *Acinetobacter* spp, and (b) a marked decrease in numbers of *Acinetobacter* infections/colonizations. Clearly, if the negative ions had had any bactericidal effect, then Kerr *et al.* would have observed a decrease in environmental isolates. This suggests that in the ICU study the reduction in observed *Acinetobacter* infections/colonizations was mainly due to physical effects rather than any bactericidal phenomena.

## Conclusion

The results presented in this paper suggest that the bactericidal action attributed to negative air ions by previous researchers may have been overestimated. With the exception of *Mycobacterium parafortuitum*, the principal cause of cell death amongst the bacteria studied was exposure to ozone, with electroporation playing a secondary role. However in the case of *Mycobacterium parafortuitum*, electroporation resulting from exposure to the electric field appears to have been the principle cause of cell inactivation. Further work is needed to determine whether this a feature common to all *Mycobacterium* species or indeed

other bacteria with a similar cell wall structure such as *Nocardia* spp.

## Methods

The bacterial strains used in the study were stored at -20°C prior to use and were then used to aseptically inoculate 100 ml of tryptone soya broth (TSB). The TSB was incubated in a shaking incubator for 24 hours at 37°C (48 hours in the case of *M. parafortuitum*). The concentration of each culture was determined by serial dilution followed by inoculation of 0.1 ml onto tryptone soya agar plates. Following incubation at 37°C for 24 or 48 hours the number of colonies was counted and the concentration of bacteria in the original culture was determined. Based on this information appropriate dilutions of the cultures were carried out to give a concentration of approximately 3000 cfu/ml. Sterile tryptone soya agar (TSA) plates were subsequently inoculated with 0.1 ml of this dilution, which was spread evenly over the surface of the media with a sterile spreader, to yield approximately 300 colonies per plate. The plates were then allowed to dry at room temperature before being used in the ion exposure experiments. For each exposure period five identical replicate plates were prepared.

Following ion exposure the agar plates were incubated at 37°C for a period of 24 or 48 hours after which time the number of colonies on each plate was counted. Control plates were treated in an identical manner with the exception of exposure to the ion source.

## Authors' contributions

LAF carried out the experimental work and prepared microbiological samples. LFG and SJS designed the test rig and participated in the design of the study. PAS, CJN and KGK participated in the design of the study. CBB conceived and coordinated to the study, drafted the manuscript and undertook the statistical analysis. All authors have read and approved the manuscript.

## Acknowledgements

The research work reported in this paper was funded by the Engineering and Physical Sciences Research Council (EPSRC) of the United Kingdom. The authors wish to thank the EPSRC for their support.

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*Menu*

## IUVA COVID-19 FAQ

▼ What is the UVC dose for killing or disabling the COVID-19 virus?

### What is the UVC dose for killing or disabling the COVID-19 virus?

Because the COVID-19 virus (SARS-CoV-2) is so new, the scientific community doesn't yet have a specific deactivation dosage. However, we know the dosage values for comparable viruses in the same SARS virus family are 10-20 mJ/cm<sup>2</sup> using direct UVC light at a wavelength of 254nm; this dosage will achieve 99.9% disinfection (i.e., inactivation) under controlled lab conditions. In real-life, the virus is often hidden or shaded from direct UVC light, reducing UVC's effectiveness. To compensate, researchers are applying dosages of 1,000 - 3,000 mJ/cm<sup>2</sup> to ensure 99.9% deactivation, the current CDC disinfection goal ([see CDC's recently published guidelines, online](#)).

CDC's recently published guidelines

- ▶ Can we use tanning beds to decontaminate PPE?
- ▶ Can we use UVC disinfection to reuse our N-95 masks as our supplies are limited?
- ▶ Can the products I find on websites such as Amazon be safely used for medical disinfection?
- ▶ Can You Use UVC for direct human disinfection?
- ▶ How do operators remain safe when using UVC lights to clean surfaces?
- ▶ Would the use of UVC at 185nm wavelength, which generates ozone, be as effective as germicidal UVC?
- ▶ Where can I find third-party testing of the efficacy of UVC lights?
- ▶ We are looking into the use UVC light to disinfect certain areas of the electric power plants. Have you done this?
- ▶ Can you tell us more about the penetration of UVC through "normal household" items?

▶ How can you tell if "UV protective" glass and glass films are actually treated to block UVA rays. Is there any

▶ We keep seeing the terms 'watts' and 'joules' in descriptions of how much UV is required to disinfect some

## Questions about far UV (200-225 nm)

▶ Is far UV (200 – 225 nm) a promising disinfection technique to address COVID-19 and other pathogen dis

▶ Is far UV (200 – 225 nm) 'skin safe'?

▶ Does far UV (200 – 225 nm) generate ozone?

*Note: The information provided here is believed to be current at the date of publication. The statements found here may not cover all applicable information. IUVA is not responsible for the conditions of use of particular UV equipment. It is the user's responsibility to determine the safe conditions of use. This information is offered in good faith and is believed to be reliable; however it provides neither warranties nor representations. IUVA disclaims any and all liability for the damages incurred directly or indirectly through the use of this document. Nothing contained herein should be considered a recommendation or endorsement. Contact your supplier for additional information.*

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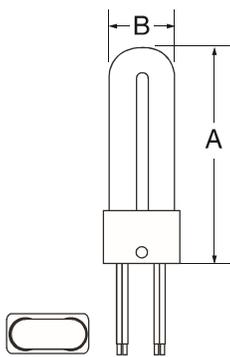
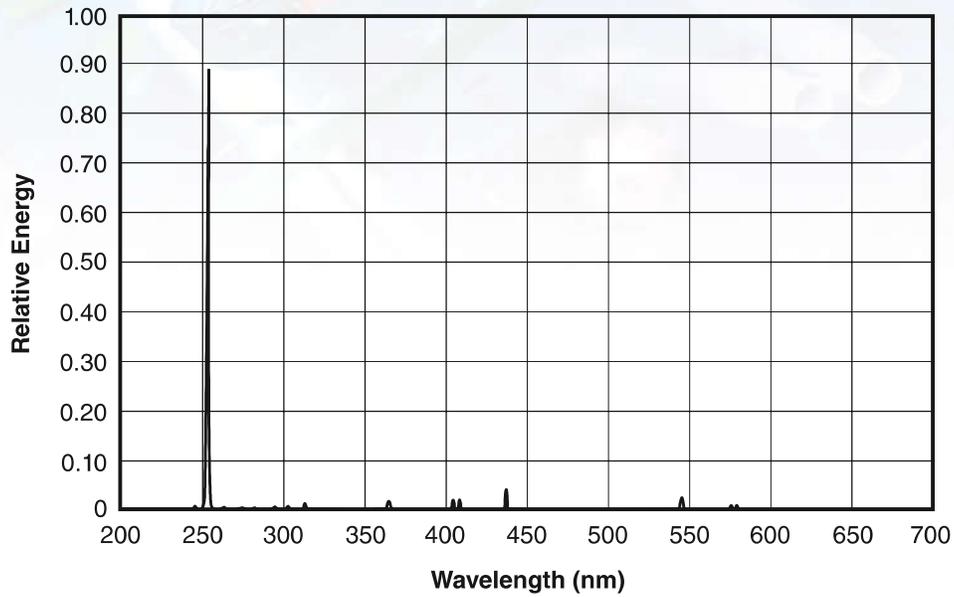
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- Specially Formulated Coating Achieves High Output Over Long Life Hours
- High Purity Lamp Construction to Stabilize UV Output and Minimizes Depreciation (Averages 20-25% Depreciation at End of Life)
- Large Production Capacity Providing Lamps with Consistent Quality and Reliable Delivery
- Flexible Design Capability for Custom Lamp Development

### APPLICATIONS

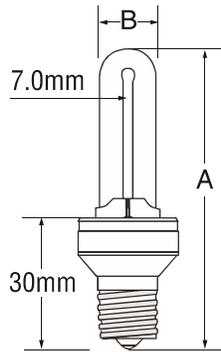
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- Wastewater
- Air Conditioning System
- Pharmaceutical Processing
- Food Processing Facility
- Packaging Materials
- Laboratory/Research
- Photochemistry
- Clean Room
- Other Sterilization and Disinfection Needs

# SPECIFICATIONS

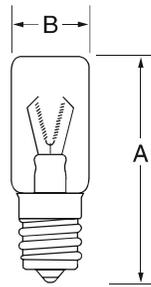
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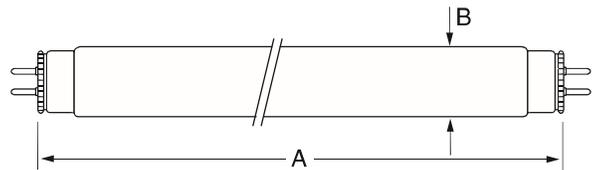
T7



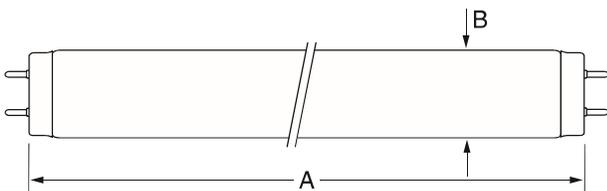
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w/Ballast  
E17 Base



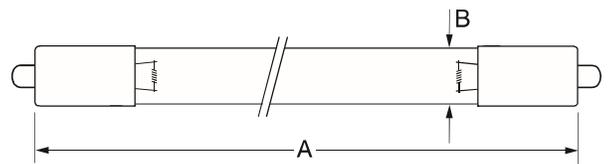
Mini Bowl  
E17 Base



T5  
Miniature Bi-Pin G5 Base

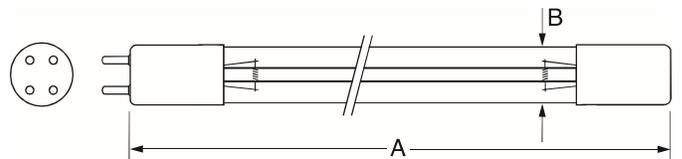
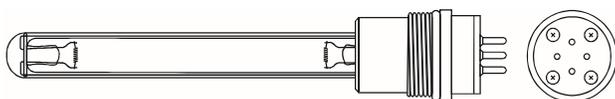


T8 and T10  
Medium Bi-Pin G13 Base



T5  
Single-Pin Base

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T5  
4-Pin Base

Watts (W)	Ordering Code	Lamp Description	Base	Volts (V)	Lamp Current (A)	Dimensions				Spectral Peak (nm)	UV Output (W)	Average Life (h)
						Length (A) (mm)	Length (A) (in)	Diameter (B) (mm)	Diameter (B) (in)			
<b>T7</b>												
2.3	3000667	GUL3	—	28	0.085	61.5	2.42	19.0	0.75	253.7	0.54	3000
<b>GTL3 Replacement with Ballast E17 Base</b>												
3.4	3000666	EGD3	E17	115	0.050	90.0	3.54	17.7	0.70	253.7	0.5	6000
<b>Mini Bowl Intermediate Screw E17 Base</b>												
3.0	3000022	GTL3	E17	10.5	0.300	63.0	2.48	20.0	0.79	253.7	0.16	3000
<b>T5 - Miniature Bi-Pin G5 Base</b>												
4.5	3000013	G4T5	G5	29.0	0.170	134.5	5.30	15.5	0.61	253.7	0.8	6000
6.0	3000015	G6T5	G5	42.0	0.160	210.5	8.29	15.5	0.61	253.7	1.8	8000
7.2	3000016	G8T5	G5	57.0	0.145	287.0	11.30	15.5	0.61	253.7	2.2	8000
16.0	3000311	G16T5	G5	50.0	0.350	287.0	11.30	15.5	0.61	253.7	3.2	8000
<b>T8 - Medium Bi-Pin G13 Base</b>												
9.5	3000006	G10T8	G13	46.0	0.230	330.0	12.99	25.5	1.00	253.7	2.7	8000
15.0	3000007	G15T8	G13	55.0	0.305	436.0	17.17	25.5	1.00	253.7	4.9	8000
25.0	3000008	G25T8	G13	46.0	0.600	436.0	17.17	25.5	1.00	253.7	6.9	8000
30.5	3000009	G30T8	G13	99.0	0.355	893.0	35.15	25.5	1.00	253.7	13.9	8000
<b>T10 - Medium Bi-Pin G13 Base</b>												
19.0	3000314	G20T10	G13	58.0	0.360	588.5	23.17	32.5	1.28	253.7	7.5	8000
39.5	3000315	G40T10	G13	106.0	0.420	1198.0	47.17	32.5	1.28	253.7	19.8	8000
<b>T5 - Single Pin Base</b>												
16.0	3000338	G10T5L	Single Pin	55.0	0.425	357.0	14.06	15.5	0.61	253.7	5.3	9000
39.0	3000312	G36T5L	Single Pin	115.0	0.425	846.0	33.31	15.5	0.61	253.7	13.0	9000
65.0	3000313	G64T5L	Single Pin	250.0	0.425	1553.6	61.17	15.5	0.61	253.7	27.0	9000
<b>T5 - Single Pin Base - Quartz</b>												
39.0	3000010	G36T5L	Single Pin	120.0	0.425	846.0	33.31	15.0	0.59	253.7	—	9000
65.0	3000014	G64T5L	Single Pin	220.0	0.425	1572.0	61.89	15.0	0.59	253.7	—	7500
<b>T5 - Single Pin Base - Ozone</b>												
16.0	3000005	G10T5VH	Single Pin	55.0	0.425	375.0	14.76	15.0	0.59	253.7	—	7500
39.0	3000011	G36T5VH	Single Pin	120.0	0.425	860.0	33.86	15.0	0.59	253.7	—	7500
65.0	3000242	G64T5VH	Single Pin	220.0	0.425	1554.0	61.18	15.0	0.59	253.7	25.0	9000
<b>T5 - 4-Pin Base</b>												
14.0	3000348	G14T5L/4P	4-Pin	40.0	0.425	287.0	11.30	15.5	0.61	253.7	3.0	8000
22.0	3000350	G22T5L/4P	4-Pin	62.0	0.425	436.0	17.16	15.5	0.61	253.7	7.0	8000
39.0	3000343	G36T5L/4P	4-Pin	115.0	0.425	846.0	33.31	15.5	0.61	253.7	13.0	9000
<b>T5 - 4-Pin Base - Quartz</b>												
16.0	3000425	G10T5L/4P	4-Pin	55.0	0.425	357.0	14.06	15.0	0.59	253.7	5.7	9000
65.0	3000423	G64T5L/4P	4-Pin	220.0	0.425	1554.0	61.18	15.0	0.59	253.7	25.0	9000
<b>T5 - 4-Pin Base - Ozone</b>												
16.0	3000354	G10T5VH/4P	4-Pin	55.0	0.425	357.0	14.06	15.0	0.59	253.7	5.7	9000

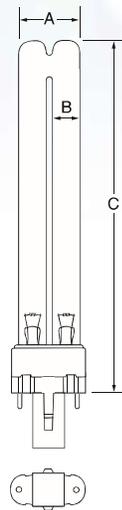
Average lamp life and output measurements taken under laboratory conditions in open air. Lamps are cycled for 2 hours 45 minutes on / 15 minutes off when testing life and output. Lamp data is for reference only. Actual lamp performance depends on system design and operating conditions.

