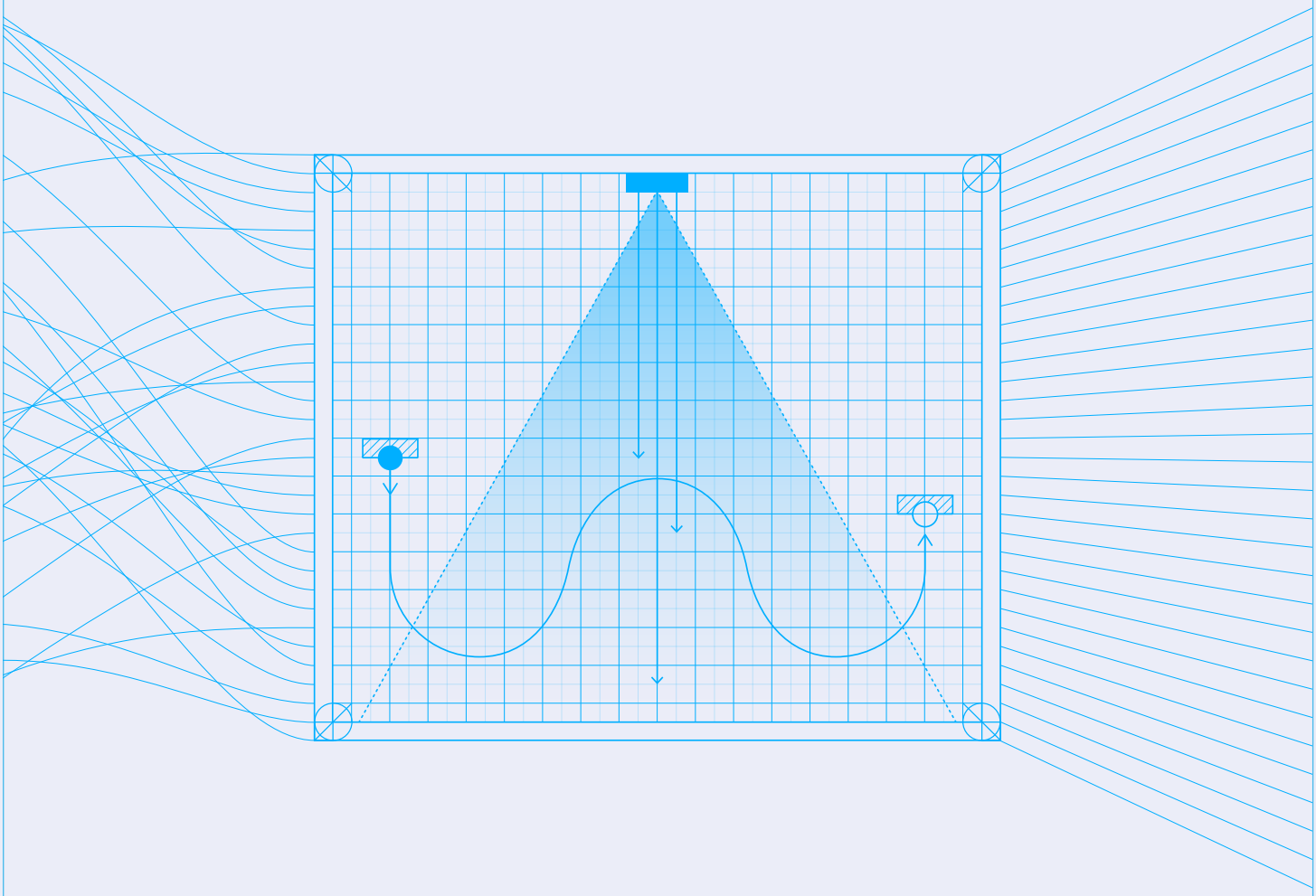
Blueprint Biosecurity



Blueprint for Far-UVC

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About Blueprint Biosecurity

Blueprint Biosecurity is a nonprofit dedicated to achieving breakthroughs in humanity's ability to suppress pathogens.

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The resulting report solely reflects the views of Blueprint Biosecurity, and does not represent the view of any other individual nor represent a consensus among subject matter experts. All opinions provided to us were the individual's own and not those of the organizations with which they are affiliated. Any errors in the report are entirely the responsibility of Blueprint Biosecurity.

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- Report Development Workshop hosted by Blueprint Biosecurity, St Andrews, June 18 2024
- Recommendations Workshop hosted by Blueprint Biosecurity, Washington DC, October 8 2024

And from presenting our work to the expert community at the following events:

- International Ultraviolet Association Americas Conference 2024, Orlando, May 20-22 2024
- 2nd International Congress of Far-UVC Science and Technology, St Andrews, June 18-21 2024
- Indoor Air 2024, International Association of Indoor Air Quality and Climate, Honolulu, July 7-11 2024
- 42nd American Society for Photobiology Biennial Meeting, Chicago, July 27-30 2024
- American Association of Aerosol Research 42nd Annual Conference, Albuquerque, October 21-25 2024
- 2024 Chemical and Biological Defense Science & Technology Conference, Ft Lauderdale, December 2-5 2024

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Executive summary

Despite the tremendous strides we have made against water-borne, food-borne, and vector-borne diseases, airborne infectious diseases remain one of humanity's biggest challenges: the COVID-19 pandemic has claimed an estimated 27 million lives. Tuberculosis kills 1.6 million people annually. One billion people are infected by influenza every year, leading to millions of serious illnesses and hundreds of thousands of deaths.

Countermeasures to infectious disease are vital, and their champions—Pasteur, Jenner, Fleming, Salk, Karikó—are rightly celebrated. But less celebrated are the interventions that operate in the background of everyday life—sanitation, pest control, food hygiene—which prevent us from encountering pathogens in the first place.

Germicidal ultraviolet light (GUV) has been used in water treatment for over 100 years. Studies in the 1940s showed the promise of treating the air above our heads with GUV—so called 'upper-room UV'—to control the spread of measles in schools and it has been used for decades to control the spread of drug-resistant tuberculosis.

Far-UVC is a new form of GUV. Because it is strongly absorbed by proteins in the outer layer of human skin and eyes, it can inactivate a wide range of pathogens with minimal penetration into and effects on human tissues. This enables higher human exposure limits, unlocking the potential for disinfecting occupied spaces continuously while achieving significantly improved air cleaning over current alternatives. Far-UVC is also silent, energy-efficient, commercially viable at scale, less vulnerable to engendering resistance than pharmaceuticals, and can be deployed in advance of an outbreak to help prevent a pandemic from occurring in the first place.

This report details a comprehensive set of recommendations to accelerate far-UVC's development and prepare it for widespread usage. The four most urgent priorities are:

1. Establish how far-UVC installations need to be designed in order to effectively suppress airborne transmission.
2. Identify different biological effects induced by far-UVC compared to solar UV and conventional GUV.
3. Understand unintended air quality impacts of far-UVC and options for mitigation.
4. Obtain high-quality evidence of real-world effectiveness.

We also identify the key building blocks for facilitating successful adoption and a longer-term research agenda to accompany adoption.

Unfortunately, the drivers of pandemic risk are going in the wrong direction. Climate change, factory farming, and human encroachment into wild habitats more than double the expected risks of pandemic pathogens jumping from animals to humans. Meanwhile, rapid advances in AI and synthetic biology leave us significantly more vulnerable to malicious actors and engineered pandemics. Experts estimate a 1 in 8 chance of a pandemic killing over 90 million people by 2050, three times the death toll of COVID-19. If we want to overcome these immense risks, the time to act is now.

A preview of chapters in the report

This report is based on an extensive review of the published literature and two years of consultation and collaboration with over 100 experts across multiple disciplines, including photobiology, atmospheric chemistry, indoor air quality, building science, environmental engineering, epidemiology, and public health. Its primary purpose is to coordinate the efforts of researchers, policymakers, entrepreneurs, funders, and other stakeholders who can accelerate far-UVC's development. It therefore begins with our *Recommendations*, the specific steps that need to be taken to validate far-UVC's real-world safety and efficacy profile.

In *Germicidal UV: introduction and history*, we cover the history of germicidal ultraviolet light from the 19th century to the present day, placing the development of far-UVC in the context of the shift in understanding of airborne disease transmission caused by the COVID-19 pandemic.

In *Far-UVC primer*, we provide a detailed glossary of important concepts that are referred to throughout the report.

In *Efficacy*, we discuss the evidence for far-UVC's efficacy and effectiveness at disinfecting indoor air. Far-UVC can measurably inactivate airborne pathogens within safe human exposure limits. However, given inconsistencies in estimated susceptibility of pathogens and the impact of dose distribution in practical applications, the degree to which far-UVC will prove superior to existing air cleaning technologies is uncertain.

The following four chapters—*Skin and eye safety*, *Evaluating cancer risk*, *Ozone and indoor air quality*, and *Ozone epidemiology*—examine far-UVC's effects on skin, eyes, and indoor air quality. The evidence indicates that it is appropriate for far-UVC eye and skin exposure limits to be meaningfully higher than the limits for longer UV wavelengths. However, further research is necessary and we expect expert bodies to update their guidance in the coming years. Far-UVC generates small quantities of ozone relative to normal outdoor levels. However, this can be mitigated, and the risks from additional ozone ought to be weighed against the benefits of disinfection.

Emitters and luminaires analyzes commercially available far-UVC lamps. Krypton chloride excimer (KrCl*) lamps are currently the primary emitter source. Fundamental innovation is not necessarily required for widespread commercial adoption, and the cost of KrCl* lamps could decrease significantly in the next 3–5 years. However, solid-state technologies like LEDs or frequency doubled blue lasers could offer several improvements over KrCl* lamps on a 5–10 year timescale.

The *Guidance, standards, and regulations* chapter examines the complex landscape governing far-UVC use and implementation and offers an overview of the consensus standards and guidance provided by a wide array of expert bodies and professional associations.

Finally, *Materials* examines how far-UVC is likely to interact with the built environment and the different materials that comprise it. While relatively little is known about far-UVC's impact specifically, exposure to other forms of UV (such as solar UV and conventional GUV) are known to cause effects on the appearance and performance of some common materials.

Scope

This report evaluates the use of far-UVC to suppress 'long-range' airborne transmission. There are other promising uses of far-UVC, including disinfection of surfaces, prevention and treatment of surgical-site infections, 'near-field' protection (for example through personal protective equipment that incorporates far-UVC), as well as the prevention of 'short-range' airborne transmission. We expect to review the prospects for short-range transmission reduction in a future update.

While there are other air cleaning technologies that play an important role in defending against airborne disease, including ventilation, filtration, and even other types of germicidal UV light, they are not within the scope of this report except insofar as they relate to far-UVC.

Recommendations

The starting point for our recommendations is the mission of Blueprint Biosecurity: achieving breakthroughs in humanity's capability to prevent, mitigate, and suppress pandemics. If far-UVC is to be effective as a pandemic prevention measure, it will need to be widely installed across indoor public spaces before outbreaks occur. That does not mean that there is no value in a more limited adoption, or that widespread adoption is needed for a viable commercial market to exist for far-UVC manufacturers. But we do expect that in order to achieve the goal of pandemic prevention, far-UVC will have to prove highly effective at suppressing the transmission of endemic respiratory illnesses—otherwise, mass adoption before outbreaks won't be justified.

The level of trust that we are asking the public to place in far-UVC is substantial. Far-UVC does not require most individuals to change their behavior to be effective, which is a key reason that it could prove so impactful. But a world in which far-UVC enjoys widespread use in public spaces is one in which only limited personal consent can be given to far-UVC exposure. The corollary of passive collective protection is that great trust is placed in the scientists who develop the technology, the professionals who install it, and most of all the institutions who set safety and efficacy standards.

The thoroughness embodied in our recommendations, in particular with regards to the need for further research, reflects the trust that we believe must be earned through rigorous science in order for far-UVC to have a substantial impact on the world.

The recommendations of this report are specific to advancing far-UVC, with preventing airborne disease transmission as the intended use case. There are other things that could materially advance the cause of airborne infection resilience, including basic research into the aerobiology and epidemiology of pathogen transmission, development of biosensors that can detect infectious aerosols in real time, and better monitoring of respiratory infections acquired in high-risk environments such as healthcare settings. A number of these may be highly impactful, but they have not been evaluated as part of this report.

The recommendations fall into three categories.

First, we offer four **research priorities**, describing the academic and clinical research that urgently needs to be pursued to validate the potential of far-UVC and facilitate adoption.

Second, we offer five **recommendations** for facilitating successful adoption at scale. These are relevant to a wide range of stakeholders, including academia, industry, government, civil society, and early adopters.

Finally, we propose a **long-term research agenda** that ought to be pursued to answer questions that are important, but that we do not believe need to be urgently resolved in order to advance the field.

Research priorities

Recommendation 1: establish how far-UVC installations need to be designed in order to effectively suppress airborne transmission.

The amount of far-UVC power that is needed for air disinfection depends on the dose-response of different pathogens to far-UVC. All of the other challenges highlighted in this report become greater the higher the dose required to be effective, making understanding this the highest priority.

Among the studies that have been conducted so far, there are order-of-magnitude differences in pathogen susceptibility to far-UVC. We need to understand what aspect(s) of experimental procedure or environment cause this variability, and its causes may not be specific to UV inactivation. Standardization of research methods into infectious aerosols may have substantial spillover benefits for other technological approaches to controlling airborne transmission.

The protein absorption that makes far-UVC safer than other forms of UV will also reduce its penetration into the protein-rich human respiratory aerosols in which airborne pathogens are contained. It is not currently known how significant this effect is, nor what additional consequences there could be on the stability of pathogens contained in the aerosol. It is also challenging to synthesize representative human respiratory aerosols in the lab.

Sophisticated modeling, such as computational fluid dynamics combined with lighting simulation, is needed to translate experimental findings into practical guidance for safe and effective deployment in diverse environments. As these research methods can be inaccessible to end users, the goal of this research has to be practical heuristics that can be endorsed by public health agencies and implemented by practitioners at scale (see Recommendation 5).

Finally, we know that environmental factors such as relative humidity affect the susceptibility of pathogens to conventional GUV, and it is reasonable to hypothesize that the efficacy of far-UVC will also be mediated by environmental factors. These must be known in order to provide effective deployment guidance.

Therefore, researchers should:

1.1 Obtain pathogen inactivation data from actual human respiratory aerosols.

1.2 Conduct controlled bioaerosol chamber studies to establish the degree to which environmental variables such as relative humidity affect the susceptibility of relevant pathogens to far-UVC.

1.3 Understand the causes of the variability in experimental results and produce standardized experimental methods.

1.4 Use relevant techniques, such as computational fluid dynamics modeling and lighting simulation, to model UV dose distribution based on different room geometries, ventilation regimes, occupancy, and the location of the infectious source.

Recommendation 2: identify different biological effects induced by far-UVC compared to solar UV and conventional GUV.

Far-UVC is absorbed by proteins in the outer layer of skin and eyes, thus reducing the harms associated with other forms of UV radiation. This is the foundation of far-UVC's promise for whole-room disinfection of occupied spaces.