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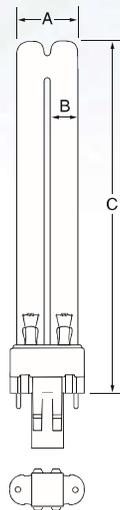
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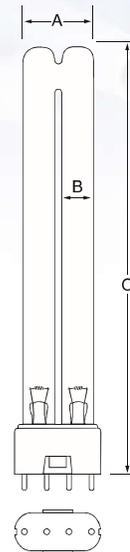
# SPECIFICATIONS



**G23 Base**



**GX23 Base**



**2G11 Base**

Watts (W)	Ordering Code	Lamp Description	Base	Volts (V)	Lamp Current (A)	Dimensions				Spectral Peak (nm)	UV Output (W)	Avg Rated Life (h)		
						Length (A)		Diameter (B)					Length (C)	
						(mm)	(in)	(mm)	(in)	(mm)	(in)			
<b>T4</b>														
5.5	3000321	GPX5	G23	35	0.180	28.0	1.10	13.0	0.51	85.0	3.35	253.7	1.2	8000
9.0	3000304	GPX9	G23	59	0.180	28.0	1.10	13.0	0.51	145.0	5.71	253.7	2.4	8000
13.4	3000323	GPX13	GX23	59	0.285	28.0	1.10	13.0	0.51	170.0	6.69	253.7	3.6	8000
<b>T6</b>														
18.0	3000324	GPL18K	2G11	58	0.375	40.0	1.57	20.0	0.51	225.0	8.86	253.7	5.5	8000
36.0	3000339	GPL36K	2G11	106	0.435	40.0	1.57	20.0	0.51	415.0	16.33	253.7	12.0	8000

Average lamp life and output measurements taken under laboratory conditions in open air. Lamps are cycled for 2 hours 45 minutes on / 15 minutes off when testing life and output. Lamp data is for reference only. Actual lamp performance depends on system design and operating conditions.

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DEFINING THE EFFECTIVENESS OF UV LAMPS INSTALLED IN CIRCULATING AIR  
DUCTWORK

Final Report

Date Published – November 2002



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Funding for the 21-CR program provided by (listed in order of support magnitude):

- U.S. Department of Energy (DOE Cooperative Agreement No. DE-FC05-99OR22674)
- Air-Conditioning & Refrigeration Institute (ARI)
- Copper Development Association (CDA)
- New York State Energy Research and Development Authority (NYSERDA)
- Refrigeration Service Engineers Society (RSES)
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ARTI-21CR/610-40030-01

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INSTALLED IN CIRCULATING AIR DUCTWORK

Final Report

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Douglas VanOsdell  
Karin Foarde



Prepared for the  
AIR-CONDITIONING AND REFRIGERATION TECHNOLOGY INSTITUTE  
Under ARTI 21-CR Program Contract Number 610-40030

## EXECUTIVE SUMMARY

Germicidal ultraviolet (UVGI) lamps have a long history of use for inactivating microbial aerosols. Most reports have focused on the control of infectious diseases, such as tuberculosis (TB), in the occupied spaces of medical facilities. Ventilation duct use of UVGI has been increasing. In-duct applications are generally more concerned with controlling environmental organisms than with controlling infectious agents. This document reports the results of a project to investigate the ability of UVGI lamps to inactivate representative environmental microbial aerosols in ventilation ducts.

### Results

During this research, UVGI lamps were experimentally demonstrated to inactivate bioaerosols composed of vegetative bacteria, bacteria spores, or fungal spores to a reproducible degree under conditions of fixed dose. Vegetative bacteria were most susceptible to UVGI, with bacteria and fungal spores being substantially more resistant. The performance equation commonly cited in the literature for UVGI inactivation was found to generally apply, provided its parameters were known.

The experimental measurements suggest that the less resistant vegetative organisms, represented by *Staphylococcus epidermidis*, can be somewhat protected by organic material coating the bioaerosol or by increases in humidity. Even with the added resistance, vegetative bacteria remained susceptible to UVGI relative to the spores. A single lamp inactivated more than 50% of the bioaerosol. Humidity and/or organic material coating the bioaerosol had much less impact on inactivation of the spore-formers, which were much more difficult to kill and required use of more lamps. For example, six lamps were required to inactivate the spore-formers to the same extent reached using a single lamp on the vegetative bacteria. Overall, the microorganism-dependent rate constant values determined during this research are similar to the applicable values.

Of the physical factors that might influence UVGI lamp performance, air temperature and flow rate, lamp design, and ballast design were found to be most significant. Isolated changes in humidity had a measurable but very small effect on lamp irradiance. Lamps designated “high output” were found to indeed provide higher irradiance than “low output” lamps, and lamps designed specifically for lower temperature operation also appeared to perform better under those conditions. However, the terminology is not completely consistent. UVGI lamps have a point of maximum output when exposed to various air temperatures and airflow rates. This optimal operating point is fundamentally related to lamp operating temperature and can be reached by various combinations of environment and heat transfer rate (principally air temperature and flow rate) that bring the lamp to the proper operating condition. The reduction in output due to overcooling at temperatures achievable in a heating, ventilation, and air conditioning (HVAC) system has been shown to be as much as a factor of two, which could have a significant effect on kill for bioaerosols.

## Conclusions And Recommendations

The key factors for the design of in-duct UVGI systems intended to disinfect moving airstreams are (1) UV lamp irradiance at operating conditions, (2) microbial resistance data, (3) having a tested and functioning bioaerosol dose model, and (4) duct wall reflectance. Given this information, UVGI system designers can make reasonable and responsible performance estimates for most in-duct applications. (The common lack of test results that provide lamp output at use temperature and airflow makes independent verification of a vendor's UVGI system design difficult.)

The good news that reliable UVGI system design is available must be tempered by an understanding of the intrinsic variance of microbial populations. Resistance variations between strains of the same organism have the potential to be substantial, but very little work has been done. In consequence of this, UVGI system designers should

- Apply safety factors to their designs, particularly as they depart from operating modes for which they have performance data and field experience.
- Know actual lamp output at the most challenging operating condition in the duct.
- Avoid relying solely on design equations to determine the performance of their systems. Actual testing with the contaminants of interest is highly recommended.
- Be extremely cautious regarding claims about UVGI systems' high levels of inactivation of pathogenic bioaerosols. While the microbiological science underlying these conclusions applies to pathogenic bioaerosols as well as environmental organisms, much greater caution is required in the former case. It would be irresponsible to claim a high inactivation rate for a pathogenic bioaerosol without substantial testing. Even with substantial testing, design failures may occur.

In the efforts to counter bioterrorism, the use of UVGI is likely to be proposed with increasing frequency. It is a low-pressure-drop, nonintrusive technology that has real potential. In that light, we recommend that test methods and guidance for performance claims regarding bioterrorism agents be developed by an experienced and broadly based group of technical experts, users, and manufacturers. Until rigorous and adequate tests have been developed and performed, claims regarding protection against aerosol bioterrorism agents are suspect. The potential errors are too large and the consequences too great.

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## 1.0 INTRODUCTION

### 1.1 Background

The use of ultraviolet (UV) lights to disinfect room air and air streams dates to about 1900. Richard Riley has numerous publications in the 1960's and 1970's that deal with UV disinfection and has published an extensive review (Riley, 1988), as has Shechmeister (1991). Early work established that the most effective UV light wavelength was the wavelength range from 220 to 300 nanometers (nm), with the peak effectiveness near 265 nm.

In current commercial practice, UV light of the appropriate wavelength is generated by electrical discharge through low-pressure mercury vapor, which is enclosed in a glass tube that transmits UV light. The resulting germicidal lamp produces UV light that has a primary wavelength of 253.7 nm. This wavelength is within the short-wave, or "C" band of UV light. It is sometimes abbreviated as UVC and is alternately known as germicidal UV or ultraviolet germicidal irradiation (both abbreviated as UVGI). UVGI has been shown to deactivate viruses, mycoplasma, bacteria, and fungi.

To date, most of the use of UVGI for airborne organism control in buildings has focused on limiting the transmission of very infectious diseases (particularly TB) in occupied spaces such as medical waiting or treatment rooms. In this application, UVGI lamps are often located in the upper part of a room (near the ceiling) and must be visually shielded from the occupied space. Mixing between the breathing zone and the irradiated zone is critical to operation, and mixing fans are sometimes used. The effectiveness of near-ceiling UVGI lamp use is fairly well known. Numerous publications are available. In a recent paper that includes an extensive list of references, Miller and Macher (2000) discuss the use and characterization of occupied-space, near-ceiling UVGI. Application principles have been published (CDC, 1994), as have guidelines for system selection and use (CDC 1999a, CDC 1999b.) The occupied space application of UVGI is not discussed further in this report.

UVGI has been used in ducts for some time, and its use is becoming increasingly frequent as concern about indoor air quality increases. Lamp manufacturers have published design guidance documents for in-duct use (Westinghouse, 1982; Phillips, 1985; Phillips, 1992). Recent papers by Schier and Fencel (1996) and Kowalski and Bahnfleth (1998) have summarized the literature and discussed in-duct applications. These and other recent papers were based on case studies and previously published performance data.

For constant and uniform irradiance, the disinfection effect of UVGI on a single microorganism population can be expressed as follows (Phillips, 1992):

$$N_t/N_0 = \exp(-k E_{eff} \cdot \Delta t) = \exp(-k \cdot Dose) \quad (1)$$

where

- $N_0$  = the number of microorganisms at the start,
- $N_t$  = the number of microorganisms after any time  $\Delta t$ ,

- $N_t/N_0$  = the fraction of microorganisms surviving,  
 $k$  = a microorganism-dependent rate constant,  $\text{cm}^2/\mu\text{W} \cdot \text{s}$ ,  
 $E_{\text{eff}}$  = the effective (germicidal) irradiance received by the microorganism,  $\mu\text{W}/\text{cm}^2$ , and  
 $Dose$  = the product of  $E_{\text{eff}} \cdot \Delta t$ ,  $\mu\text{W} \cdot \text{s}/\text{cm}^2$ .

The units shown above are common, but others are used as well, including irradiance in  $\text{J}/\text{m}^2$ . Eq. 1 describes an exponential decay in the number of living organisms as a constant level of UVGI exposure continues. The same type of equation is used to describe the effect of disinfectants on a population of microorganisms, with the dose in that case being a concentration-time product. The fractional kill after time  $t$  is  $(1-N_t/N_0)$ . While the applicability of Eq. 1 is well established for a microorganism exposed to a constant UVGI irradiance, some investigators have detected fine structure in the decay curves, including onset dose thresholds and changes in the  $k$  values at high dose (Kowalski et al., 2000.) Eq. 1 appears to be adequate for practical use in ducts.

Eq. 1 shows an apparent distribution of resistance to UVGI within a population, with a fraction of the microorganisms being killed at a low dose and others at a higher dose. This effect is thought to be due both to a distribution of resistance inherent to the microorganism population and the presence of various contaminants or multiple layers of organisms that reduce the effective UVGI to the resistant microorganisms. Practically, bioaerosols are generated from active colonies that contain numerous contaminants.

In a duct, the use of Eq. 1 is complicated by the movement of the target microorganisms in the airstream and the fact that the UVGI irradiance is not constant within the duct. In addition, the physical parameters of the duct, duct airflow, and UV installation have the potential to affect both the irradiance and the microorganisms' response to it. As is the case with upper-room UV installation design, the principal design parameters for UVGI in in-duct applications are not simple and include some uncertainty in the data available to analyze them and secondary effects. The discussion below expands on these principal and secondary variables.

Recently, the use of UV lamps inside building ventilation ducts has been increasing in both medical and nonmedical buildings for the control of environmental microorganisms. Therefore, a need has arisen to better understand the capabilities of such installations. Prior to this research, public data supporting the design and application of UV products in ducts were very limited. This research arose out of a desire, on the part of the ventilation industry, to independently evaluate the claims and design guidance of UV light vendors and manufacturers. Some UV applications are intended to disinfect the air and others are intended to disinfect surfaces. The research presented in this report was limited to in-duct use of UV lamps intended to disinfect flowing air. Only environmental (not pathogenic) microorganisms were included in the test program.

## 1.2 Project Objectives

The overall objective of this research program was to “obtain reliable scientific data through laboratory testing to determine the antimicrobial efficacy of UVC in circulating air ducts and the

potential ability of these lights to kill or inactivate common microbes and subsequently reduce indoor air microbiological contamination.”

### **1.3 Technical Approach**

To accomplish the project objective, four tasks were undertaken:

1. Identifying the key factors impacting the use of UVC lights in circulating air ducts,
2. Quantifying the impact of these factors on the ability of UV devices to kill or limit common environmental microorganisms,
3. Developing documented relationships between UVC disinfection efficacy and in-duct use and design, and
4. Communicating that understanding to the ARI-member audience through appropriate and useful test methods and design guidance.

Identification of the key factors was accomplished through a literature search and the reconciliation of published UVC performance with the experimental results obtained during the research. Quantification of the impact of these factors and the development of documented relations of UVC performance were linked through the development of a model to calculate (estimate) the UVC dose received by the organisms in the airstream and thereby link the physical and microbiological measurements and permit their generalization to other situations.

### **1.4 Report Overview**

Section 2.0 documents the apparatus and methods used during the experimental phase of this research and also describes the model used to calculate the UVGI dose received by the bioaerosols during the experiments. Section 3.0 identifies the key physical and microbiological factors for in-duct UVGI use and briefly summarizes their impact as reported in the literature and as demonstrated in experimental work from this project. Section 4.0 presents and discusses results that bear on the primary research objective. Sections 5.0 and 6.0 present conclusions and recommendations based on this research. References for the report are presented in section 7.0.

## **2.0 EXPERIMENTAL METHODS AND APPARATUS**

The experimental methods and apparatus used during this research consisted of the following:

1. Operation of a test duct with UVGI lamps installed. This operation included microbiological sampling upstream and downstream of the lamps and control and measurement of rig temperature, humidity, and flow rate,
2. Installation, operation, and irradiance measurements for the UVGI lamps,
3. Chemical byproducts measurements in the test rig,
4. Test bioaerosol generation, sampling, counting, and disinfection efficiency calculation, and
5. Use of a bioaerosol UVGI dose model to compute microbial resistance constants, which allow generalization of the efficacy results to other lamp and duct configurations and comparison to the literature.

## 2.1 Full-Scale Test Duct

The test duct used during this research is shown in Figure 1. It is a full-scale American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc. (ASHRAE) 52.2 test rig (ASHRAE, 1999) of recirculating design with airflow control over approximately 600 to 3,000 cfm (17 to 85 m<sup>3</sup>/min), temperature control between 50 and 85 °F (10 and 30 °C), and humidity (35–85% RH) control. The airflow through the test duct was measured using a calibrated ASME flow nozzle. Flow was controlled by modulating the fan motor speed using the computer interface. Control to the flow set point was automatic with operator tuning as required. Temperature and humidity control were achieved using a flow-modulated chilled water coil and modulated steam injector that were also computer-controlled. The test duct was operated at positive pressure in the sample section to prevent the infiltration of room air aerosol. Air entering the test section was filtered with High Efficiency Particulate Air (HEPA) filters to remove ambient aerosol from the airflow.

As shown in Figure 1, the test rig is set up for recirculating operation, which was the operating mode used for all bioaerosol runs. The dampers were set to draw air into the rig from the conditioned space and discharge it through the roof for the byproduct chemicals tests.

Figure 2 shows additional details of the aerosol injection region of the test duct. The test bioaerosol was injected at the location shown in Figures 1 and 2. For all experiments, the test bioaerosol was nebulized from a liquid solution using a six-jet Collison nebulizer. The upstream and downstream mixers shown have two parts: an upstream 30.5-cm (12-in.) diameter orifice in a steel plate and a downstream 15.2-cm (6-in.) diameter target disk centered on the orifice. Details of the test rig are given by ASHRAE (1999). For the initial screening tests, the test bioaerosol was injected either directly into the duct (for easily desiccated vegetative bioaerosols) or through the ASHRAE 52.2 drying tower (for more robust bioaerosols, such as the spores) and thence into the duct through a Kr-85 aerosol neutralizer. The drying tower operation is described in ASHRAE 52.2, and provides approximately a 50 s residence time for aerosol conditioning in 0.00189 m<sup>3</sup>/s (4 cfm) of clean, dry air.

Both injection techniques appeared to give adequate dispersion and mixing of the test aerosol, which had a mean aerodynamic particle diameter of 2 to 3 μm. However, use of the drying tower was considered to be better practice and all test bioaerosols were injected through the drying tower for the final tests. Further details of the microbial test aerosol preparation are provided in Section 2.6.

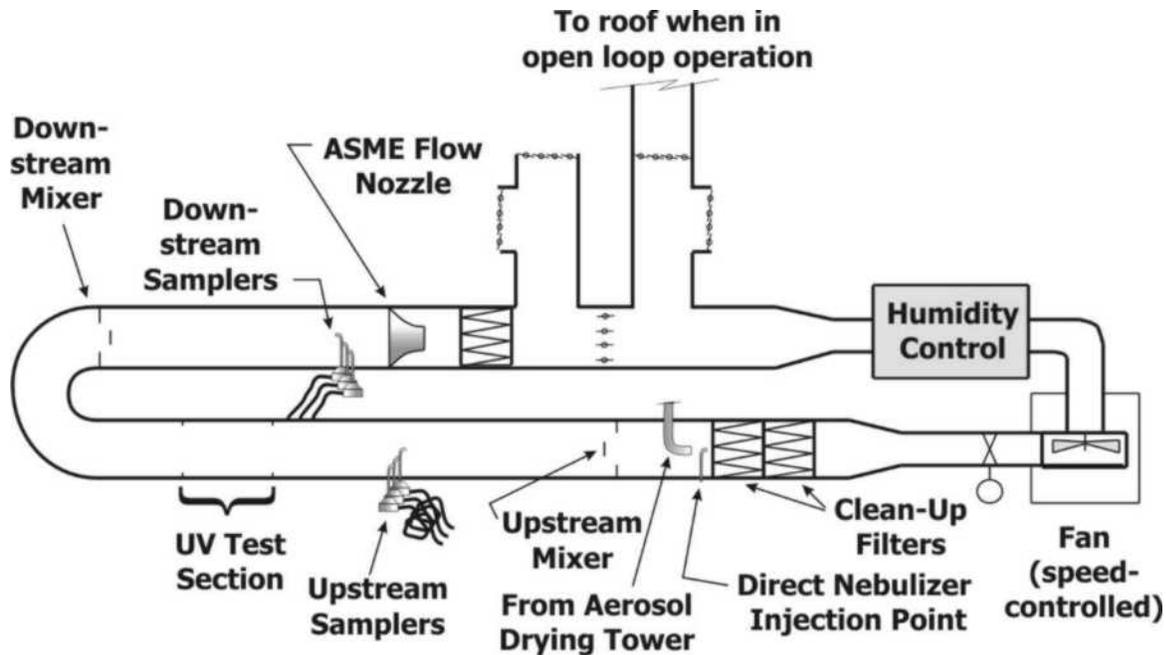


Figure 1. 52.2 test rig arranged for bioaerosol tests of UV lamps.

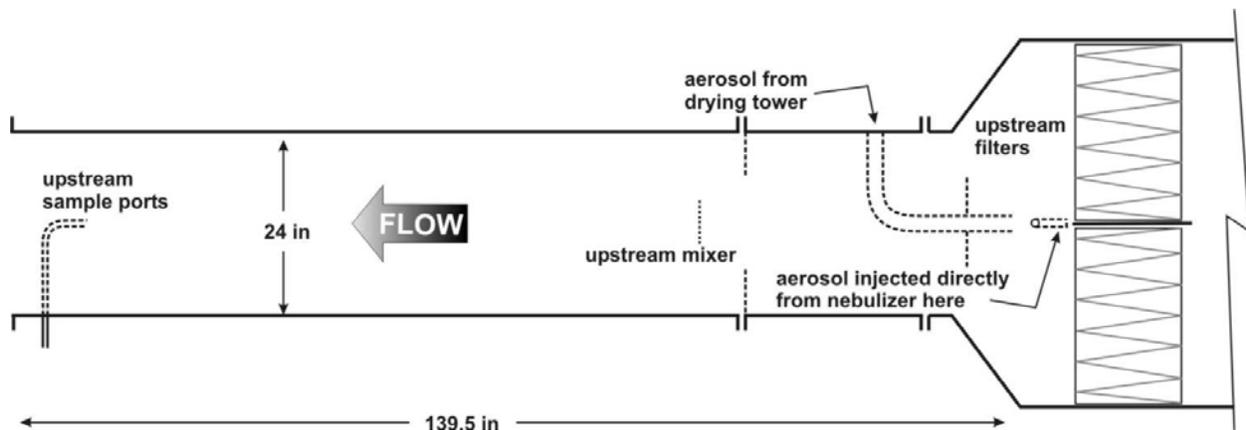


Figure 2. Detail of the aerosol injection region of the test duct.

Aerosol concentrations were measured upstream and downstream of the test section to obtain the challenge and penetrating bioaerosol concentrations, respectively. The inlet nozzles of the upstream and downstream aerosol probes were designed to yield isokinetic sampling conditions for the 28.3 L/min (1-cfm), single-stage Andersen samplers. The bioaerosol sample ports were located at points of well defined airflow and well mixed aerosol conditions, as required to achieve unbiased, representative samples. Mixing and flow at the sample locations were confirmed by following the procedures in ASHRAE 52.2, which require the same suitable flow conditions. Aerosol losses through the duct and the 180° bend, while present, were not large (typically a few percent at 2  $\mu\text{m}$  and less than 20% loss at 10  $\mu\text{m}$ ). The data reduction system compensated for these losses as described below in the quality assurance (QA) and quality control (QC) procedures.

## 2.2 UVGI Lamp Operation and Irradiance Measurement

### 2.2.1 UV Lamps and Ballasts

Two types of commercial UV lamps were utilized during this research. Because the industry does not use a standard test to measure UVGI lamp output presented in most sales literature, the lamps are difficult to characterize, except through direct measurement. Performance data are presented below. Visually, the two test lamps were identical except for the label. Both were 0.61-m (24-in) long, T5 (~ 1.6-cm (5/8-in.) diameter), single-ended lamps intended for insertion into a duct from outside. The ballasts were connected to the lamps through four-pin power connectors. Power was provided to the in-duct end of the lamp by small wires running from the power connector along the outside length of the lamp tube. A flange on the power connector was used to clamp them in place perpendicular to the duct wall. The two lamp types were

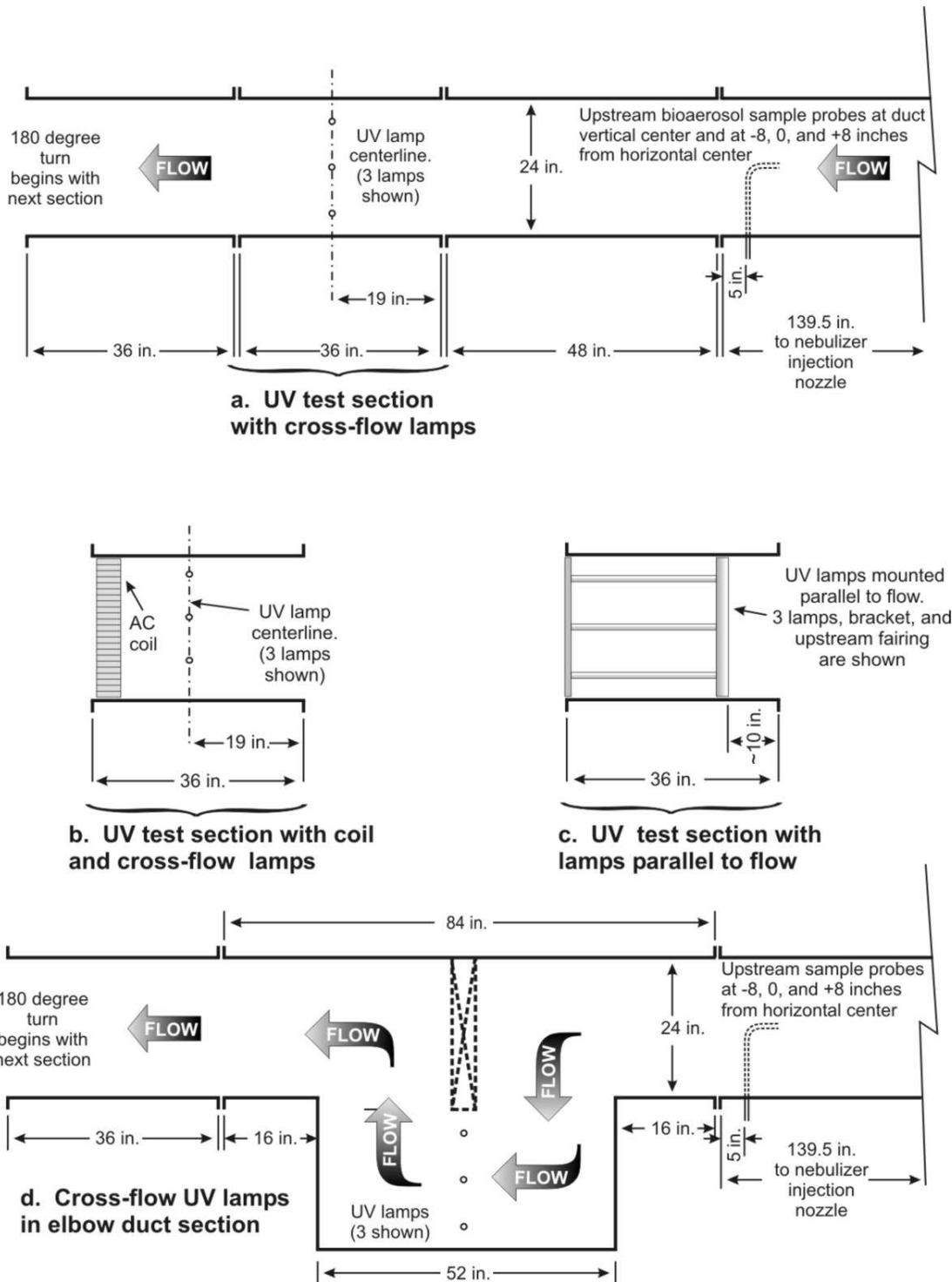
1. Low-output lamps driven by conventional magnetic ballasts. These lamps were obtained because they appeared (from inspection of vendor sales literature) to be characteristic of the low-cost end of the in-duct market. They were branded by the vendor and thus could not be independently traced back to a lamp manufacturer. Discussions with industry experts led to a characterization of these lamps as low-pressure mercury lamps driven at low power. In still air at 21°C (70°F), surface output was computed for a single lamp .
2. High-output lamps driven by electronic ballasts. Based on vendor sales literature, these lamps were designed for both higher output and also improved output at low temperature and with air flowing past them. They too were branded by the vendor and could not be independently traced. An industry expert characterized these lamps as low pressure mercury lamps that were over-driven to achieve higher output.

### 2.2.2 UV Lamp Operating Configurations

The UVGI lamps were operated (except during the alternate duct configuration tests) in the test section shown in the center of Figure 3a, with the lamps in a vertical column perpendicular to the airflow (crossflow). There were three primary lamp arrangements:

1. One lamp at duct center,
2. Three lamps, one at the center and one above and below on 20.3-cm (8-in.) centers,
3. Six lamps spaced on 10.2-cm (4-in.) centers in a vertical column.

A screen was used to obtain irradiance levels lower than those that could be obtained from a single low-output lamp. The screen consisted of three concentric layers of common window screen, rolled into a cylinder and slipped over the lamp.



**Figure 3. Lamp and test duct configurations**

### 2.2.3 In-Duct Irradiance Measurements

Bioaerosols in a duct may be exposed to UVGI light from all angles, particularly if the duct is significantly reflective to UVGI. The commonly available detector for UVGI is a solid-state

detector whose otherwise broad wavelength response is limited (using filters) to the UVC. A calibration is used to convert the voltage produced by the detector to UVGI irradiance. Some filters pass a broad UVC spectrum, while others have a narrower response.

All irradiance measurements conducted during this research utilized an International Light (IL) IL1700 Research Radiometer fitted with a factory NIST-traceable calibrated IL SED240 detector, QNDS2 filter, and W quartz diffuser. This detector has a 185 to 310 nm wavelength spectral response. With the diffuser installed, the detector has what is known as a cosine response characteristic. A true irradiance reading is obtained for light entering perpendicularly (incident angle of  $0^\circ$ ) into the detector. The detector measures UV light entering from other angles as the product of the true irradiance and the cosine of the angle of incidence. Positioned in the center of the duct and aligned in the direction of flow, the UV detector measures all UV light striking it at  $0^\circ$  and 70.7% of the UV light striking it at an incident angle of  $45^\circ$ . Very little UVGI is detected as the incidence angle widens to the sides of the detector and, of course, no UVGI from the back can be detected. All common UVGI radiometers utilize the same operating principle and have the same limitation.

Use of a standard, cosine-response UVGI radiometer in this research was desirable because it is the instruments most likely to be used in the ventilation industry. The use of this type of radiometer required investigation because a bioaerosol particle receives UVGI energy from all angles, while the UVGI radiometer does not. Should reflected UVGI from the duct walls be a high fraction of the total UVGI impinging on the bioaerosol particles, single-position measurements made with these detectors aligned with the duct axis would underestimate the total dose and be of limited value. Multi-axis measurements with a cosine-response detector would be time consuming and require consolidation of the data into a total irradiance value. (Photosensitive chemical methods have been developed for time-integrated UVGI dose measurement; however, these are also difficult to implement in the field.)

Irradiance measurements within the test duct were made at various distances from the lamps. The detector was centered in the duct at distances ranging from 25 to 100 cm (10 to 39.4 in.) from the lamp centerline and aligned along the duct centerline. The instrument cable was run through a duct wall penetration and connected to the IL1700. All lamp performance measurements were made after allowing the UV lamps to stabilize at the duct operating conditions.

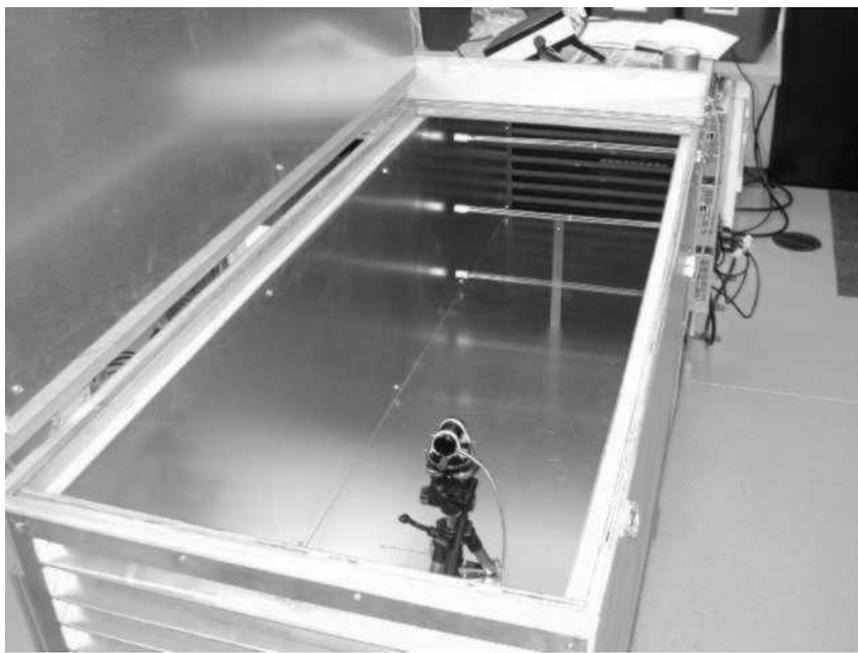
#### 2.2.4 Apparatus for All-Angle UVGI Measurements

A mocked-up duct section was utilized to measure irradiance from all possible incident angles and thereby permit evaluation of the errors attendant to use of the radiometer detectors. The duct mock-up was used in place of the test duct to allow the detector to be rapidly positioned without exposing the operator to the UVGI. The sections below describe the apparatus and methods.