While there is currently no evidence of significant harm from far-UVC, the impact of protein absorption is not yet fully understood. Protein absorption and other consequences of the higher energies of far-UVC photons will have other effects not traditionally associated with conventional GUV and solar UV exposure. Safe exposure limits for human skin and eyes are based on the propensity of different UV wavelengths to induce harmful biological effects, and therefore we should ensure that the measurable endpoints by which these harms can be detected are fully complete.

Therefore, researchers should:

2.1 Obtain a mechanistic understanding of the effects of protein absorption in the skin and eye.

2.2 Study other pathways through which the higher energies of far-UVC photons may cause relevantly different effects to longer UV wavelengths.

2.3 Use this knowledge to identify biomarkers and create action spectra that can inform the exposure limits recommended by expert bodies such as ICNIRP and ACGIH.

Recommendation 3: understand unintended air quality impacts of far-UVC and options for mitigation.

Far-UVC will generate some ozone. Our knowledge of the amount of potential harm from ozone exposure is based on epidemiological studies of outdoor ozone. In order to use this data to bound the harms of any indoor air quality effects from far-UVC, a number of assumptions need to be made. In particular, we must assume that the complex indoor air chemistry observed with the use of far-UVC is all downstream of the generation of ozone. Establishing whether this assumption is valid, or whether there are byproducts relevant to air quality that are caused by some other effect of far-UVC, should be a high priority.

It is also possible to mitigate indoor ozone generation and any byproducts not only through adequate ventilation but also through the use of catalysts or activated carbon filters. Epidemiological data suggests that reducing the concentrations of ozone and its byproducts in indoor air could have potentially significant public health benefits independent of synergizing with the use of far-UVC, and these technologies are worthy of further development.

Therefore, researchers should:

3.1 Conduct in-depth field measurements of indoor chemistry, in diverse environments representative of the spaces in which far-UVC may be deployed, that compare the effects of far-UVC to introducing the equivalent quantity of ozone. Independently estimating relevant parameters such as fluence rate, ventilation, and background ozone decay is vital to assist with interpretation and modeling of results.

3.2 Replicate lab measurements of ozone generation from different far-UVC devices to establish a robust model for predicting ozone production based on their output power and emission spectrum, and establish a standard test method.

3.3 Build on existing epidemiological research to quantify potential health effects of exposure to ozone and ozone reaction byproducts indoors.

3.4 Investigate the potential for ozone removal with catalysts and activated carbon, and identify the solutions that are most cost-effective without creating unintended consequences.

Recommendation 4: obtain high-quality evidence of real-world effectiveness.

Cluster-randomized trials (CRTs) that demonstrate reductions in infections are considered the gold standard of evidence for infection control.

CRTs have risks and drawbacks for studying transmission suppression technologies like far-UVC. Previous studies of the effectiveness of upper-room UV produced mixed results due to flaws in study design that are challenging to mitigate. It is difficult to ensure that a trial is adequately powered to detect a reduction in transmission, and underpowered studies risk undermining the field. The failure to find statistically significant results can be misinterpreted as evidence that an intervention does not work, when it is often the case that the study is not capable of providing evidence that it *does* work.

It is our judgment that without a paradigm shift in the type of evidence that is expected by public health agencies and experts in infection control, successful CRTs are necessary to catalyze widespread adoption. However, ventilation is an example of an intervention to control airborne transmission that has become widely accepted as effective without such a study. If we are wrong about the importance of CRTs in proving the real-world effectiveness of far-UVC and catalyzing adoption, our other recommendations would still stand.

Therefore, researchers should:

4.1 Ensure that clinical trials are sufficiently powered that plausible effect sizes can be detected with statistical significance.

4.2 Ensure that clinical trials of GUV employ doses that are likely to prove effective based on the experimental evidence, and ensure that building occupants remain within photobiological safety limits.

4.3 Collect data such as temperature, humidity, CO^2 concentrations, ventilation rates, pathogen concentrations and sequencing of confirmed infections, in addition to the primary infection endpoints. This will assist in the interpretation and generalizability of a CRT whether it is successful or not.

Facilitating successful adoption

Recommendation 5: create simple far-UVC deployment guidance, backed by research, that can be clearly communicated by public health agencies and other trusted institutions.

Public health communication, risk communication, and radiation safety communication are mature fields with established principles that can be applied when communicating about far-UVC. It is vital to develop informative guidance that can be implemented by practitioners and educational materials that are comprehensible to a lay audience.

In order to provide this guidance and facilitate clear communication, we need to determine which factors are critical for designing effective far-UVC applications in different spaces (see Recommendation 1).

Therefore, developers of guidance and informational materials should:

5.1 Facilitate dialogue between modelers, experimentalists, public health authorities, communication experts, and the practitioners who will have to follow the guidance.

5.2 Follow established practices in the fields of public health communication, risk communication, and radiation safety communication.

5.3 Produce guidance for far-UVC that accounts for different levels of ventilation. There cannot be a 'one size fits all' approach, and the impact of ozone-initiated secondary chemistry is particularly sensitive to levels of ventilation.

5.4 Bring the institutions trusted by the public into the guidance development process early.

Recommendation 6: improve consensus standards for airborne infection control applications and far-UVC devices.

Some consensus standards are already established, more are needed, and all of these will require future revision. 2023 saw the publication of UL 8802 *Standard for Ultraviolet (UV) Germicidal Equipment and Systems*, as well as ASHRAE 241 *Control of Infectious Aerosols*—the first standard that attempts to provide a technology-neutral framework for preventing long-range airborne transmission in a wide variety of indoor public spaces.

There are other standards that are important to the use of far-UVC, such as UL 2998 *Environmental Claim Validation Procedure (ECVP) for Zero Ozone Emissions from Air Cleaners*, that were not formulated with the properties of germicidal lamps in mind and need to be amended. Another standard that governs far-UVC use in many settings, *IEC 62471 Photobiological safety of lamps and lamp systems*, has not been updated since 2006.

A number of standard test methods exist for quantifying the efficacy of air cleaning devices against bioaerosols, and all of these will likely require revision once we understand the cause of the variability of results on far-UVC efficacy observed in the academic literature (see Recommendation 1).

Developing and revising consensus standards requires input from a wide variety of interested parties, including academic experts, trade associations, government, manufacturers, and end users, to ensure a balanced perspective. If you are reading this document, consider participating in the development of consensus standards.

Therefore, standards-setting bodies should:

6.1 Further develop and refine existing standards, particularly:

- ASHRAE 241 *Control of Infectious Aerosols*.
- ANSI/CAN/UL 8802 *Standard for Ultraviolet (UV) Germicidal Equipment and Systems.*
- ANSI/CAN/UL 8803 *Portable UV Germicidal Equipment With Uncontained UV Sources*.
- IEC/EN 62471 *Photobiological safety of lamps and lamp systems.*
- ISO 15858: *UV-C Devices - Safety information - Permissible human exposure.*
- ANSI/IES RP 27.1-22 *Photobiological Hazards From UV Lamps.*

6.2 Develop new consensus standards for:

- Ozone generation from germicidal applications, using new testing methodologies that account for the particular properties of far-UVC devices.
- Manufacturing and labeling of GUV devices that provide consistent information to consumers on expected lifetime, UV power output and emissions spectrum, and photobiological exposure limits based on the device emissions spectrum.

6.3 Modify standards relevant to particular industries and settings, such as the Facilities Guidelines Institute standards for hospitals and other healthcare settings in the United States, to facilitate the use of GUV in high-infection-risk spaces.

6.4 Develop improved standard testing methodologies for air cleaners that claim to remove infectious aerosols. These can be incorporated into standards such as ASHRAE 241, and can also be used by government agencies such as the US EPA who have the authority to regulate marketing claims.

Recommendation 7: ensure that far-UVC is installed in accordance with consensus standards, as part of a layered approach with other engineering controls such as adequate ventilation and filtration.

Layered interventions with different mechanisms are more resilient to the diversity of potential biological threats, and the use of multiple different approaches is a basic principle of biosecurity. Different technologies have different strengths and weaknesses, and far-UVC is likely to be more effective at mitigating some airborne infection threats than others. Adequate ventilation and the use of mechanical filtration (e.g. HEPA or MERV-13) have other potential health benefits as well as mitigating some of the possible risks of far-UVC use.

Not all far-UVC devices are the same, and emissions of non-far-UVC wavelengths from KrCl* lamps and LEDs pose different photobiological and photochemical risks. Not all use cases are the same either, and the potential risks of far-UVC use are higher in places with longer occupant dwell times and lower standards of ventilation, and without professional facilities management.

We expect some of these recommendations to be superseded by the further development of consensus standards and deployment guidance, but they represent what we believe is prudent today.

Therefore, we recommend to those considering installing far-UVC today:

7.1 Purchase devices that have been certified to appropriate product standards such as IEC/EN 62471 or ANSI/CAN/UL 8802. In some jurisdictions certain standards are mandatory.

7.2 When assessing photobiological exposure limits, either in a test lab or the field, the combined effect of all the emissions of the device—not just the peak 222-nm emissions of a KrCl* lamp—must be factored in.

7.3 Ensure that ventilation is sufficient and working as intended before considering the installation of far-UVC. Far-UVC is not an alternative to minimum acceptable ventilation standards.

7.4 Typically avoid mounting fixtures on walls, and where possible mount devices on the ceiling facing down to provide an additional safety margin for eye exposure. However, the best approach to safe and effective installation does depend on the specifics of room geometry and the behavior of occupants.

7.5 Carefully weigh the benefits and risks of the use of far-UVC in private residences.

Recommendation 8: improve performance and affordability of far-UVC emitters.

Far-UVC emitters are currently expensive and may not be cost-effective outside of high-risk settings, such as healthcare facilities. As disinfection is ultimately a product of dose and the susceptibility of the pathogen (see Recommendation 1), the relevant criteria for cost-effectiveness is the cost per mW of far-UVC output that can be safely installed in a space.

Reducing cost per mW can be achieved through a number of means. Increased production of far-UVC lamps for other applications will help achieve economies of scale, and this additional source of far-UVC emitter production could also be repurposed in a future pandemic. The far-UVC industry is currently very small, and substantial reductions in cost per mW are feasible merely from scale.

There is potential for direct cost reduction of key lamp components, as well as prospects for wholly new emitter technologies based on semiconductor technology such as LEDs or frequency-doubled blue lasers. But there are other strategies for addressing the cost per mW of useful power output that do not require fundamental innovation, and some product features may have the capability of addressing multiple challenges—safety, cost, energy efficiency—simultaneously.

We have seen marked improvements in far-UVC emitters over the last decade, and we believe that the improvements in features and cost necessary for widespread deployment will happen if there is the prospect of a market to sustain the industry and attract investment.

Therefore, industry should:

8.1 Improve diffuser technology, as this has the potential to reduce cost per mW and increase the energy efficiency of fixtures designed for lower ceiling heights.

8.2 Evaluate the use of proximity sensors or cameras, combined with the capability to dim or boost output, allowing for dynamic output regulation based on room occupancy.

8.3 Develop cost-effective filters that are as transparent as possible to far-UVC wavelengths and opaque outside that range. Such filters are useful for multiple different types of far-UVC sources, and filters can be a significant cost component of lamps.

8.4 Extend lamp lifetime to reduce both maintenance and effective cost per mW.

8.5 Develop next generation emitters, such as semiconductor technology. These may outcompete current sources on cost, and also provide different features for different applications.

8.6 Develop markets for far-UVC outside of indoor air disinfection. Examples that we have seen proposed, but we have not evaluated as part of this report, include water treatment, surface disinfection, healthcare tools, agriculture and food production, food processing, pest control, and scientific equipment.

Recommendation 9: create cost-benefit analysis frameworks for deploying far UVC

Far-UVC technology is not meant to be used everywhere or in every situation. Its adoption should be thoughtful and guided by evidence. Many stakeholders, especially institutional decision-makers, will base their investment decisions on a careful cost-benefit analysis.

These decisions, whether made by private businesses, healthcare facilities, or government agencies, require evaluating a complex mix of both tangible (hard) and intangible (soft) costs and benefits. These include costs such as upfront capital, ongoing operational costs, and deployment risks, and expected gains such as fewer infections, reduced absenteeism, increased productivity, and lower healthcare costs.

In situations where achieving airborne infection control is (or becomes) required, and decision-makers use far-UVC to substitute for other methods, then the benefits can take the form of cost and/or energy savings from reducing the use of less efficient methods. Developing frameworks that compare the costs and benefits of different solutions is necessary.

Therefore, to support more informed decision-making, researchers and building design professionals should:

9.1 Develop cost-benefit analysis frameworks and tools tailored to specific deployment settings, starting with high-risk, high-impact environments like healthcare facilities, public gathering spaces, and schools.

9.2 Regularly update these frameworks and tools with the latest research and data on far-UVC technology's benefits, costs, and implementation needs.

9.3 Ensure that these cost-benefit analysis tools are adaptable for use with other disinfection and air-cleaning technologies, enabling fair and consistent comparisons across different solutions.

Long-term research agenda

Recommendation 10: conduct long-term safety studies in diverse populations.

Post-approval studies play an important role in ensuring the long-term safety of pharmaceuticals, and the same principle applies to far-UVC. As with pharmaceuticals, it would be an excessive application of the precautionary principle to require long-term studies of hypothetical side effects before implementing an efficacious innovation that saves lives. However, that does not mean that such studies are unnecessary.

Long-term safety studies for chronic far-UVC exposure will ideally have longer duration and follow-up than a CRT designed to prove effectiveness. The challenges of powering a CRT to detect reductions in infections limits the types of facilities (and therefore people) that can be feasibly studied. To obtain data in diverse populations that may have different sensitivities to far-UVC exposure, long-term safety will likely need to be studied in settings that may not be suitable for studying effectiveness.

Therefore, researchers should:

10.1 Identify practical study designs for assessing the long-term effects of chronic exposure to far-UVC.

10.2 Commence these studies as soon as is practical. This requires a combination of long-term commitment on the part of the participating buildings and occupants in order to justify the set up of the study, as well as knowledge of the relevant biological endpoints that should be included in such a study (see Recommendation 2).

Recommendation 11: study the effects of far-UVC on materials ubiquitous in the built environment.

Far-UVC will interact in some way with materials commonly found in the built environment, and there is a need to prioritize what materials to study. This prioritization exercise should account for both potential mechanisms and the importance, ubiquity, and intended lifespan of the material.

Substantial changes will be necessary to make buildings healthier. We have re-engineered the built environment many times, from the sanitation revolution, to fire safety, to reducing energy usage. Periodic renovations and retrofits are an opportunity to address multiple problems simultaneously. If far-UVC is found to have undesirable effects on common materials, there are potential mitigation strategies.

Therefore, researchers should:

11. 1 Conduct controlled exposure studies in the lab on common materials, cosmetics, clothing, and plants, not just for aesthetics and performance but also for potential off-gassing of harmful compounds.

11.2 Conduct long-term studies in the real-world environment under realistic exposures to quantify whether any hypothesized effects occur in complex products and environments.

11.3 Study potential mitigation strategies including: using coatings or sealants that are far-UVC resistant, identifying sensitive materials, and producing practical guidance for reducing the exposure of materials that prove to be particularly sensitive.

Recommendation 12: obtain a deeper understanding of the mechanisms by which far-UVC inactivates pathogens.

Understanding the mechanisms of pathogen inactivation will help us predict far-UVC's efficacy against a broad range of threats without testing every pathogen individually.

A wider biosecurity goal is facilitating real-time feedback on the effectiveness of interventions that reduce the concentration of infectious aerosols, as proposed in the ARPA-H BREATHE program. However, it is currently not possible to reliably distinguish between infectious and

inactivated pathogens without performing time-consuming bioassays. If we understood mechanisms of inactivation better, this could potentially provide targets for novel biosensors that would be more practical to widely deploy.

Therefore, researchers should:

12.1 Study how far-UVC inactivates microbes, including the likely possibility that this occurs through multiple mechanisms.

12.2 Identify targets that could be used for novel biosensors that would distinguish between infectious and inactivated pathogens.

Why are we interested in accelerating the development of far-UVC?

Even after a global pandemic that spread largely via aerosols, tools to suppress the spread of airborne disease remain deeply neglected. This neglect is particularly concerning given the exceptional efficiency of airborne transmission in the wrong circumstances. The Omicron variant of SARS-CoV-2 demonstrated how rapidly airborne pathogens can spread, doubling cases approximately every 2-3 days and infecting an estimated 125 million people globally within just 10 weeks of its identification. Measles, the most infectious human pathogen, is also airborne.

Existing solutions are critical but insufficient

Current approaches to controlling airborne disease transmission fall broadly into three categories: medical countermeasures like vaccines and therapeutics, personal protective equipment like masks, and engineering controls like ventilation, filtration, and upper-room UV. Each of these technologies plays a critical role in pandemic prevention and mitigation but has significant limitations and vulnerabilities.

Medical countermeasures

Vaccines and therapeutics are vital, but they take time to develop and distribute. Even with record speed, COVID-19 vaccines were not available for a year, and many countries waited significantly longer. Some people, including the immunocompromised, infants, and the elderly, may not be fully protected even when vaccines are available.

Personal protective equipment

High-quality respiratory protection, like N95s and elastomeric respirators, is essential for individual protection against airborne pathogens when properly worn, and we are working on increasing the amount and quality of personal protective equipment that will be available when the next pandemic strikes. However, masks also face significant challenges, including cost, comfort, communication difficulties, and compliance fatigue, so we cannot rely on them alone for population-wide protection during prolonged outbreaks. Masks also pose particular challenges for specific populations, such as young children and individuals with certain medical conditions or disabilities, and in settings where clear communication is essential.

Engineering controls

We are optimistic about the potential for engineering controls to reduce the burden of endemic disease. Once installed, they protect everyone in the space without requiring individual action or compliance. However, existing solutions face severe limitations.

Ventilation and filtration

Ventilation and filtration are proven methods of reducing indoor airborne pathogen concentrations and we are enthusiastic about them being implemented where practical. But they can fall short in specific situations: many HVAC systems cannot meet current air cleaning standards, especially in crowded, aging, or energy-constrained buildings. Portable air cleaners (PACs) with high-efficiency particulate air (HEPA) filters are effective and offer meaningful protection in many spaces, and we are encouraged by recent innovations like the Corsi-Rosenthal box that potentially offer a cheaper, quieter, and more energy-efficient solution. But in many high-risk indoor public spaces and scenarios, these interventions will likely remain insufficient for comprehensive protection. When operated at the high levels needed for substantial protection, they can generate noise and drafts that can make spaces uncomfortable, causing users to turn them down or off.

These limitations become especially apparent when considering the air cleaning levels needed for highly infectious diseases. ASHRAE Standard 241 (Control of Infectious Aerosols, 2023) recommends clean air delivery rates that can far exceed both CDC guidelines and the ventilation capacities of a typical building's systems. A restaurant, for example, would require an additional 60 CFM / person, of clean airflow beyond standard ventilation systems to achieve the protection levels recommended by ASHRAE 241, comparable to the requirements of a modern operating theatre (see *Guidelines, standards, and regulations* section). Achieving this through ventilation or filtration could create disruptive gusts, noise, and unsustainable energy costs.

Upper-room and in-duct UV

Conventional upper-room 254-nm UVC systems have been deployed for decades in tuberculosis wards and operating theatres, and their use is recommended by CDC/NIOSH and WHO. These systems can achieve ASHRAE 241 standards but their key challenge is scalability: in order to be effective, UV intensity in the upper room must be much higher than safe human exposure limits, requiring expert installation and occupant awareness of the overhead hazard. In-duct UV systems disinfect air through HVAC systems but share the same challenges as ventilation and filtration: high energy use, noise, and limited effectiveness in spaces without ducted systems.

The promise of far-UVC

Far-UVC has a number of traits that make it extremely promising as a highly scalable and effective air cleaning technology. It will not be a panacea, however, and the remaining chapters of this report evaluate its safety and efficacy profile in significantly more detail with a critical eye for key gaps and uncertainties. Far-UVC's promising attributes include the following:

- **Far-UVC can inactivate a wide range of pathogens:** studies have shown strong inactivation against viruses like influenza and coronaviruses, bacteria such as *Staphylococcus aureus* and *Pseudomonas aeruginosa*, and even pathogenic fungi like *Candida auris*, suggesting far-UVC may be useful against both familiar and novel threats (see *Efficacy* section).

- **Far-UVC installations can be made safe by design:** far-UVC is absorbed by proteins in the outermost layers of the skin and eyes, allowing for higher safe exposure limits and safe operation in occupied spaces (see *Skin and eye safety* section).

- **Far-UVC is energy-efficient:** modeling shows that far-UVC can be up to 450 times more efficient than ventilation and 40 percent more efficient than air purifiers in delivering clean, disinfected air (see *Efficacy* section).

- **Far-UVC is silent and practical:** far-UVC runs silently, requires far less space than portable air cleaners or ventilation ductwork, and is relatively simple to install.

- **Far-UVC is showing promise for preventing fomite and short-range transmission as well:** while this report focuses on long-range airborne transmission, far-UVC also inactivates pathogens in the concentrated plumes that drive short-range transmission and those on contaminated surfaces.

- **Far-UVC may help combat antimicrobial resistance:** far-UVC has been shown in laboratory studies to inactivate drug-resistant bacteria on surfaces and in air. It holds promise as a supplemental tool to reduce the burden of antimicrobial-resistant pathogens in high-risk settings.

Deployment of far-UVC could be highly cost-effective

The science of far-UVC, and the availability of commercial far-UVC emitters, has rapidly evolved since the onset of the COVID-19 pandemic. Many uncertainties, highlighted in this Blueprint, remain. However, preliminary analyses suggest far-UVC could prove highly cost-effective on a few different dimensions:

1. One analysis of the costs and benefits of implementing ASHRAE 241 air cleaning targets estimated a 10-to-1 return on investment, even when considering only seasonal illnesses.
2. An analysis of the use of far-UVC in indoor public spaces in Switzerland estimated a benefit cost ratio (BCR) of 30–290x in a normal winter respiratory illness season, and higher in pandemic scenarios.
3. Finally, another analysis of the use of conventional UVC in aircraft cabins found a 1,000 percent annual return on investment and a cost of $10,000 per life saved. This analysis focused only on reducing the transmission of endemic influenza and SARS-CoV-2. Far-UVC could offer similar benefits with fewer safety and operational constraints.

Additional rigorous cost-benefit analyses need to be developed and tailored to different contexts and use cases, and updated as both our scientific understanding of far-UVC and the costs of commercially available devices evolve (see Recommendation 9). But these early results are highly encouraging.

Far-UVC technology is at a critical inflection point

There are clear opportunities to rapidly accelerate the development of far-UVC with attainable levels of funding. The Recommendations of this report have been formulated to direct funding and effort towards the most important priorities. They reflect not only the level of trust that the public would be asked to place in this technology, but our ambition to support deployment at the scale necessary to save millions of lives.

We believe that public and philanthropic funding on the order of $100 million will be needed over the next five years to provide the standard of evidence that public health agencies will expect before considering widespread deployment. This is substantial relative to the current investment in the field but achievable. It is an amount routinely invested in promising biomedical research—not a Human Genome Project, Apollo Program or Operation Warp Speed.

Far-UVC represents one of the highest leverage funding opportunities in airborne disease and pandemic prevention that we are aware of. With strategic, coordinated investment, far-UVC technology could transform how we approach preventing airborne disease in the built environment, potentially averting millions of deaths, billions of infections, and trillions in economic costs before the next major pandemic strikes.

1. Germicidal UV: introduction and history

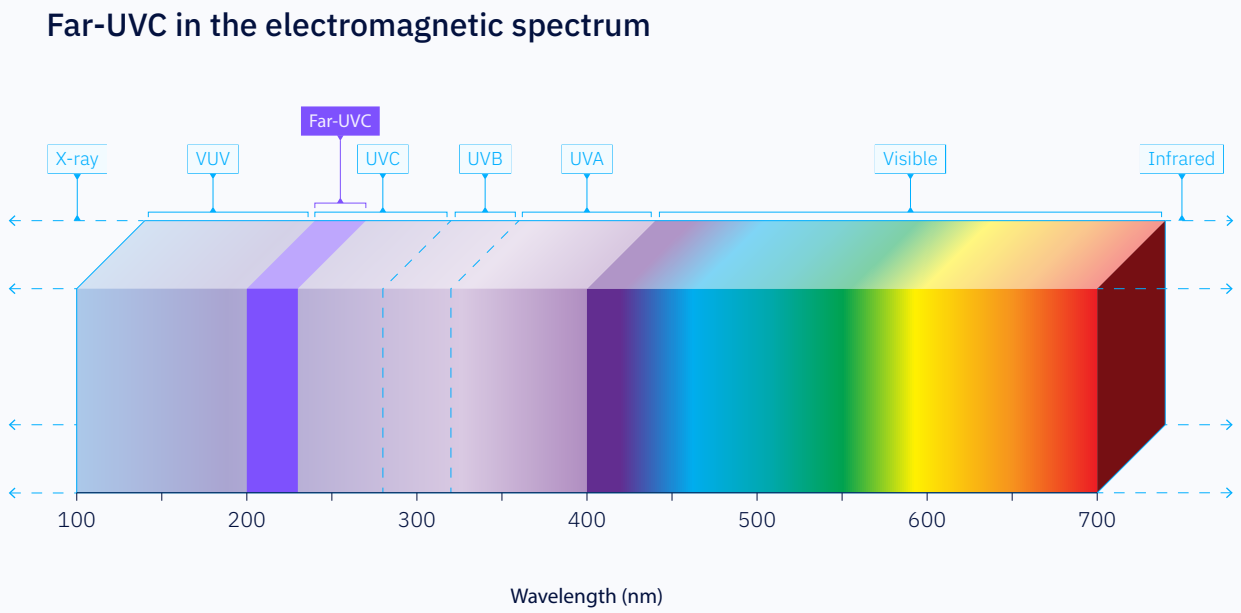

FIGURE 1.1. The electromagnetic spectrum is defined by wavelength.

Invisible colors

The discovery of ultraviolet (UV) light dates back to the early 19th century when Johann Wilhelm Ritter first detected an invisible form of radiation beyond the violet end of the visible spectrum in 1801[1]. He observed that this radiation caused silver chloride-coated paper to darken faster than visible light, indicating a high-energy, chemically reactive form of light, which he initially called 'deoxidizing rays' before the term 'ultraviolet' was later adopted.

Much like visible light, UV contains different 'colors', and despite being invisible to the human eye, these different colors are just as distinct as red and blue (Figure 1.1). What we are familiar with as 'blacklights', widely used for everything from zapping mosquitoes to illuminating nightclubs, are actually part of a set of invisible colors we call 'UVA'. By contrast, it is the invisible colors we call 'UVB' that are believed to be the primary cause of most skin cancers. 'UVC' is the name of another set of invisible colors further away from visible light in the UV spectrum, of which far-UVC is a subset.

The discovery of UV's germicidal properties

By the late 19th century, researchers began exploring both the physical and biological effects of light. In 1877, Arthur Downes and Thomas P. Blunt made a key observation when they demonstrated that sunlight—which contains UVA and UVB—could inhibit bacterial growth[2–5]. They exposed test tubes containing bacteria to direct sunlight and observed that microbial growth was significantly reduced or entirely prevented. They further determined that the germicidal effect depended on the intensity and duration of exposure, with shorter wavelengths of sunlight proving most effective. In 1890, Robert Koch demonstrated the lethal effect of sunlight on *Mycobacterium tuberculosis*, hinting at UV's potential for combating diseases like tuberculosis[6]. These discoveries laid the foundation for our understanding of the bactericidal effects of light, including what would later be recognized as the ultraviolet section of the electromagnetic radiation spectrum.

Building on this knowledge, Niels Ryberg Finsen became one of the first to harness UV for medical applications. In the late 19th century, he pioneered the use of concentrated UV therapy to treat skin tuberculosis *(lupus vulgaris)*, a chronic infection caused by *Mycobacterium tuberculosis*[7–9]. Finsen's therapy, which earned him the 1903 Nobel Prize in Medicine, relied on carbon arc lamps and quartz lenses to generate and focus UV light, leading to documented clinical success[10].

At the same time, laboratory studies were beginning to show that UV had direct bactericidal properties. Early microbiologists such as Valdemar Bie and Sofus Bang, working under Finsen, reported that wavelengths below 300 nm were particularly effective at inactivating bacteria[5,11,12]. By 1903, Bang had demonstrated that concentrated UV from an arc lamp could kill *Mycobacterium tuberculosis* within minutes, providing some of the earliest direct evidence of UV's germicidal effects[13].

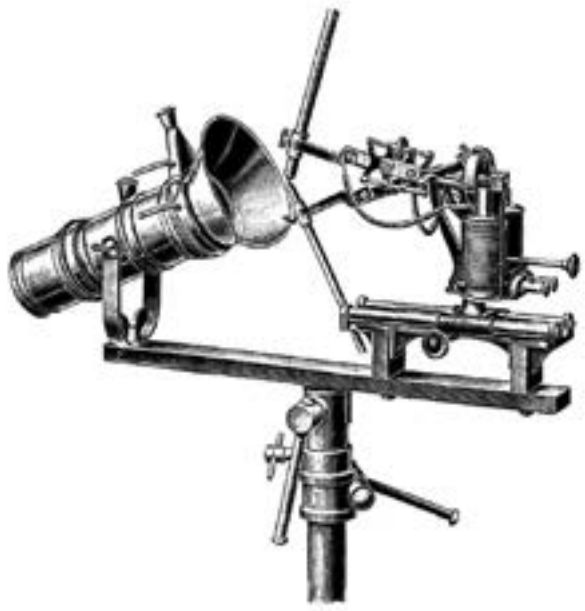

FIGURE 1.2. Carbon arc lamp used by Finsen in early UV research. Later called the Finsen lamp, it worked by creating a high-intensity light source that was filtered and focused to concentrate specific wavelengths, primarily in the UVA and UVB ranges. Source: Science Source.