##### 2.2.4.1 Duct mock-up and all-angle UVGI measurements

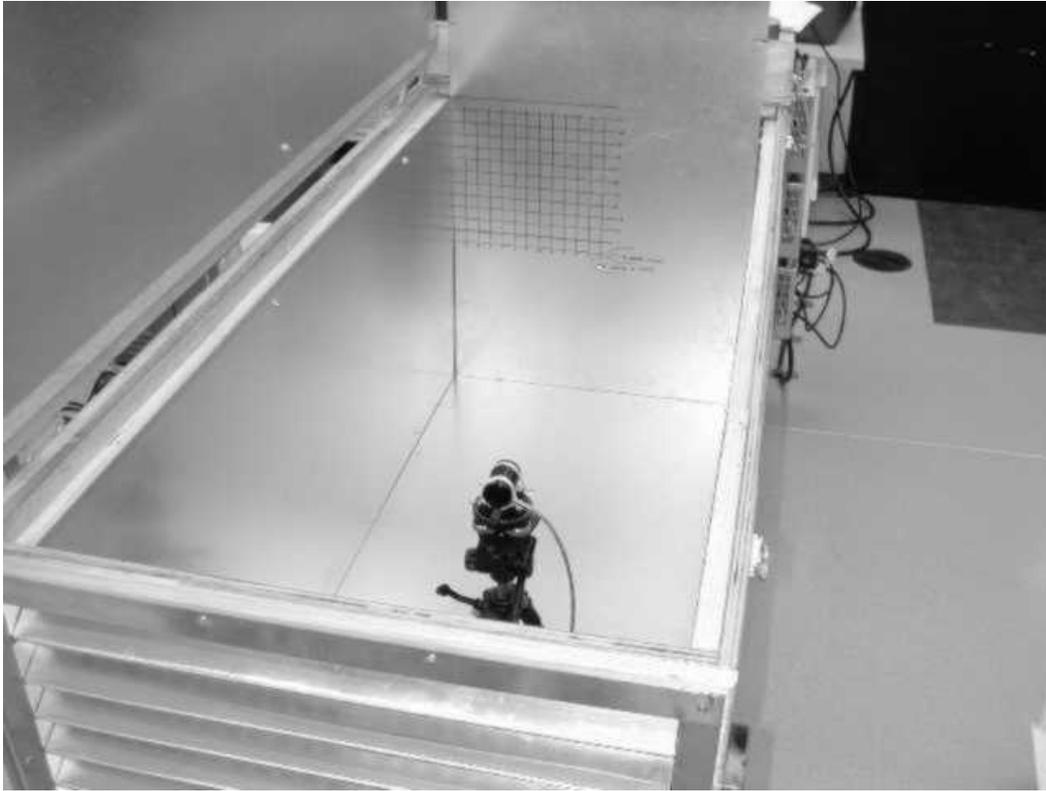
The duct mock-up was a 61- by 61-cm (24- by 24-in.) interior dimensions duct section in which the light from the lamps could be blocked to allow the radiometer to be adjusted without exposing the operator to the UV light. The wall materials could also be easily changed for

reflectance measurements. The mock-up consisted of a box with a replaceable lining. The ends of the apparatus were covered with grills to prevent accidental exposure. Access into the box was through two full-depth doors that formed the top of the duct mock-up. The three test lamps were installed in a vertical column on the right side of the apparatus as shown in Figure 4. Because the lamps needed to be kept at a stable operating temperature, they could not be turned off without allowing for a substantial warm-up period. Accordingly, they remained on throughout the experiment, and a sheet metal guillotine damper (seen in Figure 5) was lowered before the access door was opened. The relative irradiance level was primary in this experiment, not the absolute level, and therefore temperature control beyond a stable operating condition was not required. For all of these experiments, three low-output lamps powered by magnetic ballasts were positioned perpendicular to the “flow direction” in the duct mock-up, as shown in Figure 4. One lamp was at the horizontal center, one 20.3 cm (8 in.) above, and one 20.3 cm (8 in.) below.



**Figure 4. Lamps shown in an inside view of duct mock-up apparatus.**

The radiometer and detector were described above. The detector was mounted on a rotating and hinged protractor-equipped base and tripod that allowed the detector to be moved vertically and aligned in any horizontal or vertical angle. A pencil laser (mounted below, and aligned with, the detector axis of measurement) was used to accurately position it using the grid on the damper. While not obvious in the photographs, a  $22^\circ$  acceptance angle cone was mounted on the front of the detector to prevent confusion when interpreting the all-angle measurement data.



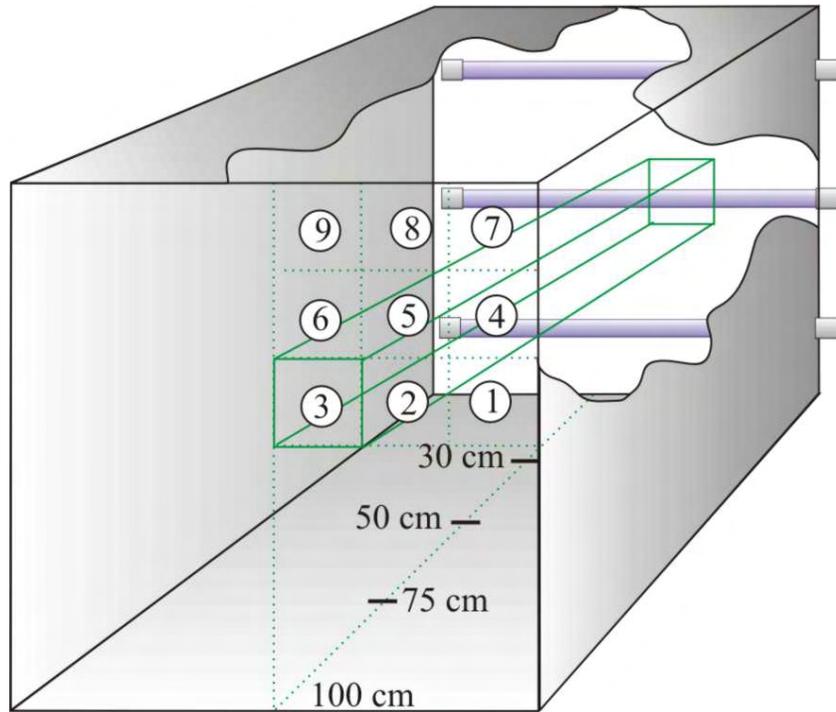
**Figure 5. Inside of duct mock-up with guillotine and locator grid.**

The UV detector was positioned at 50 and 100 cm (19.7 and 39.4 in.) from the plane of the lamps at the center of the test duct. Irradiance measurements were made every  $22.5^\circ$  around both a horizontal and a vertical plane, with  $0^\circ$  being the perpendicular from the plane of the lamps to the detector face.

#### 2.2.4.2 Measurement of irradiance with lined duct

Different materials reflect UV light to a different extent, as further discussed below. The apparatus was used to allow the measurement of irradiance for both relatively high- and low-reflectance materials. The purpose was to determine effect of reflectance on the uniformity of irradiance in the duct and the level of irradiance measured using the radiometer.

Figure 6 is a schematic drawing of the measurement locations in the duct mock-up. The irradiance from three low-output lamps was measured at static flow conditions, which allowed comparison within this data set. The measurements were made within a single quadrant of the duct mock-up. This quadrant was divided into 9 segments, each 10.2 (4 in.) on a side (this is the same grid used in the performance model). Measurements were made with the radiometer detector facing the lamps at 30, 50, 75, and 100 cm (11.8, 19.7, 29.5, and 39.4 in.) from the plane of the lamps. The same set of measurements was made for new galvanized metal and for photographic black flock paper.



**Figure 6. Reflectance measurement position grid.**

## 2.3 Chemical Byproducts Experiments

All chemical byproducts measurements were conducted in the full-scale test duct at ambient temperature and humidity and at a flow rate of  $0.472 \text{ m}^3/\text{s}$  (1,000 cfm.) To prevent buildup of any byproducts, the test duct was operated in open-loop mode, without recirculation. Inlet air was drawn from conditioned space, filtered ahead of the test section, and discharged through a roof vent. Six lamps were mounted and operated in crossflow on 10.2-cm (4-in.) centers. They were allowed to operate for at least 15 minutes prior to beginning each test.

### 2.3.1 Ozone Measurement

Specific types of UV lamps are used to generate ozone in laboratory settings, which raises questions about ozone production by UVGI lamps. The 185-nm emission line for mercury is generated in low-pressure discharge lamps and would generate ozone in air. However, most low-pressure UVGI lamps intended for use in air utilize glass that does not pass the 185-nm

wavelength; this eliminates/minimizes ozone production. Medium-pressure mercury lamps, on the other hand, may generate ozone. The intent of the ozone generation experiment was to validate the assertion that no measurable ozone was produced under normal operating conditions. The challenge of this experiment lay in the large amount of air required to operate normally and hence the expected low concentration difference between inlet and outlet concentrations.

The potential for UVGI lamps to produce ozone under operating conditions was investigated using an ambient ozone analyzer (Thermo-Environmental Instruments, Inc., Model 49 UV Photometric O<sub>3</sub> Analyzer, 0–50 ppm range) to measure ozone upstream and downstream of the six UVGI lamps. The gas sample was drawn directly into the instrument. The upstream sample was obtained at the bioaerosol upstream port (2.13 m (7 ft) upstream of the lamps), and the downstream samples were centered approximately 46 cm (1.5 ft) downstream and at the downstream bioaerosol sample port [6.1 m (20 ft) downstream.] Tests were run with both the low- and high-output lamps.

### 2.3.2 Volatile Organic Chemical Byproducts Measurement

Similarly to the issues regarding ozone production by UV lamps UV light under some conditions is also known to induce chemical reactions in some volatile organic chemicals (VOCs). The goal of this experiment was to operate the UVGI lamps under normal duct operating conditions, challenge them with a “cocktail” of VOCs, sample upstream and downstream, and look for chemicals downstream that were not found upstream. As with the ozone experiment, the major experimental challenge was the level of dilution, which would make detection of low levels of byproducts very difficult.

The test duct was operated at 0.472 m<sup>3</sup>/s (1,000 cfm), 22°C (72 °F), and 50% RH in open loop mode. A VOC cocktail consisting of approximately equimolar fractions of toluene, hexane, 2-butanol (commonly known as methyl ethyl ketone, or MEK), and iso-butanol was injected into a total vaporizer at 0.2 mL/min. The vaporized liquid entered a 57 L/min (2-cfm) hot airstream that was injected into the test duct, producing an approximately 1.5 ppm VOC challenge.

Air samples were drawn through adsorbent sampling tubes at 0.2 L/min for 90 min to obtain upstream and downstream samples of the duct air. The sample tubes were located at the duct center at the location of the bioaerosol upstream and downstream sample ports. Duplicate samples were drawn; the total duct operating time was approximately 3.5 hours. A field blank adsorbent tube was also obtained.

The sample tubes were returned (chilled) to the commercial analysis company, where they were desorbed into a GC/MS and the concentrations of total and individual VOCs were measured.

## **2.4 Microbiological Measurements**

### 2.4.1 Bioaerosol Disinfection Efficiency Measurement

The basic measurement of bioaerosol kill by UVGI irradiation was based on the following experimental procedures:

1. Establishment of stable flow and lamp operation in the test duct at the test conditions,

2. Generation of a stable test bioaerosol of the desired test organism,
3. Collection of upstream and downstream bioaerosol samples, using one-stage Andersen biological samplers,
4. Growth and enumeration of microbiological samples, and
5. Data reduction to compute the kill efficiency of the UVGI lamp configuration.

The same general procedures were used for each bioaerosol kill measurement.

#### 2.4.2 Test Duct Operation

The test duct was operated as described in Section 2.2.

#### 2.4.3 Test Bioaerosol Generation

Except for a brief investigation of solid aerosols, all test bioaerosols used during this research were nebulized from an aqueous organism suspension and injected into the test duct. Depending on the organism, the bacterial and fungal challenge suspensions were prepared by either

1. Inoculating the test organism onto solid media, incubating the culture until mature, wiping a wetted sterile swab across the surface of the pure culture, and eluting from the swab into sterile high-purity deionized [18 megaohm (M $\Omega$ )/cm resistance] water to obtain 15% transmission on the spectrometer, or
2. Purchasing a spore suspension from a commercial vendor and diluting it in sterile water.

Both correspond to a concentration of approximately  $1.5 \times 10^7$  CFU/mL. The microorganisms were resuspended in sterile 18 M $\Omega$ /cm deionized water. Very high purity water was used to minimize the particles from sources other than the organisms themselves (e.g., dissolved solids). For the broth suspensions, the organisms were prepared following the same protocol, but were resuspended in trypticase soy broth instead of 18 M $\Omega$ /cm deionized water. However, in an attempt to minimize particulate contamination from the broth, the broth was prepared in 18 M $\Omega$ /cm deionized water.

The challenge organism suspensions were aerosolized using a Collison modified MRE-type six-jet nebulizer (BGI, Waltham, MA) at 100 kPa (15 psi) air pressure. The Collison generates droplets with an approximate volume mean diameter of 2  $\mu$ m. The particle diameter after the water evaporates depends upon the solids content of the suspension. Particle size is determined by the size of the suspended particles (if singlets).

During bioaerosol generation, an optical particle counter (OPC) was used to monitor particle levels in real time to enable control of the experiment within the test duct. The output was sent to a RS-232 serial port and was recorded on a computer. The OPC was not used for the efficiency measurements because it cannot distinguish viable from non-viable organisms.

#### 2.4.4 Bioaerosol Sampling

Bioaerosol samples were collected by sequential sampling with the one-stage Andersen bioaerosol samplers. Simultaneous triplicate samples were obtained during each upstream or downstream sampling session, and two upstream and two downstream sample sessions were run on any single experimental condition. The bioaerosol sampling lines were 1.4 cm ID stainless

steel lines (5/8-in. OD tube) with dimensions chosen to minimize particle losses during sampling. The sample lines upstream and downstream had the same configuration in terms of diameter, horizontal and vertical lengths, and number of bends limit upstream and downstream differences. Full flow quick-connects were used to connect the bioaerosol samplers to the upstream and downstream sample lines. Sampling nozzles of the appropriate entrance diameter were placed on the ends of the sample probes to maintain isokinetic sampling for the test flow rate.

The one-stage Andersen samplers (Graseby Andersen, Atlanta, GA) are 400-hole multiple-jet impactors operating at 28.3 L/min (1 cfm.) During sampling, air is impinged on agar in petri dishes positioned beneath the stages. Only culturable, viable microorganisms were measured.

#### 2.4.5 Microbiological Growth and Enumeration

The bioaerosol sampler plates were allowed to grow and colony-forming units (CFUs) were enumerated. The growth media used were trypticase soy agar (TSA) for the bacteria and Sabouroud's dextrose agar (SDA) for the fungi. CFUs were counted shortly after moderate growth became apparent.

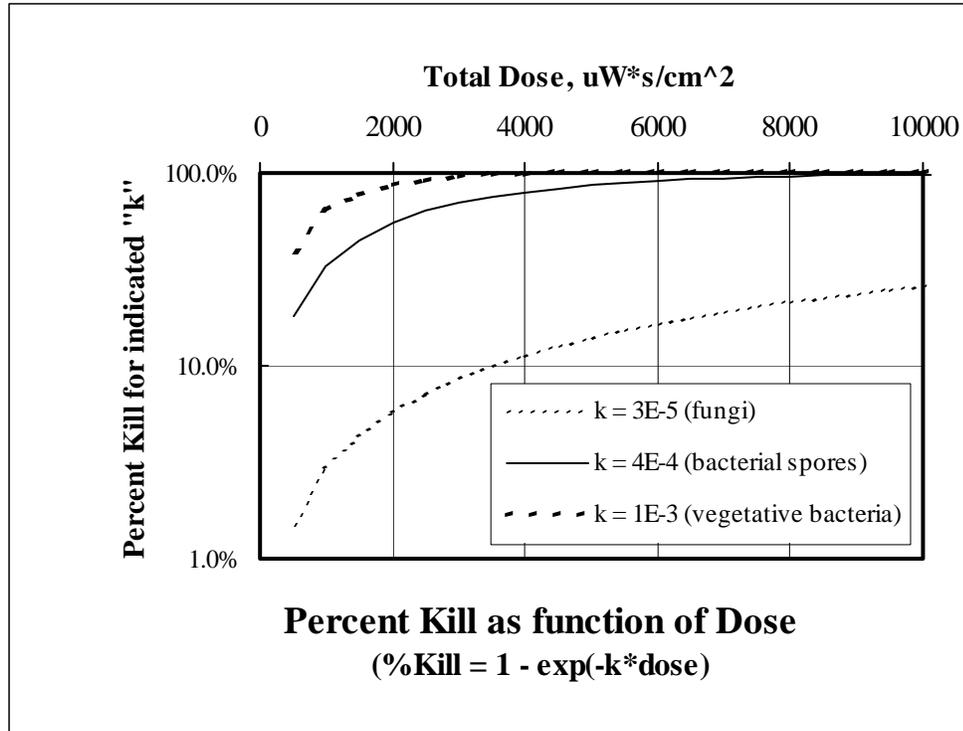
A "positive-hole" correction is applied to adjust for the probability that more than one viable microorganism is collected through a sampling hole and combined with other microorganisms to produce a single CFU (Macher, 1989). The impactor cut-point ( $d_{50}$ ) is 0.65  $\mu\text{m}$  for the one-stage Andersen.