The early 20th century saw further refinements in understanding UV's germicidal potential. Frederick L. Gates published the first precise bactericidal 'action spectrum'—a graph showing the efficacies of different UV wavelengths at killing bacteria[5,14–16]. Gates demonstrated that UV wavelengths around 265 nm (within the UVC section of the UV spectrum) were the most effective for inactivating bacteria. He also tested far-UVC (225 nm) and found it to be bactericidal, but technical limitations at the time, including the low power of UV sources available at that wavelength, made it difficult for him to fully evaluate shorter wavelengths and establish a complete action spectrum.

Advancements in mercury-vapor lamp technology provided a new means of generating UV, and in particular UVC. The first mercury-vapor lamp to achieve widespread success was invented in 1901 by American engineer Peter Cooper Hewitt[17]. His initial design, while effective, produced a bluish-green light that limited its applications. In 1903, Hewitt introduced an improved version with enhanced color qualities, making it more suitable for industrial use[18]. Beyond illumination, the UV emitted by mercury-vapor lamps was soon recognized for its potential in disinfection. By 1910, the technology was being applied to water treatment, marking the beginning of UV-based sterilization methods that would later expand to air and surface disinfection[19]. While Finsen carbon arc lamps and quartz lenses created mostly UVA and visible light, these lamps were able to create UVC, with a peak at 254 nm, much closer to the wavelength that Gates would show to be the most effective at inactivating bacteria.

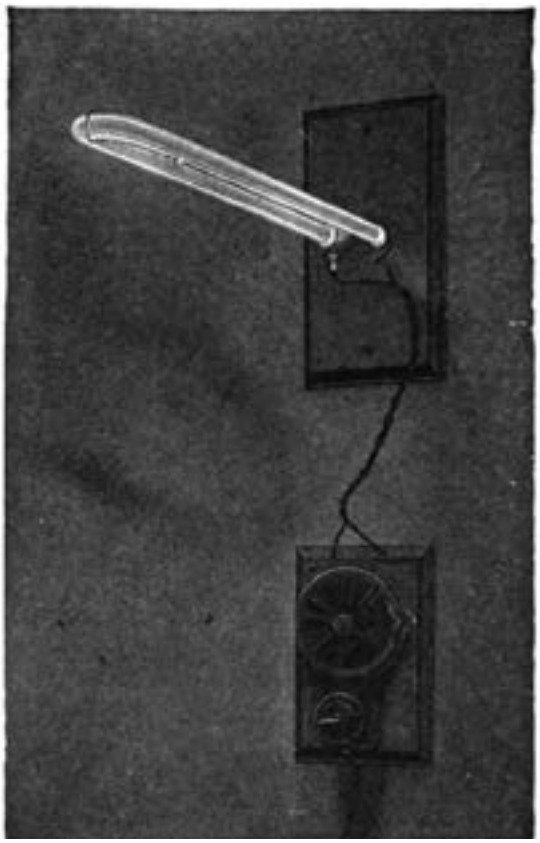

FIGURE 1.3. Peter Cooper Hewitt's mercury-vapor lamp. Source: Alamy.

In the 1930s, physician Mildred Weeks Wells and engineer William Firth Wells began investigating how respiratory diseases spread. The husband-wife research team brought complementary expertise: Mildred's knowledge of infectious diseases and their transmission paired well with William's background in air and water quality engineering. Through careful experimentation and mathematical modeling, they showed that when people cough, sneeze, or breathe, they emit droplets across a spectrum of sizes[20,21]. Larger droplets quickly fell to the ground, while smaller ones could evaporate before settling, leaving behind 'droplet nuclei' that could float in the air for extended periods. This size-based behavior, later known as the Wells curve, explained why some diseases could spread through the air over considerable distances—a mechanism that differed from the dominant theory of direct droplet spread.

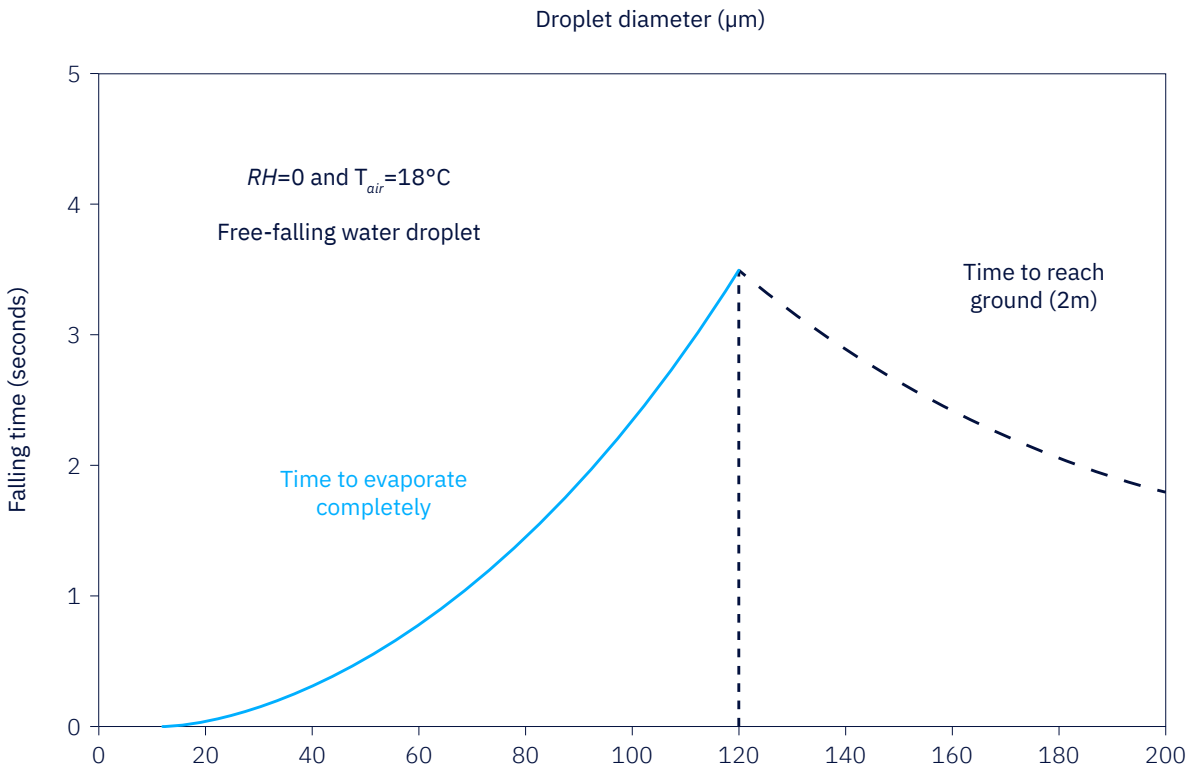

FIGURE 1.4. The Wells curve describes how respiratory droplets behave after being exhaled—larger droplets fall to the ground due to gravity, while smaller droplets rapidly evaporate, leaving behind airborne droplet nuclei that can carry infectious pathogens[20].Excerpted from Yu, 2016[22].

As the Wells developed their theoretical understanding of airborne disease transmission, hospitals began testing UV for infection control. In 1936, Deryl Hart at Duke University Hospital sought to address persistent post-operative infections that conventional sterilization techniques had failed to prevent[5,23]. Initially, Hart's team experimented with therapy UV lamps and carbon arc lamps, but these proved ineffective at reducing bacterial contamination in the operating room.

Seeking a more effective solution, they collaborated with Westinghouse Lamp Company, which supplied specially designed low-pressure mercury-vapor lamps optimized to emit germicidal UVC (commonly referred to as GUV) at 254 nm. The installation of these high-intensity UVC lamps dramatically reduced airborne bacteria, and their early studies showed a complete elimination of infections in UV-treated thoracoplasty cases, compared to a 33% infection rate in untreated cases. However, the use of UVC required strict safety measures—surgical staff had to wear goggles, tightly woven cloth hoods, and even sun helmets to protect their skin and eyes from overexposure.

Over time, the effectiveness of UVC disinfection became even more evident. A 1960 study by Hart, analyzing over 4,500 operations, found that UVC reduced post-operative wound infection rates in clean surgeries from 11.62% to just 0.24%, reinforcing its value as a powerful tool for infection control[24]. The results were so compelling that other hospitals quickly adopted similar systems, and Duke University Hospital continues to use UVC disinfection in its surgical suites to this day.

These results also led hospitals to explore UV applications in other settings[5]. At Boston's Infants' and Children's Hospital, UV barriers created cubicle-like divisions designed to prevent respiratory cross-infections between patients[25]. Del Mundo and McKhann reported infection rates of 12.5% in control wards compared to 2.7% in wards with UV barriers[26]. Other hospitals documented similar reductions using both UV barriers and upper-room installations, where UV lamps irradiated only the air above people's heads[27–34]. This design allowed warm air currents, generated by people's body heat, and natural air mixing to carry exhaled pathogens into the upper UV-irradiated zone, where they were rapidly inactivated before they could be inhaled by others.

The Wells saw an opportunity to combine their understanding of airborne transmission with UV technology. In 1937, they launched a controlled study at Germantown Friends School near Philadelphia, where measles regularly swept through classrooms despite strict surface cleaning and student separation protocols. Their controlled studies compared classrooms with UV lamps installed near the ceiling, irradiating the upper unoccupied section of the room, to identical untreated rooms. In the UV-equipped classrooms, measles cases dropped by 13.3% compared to control rooms, even as cases continued spreading in nearby districts[35].

FIGURE 1.5. Picture from the Wells' study using GUV in the upper room portion of school classrooms[35]. The metallic chandelier holds UV lamps. These lamps point upwards, irradiating the unoccupied upper section of the room, relying on air circulation to move pathogens up and clean air down.

By 1943, the US Navy launched a systematic four-year study at its training centers, where respiratory diseases posed a serious problem among recruits. The Navy installed GUV systems in alternate barracks housing over 5,000 recruits, documenting all sick bay admissions while monitoring bacterial levels in the air. In the first year at Sampson Naval Training Center, high-intensity UV systems reduced respiratory infections by 25%, though low-intensity systems showed no effect[36].

However, the medical and public health community was resistant to the idea that pathogens could spread through the air. This resistance was rooted in a paradigm shift led by epidemiologist Charles Chapin in the early 1900s[37]. For much of history, the dominant belief was miasma theory—the idea that diseases were caused by exposure to 'bad air' from decaying matter. This view was gradually overturned in the 19th century by germ theory, which demonstrated that specific diseases were caused by microscopic organisms, not miasma. Chapin, a strong proponent of vaccination, pandemic response, and germ theory, argued that diseases primarily spread through direct contact and large droplets[38]. His work helped advance public health and infection control through hygiene, isolation, and sanitation. However, the shift went too far, and the medical community became skeptical that any airborne transmission of diseases existed, despite emerging evidence to the contrary.

The most rigorous evidence for UV's effectiveness against airborne infection came from studies designed by William Wells and carried out by Richard Riley at a Veterans Administration Hospital TB ward between 1954 and 1961[39–41]. The ward's ventilation system exhausted air from TB patient rooms through chambers housing guinea pigs. Some chambers received UV-treated air, while others received untreated air. Over the two-year period, while many guinea pigs breathing untreated ward air developed TB infections, none of the animals breathing UV-irradiated air were infected. Additional compelling evidence came during the 1957 Asian influenza pandemic, when McLean and colleagues observed infection rates of only 1.9% in UV-irradiated wards compared to 18.9% in non-irradiated wards, demonstrating UV's effectiveness against viral as well as bacterial pathogens[41].

This research shifted scientific consensus and ultimately led to the widespread acknowledgment of airborne TB transmission. GUV was adopted as a TB control measure in high-risk settings. However, many experts remained reluctant to generalize these findings to other respiratory diseases[37]. Despite its successes, interest in GUV air disinfection waned. Antibiotics revolutionized TB treatment, and a wave of new vaccines targeted diseases like diphtheria, polio, influenza, and even TB itself[5]. Public health officials saw these advances as the future of disease control and largely dismissed GUV as unnecessary, even for diseases accepted to have airborne transmission. Enthusiasm was further dampened by concerns about potential health effects from UV exposure and the ozone produced by early lamp designs[5]. Additionally, follow-up studies failed to replicate the Wells' dramatic reductions in disease transmission in classrooms[5]. These studies suggested that continued transmission outside of classrooms, particularly in school buses and other communal spaces that were not protected by UV, limited the effectiveness of local UV interventions: in areas with high background infection rates, students who avoided an infection in a UV-protected classroom often caught it somewhere else instead[42,43].

The technology would not see widespread revival until the late 1980s, when an unexpected rise in TB cases and the emergence of drug-resistant strains renewed interest in environmental controls for airborne infection. Thankfully, Riley had been working amidst the skepticism to refine the practice and modeling required to utilize germicidal UV effectively. Riley and colleagues conducted detailed studies of UV air disinfection in model rooms[5]. These experiments revealed critical factors affecting performance: air mixing between the lower occupied space and upper UV-treated zone proved essential, with temperature gradients and ceiling fans significantly impacting this mixing[44–46]. Their work also showed that high relative humidity reduced UV's effectiveness, with sharp declines in pathogen kill rates above 60–70% humidity[47].

Building on the Wells' earlier work, Riley developed a model to quantify the probability of airborne infection in an indoor space. The model, commonly called the Wells-Riley model, estimates infection risk based on factors such as the number of infectious individuals, room ventilation, duration of exposure, and the pathogen's transmission characteristics[48]. It remains a foundational tool for understanding airborne disease spread and evaluating mitigation strategies, including the effectiveness of UV air disinfection[49,50].

As the limitations of conventional TB control measures became clear, renewed studies on GUV demonstrated its ability to supplement other infection control strategies. Studies revisiting upper-room UV systems confirmed their efficacy in high-risk settings, such as hospitals, operating rooms, homeless shelters, and correctional facilities, led in large part by Riley and Ed Nardell, a physician at Brigham and Women's Hospital[51–57].

This period saw a shift in institutional support, with the US Centers for Disease Control and Prevention (CDC) incorporating GUV into its guidelines for TB control in healthcare settings, marking a turning point for the technology's acceptance[58]. Results of Escombe et al., 2009 confirmed that upper-room GUV radically reduced the amount of infectious TB present in the air[59].

Additional research throughout the 1990s refined GUV application, focusing on optimizing fixture design, air circulation, and safety measures to minimize UV exposure risks. Computational fluid dynamics models allowed researchers to predict airflow patterns and ensure effective disinfection while protecting room occupants. These advancements reinforced GUV as a viable intervention, particularly where mechanical ventilation improvements were cost-prohibitive. But outside of the specific use case of spaces that are high risk for TB, there was historically relatively little interest in the use of upper-room UV. To this day, the official guidance on the use of upper-room UV provided by CDC/NIOSH is explicitly for its use in controlling the spread of tuberculosis[58].