#### 2.4.6 Microbial Kill Estimates for Experimental Planning

An individual microbiological kill effectiveness test as described above provides the kill in the form of the ratio  $N/N_0$  for a given organism, configuration, lamp, and operating condition (flow, temperature, RH.) Eq. 1 can be used to generalize effectiveness measurements for various UV lamps and microorganisms by computing the microbial resistance constant,  $k$ , provided all the other terms are known. This requires that the parameters in Eq. 1 be in a measurable range. The radiometer described above had a sufficient dynamic range to measure  $E_{eff}$  from as many lamps as was practical to place within the test duct. The exposure duration,  $\Delta t$ , was calculable as the bioaerosol path length having meaningful UVGI exposure, divided by the duct velocity.

For the microbial kill experiments, it was important to stay within a measurable range of kill. An experimental condition that achieves 100% kill cannot be analyzed because the fraction of UVGI radiation that is "wasted" cannot be evaluated. Similar, zero kill (below the detection limits of the impactors) experiments were not useful. The tests were conducted in an expected kill range of roughly 10–95%, although dilutions and long sampling runs extended the range somewhat. For these reasons, the initial step in setting up the UV lamp systems for these experiments was to estimate the microbial kills for different configurations of UVGI lamps and duct velocities.

Eq. 1 was used to estimate the UVGI dose required in a duct to achieve a measurable microbial kill. Figure 7 plots Eq. 1 for a reasonable range of  $k$ . Fungi are the most resistant to UVGI, while vegetative bacteria are easily killed. Figure 7 shows that achieving UVGI doses in the 500 to 2,500  $\mu\text{W}\cdot\text{s}/\text{cm}^2$  range should provide adequate range for the bioaerosol experiments.



**Figure 7. Computed % kill as a function of dose and microbial resistance**

Lamp output was directly measured in a duct, and doses in the range of approximately 1,500 to 2,500  $\mu\text{W}\cdot\text{s}/\text{cm}^2$  were found to be readily achievable with three low-power lamps in a 2-m (78.9 in.) exposure distance [1 m (39.4 in.) on either side of the lamps.] The irradiance field was found to be relatively uniform. The initial bioaerosol experiments were then designed with confidence. Later experiments were designed applying more sophisticated models of bioaerosol dose in a flow air duct that are described in the next section.

Figure 7 also shows that increasing dose (with more or better lamps or ballasts or by reducing velocity) does not proportionately increase kill and will have very little effect at some dose levels. This also means that a modest dose can do nearly as well as a high dose and achieve high kill through recirculation and repetitive exposure.

#### 2.4.7 Data Reduction

Measurements were made with the UVGI lamps both “ON” and “OFF” to account for losses in the duct downstream of the lamps. A single measurement run consisted of two sets of three simultaneous, 1-min, one-stage Andersen upstream samples averaged to give the upstream challenge count,  $C_{\text{up}}$ , and two sets of three downstream samples averaged to provide the downstream challenge count,  $C_{\text{down}}$ , for both the “ON” and “OFF” cases. The fractional penetrations,  $P_{\text{ON}}$  and  $P_{\text{OFF}}$ , were computed as the respective  $C_{\text{up}}/C_{\text{down}}$ . The inactivation efficiency was then computed as shown in Eq. 2:

$$\text{Inactivation efficiency} = 100 \cdot (1 - P_{\text{ON}}/P_{\text{OFF}}) \quad (2)$$

The two replicate runs were compared and averaged to arrive at a final test result for the device, challenge organism, and operating conditions. The dose exposure model was then used to compute the  $k$  constant for the organism used in the test.

#### 2.4.8 Bioaerosol QA/QC

Multiple quality assurance and control tests were used to maintain high-quality data. The first line of checks were physical system checks to ensure that the test conditions were as specified. All measurement instruments were calibrated and maintained as suggested by the manufacturer. System configuration was checked prior to each test to ensure that the test bench was set up as specified. Temperature, humidity, duct flow rate, and UVGI irradiance were measured prior to and following each test.

No standard UV germicidal device exists to allow calibration of the microbiological aerosol efficiency measurement in the test duct. However, the test duct can be “zeroed and spanned” by testing at the 0% and 100% penetration points. In previous filter tests, these tests have been found critical for maintaining high-quality data. The 100% penetration tests were performed to provide a relatively stringent check of the adequacy of the overall duct, sampling, measurement, and aerosol generation system, as well as organism viability. These tests were performed as normal disinfection efficiency tests, except that the UVGI lamps were not turned on. A perfect system would yield a measured penetration of 100% for the test bioaerosol. Deviations from 100% efficiency occur because of particle losses in the duct, losses within the test device, differences in the degree of aerosol uniformity (i.e., mixing) at the upstream and downstream probes, differences in particle transport efficiency in the upstream and downstream sampling lines, and loss of viability of the test organism. Results from the no-light tests ( $P_{\text{OFF}}$  correction factors) were used during data analysis to correct the efficiency measurements obtained during the filter tests.

The 100% efficiency test was conducted by installing a HEPA filter in the test duct to ensure that near-zero penetration was measured under the sampling conditions. Measurement of significant penetration generally indicates sampling line leaks or sample handling problems.

Additional bioaerosol QC tests ensured the purity of the test organism suspension and the sterility of the media and sterile water.

### **2.5 In-Duct Irradiance Model and Dose Calculations**

While the UVGI irradiance and the microbial kill achieved can be measured as described above, an important part of the data analysis is the development of a model that allows generalization of a few measurements to the larger world of air ducts. This section describes the model used in this research. The general approach applied was to begin with a model for irradiance in a duct and compare that model to measurements made with the radiometer. The model was then calibrated such that it predicted the irradiance measured at a few locations. A set of reasonable bioaerosol trajectories in the duct were then proposed, and the dose received by a particle following each trajectory was computed step-wise over a distance of 1 m (39.4 in.) This trajectory dose was used with Eq. 1 to compute bioaerosol survival fractions on that trajectory.

The survival fractions were averaged over all of the trajectories to obtain an average survival, from which an average  $k$  could be computed.

### 2.5.1 UVGI Irradiance Field in an Air Duct

UV light is electromagnetic radiation, and as such is governed by well known physics. All photocatalytic reactions (smog formation, for instance) and radiant heat transfer follow the same physical laws. The energy passing through a fixed area decreases according to the inverse square law, because the irradiance reduction is inversely proportional to the square of the distance from the source. This assumes a point source and point receptor.

Because commercial UVGI lamps are generally cylindrical sources with finite length and diameter, the inverse square law is not easily used to compute the UVGI irradiance field within a duct. An arbitrary location in the duct is irradiated by the whole length of the lamp as well as by reflected light. In practice, the UVGI field falls off roughly as the inverse of distance from the lamps. For most lamps and ducts, this means that the biological inactivation takes place within 0.5 to 1.0 m (20 to 40 in.) of the lamps.

Note: Terminology is not uniform within the literature. Phillips (1992) uses “irradiance,” which is defined with units of power per unit area (e.g.,  $\mu\text{W}/\text{cm}^2$ ), as does Ryer (1998) in his Light Measurement Handbook. Irradiance is a different quantity than “intensity,” which is an angular measure with units of power per steradian (e.g.,  $\text{W}/\text{sr}$ ) as stated by Ryer (1988). The UV literature, however, sometimes uses “intensity” with the meaning and units of “irradiance.” Confusion will be minimized by carefully observing the units that accompany the numbers.

Use of the view factor calculation is an available alternative. Because photocatalytic reactions and radiant heat transfer have both been studied extensively, means to compute the energy passing through a unit area have been developed for numerous geometries, including a cylindrical source and planar receptor. Use of these equations is cited by Phillips (1992) and Kowalski and Bahnfleth (2000). The intensity of the lamp is assumed to be constant over its length, and the irradiance can then be calculated at any location in the duct.

The direct UVGI irradiance field at any position can be calculated for cylindrical lamps by using a view factor equation originally developed for radiant heat transfer from cylinders (Modest, 1993). The applicable view factor is that for a “differential planar element to a right-circular cylinder of finite length,  $l$ , and radius,  $r$ ; normal to element that passes through one end of the cylinder and is perpendicular to the cylinder axis.” The length of the normal between the cylinder and the element is  $h$ . For an arbitrary differential planar element and a UV lamp, the view factor must be computed for each end of each lamp because the perpendicular passes through one end of the cylinder. The sum of the view factors for the two ends of the lamp gives the total view factor for the lamp. The product of the lamp view factor and the lamp surface intensity gives the total UVGI energy predicted to pass directly through point P.

$$VF = \frac{L}{\pi H} \left[ \frac{1}{L} \tan^{-1} \frac{L}{\sqrt{H^2 - 1}} + \frac{X - 2H}{\sqrt{XY}} \tan^{-1} \frac{\sqrt{X(H-1)}}{\sqrt{Y(H+1)}} - \tan^{-1} \sqrt{\frac{H-1}{H+1}} \right] \quad (3)$$

where

$VF$	=	view factor, either left or right end of lamp,
$l$	=	lamp segment length,
$r$	=	lamp radius,
$h$	=	perpendicular distance from lamp to point, P, in duct,
$L$	=	$l / r$ , dimensionless lamp length,
$H$	=	$h / r$ , dimensionless distance,
$X$	=	$(1 + H)^2 + L^2$ , and
$Y$	=	$(1 - H)^2 + L^2$ .

$$VF_{entire\ lamp} = VF_{left} + VF_{right} .$$

$$\text{Irradiance at P} = \text{lamp UVGI surface irradiance} \cdot VF_{entire\ lamp} ;$$

$$\text{Irradiance at P} = (\text{energy into lamp} \cdot \text{UVGI output efficiency}) / \text{lamp surface area} .$$

The calculation was repeated for each lamp and the irradiance added to obtain total irradiance at each point. Dose calculations have been reported using an assumption of total mixing with irradiance computed at a fixed distance to use of the view factor equation to compute irradiance at multiple points along an assumed or computed particle trajectory.

The view factor calculation does not account for reflected light. As stated above, the literature (in most cases) accounts for reflectance from ducts by multiplying the direct irradiance by a factor that depends on the duct wall material. Kowalski and Bahnfleth (2000) mention use of a ray-tracing method that allows direct calculation of the UVGI energy reflected from a surface. Ray tracing appears to have some potential advantages as a more exact calculational tool, but the discussion is limited and its overall usefulness relative to more conventional techniques is not clear. Precision in the UVGI calculations may not limit the design of UVGI systems.

Practically, UVGI lamp output is known to be affected by a number of factors, as discussed below. The data appear to have trends, but the published literature is incomplete. Because this project deals with the practical issue of how to ensure that germicidal UVGI installations work with high reliability, measurements are needed. These measurements can then be correlated with the microbiological effects.

## 2.5.2 Particle Movement in Air Ducts

### 2.5.2.1 Duct airflow pattern

The mean velocity of the ventilation air in the vicinity of UVGI devices is commonly in the range of 7.1 to 14.2 m/min (250 to 500 ft/min.) In a ventilation air duct that has dimensions of feet, this velocity results in turbulent flow with a Reynolds number of 20,000 to 50,000. Depending on the upstream duct design, fittings, and obstructions, this flow may be fairly evenly distributed in the duct, or significant flow variation may be present. For UVGI, a fairly uniform velocity distribution across the duct is desirable because it generally provides uniform particle loading, and irradiating all of the microorganism particles is the goal. As in many other ventilation applications, an asymmetric flow profile could lead to surprises in UV disinfection.

Normal duct flow characterization using Pitot tube traverses or averaging flow measurement devices is sufficient.

While a turbulent flow condition suggests that considerable mixing is taking place, this may not be so. The turbulence found in air ducts at moderate Reynolds numbers is composed of moderate-sized eddies that may travel several meters down a duct without a great deal of mixing. For example, the author has observed, in a low-speed wind tunnel, neutral density bubbles moving about within, but staying in, the 15.2-cm (6-in.) square eddy created by an egg-crate flow straightener over a distance of approximately 8 m (25 ft.) The eddies are not stable, and form, die, and re-form constantly, which causes some mixing. However, the actual flow path of an arbitrary “particle” of fluid is a random path around a relatively straight average line of flight, and mixing is limited over the effective range of UVGI lamps. Flow smoothing devices such as turning vanes tend to further reduce mixing.

While the flow is indeed turbulent, to assume that all particles of the fluid are mixed in a short distance is a mistake. A fluid particle in the corner as the flow enters the UV-irradiated portion of the duct is likely to stay there as it moves through. For critical applications, the  $E_{eff} \Delta t$  product (or dose) must be adequate to kill (or achieve the target level of reduction) in the furthest corner of the duct. This is consistent with the advice offered by Phillips (1992): “The critical factor is to reach the (dose) threshold at the wall. There the irradiation level is at minimum ....”

#### 2.5.2.2 Particle dynamics in airflow

Environmental microbiological particles, while having various shapes and surface characteristics, follow the same flow dynamics that other airborne particles follow. They are generally in the range of 1 to 4  $\mu\text{m}$  in aerodynamic diameter. Aerosol particles of this size in a flow field at duct velocities [127 cm/s (250 ft/min)] generally follow the main fluid flow path, deviating as the vector sum of their momentum and drag force require. The settling velocity of such particles is on the order of 0.003 to 0.05 cm/s, and the particle velocity is dominated by the duct airflow. Most will follow the flow closely. (Other forces on particles are less important in the open duct.) Particle momentum leads to particles’ modest deviation from the turbulent eddy movement. Particles near the boundaries of an eddy, however, may deviate sufficiently to enter the next eddy. This results in particle mixing as well as air mixing, as does the death and formation of eddies. As mentioned in the previous section, however, the mixing is not as great as might be assumed.

Particles are not always evenly distributed across a duct. Micrometer-sized particles are known to stratify in the elbows of ducts, and the bottoms of ducts collect most of the dust deposit. Common particle sampling guidelines suggest that 10 duct diameters are required to recover a mixed velocity and particle distribution condition downstream of a flow obstacle. Kowalski and Bahnfleth (2000) assert that “sufficient mixing will occur at these velocities to temper the effects of a non-uniform velocity profile” and make other statements endorsing the assumption of complete mixing within the UV irradiated zone. Their exact meaning is not clear, but the authors are probably justifying the use of an average velocity rather than implying that an air duct is a “completely mixed reactor.”

From a UV system design viewpoint, unless the particles are so concentrated that they appreciably attenuate the UV radiation, particle position in the duct does not matter so long as the entire duct is irradiated to the threshold dose. (Position would be an issue under dusty conditions; under such conditions, the UV lamps would become soiled and the installation would be limited by soiling, not by its design.) Thus no assumption of particle position is required—provided the duct is fully irradiated with a dose adequate to achieve the desired performance.

On the other hand, particle position would be very important if a UV light or UV device was installed such that it was not symmetric in the duct. Under such conditions, particle stratification could lead to significant differences in performance because some microorganisms would receive a larger dose than others by virtue of their transit path.