The COVID-19 pandemic, and the emergence of far-UVC

As described above, for much of the 20th and early 21st century there was skepticism that respiratory illnesses are spread by airborne transmission. During the COVID-19 pandemic, this led to slower adoption of measures widely accepted to be effective against airborne transmission of disease, including upper-room UV, ventilation, and filtration of indoor air. This has been described in detail by many of the researchers responsible for overturning the consensus during the pandemic[37,60].

Before the COVID-19 pandemic, Professor David Brenner, Director of the Center for Radiological Research (CRR) at Columbia University, was investigating shorter wavelengths of UVC for disinfection in surgical settings[61,62]. The aim was to identify new sources of UVC that could effectively kill pathogens without the risks of overexposure associated with conventional germicidal UV from low-pressure mercury vapor lamps. Brenner and his colleagues, especially Manuela Buonanno and David Welch, began exploring the potential of far-UVC, a subset of the UVC spectrum. They hypothesized that these shorter UVC wavelengths could inactivate microbes without penetrating human skin or eyes, due to their strong absorption by proteins[63].

Initial studies demonstrated that far-UVC could efficiently inactivate drug-resistant bacteria without damaging more sensitive human tissues. Early studies used krypton-bromine excimer (KrBr*) lamps emitting primarily at 207 nm, which effectively inactivated bacteria while being less harmful to human skin compared to 254-nm germicidal UVC. However, krypton chloride excimer (KrCl*) lamps, emitting primarily at 222 nm, became more widely available and offered a higher output intensity, making them more practical for real-world applications[64].

In 2018, researchers at the CRR demonstrated the first proof of concept that far-UVC could effectively inactivate viruses that cause respiratory illness, showing over 95% inactivation of aerosolized H1N1 influenza virus with low doses of 222-nm far-UVC[65,66]. This established the potential for the application of 'whole-room' far-UVC irradiation in indoor public locations.

When COVID-19 emerged in 2020, research rapidly expanded to examine both efficacy and safety. Studies at the CRR demonstrated far-UVC's effectiveness against coronaviruses, while Ewan Eadie and colleagues at the photobiology unit at Ninewells Hospital at the University of Dundee showed that properly filtered far-UVC produced no skin erythema (sunburn) even at a dose more than 500 times higher than the consensus exposure limits at the time[67,68]. The research conducted at Ninewells confirmed that filtering out longer UV wavelengths from far-UVC emitters was important—unfiltered sources could cause erythema, while properly filtered devices showed no acute effects[69].

In 2022, the first study of far-UVC at room-sized scale was published, showing remarkable efficacy against airborne bacteria[70].

However, despite the technology's promise, widespread adoption of far-UVC during the COVID-19 pandemic faced several significant barriers. Early in the pandemic, the medical community had not yet accepted airborne transmission, which meant that prevention efforts focused primarily on surface disinfection and droplet precautions rather than air disinfection technologies[37,60]. Even after airborne transmission was widely accepted, implementation guidance remained unclear for far-UVC. Unlike upper-room GUV systems, which had decades of established protocols, there was limited practical experience, no authoritative guidance, and no agreed-upon standard for assessing the safety and efficacy of whole-room far-UVC deployment.

In addition, there are not yet any large-scale clinical trials demonstrating far-UVC's real-world effectiveness at blocking human-to-human transmission. This hinders acceptance of and advocacy for the technology by many important institutions, who are understandably risk-averse at the prospect of increasing the public's exposure to any form of radiation. It is also unreasonable to expect laypeople to establish the veracity of manufacturers' claims about efficacy and safety. Many air cleaning technologies have been offered to the market that are at best ineffective and at worst actively harmful, and there is a need to produce an evidence base that secures the endorsement of safe and effective technologies from trusted institutions.

This document represents our assessment of the state of knowledge of the use of far-UVC to control the transmission of airborne disease. While there is uncertainty, as there is with any technology at this early stage, the potential for far-UVC is supported by quality research. The amount of available funding and the number of researchers, engineers and policymakers engaged with the technology is not commensurate with its tremendous potential.

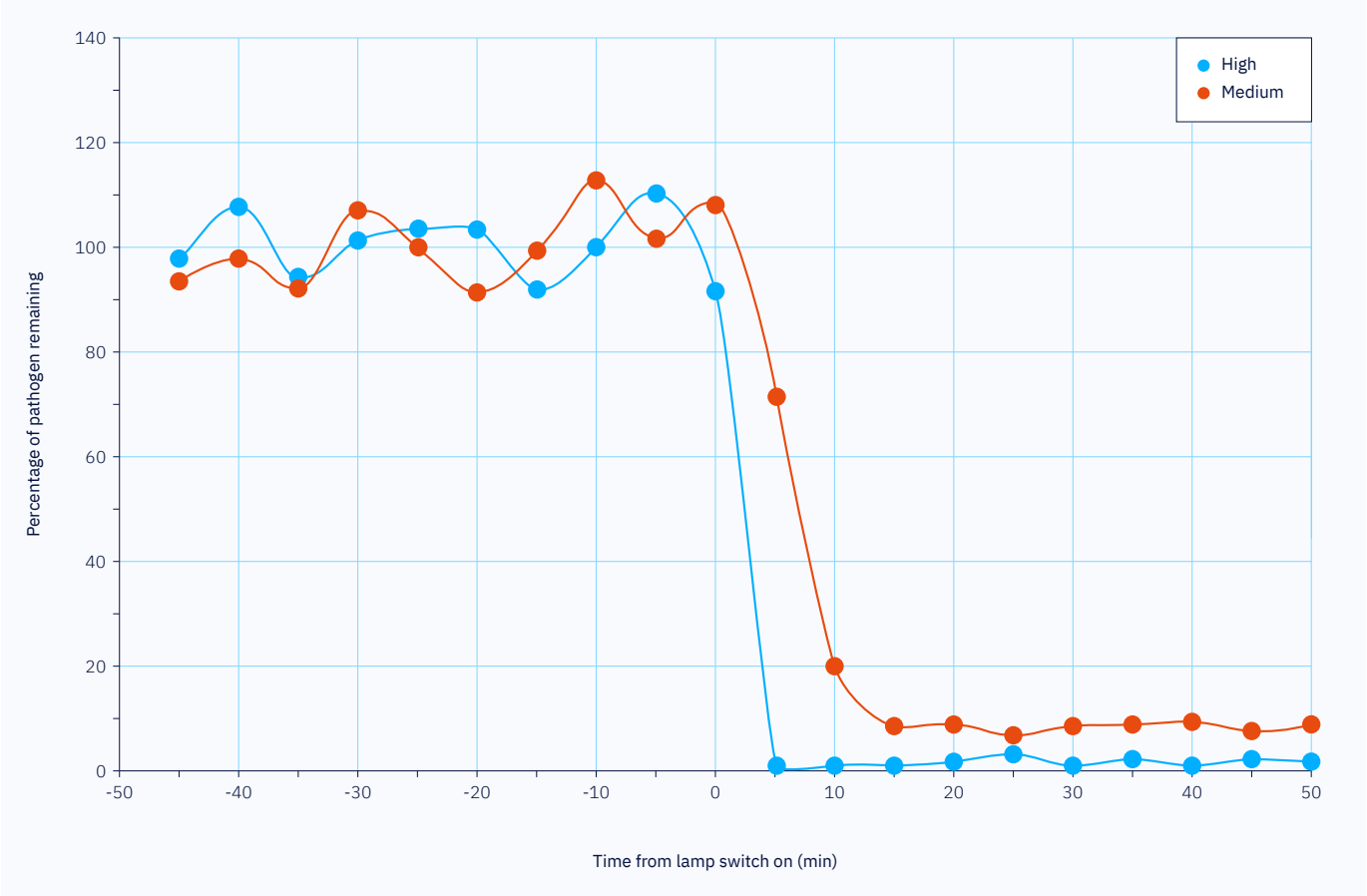

FIGURE 1.6. Far-UVC (222 nm) exposure dramatically reduced airborne *Staphylococcus aureus* in a room-sized chamber, achieving up to 98.4% pathogen reduction at exposure levels consistent with safety guidelines[70].

Further reading

- The History of Ultraviolet Germicidal Irradiation for Air Disinfection by Nicholas Reed, 2010
- Air-Borne by Carl Zimmer, 2025

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2. Far-UVC primer

This section provides foundational concepts and terminology used throughout the rest of this document. The section ensures all readers have access to the same background knowledge, but readers need not read it all at once. Readers familiar with these concepts are welcome to skip sections, or proceed directly to later sections if comfortable with UV physics and terminology and return to relevant sections when additional context would be helpful.

The electromagnetic spectrum

Radiation is energy transmitted through space as a particle or wave. The electromagnetic (EM) spectrum encompasses all energy that is transmitted in the form of electromagnetic waves. The fundamental unit of electromagnetic radiation is a photon.

Visible light is one section of the EM spectrum, although other sections of the EM spectrum are often colloquially referred to as 'light'. 'Ultraviolet light' is one such example of this, and devices that produce ultraviolet (UV) radiation are often called 'lamps'. In this document, we reserve the term 'light' to mean 'visible light' for clarity and consistency, although we do refer to 'lamps' that produce UV radiation.

Photons have characteristics of both waves and particles, depending on how they are measured (this is known as 'wave-particle duality').

The energy of a photon with wavelength λ (in meters) is given by:

Equation 2.1

$$E = \frac{hc}{\lambda}$$

where h is the Planck constant (6.626×10^{-34} Js), and c is the speed of light in a vacuum (2.99×10^{8} ms^{-1}). Note that photon energy is inversely proportional to the wavelength, so short wavelength X-ray or gamma ray photons transmit more energy than longer radio waves or microwaves. Listed here in ascending order of wavelength, the EM spectrum includes: gamma ray, X-ray, ultraviolet, visible light, infrared, microwaves, and radio waves[1]. Figure 2.1 shows the ultraviolet and visible portion of the spectrum, which is most relevant for understanding far-UVC applications.

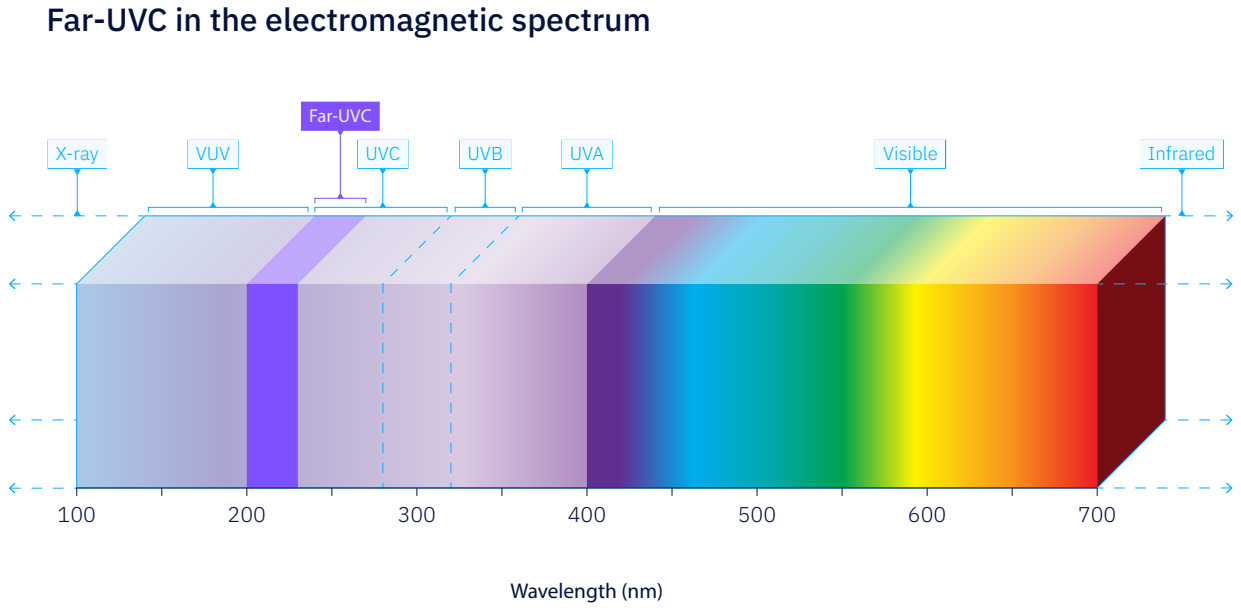

FIGURE 2.1. The electromagnetic spectrum is defined by wavelength.

Ultraviolet radiation

Many of the EM bands are well known to the general public, including visible light, microwaves, X-rays, and the focus of this section, ultraviolet (UV) radiation. UV is further divided into partially overlapping subcategories:

UVA (320–400 nm) has the longest wavelength and lowest photon energy within the UV spectrum. UVA is an important component of the sunlight that reaches the earth's surface. It penetrates more deeply into the skin than other UV subtypes. Excess UVA exposure is a risk factor for erythema (sunburn), cataracts, some forms of skin cancer, and skin aging.

UVB (280–320 nm) is shorter in wavelength, thus higher energy per photon, than UVA. Similar to UVA, it is a component of sunlight and excess exposure is strongly associated with erythema, skin cancer, cataracts, eye surface pathologies (e.g., pterygium and pinguicula), and skin aging. Moreover, it can damage cellular DNA.

UVC (200–280 nm) has shorter wavelengths and higher energy than UVA and UVB. While it is a component of solar radiation, UVC is almost fully absorbed by the Earth's stratospheric ozone layer[2]. UVC efficiently damages the DNA of microorganisms, such as bacteria and viruses. UVC lamps, and particularly mercury vapor lamps that primarily emit 254-nm UVC, have been used in various germicidal applications for over a century. 254-nm UVC penetrates skin and eyes less efficiently than UVB, although 254 nm exposure limits are not significantly different to 280–300-nm UVB.

Far-UVC (200–235 nm) is a subset of UVC that penetrates human skin and eyes less as compared to longer UVC wavelengths. This results in lower risk of tissue damage and thereby allows for higher human exposure limits (see *Skin and eye safety* section)[3,4].

Vacuum ultraviolet (VUV, definitions range from 10 to ~120 nm at the low end to 180–200 nm at the high end) can be considered a subset of UVC, or considered its own section of the EM spectrum. At wavelengths below 200 nm, there is a marked increase in the absorption of photons by O_2 molecules, resulting in significant ozone (O_3) generation. VUV is so named because it can only propagate effectively in a vacuum or in an environment free of oxygen (such as in a controlled gas like argon): in environments with oxygen the photons are quickly absorbed. VUV is not used for disinfection in occupied spaces due to this absorption by oxygen and generation of ozone, but it has niche applications in industrial and laboratory settings including municipal wastewater treatment[5].

Extreme ultraviolet (EUV, 10–121 nm) can be considered as a separate section, or it can be seen as overlapping with VUV, or as a subset of VUV. It is used in industrial applications, for example in semiconductor manufacturing.

Ionizing and non-ionizing radiation

Radiation is conventionally categorized into ionizing and non-ionizing radiation. This categorization has significant implications for how the risks of radiation exposure are managed, and how they are understood by the general public.