### 2.5.3 In-Duct UV Dosage Model

The model was implemented in a spreadsheet. It was based on the following:

1. Estimation of lamp surface output from the irradiance measured at a few points near the duct centerline, and use of that output in the view factor equation.
2. Use of the view factor equation to compute the UV irradiance at 100 points along each of 9 straight-line particle trajectories. These 9 trajectories were the centerline of the squares in 1 quadrant, much as was shown in Figure 6. The product of the time step duration and the irradiance at each point, added up over the full length of the trajectory, gave the calculated dose for that trajectory. This amounts to an assumption of complete mixing within the cell for each time step, but not from time step to time step. A 0.01-s time step was used, which gave about 80 irradiance calculations along each trajectory in a 1-m travel distance for each of the 9 cells. By symmetry, the irradiance in each quadrant is identical, so the 9 cells represented the entire duct. For the dimensions of the test duct, each trajectory was the centerline of a 10- by 10- by 100-cm (4- by 4- by 39.4-in.) cell.
3. The assumption is made that the microbial particles are uniformly distributed among these 36 trajectories.
4. Calculation of the accumulated dose for microbial particles passing through each of the 100 UV calculation points as they travel along the 36 trajectories.
5. Calculation of expected kill from literature values of microbial resistance to UV.

The results from the model were then compared to the measured microbial kill within the test rig.

The model is currently as sophisticated as is appropriate for a spreadsheet model. It seems to be working well, in that the estimated lamp efficiency values and the estimated microbial kill values agree reasonably well (~20%) with literature values.

## 3.0 KEY FACTORS

### 3.1 Physical Factors Results and Discussion

#### 3.1.1 Effect of Duct Air Conditions on UVGI Lamp Output

##### 3.1.1.1 Duct air temperature and velocity

Phillips Lighting (1992) states that the resonance line at 254 nm in a low-pressure mercury lamp is strongest at a particular vapor pressure that occurs in their proprietary UVGI lamps at a still air ambient temperature of about 68 °F (20 °C). For their lamps, this gives a lamp wall temperature of about 104 °F (40 °C), and the UVGI output is greatest at that temperature. At both higher and lower temperatures, the UVGI output is reduced, with the output at 50 °F (10 °C) being about 88% of that at 68 °F (20 °C). In their product literature, Westinghouse (1985) concurs. They note that the output of their UVGI lamps, like all other gaseous discharge lamps, diminishes as the temperature increases or decreases from the design temperature, which for the Westinghouse lamps is stated to be 80 °F (27 °C) in still air. They note that the output of one lamp at 40 °F (5 °C) is only two-thirds of its output at 80 °F (27 °C.)

Westinghouse (1985) also notes that low temperatures can reduce the operating life of the lamps and that another of their proprietary lamps responds differently. Another lamp, when operated at its highest current input, is said to be much less sensitive to ambient temperature changes. Phillips Lighting (1992) makes a similar argument, saying that their medium-pressure mercury lamp, relative to the low-pressure lamp, has a higher power density and higher wall temperature and is less sensitive to ambient temperature fluctuations. High lamp output at the lower temperatures encountered in ducts is a central argument for the “high output UVGI emitter” presented by Scheir and Fencel (1998). Westinghouse (1985) further notes that low operating temperatures reduce the operating life of their lamps.

The effect of airflow on UVGI lamp output is increased heat transfer that is due to the moving air. If the air moving past the lamp is ambient or cool, the lamp may be cooled below its optimum operating point, which reduces output. If the air is warm, the lamp may be heated above its optimum, which also reduces output. Lamps in the return air of building ventilation systems are likely to be slightly cooled below their optimum temperature, while lamps downstream of a cooling coil could be considerably cooled due to a combination of airflow and low temperature. Lamps designed for operation at low temperatures should also be resistant to airflow effects.

The results obtained during this research for irradiance as a function of air temperature and velocity are shown in Figures 8 and 9 for “low” and “high” output lamps, respectively. Both figures show duct center irradiance at 50 cm (19.7 in.) as a function of duct velocity for each of three duct air temperatures. The overall shape of the curves appears to be the same in all cases.

At a fixed temperature, the irradiance produced by both kinds of lamps was found to increase from a minimum at no flow (and consequent high lamp temperature) to a maximum at a

particular flow rate (the optimum lamp operating temperature), and then to decrease as the flow rate increased further and the lamp was cooled more than was optimum. Airflow has its strongest impact at the lowest temperatures, where the available temperature difference (between the air and the lamp wall) to drive heat transfer is greatest. The apparent reduction in the effect of flow rate at high flow rates [decreasing negative slope of the lines above 2 m/s (400 ft/min)] presumably occurs because the temperature difference between the lamp wall and the air is being reduced and the heat transfer is consequently being reduced.

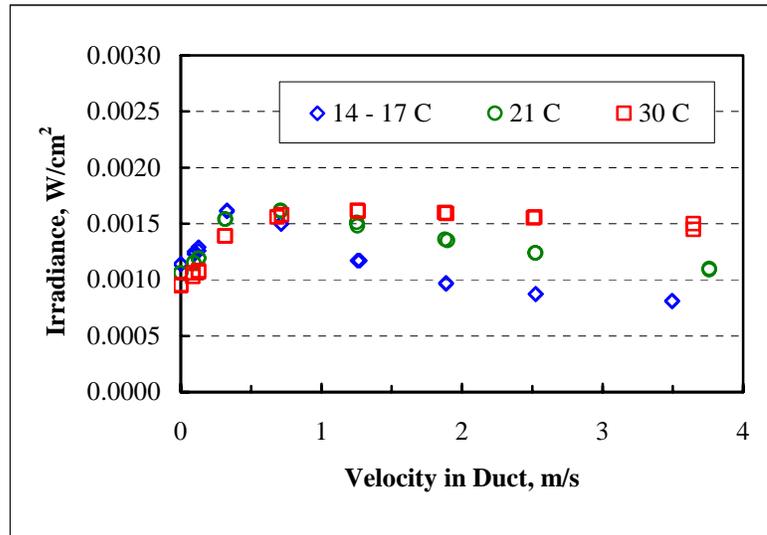


Figure 8. Effect of air velocity and temperature on “low” power lamp irradiance at 1 m

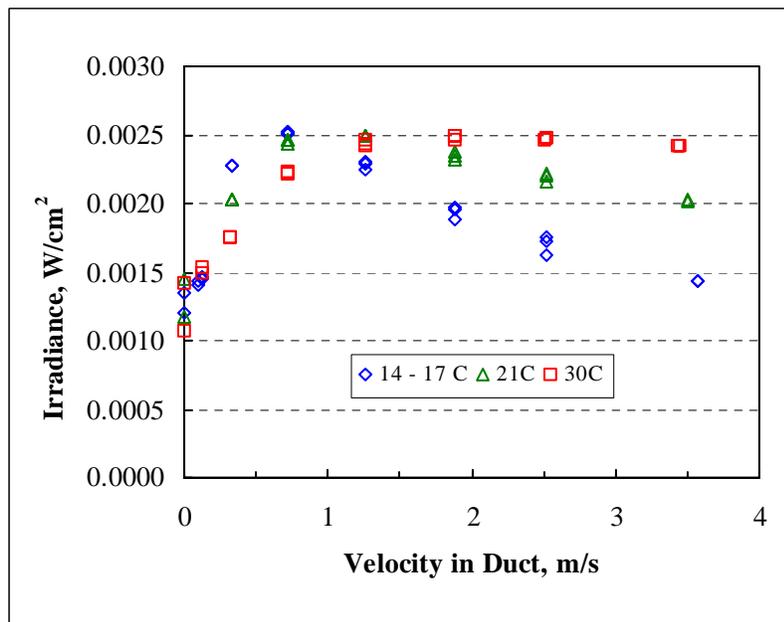


Figure 9. Effect of air velocity and temperature on 1-m irradiance for “high” output lamps.

This behavior is consistent with literature reports and discussions with manufacturers, and the shapes of the curves are similar to those presented by Phillips (1992). UVGI lamp output

reaches a maximum output at an operating temperature that is determined by lamp mercury content, fill gases, electrical energy throughput, and other electrical considerations. For any given lamp/ballast combination, the lamp operating temperature is most affected by flow rate and temperature.

### 3.1.1.2 Relative humidity

The literature regarding the effects of humidity on UVGI does not present a completely consistent picture. Water molecules are known to adsorb UV light, so increased humidity should reduce the transmittance of UVGI. The effect should be proportional to absolute humidity (mass of water per volume of dry air), rather than to relative humidity. Within the normal ranges of temperature and humidity in ventilation systems, the effect would be expected to be modest because water makes up only a small fraction of the total gas, and that fraction changes little. As noted by Kowalski and Bahnfleth (2000), “studies on this matter are contradictory and incomplete.” Scheir and Fencl (1998) state that “humidity is an attenuator to UVGI energy.” Phillips Lighting (1985) provides a graphical correction factor to increase the number of lamps that are required when the relative humidity exceeds 60%. On the other hand, Phillips Lighting (1992) does not include this correction factor. The effect of humidity may be different for surface disinfection than for bioaerosol treatment. Relative humidity above approximately 60% has been shown to affect the surface properties of powders (Zimon, 1970); this effect is perhaps due to surface condensation in the interstitial spaces, which might lead to sufficient water to attenuate the UVGI. An effect on direct transmittance seems unlikely.

To investigate the effect of air humidity on irradiance, the radiometer UV detector was positioned at a convenient location 1.36 m (4.46 ft) from the lamp centerline, at the center of the duct. The longer path length from lamp to detector (relative to that used in the study of the effects of flow rate) was deliberate. The same detector and filter arrangement was used. The test rig was operated at 0.472 m/s (1,000 cfm) and 24°C (74°F), and the relative humidity varied from 35 to 85% RH in three levels in a random order.

The results of this investigation are shown in Figure 10. The effect of humidity is modest but detectable. Over this short path length, UVGI irradiance decreases linearly with absolute (not relative) humidity, as would be expected for the direct interaction of water molecules and the UV photons. References in the UVGI literature stating that the effect of humidity are unclear may be the result of liquid droplet carryover in some field installations, which would result in much greater attenuation of the UVGI. However, for droplet-free air, the effect of humidity appears straightforward, predictable, and modest.



their maximum output point if the air is at 30°C (85 °F), but would be below their peak output at 15°C (58 °F.)

The high-output lamps whose irradiance is presented in Figure 9 were characterized by the manufacturer as lamps designed to operate at lower temperatures than conventional lamps; they appear to have an operating characteristic consistent with that design. At 15°C (58 °F), the maximum output of about  $2.50 \times 10^{-3} \text{ W/m}^2$  was reached at about 0.76 m/s (150 ft/min), while the maximum at 21°C (70 °F) was reached at 1.27 m/s (250 ft/min) and the maximum irradiance at 30°C (85 °F) was reached at about 2.03 m/s (400 ft/min.)

One of the factors about this lamp design that enables it to operate at lower temperatures may be the higher power load being put into a lamp, relative to the physical size. The “high” power lamps are thought to be low-pressure lamps operated at higher power loads than the “low” output lamps. Phillips (1992) notes that medium-pressure lamps, operated at appropriate electrical throughput, have a higher wall temperature and are more resistant to cool temperatures than are low-pressure lamps.

### 3.1.3 Effects of Lamp Soiling

As with all lamps, germicidal UV lamps that become soiled suffer a reduction in the delivered irradiance. Phillips (1992) comments that the lamps “must be cleaned at a frequency depending on the degree of grime” in the air to which they are exposed, and the literature concurs. The installation of lamps downstream of filters is generally recommended. Phillips Lighting (1985) states that cleaning polishes may adsorb UVGI and recommends that they not be used to clean and polish reflectors. Westinghouse (1985) recommends using alcohol or ammonia and water and a clean, lint-free cloth for cleaning; leaving a residue should be avoided.

### 3.1.4 Reflectance

The importance of duct surface reflectance is that the effective irradiance delivered to the microorganisms is increased through reflected UV energy. Reflectance specific to UVGI is not the same as that to visible light. Phillips (1985) and others provide reflectance values for a number of clean materials. Phillips Lighting (1992), in their design procedure for an axial lamp mount in a circular duct, takes explicit credit for a reflective duct. They reduce the length of duct necessary to treat air by  $(1-R)$ , where  $R$  is the reflectance of the duct. Phillips (1985) also allows credit for reflectance in their design procedure, though using a different and apparently more conservative design correction. Westinghouse (1982) notes that reflective materials should be used, but does not appear to take explicit credit for reflectance in their design procedure. Given the high likelihood of eventual soiling in a ventilation system, caution is advised with regard to taking credit for reflectance. Table 1 repeats some duct reflectance values from the literature.

**Table 1. Reflectance of Various Materials**

<b>Material</b>	<b>Reflectance</b>
Aluminum, untreated surface	40–60%
Aluminum, sputtered on glass	75–85%
Stainless steel plate	25–30%
Chromium plating	39%
Aluminum paint	40–75%
Various white oil paints	3–10%
White baked enamel	5–10%
Magnesium oxide	75–88%
White plastering	40–60%
White note paper	25%
Typical duct liner	1%

Note: Reflectance values for a typical duct liner are taken from Schier and Fencil, 1996; values for all other listed materials are taken from Phillips, 1985.

The values in Table 1 show that reflectance for UVGI is not the same as for visible light. Aluminum is frequently mentioned as providing a desirable reflector surface. A reflectance value for a galvanized steel duct was not found in the literature, but experience with UVGI lamps in galvanized ducts indicates that galvanized steel has some reflectance for UVGI.

The intent of the reflectance measurements made during this research was to evaluate the impact of reflectance on centerline irradiance measurements and the relative contribution of direct UVGI and reflected UVGI on those measurements. The materials used were new galvanized sheet metal and black photographic flock paper, and the basic measurement was irradiance as a function of position in a mocked-up duct. Duct reflectance was investigated in the duct mock-up apparatus by measuring the irradiance from low-power lamps with the duct lined with two materials—new galvanized steel and photographic background black flock paper. Three low-power lamps were mounted horizontally across the duct: one at the center, and one each 20.3 cm (8 in.) above and 20.3 cm (8 in.) below the center. Each lamp was driven by a magnetic ballast and was allowed to reach an equilibrium output before measurements were started. All measurements were made at the same static airflow conditions, and therefore the results can be compared within this data set. Figure 6, above, shows the layout of the measurement points. The same set of measurements was made for both duct lining materials.

Figure 11 shows the results of this measurement for the galvanized steel-lined duct, and Figure 12 shows the results for the black flock paper-lined duct. In both cases, the measurement points were laid out as shown in Figure 6, with points 1, 2, and 3 being the row nearest the center, with point 1 to the outside of the duct and point 3 nearest the center. The “sawtooth” shape of the curves, most visible nearest the lamps, results from the point near the outside of the duct receiving less irradiance than the points near the center. In both figures, the “sawtooth” shape is more prominent near the lamps, because measurements near the lamps are dominated by irradiance directly from the lamps, while farther away, more reflected light reaches the detector.

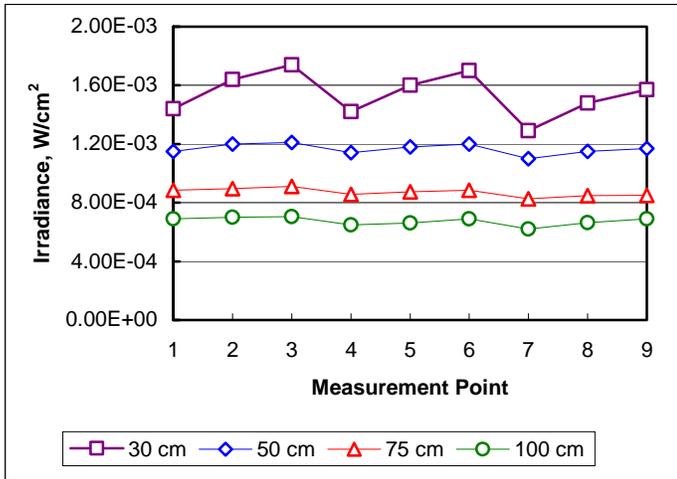


Figure 11. Irradiance distribution in galvanized duct.

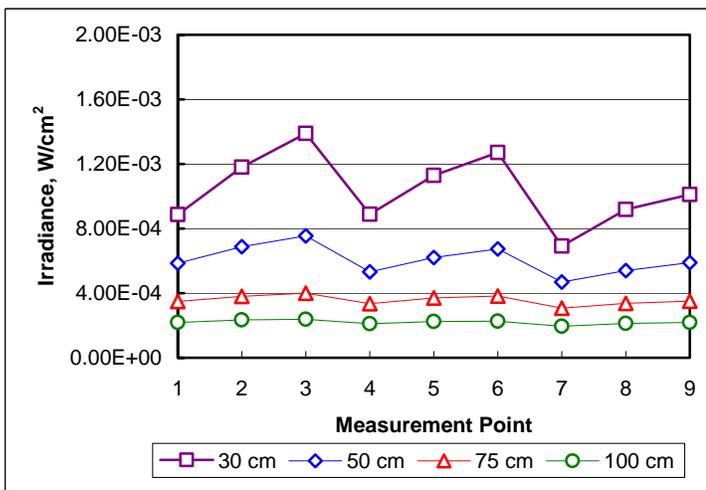
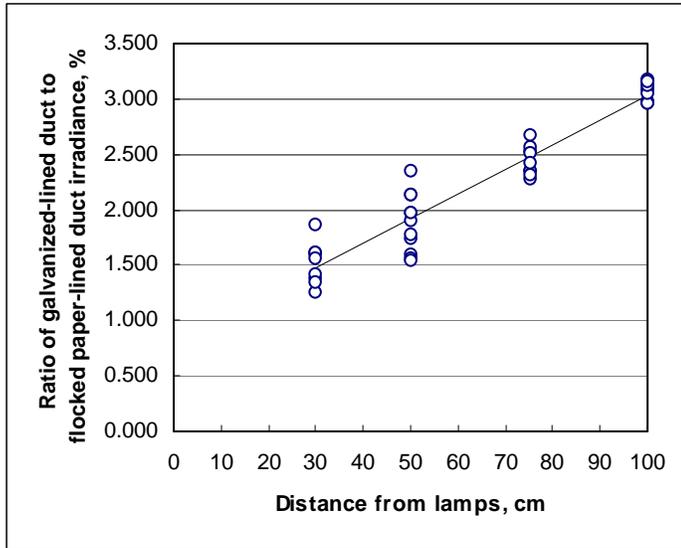


Figure 12. Irradiance distribution for black flock paper-lined duct.

The “sawtooth” is more prominent for the black flock paper-lined duct because essentially all irradiance comes directly from the lamps.

Figure 13 shows the ratio of total irradiance in the galvanized duct to total irradiance in the black flock paper-lined duct for all measurement points. It shows that reflected light contributes an increased fraction of the total irradiance as distance from the lamp increases. Simultaneously, because of the increase in distance, the total irradiance decreases. The straight line drawn in Figure 13 is a fit to all the data. The fractional increase due to reflectance is substantial, with the total irradiance increased by a factor of roughly 1.5 at 30 cm (11.8 in.) and by a factor of 3 at 100 cm (39.4 in.) Over the distance studied, the overall effect is near linear with distance from the lamps.



**Figure 13. Ratio of galvanized to black flock irradiance.**

These results apply to lamps mounted across the duct in a crossflow configuration with a clean galvanized duct. Reflectance effects should be different with the lamps mounted parallel to the flow.

### 3.1.5 Enclosure and Location Factors

Objects that are physically in the path of the UV radiation provide shadows and protected regions in which microorganisms will not be effectively irradiated. Blockage near the UV lamps is especially important because the high-irradiance regions require the shortest exposure duration.

Other location factors primarily relate to the temperature and airflow dependencies discussed above.

#### 3.1.5.1 Irradiance effects

Irradiance at a fixed distance from the UVGI lamps was routinely measured for most operating conditions. Access was not available to make a measurement with the lamps oriented parallel to the flow, but irradiance was measured for the crossflow configurations. The position of the detector relative to the lamps depended on accessibility. Direct comparison is available for the crossflow case with and without the coil with the detector at 50 cm (1.64 ft) upstream from the lamps. Measurements were made for three “high” output lamps with the coil not installed in the duct (the standard configuration), with the lamps between the coil and the detector (coil downstream of the detector), and with the coil moved upstream to be between the lamps and the detector. The air temperature was 22°C (71°F) and the relative humidity 55–59%. Results are given in Table 2.

**Table 2. Irradiance Measurements with Coil in Duct**

<b>Condition</b>	<b>Measured irradiance, W/cm<sup>2</sup></b>
No coil installed – open duct	0.00252
Coil downstream	0.00260
Coil between lamp and detector (1)	0.00000198
Coil between lamp and detector (2)	0.00000642
Coil between lamp and detector (3)	0.00000161

The first two data rows of Table 2 show that the aluminum coil did not reflect a significant amount of UVGI energy back to the detector; the irradiance measurement with and without the coil is essentially the same. Relative to the top two rows, the three bottom rows show that less than 1% of the UVGI that would be expected to reach the detector penetrates the coil. The cases with the coil blocking the UV are minor variations of the same measurement. For (1), the detector was located exactly at the duct centerline. For (2), the detector was slightly [1.9 cm (0.75 in.) to change the ‘view’ before making the measurement. For (3), UVGI leakage around the coil was essentially stopped by sealing the visible light leaks around the coil. Comparison of (1) and (2) shows that the irradiance measurement is sensitive to detector position with the coil installed, though most UVGI is stopped in both cases. Light reflecting around the outside edges of the coil is shown to be a substantial fraction of the total UVGI measured by (3).

### 3.1.5.2 Results for microbial kill in various configurations

Table 3 compares disinfection efficiency for the four lamp/duct configurations with the same number of lamps in each. Using two different microbial challenge aerosols (*Bacillus subtilis* and *Aspergillus versicolor*), penetration was evaluated with and without the lamp bank turned on. *Staphylococcus epidermidis* was not used as a challenge because three lamps were expected to produce too high a dose to provide useful data.

The data were analyzed by comparing results for each alternative arrangement to the crossflow lamp arrangement results; this analysis was required because the alternative configurations were not compatible with the previously developed model that had been used to estimate dose. Comparison using the model to compute *k* values was not possible because the model was developed for the case of a straight, open duct and was of uncertain usefulness for the other cases.

**Table 3. Effect of Duct Configuration on Microbial Kill**

<b>Lamp Configuration</b>	<b>Number of Lamps</b>	<b><i>Bacillus subtilis</i> % Kill</b>	<b><i>Aspergillus versicolor</i> % Kill</b>
Straight duct, crossflow lamps	6	99	56
	3	76	20
Straight duct, parallel lamps	6	67	40
	3	30	17
Straight duct, coil	6	92	43
	3	55	23
Elbow duct	6	91	62
	3	55	30

## 3.2 Chemical Byproducts Results and Discussion

### 3.2.1 Ozone

The results of the ozone production measurements for six high-output lamps mounted in crossflow at 0.76 m/s (150 ft/min) were nil. The ozone concentration downstream of the lamps was not different from that upstream. In both cases, the readings were about 28 ppb. Therefore, under flow conditions, ozone production was not detectable.

### 3.2.2 Effect of UV lights on VOCs

The experiment used was described in Section 2.3.2. As with the ozone production experiment, the test was conducted at a normal operating flow rate, not in still air. The results show that no significant change in the concentration of the injected chemicals was detected, and no chemical byproducts were clearly detected. Figure 14 includes the results from two upstream and two downstream tests with the UV lights on; it visibly shows that the upstream and downstream tests were not different. Table 4 shows the chemicals identified in byproduct sampling.

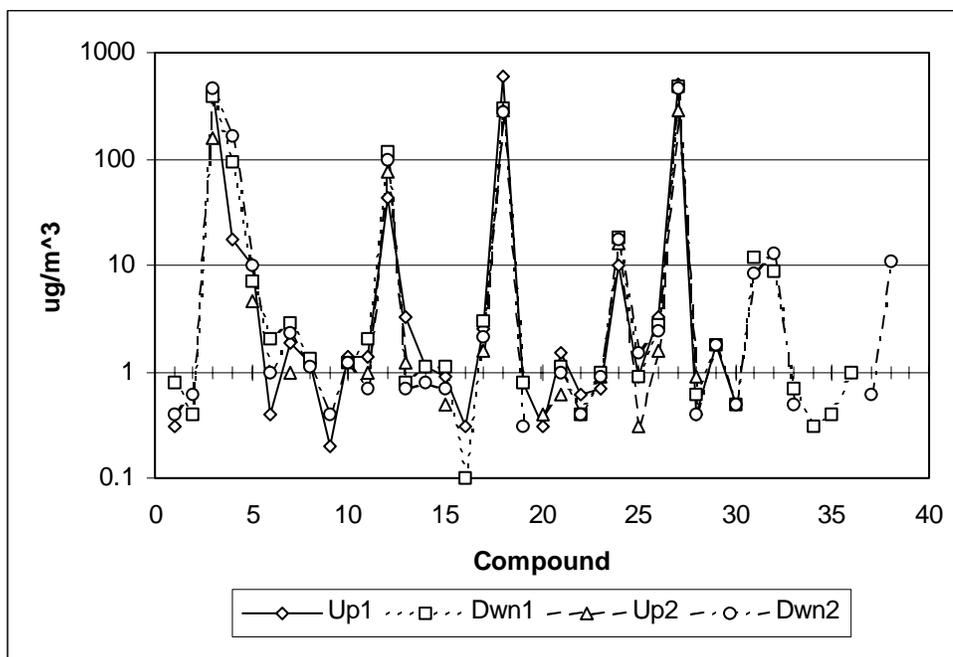


Figure 14. Results of chemical sampling up- and downstream of lamps

**Table 4. Chemicals Identified in Byproduct Sampling**

<b>ID</b>	<b>Chemical Name</b>	<b>CAS No</b>	<b>ID</b>	<b>Chemical Name</b>	<b>CAS No</b>
1	1-Hexanol, 2-ethyl	104-76-7	21	Nonyl aldehyde (Nonanal)	124-19-6
2	1-Pentene, 4-methyl	691-37-2	22	Octanal*	124-13-0
3	1-Propanol, 2-methyl (Isobuty alcohol)	78-83-1	23	Pentane, 2-methyl	107-83-5
4	2-Butanone (Methyl ethyl ketone, MEK)	78-93-3	24	Pentane, 3-methyl	96-14-0
5	2-Propenal, 2-methyl*	78-85-3	25	(2,6,6-Trimethyl-bicyclo[3.1.1] hept-2-ene	80-56-8
6	Acetic acid	64-19-7	26	Propanal, 2-methyl (Isobutanal)*	78-84-2
7	Benzaldehyde	100-52-7	27	Toluene (Methylbenzene)	108-88-3
8	Benzene	71-43-2	28	Tridecane	629-50-5
9	Benzene, 1,2,4-trimethyl	95-63-6	29	Xylene (para and/or meta)	106-42-3
10	Benzene, ethyl	100-41-4	30	Xylene, ortho	95-47-6
11	Benzoic Acid*	65-85-0	31	2-Propanol (Isopropanol)	67-63-0
12	Cyclopentane, methyl	96-37-7	32	3-Buten-2-one*	78-94-4
13	Decanal	112-31-2	33	1-ethyl-4-methyl (4-Ethyltoluene)	622-96-8
14	Decane	124-18-5	34	Heptanal (Heptaldehyde)	111-71-7
15	Dodecane	112-40-3	35	Heptane, 2,4-dimethyl	2213-23-2
16	Ethanol	64-17-5	36	Pentane, 2,2,3,4-tetramethyl*	1186-53-4
17	Ethanone, 1-phenyl (9CI)	98-86-2	37	3-Buten-2-one, 3-methyl*	814-78-8
18	Hexane*	110-54-3	38	Acetone	67-64-1
19	1-Methyl-4-(1methylethyl)cyclohexene)	138-86-3	39	Butane, 2-methyl (Isopentane)	78-78-4
20	Methylene chloride (Dichloromethane)	75-09-2			

### 3.3 Microbiological Factors Results and Discussion

Dosage is defined as the UVGI irradiance received by the microorganism times the duration of exposure. This is the  $E_{eff} \bullet \Delta t$  product of Eq. 1.  $E_{eff}$  is a function of duct position. The most common unit for dosage is  $\mu\text{W}\bullet\text{s}/\text{cm}^2$ , but  $\text{W}\bullet\text{s}/\text{cm}^2$  and  $\text{J}/\text{cm}^2$  are also used.

Given the theoretical ability to compute particle trajectories, the irradiance received by each microorganism particle of each size at any given time appears simple, at least in concept, to predict the disinfection performance of a UVGI installation. To compute the dose,  $E_{eff} \bullet \Delta t$  can be integrated over the particle path, and the fraction of microorganisms killed can then be estimated. But numerous microbiological considerations must be taken into account.

Although there are multiple tables available that list a series of organisms and the respective dosages or energies required to kill them, a careful review of the literature shows that these tables may unintentionally oversimplify the required dosages. Many of the data in these tables come from the Westinghouse data, which are based on a series of experiments done primarily in the 1940's. Many of these are truly elegant experiments; however, their goal was a fundamental understanding of UV and not an evaluation of UVGI lamps. More recent summaries of UVGI kill (Kowalski and Bahnfleth, 2000) have perpetuated the problem. There were few common conditions under which the experiments were performed (i.e., some organisms were irradiated while airborne, others when seeded on agar plates or suspended in buffer). These differences may explain a great many of the seemingly inconsistent kill dosages reported for the same organism.

#### 3.3.1 Test Organism Selection

Preliminary microbiological tests were conducted with seven organisms: *Serratia marcescens*, *Staphylococcus epidermidis*, *Pseudomonas fluorescens*, *Bacillus subtilis*, *Aspergillus versicolor*, *Penicillium chrysogenum*, and *Cladosporium sphaerospermum*. The first four are bacteria, and the last three fungi. These organisms cover a broad range of susceptibility to UV. *S. marcescens*, *S. epidermidis*, and *P. fluorescens* are vegetative bacteria that are readily killed. *B. subtilis* is a spore-forming bacteria, and the three fungi (*A. versicolor*, *P. chrysogenum*, and *C. sphaerospermum*) also form spores. We expected these organisms to be difficult to kill. Most of the tests were conducted at approximately 24 C (75°F), 50% RH, with the test rig at either 0.472 or 0.694  $\text{m}^3/\text{s}$  (1,000 or 1,470 cfm) for an approach velocity of 1.27 or 1.87 m/s (250 or 368 ft/min.) All of the tests utilized one or more "standard" UV lamps [nominal 1.6-cm (5/8-in.) diameter, single-ended, 61-cm (24-in.) length] powered by a large and heavy preheat, self-start ballast and mounted perpendicular to the airflow.

Depending on the experiment, the lamps were configured in one of four different ways. The goal was to irradiate all the organisms and remain in an experimental range with reliably measurable kill. For the three fungi and the *B. subtilis*, we used either six lamps in a single column on 10.2-cm (4-in.) spacing or three lamps in a single column on 20.3-cm (8-in.) spacing for high and low irradiance levels, respectively. For the three vegetative bacteria, we used either one lamp at the duct center or one lamp covered by three layers of common building screen. In each case, the irradiance was measured at a known distance from the lamp (on the lamp centerline), and that irradiance value was used to calibrate the model. The model was then used to compute the mean

dose for all the trajectories, and the  $k$  factor was computed from this mean dose and measured microbial kill. A given lamp configuration produces a higher dose at 0.472 m<sup>3</sup>/s (1,000 cfm) than at 0.694 m<sup>3</sup>/s (1,470 cfm), as is reflected in Table 5. This large duct experiment is not a preferred way to determine  $k$  factors because the distribution of dose is fairly broad across the different trajectories. Computing  $k$  values allowed us to compare our full-scale work to the published literature; the agreement is fairly good in most cases.

As can be seen in Table 5, with this range of irradiance, all organisms can be brought into the measurable range. Each result is for an individual test or run and they are generally in good agreement. Use of the screened lamp was not fully satisfactory, but it dramatically demonstrated the susceptibility of some organisms and the need for the microbiologically informed testing that was conducted in this project.