Ionizing radiation consists of EM waves with much higher photon energies than far-UVC, such as extreme UV, X-rays and gamma rays, as well as alpha particles, beta particles and neutrons emitted by atoms undergoing radioactive decay.

Ionizing radiation is so called because it has sufficient energy to remove tightly bound electrons from atoms, creating positively charged ions. These ions can directly interact with and damage essential structures in cells such as DNA, or they can result in free radical production that indirectly damages DNA or other cellular structures. Ionizing radiation exposure is strongly associated with DNA mutation, which can lead to cancer, or at high doses can cause cell death[6,7]. Because of these risks, exposure to ionizing radiation is carefully regulated and controlled in medical and industrial settings.

EM waves with lower energy levels, such as radio waves, microwaves, infrared, visible light, UVA, UVB, and UVC are classified as non-ionizing radiation. While non-ionizing EM photons do not have enough energy to ionize most atoms, they can still cause biological effects, for example through heating or photochemical reactions.

While categorized as non-ionizing radiation, far-UVC photons do have sufficient energy to ionize some atoms. For example, a 222-nm far-UVC photon has 5.58 electron volts (eV) of energy, which is sufficient to ionize alkali metals, and photons in the UVB range are capable of ionizing caesium[8]. In this sense, far-UVC radiation can be 'ionizing' by a literal definition of the term. However, due to its inability to ionize atoms typically found in biological systems, far-UVC is classified as non-ionizing radiation.

Key definitions

TABLE 2.1. Key units and definitions.

Concept	Definition[9]	Unit
Radiant energy	The total energy emitted, transferred, or received as radiation in a defined period of time.	Joules (J)
Radiant power (or **radiant flux**)	**Radiant energy** emitted, transferred, or received per unit of time.	Watts (W) or J/s
Irradiance	**Radiant power** incident from all upward directions on a small surface divided by the area of the surface.	W/m²
Fluence rate (or **spherical irradiance**)	**Radiant power** incident from all directions onto a small sphere divided by the cross-sectional area of that sphere.	W/m²
Fluence (or **radiant exposure**)	**Radiant energy** incident from all directions on a small sphere divided by the cross-sectional area of that sphere.	J/m²
Dose (informal)	**Fluence** (or equivalent when considering **irradiance** rather than **fluence rate**).	J/m²

Irradiance and **fluence rate** both measure the amount of **radiant power** over a small area. As such, they are typically used to measure how much far-UVC radiation a person or space is exposed to. The difference is illustrated in Figure 2.2 below—**fluence rate** encompasses **radiant power** emitted onto the area from all directions, whereas **irradiance** includes only **radiant power** from one direction above a surface (for example, from the sun to the surface of the earth).

For UV disinfection of air and water, **fluence rate** is generally the applicable concept. For human skin and eye exposure, and for disinfection of surfaces, the applicable concept is generally **irradiance**.

In the most general terms, energy is the integral of power over time. Thus, **fluence** is the integral of **fluence rate** over time. In the simplest case, exposure to a uniform **fluence rate** of 1 W/m² means that every second the fluence delivered is 1 J/m².

Research and guidance on the use and safety of germicidal UV often refers to the concept of **dose**. In the context of UV air disinfection, **dose** is based on **fluence rate** and has the same definition as **fluence** or **radiant exposure**. In the context of assessing UV exposure to human skin and eyes, **dose** is based on **irradiance**.

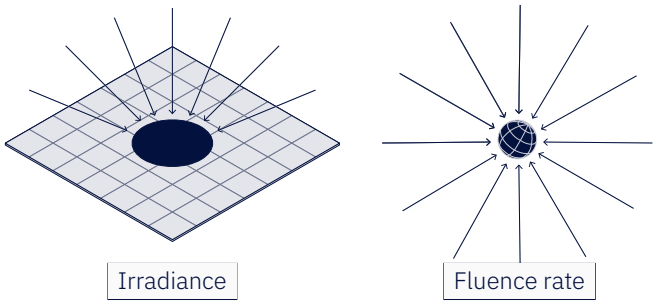

FIGURE 2.2. **Irradiance** versus **fluence rate**. Source: Ultraviolet Germicidal Irradiation Handbook[5].

Angular dependence

An important concept in electromagnetic radiation is angular dependence, which we experience in our everyday exposure to solar UV. Radiation has direction, and when the angle of incidence is perpendicular to the surface of an object the **radiant power** is spread over the smallest possible surface area. As the angle of incidence departs from the perpendicular, the same **radiant power** is spread over a larger surface area, so each part of that surface receives less radiation.

Angular dependence is mathematically described by Lambert's cosine law, meaning **radiant power** is proportional to the cosine of the incident angle on the surface.

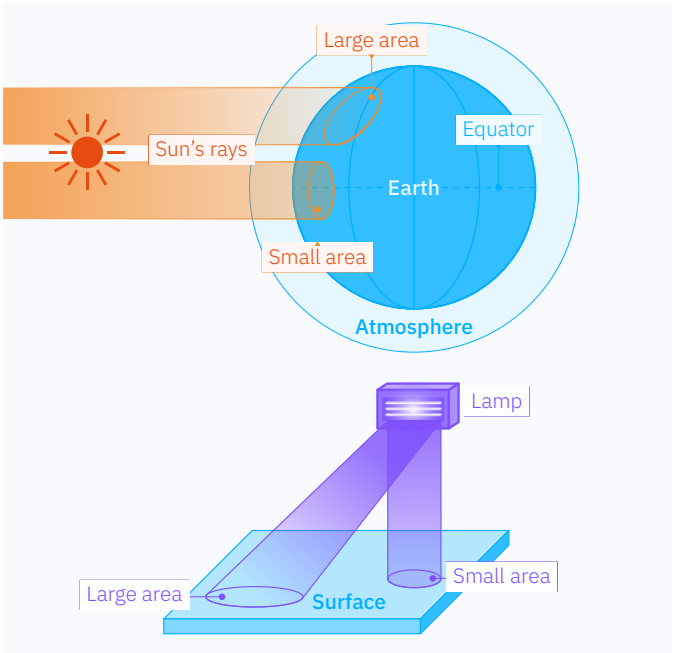

FIGURE 2.3. Illustration of angular dependence. Source: Peter Halasz on Wikipedia, licensed under CC BY-SA 2.5[10].

Inverse square law

The inverse square law describes the relationship between **fluence rate** (or **irradiance**) and distance from an ideal point source in a system where no UV-absorbing compounds are present. An ideal point source is one that is assumed to emit photons uniformly in all directions. For this source type, **fluence rate** will decrease with the square of distance from the source. In other words, when an object's distance from the radiation source is doubled, the **fluence rate** reduces to ¼, and so forth as illustrated in Figure 2.4.

Because air is a weak absorber of UVC radiation, the inverse square law provides a rough understanding of the spatial distribution of **fluence rate** as related to UVC source location, even though real UVC sources are not ideal point sources. The practical import of the inverse square law is that **fluence rates** are significantly higher closer to the source, so that (for example) the areas directly next to a UVC lamp will have much higher **fluence rates** than those further away.

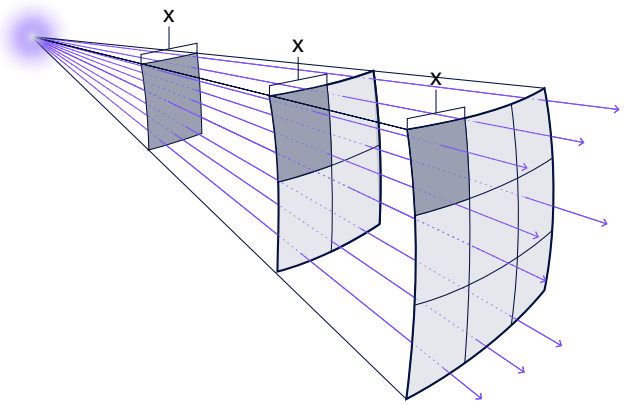

FIGURE 2.4. Inverse square law. The **fluence rate**—represented pictorially by the density of the rays emitted from the point source—decreases with the square of the distance from the UV source. Source: Borb on Wikipedia, licensed under CC BY-SA 3.0[11].

Chromophores

A chromophore is any molecule, or part of a molecule, that absorbs a specific wavelength of electromagnetic radiation. For example, O_2 is a chromophore of UV radiation of wavelengths less than 242 nm, whereas food colorings contain chromophores of visible light. An important chromophore for far-UVC is the peptide bonds that link amino acids to form proteins[12], and there are many UV chromophores in commonly used materials (see *UVC impact on materials* section).

Action spectra

An action spectrum describes the relative effectiveness of different wavelengths of radiation at producing a specific biological effect, such as DNA damage, pathogen inactivation, or sunburn. In the context of UV radiation, the action spectrum is crucial, because various wavelengths are differentially absorbed by tissue, cells, and cellular components such as DNA, proteins, and lipids. An action spectrum is determined through experimental studies that measure a biological response to various wavelengths of UV radiation, ideally using tunable, (nearly) monochromatic sources of radiation with a single wavelength.

Action spectra help us understand how different wavelengths of radiation affect biological systems, including human skin, cell types, or tissues, as well as microbial pathogens. For example, Figures 2.5 and 2.6 show the action spectra for the propensity of proteins and nucleic acids to absorb photons of different wavelengths. Higher relative absorption means that it is more likely that a photon will interact with a molecule and produce a photochemical and/or photobiological effect.

The protein and nucleic acid absorbance spectra show that absorption properties can be highly nonlinear, and are not simply a function of wavelength and photon energy. When we then consider complex phenomena like skin cancer, the action spectra can be non-intuitive. For example, the action spectrum for non-melanoma skin cancer peaks sharply in the UVB range[3].

The cancer risk of UVB is orders of magnitude higher than that of UVC and UVA. This is due to the interaction of certain properties of UVB (penetration depth, propensity to damage DNA) with properties of the body (skin structure and the photon absorption properties of nucleic acids and proteins) (see *Evaluating cancer risk* section).

It is therefore not the case that we can use simple heuristics like "higher energy photons are always more dangerous," or "lower energy photons are less effective at killing pathogens." It depends on a combination of complex photochemical properties of particular macromolecules in specific cells, tissues, and complex biological systems.

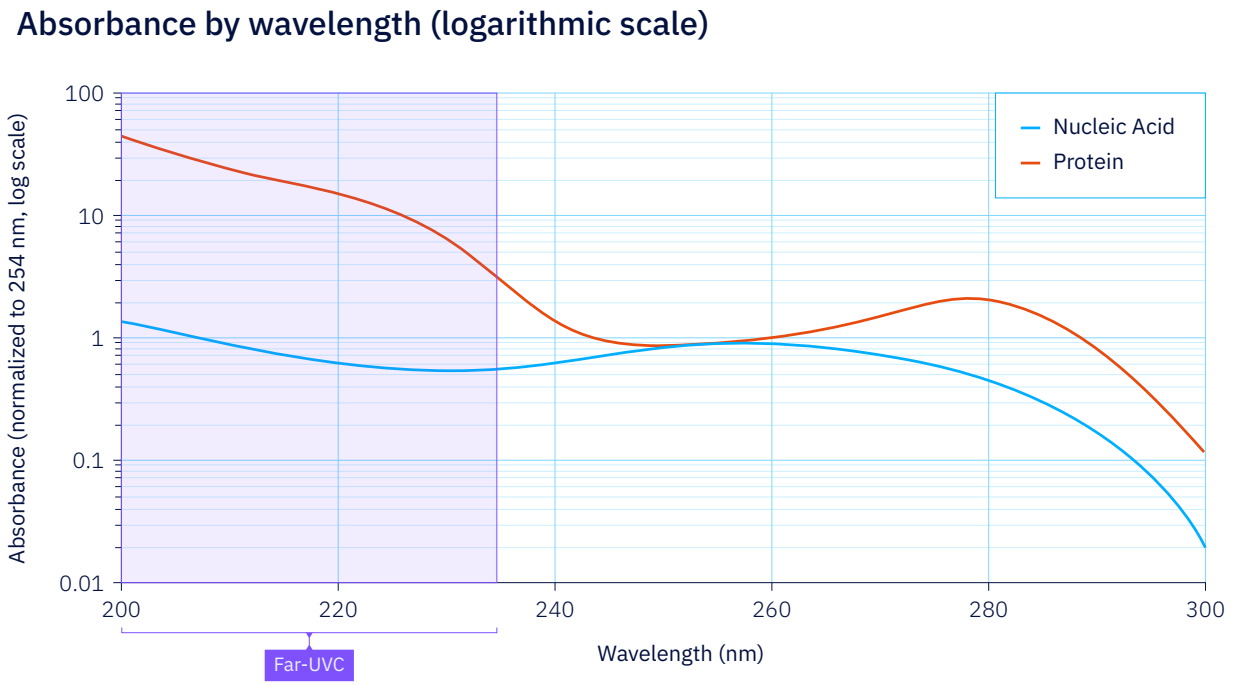

FIGURE 2.5. Excerpted from IUVA, 2021[1]; original sources Setlow and Doyle, 1957[13] and Voet et al., 1963[14]. Nucleic acid and protein absorbance spectra by wavelength, normalized to 254 nm on a logarithmic vertical scale.

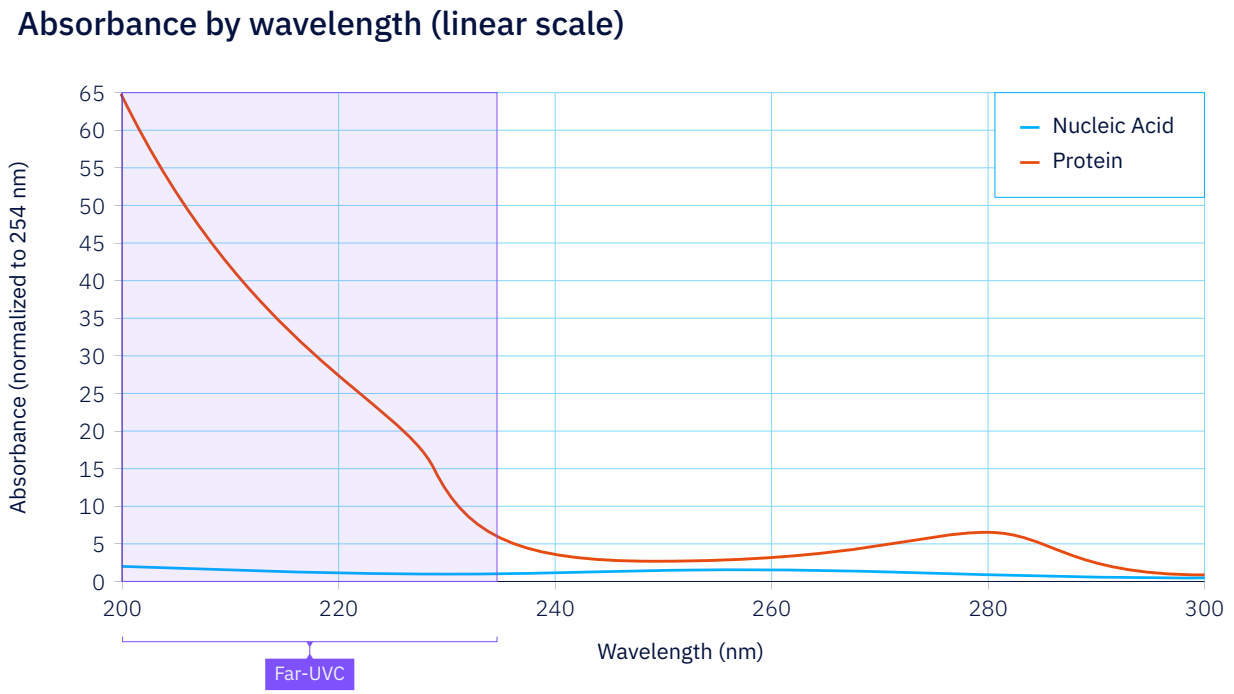

FIGURE 2.6. Excerpted from IUVA, 2021[1]; original sources Setlow and Doyle, 1957[13] and Voet et al., 1963[14]. Nucleic acid and protein absorbance spectra by wavelength, normalized to 254 nm on a linear vertical scale.

UV exposure limits

Exposure limits for UV radiation are based on action spectra. These action spectra are provided by organizations such as the American Conference of Governmental Industrial Hygienists (ACGIH) and the International Commission on Non-Ionizing Radiation Protection (ICNIRP). These action spectra were originally established in the 1970s, although ACGIH updated theirs in 2022 based on recent research into far-UVC skin and eye safety[15].

The ACGIH exposure limits are called Threshold Limit Values (TLVs). ICNIRP exposure limits are sometimes colloquially referred to as 'TLVs', but this is a trademarked term by ACGIH for all of their recommendations across industrial hygiene, not just UV exposure limits.

Both ICNIRP and ACGIH recommend a *spectrally weighted* UV exposure limit of 3 mJ/cm² over 8 hours in a 24-hour period. Each wavelength is assigned a spectral effectiveness value S(λ) that reflects its relative biological impact. The actual (unweighted) UV dose at each wavelength is multiplied by its S(λ) value, and these weighted doses are summed to calculate the total spectrally weighted dose. In practice, this means that wavelengths considered to be more hazardous count more towards the exposure limit. This total spectrally weighted dose must remain below 3 mJ/cm² to stay within the exposure limit.

As with other action spectra, S(λ) is conventionally presented in a normalized form such that the maximum value (1) is observed at the wavelength that has the greatest potential to cause the relevant effect. For example, the ACGIH Eye and ICNIRP values of S(λ) are based on the photokeratitis (inflammation of the cornea) action spectrum. A higher S(λ) means that UV at that wavelength contributes relatively more towards the 3 mJ/cm² weighted exposure limit.

As can be seen from Figures 2.7 and 2.8, S(λ) for different UVC wavelengths ranges across more than 1.5 orders of magnitude according to ICNIRP, and more than 2.5 orders of magnitude according to ACGIH. Some UVC wavelengths therefore contribute dramatically more towards the 3 mJ/cm² limit than others, with far-UVC wavelengths contributing the least.

This dynamic can be illustrated in the exposure limits for the most widely-used far-UVC source, a krypton chloride excimer (KrCl*) lamp. These lamps produce predominantly, *but not exclusively*, UVC with a wavelength of 222 nm. For some optically-filtered KrCl* lamps, the exposure limit may be close to a hypothetical source that emitted only 222 nm. However, for others—especially unfiltered or poorly filtered lamps—the exposure limit can vary significantly, due to the fact that they also emit higher wavelengths with a much larger S(λ).

Though these *unweighted* emission spectra appear similar on a linear scale, even a small emission at (for example) 259 nm in the case of the unfiltered lamp has a profound effect on the spectrally weighted exposure. This is because relative spectral effectiveness of 259 nm, according to the ACGIH Eye S(λ) as displayed in Figure 2.7, is over 50 times higher than that of 222 nm, and therefore exposure to the same dose of 259-nm radiation counts 50 times more towards an individual's exposure limit. If a person was subject to the same irradiance from each of these two lamps, they would exceed exposure limits for the unfiltered lamp 3 times faster than for the filtered lamp.

While far-UVC has lower spectral effectiveness than higher wavelength UVC, and therefore higher safe exposure limits, it is nevertheless still crucial to adhere to these exposure limits. It is remaining within exposure limits that makes any form of electromagnetic radiation safe, rather than any wavelength being universally safe or unsafe.

What makes far-UVC promising is that studies have demonstrated significant pathogen inactivation while remaining well within human exposure limits. This means a dose that is safe for human exposure can still effectively reduce airborne infectious particles (explored further in the *Efficacy* section).

Exposure guidance: ACGIH and ICNIRP (logarithmic scale)

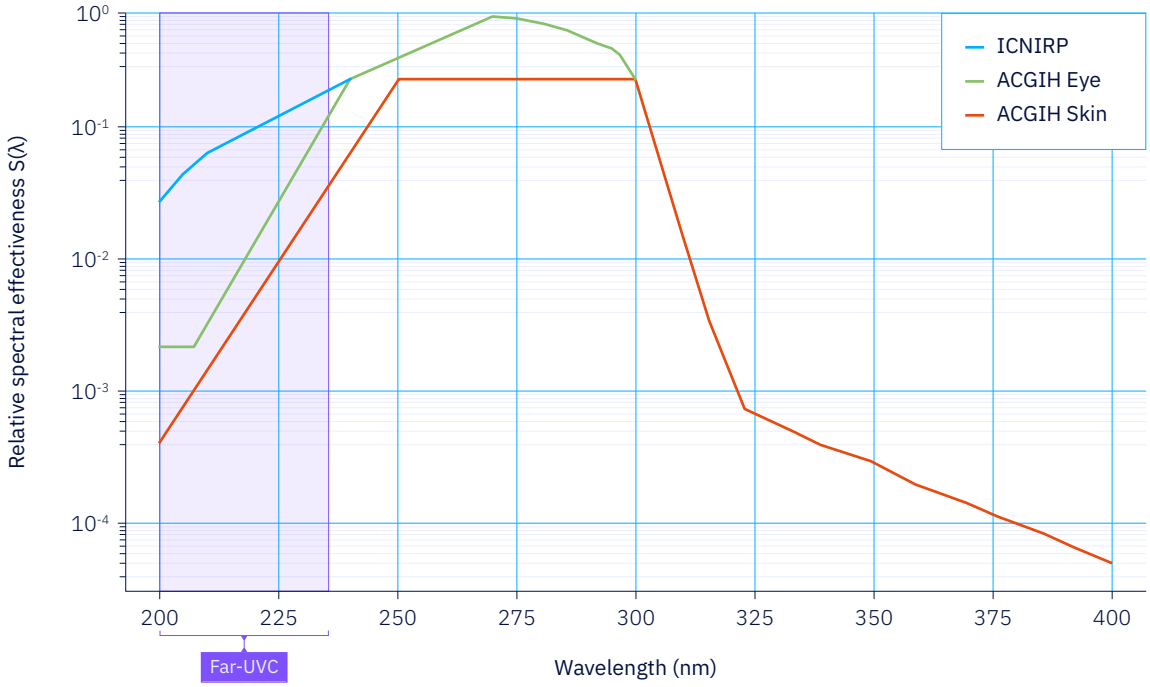

FIGURE 2.7. Excerpted from Görlitz et al., 2024. ACGIH and ICNIRP relative spectral effectiveness by wavelength on logarithmic vertical scales[3].

Exposure guidance: ACGIH and ICNIRP (linear scale)

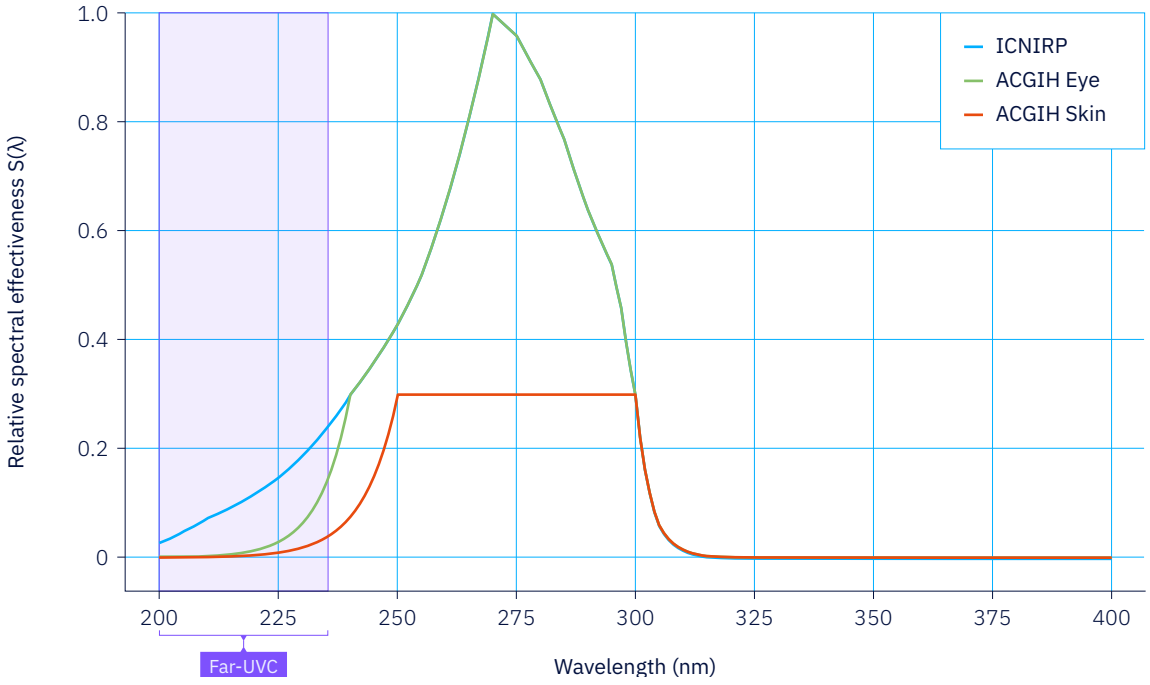

FIGURE 2.8. Excerpted from Görlitz et al., 2024. ACGIH and ICNIRP relative spectral effectiveness by wavelength on a linear vertical scale[3].

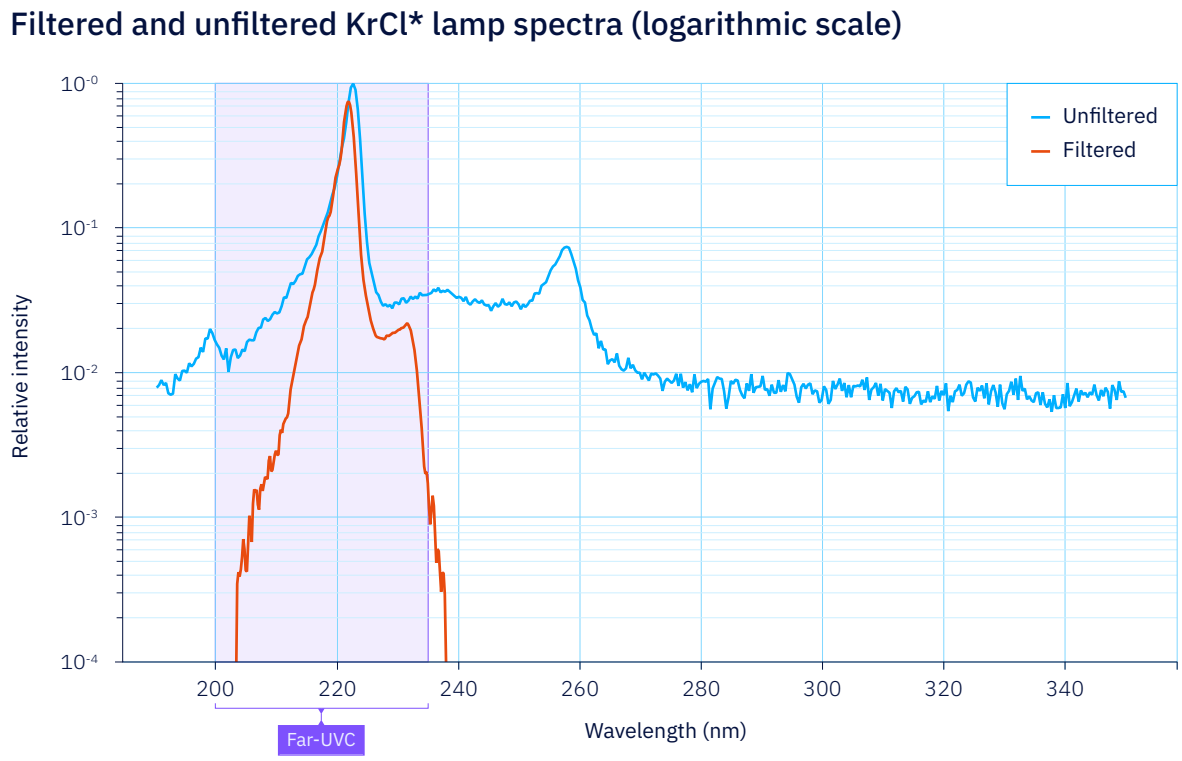

FIGURE 2.9. Comparison of the weighted and unweighted relative intensity emitted by two different far-UVC fixtures, logarithmic scale. Data from the OSLUV Project[16].

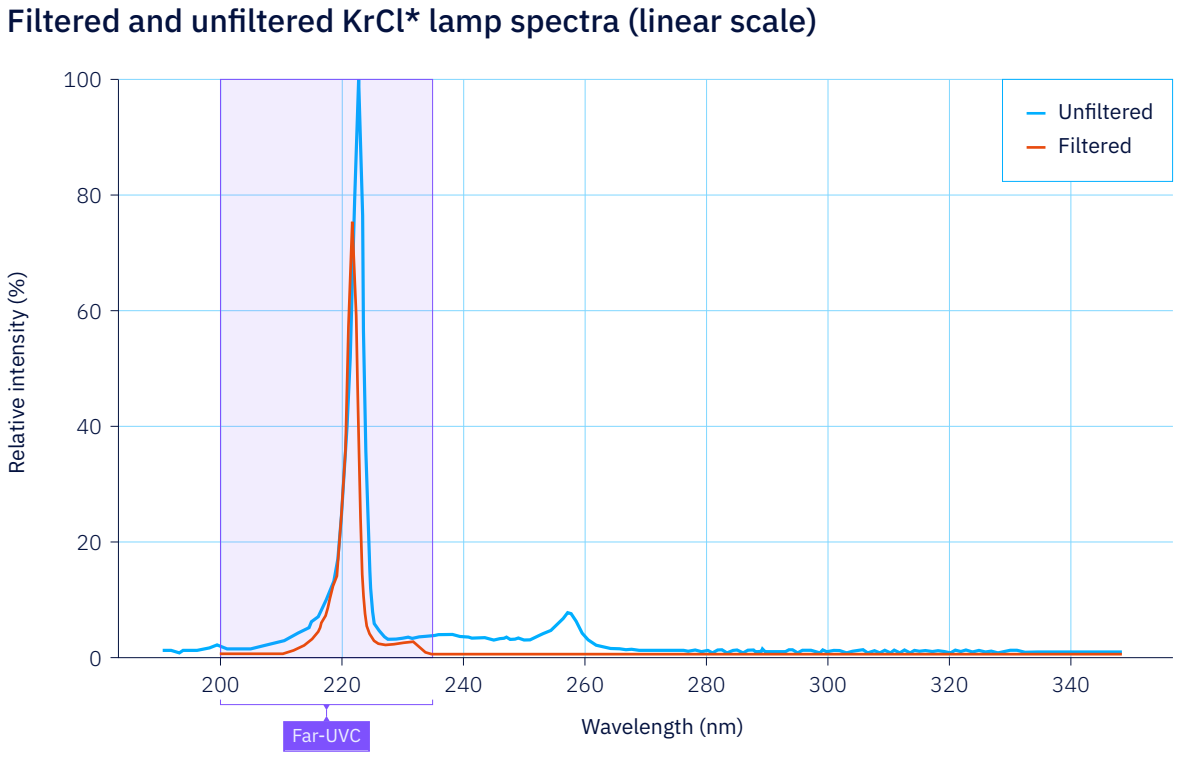

FIGURE 2.10. Comparison of the weighted and unweighted relative intensity emitted by two different far-UVC fixtures, linear scale. Data from the OSLUV Project[16].