**Table 5. Results of Preliminary Microbial Kill Experiments**

<b>Organism</b>	<b>Number of Lamps</b>	<b>Dose <math>\mu</math>W-sec/cm<sup>2</sup></b>	<b>% Kill</b>	<b><math>k</math> Factor</b>
<i>Serratia marcesens</i>	1 lamp	1341	99	3.31E-03
	1 lamp, 3 screens	369	97	9.16E-03
	1 lamp, 3 screens	369	98	9.99E-03
<i>Staphylococcus epidermidis</i>	1 lamp	984	81	1.68E-03
	1 lamp	984	83	1.77E-03
	1 lamp, 3 screens	329	22	7.71E-04
	1 lamp, 3 screens	329	9	2.99E-04
<i>Pseudomonas fluorescens</i>	1 lamp	1,341	98	2.90E-03
	1 lamp, 3 screens	369	99.7	1.58E-02
<i>Bacillus subtilis</i>	6 lamps	7,509	80	2.17E-04
	6 lamps	7,509	90	3.06E-04
	3 lamp	3,197	48	2.07E-04
	3 lamp	3,197	53	2.34E-04
<i>Aspergillus versicolor</i>	6 lamps	7,509	83	2.35E-04
	6 lamps	7,509	66	1.45E-04
	3 lamps	3,197	5	1.51E-05
	3 lamps	3,197	16	5.49E-05
<i>Penicillium chrysogenum</i>	6 lamps	7,509	10	1.40E-05
	6 lamps	7,509	17	2.46E-05
	3 lamps	3,197	0.00	—
	3 lamps	3,197	1	4.57E-06
<i>Cladosporium sphaerospermum</i>	6 lamps	7,509	16	2.39E-05
	3 lamps	3,197	3	8.24E-06

Note: “3 screens” indicates that the single lamp was wrapped with three layers of household screen to reduce irradiance.

The experimental conditions achieved were adequate to conduct useful tests with all of the microbial organisms. That is, the results fell within a useful experimental range and provided the

needed information. The vegetative bacteria (*S. marcescens*, *S. epidermidis*, and *P. fluorescens*) were readily killed. The spore-forming bacteria (*B. subtilis*) was, as expected, much more resistant. The three fungi whose spores were irradiated (*A. versicolor*, *P. chrysogenum* and *C. sphaerospermum*) were also relatively resistant, although significant kill was achieved by arranging sufficient UVGI irradiance. The results of the experiments are in general agreement with previously published microbial kill data and design information. Therefore, we selected the vegetative bacteria *S. epidermidis*, the spore-forming bacteria *B. subtilis*, and the fungus *A. versicolor* for the final testing of the lights.

### 3.3.2 Growth of Organisms in the Dark

All of the plates collected during the testing were incubated in the dark. UV light kills cells by damaging their deoxyribonucleic acid (DNA). The exact mechanism differs between vegetative cells and spores. Exposure to UV causes the formation of thymine dimers by phototransformation in vegetative cells and by spore photoproduct in spores. This results in a distortion of the DNA strand; replication is then halted. Because cells are commonly exposed to UV, they have developed a number of repair mechanisms to counteract the UV-induced damage.

Although the phenomenon of UV repair was reported in the late 1940's and 1950's (Kelner, 1949; Dulbecco, 1955), little was understood about the cellular repair mechanisms for UV damage until the 1960's. Fungal spores, bacteria, and plant, animal, and viral DNA may all be repaired through photoreactivation with near-UV or visible light. Photoreactivation is only one of the repair mechanisms available to cells to repair damaged DNA. In addition to light repair, excision repair, also known as dark repair, and the so called SOS repair mechanism are important. Spores have an additional mechanism of spore photoproduct repair (Setlow, 1992). The enzyme spore photoproduct lyase is activated during germination to repair damage that occurred to the dormant spore (Sun et al., 1994). Excise repair is exactly what it sounds like: the cell cuts out the segment of DNA with the thymine dimer and synthesizes the new strand to replace it. The SOS mechanism is particularly interesting because it results in the misincorporation of DNA bases, which leads to an increased mutation rate for the cell. This mechanism is responsible for the high mutation rate seen in cells irradiated with UV. Scientists have used UV to induce mutations in cells for many years. Mutated cells are by no means dead; while some mutations are lethal, most are not. It is thought that one explanation for the evolutionary development of the SOS mechanism is that at a cellular level, mutation is preferable to death. In human cells, this may assist in the development of skin cancers. However, in microorganisms, some of these mutations may be beneficial or neutral to the cell. The dosages required to kill an organism are affected by these repair mechanisms, and the dominant mechanism for a cell can profoundly affect an organism's response to UVGI. An additional repair mechanism exists for viruses. The infected cell may repair the damaged viral DNA for the virus if the infected cell contains the repair enzymes.

### 3.3.3 Recovery of Damaged Test Organisms

The selection of the appropriate recovery media was another factor that needed to be addressed. Few of the early experiments that are frequently cited for determining the kill dosages of a specific organism address the fact that damaged organisms will not grow well on standard media

after irradiation or any other antimicrobial treatment. The standard bacteriological and mycological media are generally considered too nutrient rich to effectively recover the damaged organism. Recovery of environmentally stressed organisms using minimal nutrient media can be more accurate.

Damaged organisms are notoriously difficult to isolate on standard nutrient media. Therefore, the selection of the recovery media was thought to be an important factor in this study. We used media typically used to recover damaged organisms and compared the results with the more standard media. No differences were found for recovery, so trypticase soy agar was used for the bacteria and Sabouroud's dextrose agar was used for the fungi.

### 3.3.4 The Influence of Aerosol Composition on Bioaerosol Tests

Most of the UV inactivation studies done with airborne microorganisms (bioaerosols) have used pure cultures of one particular organism that were suspended in a liquid diluent and then nebulized. The overall composition of the aerosol, including the bioaerosol, is important. Bacteria suspended in phosphate buffer were more susceptible to UV inactivation than those suspended in broth (Sharp, 1940). Presumably, the more complex organic constituents of broth provide a physical protection or shield from the UV rays. Also, bacteria in broth are less stressed and may be naturally more resistant. On the other hand, Beebe (1958) found that in some cases, dry aerosols of the organism were more resistant to UV than wet (nebulized liquid) aerosols.

Other aerosol characteristics are also important. Bacteria in droplet nuclei are more readily killed than aggregates. Dust or other particulates associated with the bioaerosol decrease the efficacy of the UV because of the UV's poor penetration.

Because our goal was to develop a method to test and compare the lights in a test duct, some of these issues needed to be addressed. To address the influence of the confounding/potentially protective aerosol characteristics, the test organisms were generated in both water and broth as described in section 2.4.3. The water media was used as the baseline, and the broth for all of the potentially protective circumstances.

### 3.3.5 The Influence of Relative Humidity on Test Organisms

There is little or no agreement in the literature regarding an organism's sensitivity to UV and the moisture content of the cell. Attempts to correlate the susceptibility of an organism to UV at different RHs have yielded inconsistent results. Some organisms have shown decreased kill at RHs greater than 60–70%, while others have not. Studies with *S. marcescens* showed decreased kill at RHs both greater than 70% and below 40%.

The duct's RH did not appear to affect particle viability (the upstream and downstream counts with the lamps off were about the same) or particle size (by optical particle counter) between the upstream and downstream measuring points. Because physical equilibrium with water vapor is achieved quickly for micrometer-sized particles, this is not surprising.

To help elucidate the RH effect on organisms within the confines of this project, experiments were performed at 55% RH and 85% RH with both water and broth suspensions.

## **4.0 UVGI EFFICACY IN AIR DUCTS**

### **4.1 Measured Efficacy and Discussion**

During this research, UVGI lamps were found to inactivate vegetative bacteria, bacteria spores, and fungal spores to a reproducible degree under conditions of fixed dose. Further, Eq. 1 was found to generally apply, provided the dose and the  $k$  value were known. This section summarizes the bioaerosol measurements that were the key findings of the research.

Table 6 summarizes the final results for our three primary test organisms. The test organisms were suspended in either water or broth to examine the impact of the potentially protective effect of organic matter (dirt, debris, etc.) on the organisms in these tests. The experiments were performed at either 55% RH or 85% RH to assess the impact of the potentially protective effect of humidity on the organisms within the confines of this test method. While the focus of this project was not to elucidate the nature of these organisms, understanding the various effects was critical in developing the test and interpreting the results.

**Table 6. Percent Kill of the Three Test Organisms at Varying Test Conditions**

Organism	Lamp Intensity	Water Suspension				Broth Suspension			
		55% RH		85% RH		55% RH		85% RH	
<i>Staphylococcus epidermidis</i>		# lights		# lights		# lights		# lights	
		1	<1	1	<1	1	<1	1	<1
	Low	99.1	69	74	15	86	19	63	0
	High	> 99	83	82	25	ND	ND	ND	ND
<i>Bacillus subtilis</i>		# lights		# lights		# lights		# lights	
		6	3	6	3	6	3	6	3
	Low	83	32	83	35	84	49	89	46
	High	99	76	98	75	99	83	99	85
<i>Aspergillus versicolor</i>		# lights		# lights		# lights		# lights	
		6	3	6	3	6	3	6	3
	Low	23	7	36	15	47	25	47	14
	High	56	22	74	28	ND	ND	ND	ND

ND = Not Done

#### 4.1.1 Vegetative Organisms

The results suggest that the vegetative organisms, represented by *S. epidermidis*, became more resistant to UV as the protective factors increased. Additionally, the results suggest that the effect may be additive for the vegetative organisms. When comparing a 99.1% kill with one light at 55% RH to a 74% kill with one light at 85% RH, as seen in Table 6, increased humidity appeared to be protective. A similar result was seen for the organisms generated in the broth suspension. At 55% RH, there was an 86% kill with one light, but at 85%, there was a 63% kill. The increased amount of organic matter in the broth also appeared to be protective. And where both elevated humidity and organic matter (85% RH, broth suspension) were present, only 63% of the organisms were killed with one light.

#### 4.1.2 Spore-Forming Bacteria and Fungi

Humidity and suspension composition had much less impact on the spore-formers. It should be noted that six lights were required to inactivate the spore-formers to levels similar to those seen with the vegetative bacteria. As stated before, the spore-formers were chosen because they are much more difficult to kill. For the *B. subtilis* spores, there did not appear to be a difference in the percentage killed, regardless of protective factor. For the fungal spore, *A. versicolor*, there may have been a small effect. The difference may not be significant in practice; we will investigate it further as part of the EPA supplemental project. However, this difference is very interesting because it may point to some fundamental properties of microbial aerosols that cannot be readily investigated, except in the aerosol state. When microbial aerosols are in contact with

agar, as has been the case in most of the reported literature, it would not be possible to investigate the impact of air RH. Additional tests with a dilute protein solution are under way. These tests will help elucidate the importance of humidity on kill for the different classes of organisms.

It is critical to understand the effect of humidity on the various organisms. The literature is contradictory on the subject. The physical factors work has shown that the lights are not affected. This makes it critical to clarify the impact of humidity on the organisms.

#### 4.1.3 Summary of Calculated Microbial Resistance Constants

One of the more confusing aspects of comparing the effectiveness of different UV lights is that manufacturers report different dosages, and therefore, correspondingly different kills. As can be seen in Table 6, the impact of the high-intensity lights was that a higher percentage of organisms was killed due to the higher dose. As a check on the test method we developed, it was necessary to compare our results to those of the same or similar organisms reported in the literature. To separate out organism effects from light effects, we calculated the  $k$  value.

The  $k$  value was calculated as shown in Eq. 1 and is defined as the microorganism-dependent rate constant. Table 7 shows the  $k$  values ( $\text{cm}^2/\text{mW}\cdot\text{s}$ ) calculated from data reported in Table 6 for the three primary organisms in the study at both 55% RH and 85% RH. Only the data from the tests in which the organisms were suspended in water were reported.

**Table 7. Summary of  $k$  Values for Test Organisms**

Test Organism	$k$ values, $\text{cm}^2/\text{mW}\cdot\text{s}$ , at indicated humidity	
	55% RH	85% RH
<i>Staphylococcus epidermidis</i>	$2.4 \pm 2.0$ (n=18)	$0.8 \pm 0.2$ (n=12)
<i>Bacillus subtilis</i>	$0.2 \pm 0.06$ (n=14)	$0.2 \pm 0.06$ (n=14)
<i>Aspergillus versicolor</i>	$0.03 \pm 0.02$ (n=12)	$0.06 \pm 0.03$ (n=12)

n = number of tests

As anticipated, the  $k$  values for the three test organisms demonstrate that the vegetative bacteria was the most readily killed, while the fungal spore was the most difficult to kill. The values we determined are similar to those reported in the literature. The literature-reported values for organisms similar to *S. epidermidis* range from 0.4 to 1.9  $\text{cm}^2/\text{mW}\cdot\text{s}$ . For *B. subtilis*, the values range from 0.1 to 0.9  $\text{cm}^2/\text{mW}\cdot\text{s}$ . The reported  $k$  values for various Aspergilli range from 0.01 to 0.05  $\text{cm}^2/\text{mW}\cdot\text{s}$ .

## 4.2 Design of UVGI Systems for Air Ducts

Most of the guidance available from the published literature and manufacturers of UVGI systems appears to have its origin in the design guidance provided by the UV lamp manufacturers Westinghouse (1982) and Phillips (1985, 1992). The guidance is based on the validity of Eq. 1, detailed knowledge of lamp performance under different conditions, and use of either a variation

on the inverse-square law or the view factor equation to estimate the effective irradiance in the space. With the caveat that the microbial resistance constants  $k$  should not be taken to have great precision, the present research generally confirms these design methods. Conversations with various manufacturers have indicated that some have independently developed models that have the general characteristics of the model we developed to support this research.

Any design method must begin with knowledge of the output of the UVGI lamps at the expected operating conditions. For branded lamps, that information is available (at least in part) from the manufacturer. For an unbranded lamp, few assumptions can be made; the irradiance must be measured at operating conditions. The design models can then be used.

## 5.0 CONCLUSIONS

We can draw several conclusions from our studies.

The key factors for the design of in-duct UVGI systems intended to disinfect moving air streams are (1) UV lamp irradiance at operating conditions, (2) microbial resistance data, (3) functioning bioaerosol dose model and (4) duct wall reflectance.

UVGI lamps have a point of maximum output when exposed to various air temperatures and airflow rates. This optimal operating point is fundamentally related to lamp operating temperature and can be reached by various combinations of environment and heat transfer rate (principally air temperature and flow rate) that bring the lamp to the proper operating condition.

Different UVGI lamps have different characteristics, and the optimum cooling rate may be different for each.

The reduction in output due to overcooling at temperatures achievable in an HVAC system has been shown to be as much as a factor of two, which could have a significant effect on kill for bioaerosols.

The variance in  $k$  values reported in section 4.1.3 for the present work (results reported as mean and standard deviation) and the ranges of reported  $k$  values should be considered to be measures of real variability in microbial resistance until proven otherwise. Humidity and organic matter were shown in sections 4.1.1 and 4.1.2 to have protective effects that would be difficult to distinguish from natural variation in an environmental organism, and significant variation should be expected. Resistance variations between strains of the same organism have the potential to be substantial. Very little work has been done. In consequence of this, UVGI system designers should

- Apply safety factors to their designs, particularly as they depart from operating modes with which they have performance data and field experience.
- Avoid relying solely on design equations to determine the performance of their systems. Actual testing with the contaminants of interest is highly recommended.
- Be extremely cautious regarding claims about UVGI systems' high levels of inactivation of pathogenic bioaerosols. While the microbiological science underlying these

conclusions is thought to apply to pathogenic bioaerosols as well as environmental organisms, much greater caution is required in the former case. Claiming a high inactivation rate for a pathogenic bioaerosol without substantial testing would be irresponsible. Even with substantial testing, design failures may occur.

## 6.0 RECOMMENDATIONS

Because lamp output varies so strongly with temperature and airflow rate, lamp output measurements should either be made at the expected use conditions or extrapolated to those conditions.

In the efforts to counter bioterrorism, the use of UVGI is likely to be proposed with increasing frequency. It is a low-pressure-drop, nonintrusive technology that has real potential. In that light, we recommend that test methods and guidance for performance claims regarding bioterrorism agents be developed by an experienced and broadly based group of technical experts, users, and manufacturers. Until rigorous and adequate tests have been developed and performed, claims regarding protection against aerosol bioterrorism agents are suspect. The potential errors are too large and the consequences too great.

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