Germicidal UV applications

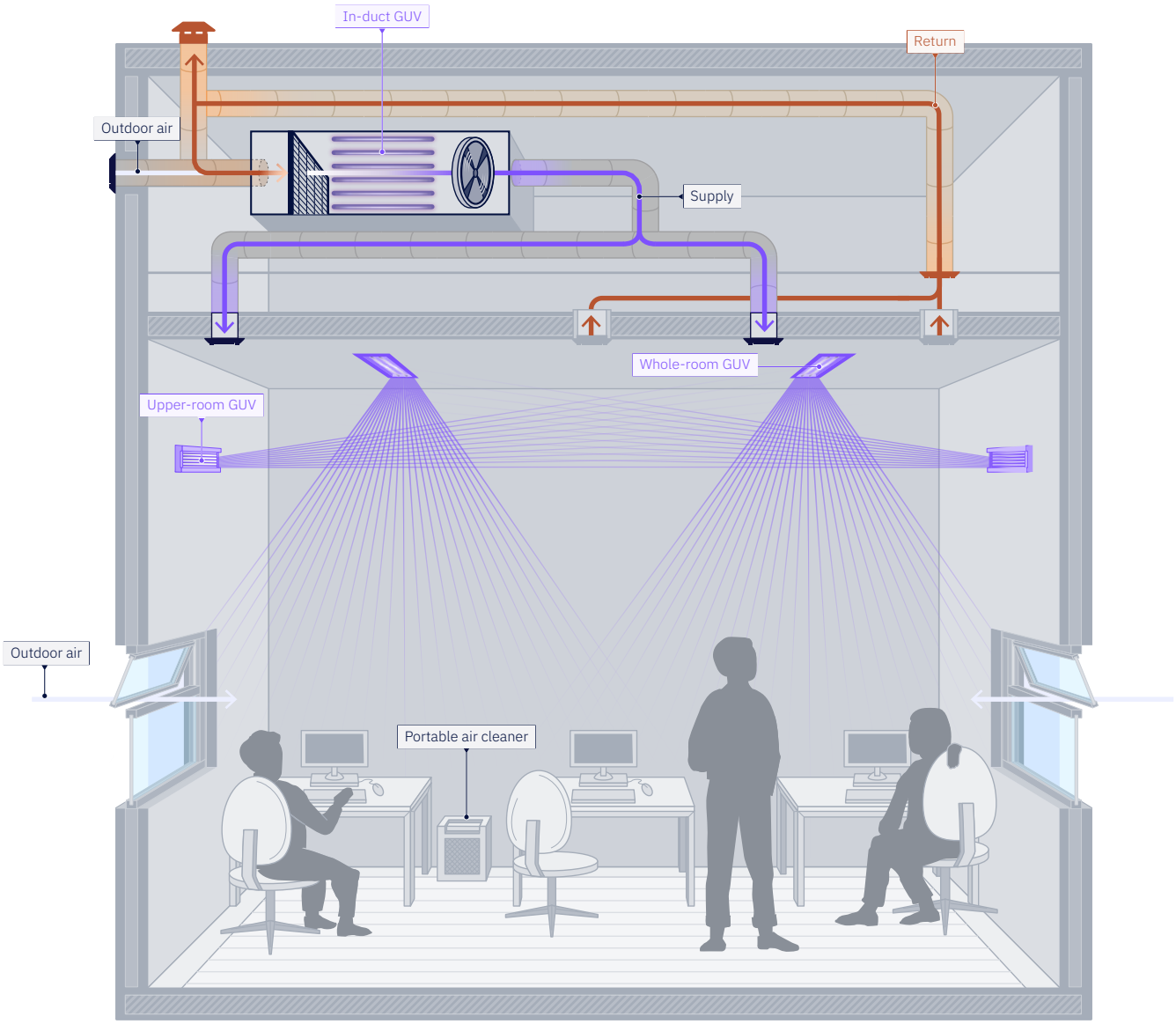

FIGURE 2.11. Strategies for disinfecting indoor air with GUV. Figure by Blueprint Biosecurity.

There are three primary strategies for deploying germicidal ultraviolet radiation (GUV) which are outlined in Figure 2.11. These are upper-room GUV, in-duct GUV, and whole-room GUV.

Upper-room GUV fixtures are mounted high on walls or ceilings. They direct UVC radiation into the upper portion of the room, away from occupants. Air circulation within the room—generated from natural convection, HVAC, or fans—moves air between the lower occupied space and the irradiated zone in the upper room. Upper-room GUV must be carefully designed and installed by professionals to ensure safety for occupants.

Whole-room GUV systems disperse UV throughout the entire space, providing continuous disinfection of air and surfaces in occupied areas. Far-UVC is the only form of UV thought to be viable for whole-room disinfection of occupied spaces, due to the higher exposure limits recommended by ACGIH and ICNIRP.

In-duct GUV systems are installed within HVAC systems to disinfect recirculating air, thus reducing the concentration of airborne pathogens in a room through dilution. In-duct systems are conceptually similar to outdoor air ventilation and filtration of recirculating air.

Effectiveness, efficacy, and susceptibility

Finally, there are three different concepts that need to be distinguished when discussing how well far-UVC works.

Effectiveness refers to how well far-UVC reduces disease transmission in real-world settings. For example, if far-UVC were installed in a school classroom or an urgent care waiting room, how many illnesses would it prevent?

Efficacy refers to how well far-UVC inactivates pathogens under controlled conditions. This is typically measured as a reduction (or log reduction) in pathogen concentration compared to controls. In the context of air disinfection it can be expressed in terms of equivalent air changes per hour (eACH) or clean air delivery rate (CADR).

Susceptibility, typically expressed as a k or z value, describes how sensitive a particular pathogen is to far-UVC exposure. It is measured by the pathogen's dose-response relationship to UV and can vary significantly between different pathogens and in different experimental conditions.

For more discussion on these topics, see the *Efficacy* section.

Further reading

- Far UV-C Radiation: Current State-of Knowledge
- Ultraviolet Germicidal Irradiation Handbook: UVGI for Air and Surface Disinfection
- Far UV-C radiation: An emerging tool for pandemic control

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3. Efficacy

Summary

Far-UVC can inactivate a very wide range of pathogens. Some important pathogens (such as coronaviruses) appear to be especially susceptible. But the critical question is not whether far-UVC inactivates pathogens, but *how well*; in other words, *how much* far-UVC do we need to render an occupied indoor space safe.

This section provides an analytical framework and explains the evidence for far-UVC's promise as an air cleaning technology. The most widely quoted study is Eadie et al., 2022[1], where a realistic use of far-UVC reduced airborne bacteria by 98.4% in a room-sized chamber. This section highlights the wider evidence base and the importance of gaining a better understanding of things we're currently uncertain about, as well as the implications of these uncertainties on the practical application and overall promise of far-UVC for suppressing airborne transmission.

Clinical trials studying how many airborne infections can be prevented by environmental or 'place-based' interventions like far-UVC are difficult to adequately power, due to challenges associated with studying places rather than people and transmission rather than illness. This section explains those challenges and introduces an alternative engineering approach that has been developed to quantify how well air cleaning technologies work, placing the evidence for far-UVC's efficacy in the context of that engineering framework.

We do not believe that an engineering and modeling approach is a complete substitute for demonstrating effectiveness through clinical trials, and the challenges associated with these need to be overcome. However, the engineering approach is necessary to design effective far-UVC installations in a wide range of environments, as would need to be done for a clinical trial, and also to interpret and extrapolate the results of any clinical trial to different environments.

Crucial considerations

- From experimental data to date, there is order of magnitude variation in susceptibility to far-UVC of different aerosolized pathogens and in different experiments.
- If the higher estimates of pathogen susceptibility are true, far-UVC may compare favorably to existing technological approaches on cost, energy efficiency, and the total amount of achievable disinfection.
- However, if the lower estimates of pathogen susceptibility are correct, then far-UVC compares less favorably to existing technological approaches such as ventilation and filtration, and practical application will be more challenging, as larger doses of far-UVC would be required to be efficacious.
- Most research into far-UVC germicidal efficacy to date has been conducted in pathogens aerosolized in media that are importantly different to human respiratory aerosols. In particular, we'd expect the protein content of human respiratory aerosols to attenuate the efficacy of far-UVC to some degree relative to a water-based medium, but the extent of this is currently unknown.
- Human-to-animal studies are one option with historical precedent for studying efficacy of far-UVC against pathogens contained in respiratory aerosols.
- Assumptions about air mixing and the impact of ventilation regimes are likely to affect how we translate experimental findings to practical applications, and more research is needed in this area.
- While this report is primarily focused on long-range airborne disease transmission, if far-UVC efficacy for short-range transmission proves favorable, the value of far-UVC potentially increases significantly.
- Resolving these uncertainties is critical to producing practical deployment guidance

Analysis

Key concepts

Disease transmission

We can broadly categorize respiratory disease transmission into three modes: **long-range airborne**, **short-range airborne**, and **droplet transmission**. Diseases can be primarily transmitted through one of these modes or through multiple modes, and the precise boundaries between these modes of transmission are an area of active research and debate[2].

Long-range airborne transmission occurs when pathogens are mixed into and diluted by the air in a space. In this scenario, viable pathogens emitted by an infectious person disperse throughout the space and become relatively evenly distributed due to air currents and mixing. The time between emission and potential inhalation by others is typically minutes or longer. To model transmission risk in these cases, we use the average concentration of pathogen in the room air based on an assumed even distribution.

Short-range airborne transmission occurs when someone inhales a concentrated plume of respiratory aerosols directly from an infectious person's exhaled breath, cough, or sneeze before it has been substantially diluted by room air. Rather than being characterized by average room concentrations, this form of transmission involves exposure to localized high concentrations of pathogens over very short time periods (seconds). A common example would be being downwind of someone's cough or in their exhaled breath plume.

Speaking, coughing, or sneezing generates droplets and disperses them into the air. **Droplet transmission** traditionally refers to larger respiratory particles that travel ballistically (like tiny projectiles) rather than floating in air currents, typically depositing within 1–2 meters. However, the distinction between 'droplet' and 'airborne' is increasingly recognized as oversimplified, with particle size, environmental conditions, and air flows all affecting how long particles remain airborne.

The dominant mode of transmission in any given scenario depends on multiple environmental and built environment factors including ventilation design, air mixing, room layout, and occupant density. Consider two illustrative examples: in a classroom with ceiling fans providing good air mixing but minimal ventilation (low air changes per hour), one student's sneeze will initially create a concentrated plume that could cause short-range transmission to nearby students. However, over minutes those particles will become mixed throughout the room air, transitioning to a relatively uniform concentration that poses a long-range transmission risk to all occupants. In contrast, in a space with a strong directional ventilation system that quickly removes air from the space, someone's sneeze might create a concentrated plume that is carried by the air flow toward anyone downwind before being removed by the ventilation system. In this case, the short-range transmission risk to people in the path of the ventilated plume may dominate over long-range transmission risk from well-mixed room air.

In this section, we focus primarily on analyzing far-UVC's potential effectiveness against long-range airborne transmission, where we can reasonably model pathogen concentrations and decay rates averaged across a space. The potential effectiveness against short-range transmission, where pathogens may have minimal exposure time to far-UVC before being inhaled, requires different analytical approaches (see for example Henriques et al., 2025 which compares short- and long-range transmission[3]).

Pathogen decay

The rate of exponential decay is defined in the equation

Equation 3.1

$$N_t = N_0 e^{-\lambda t}$$

with N_t = quantity at time t
N_0 = starting quantity
λ = rate of decay per unit time
t = time

Or if we express the fraction of a population that survives (S)= N_t/N_0 after time t

Equation 3.2

$$S = e^{-\lambda t}$$

This gives the remaining fraction of a population (such as a pathogen) after time t. There are a number of potential sources of decay of viable airborne pathogens in the absence of deliberate attempts to control it–for example, the deposition rate at which aerosol particles hit a surface or fall to the floor, as well as environmental influences like temperature, pH, and relative humidity[4].

Where there are two or more sources of decay, these are additive to the total rate of decay. For example:

$\lambda_{deposition}$ = decay due to deposition

$\lambda_{environment}$ = decay due to environment

λ_n = decay constant due to n

λ_{sum} = total effect of all sources of decay

$\lambda_{deposition} + \lambda_{environment} + \ldots \lambda_n = \lambda_{sum}$

Steady-state concentration

In a scenario where there is a contaminant that is being emitted into a space at a constant rate, the average concentration of the contaminant in a space will tend towards a steady state. This steady state is simply the rate of pathogen emission divided by the total decay, where both the pathogen generation and the decay constant have the same unit of time. The steady-state concentration C_{ss} is calculated as:

Equation 3.3

$$C_{ss} = \frac{E}{\lambda_{sum} \cdot V}$$

with E = emission rate (quantity of contaminant emitted hr^{-1})
λ_{sum} = starting sum of all sources of decay (hr^{-1})
V = volume of space

Therefore, if we know the change in C_{ss} when there is no change in emission rate, we can calculate the relative change in λ_{sum}. Equation 3.3 shows that every halving of C_{ss} requires λ_{sum} to double, and that getting additional decreases in C_{ss} requires higher and higher λ_{sum}.

Reduction in C_{ss}	Required change in λ_{sum}
50%	2x
75%	4x
90%	10x
95%	20x
99%	100x

Air changes per hour

Air changes per hour (ACH) is a simple calculation of how many times per hour the entire volume of air in a given space is replaced. It is defined as[5]:

Equation 3.4

$$\mathrm{ACH} = \frac{Q}{V}$$

with Q = volumetric air flow rate per hour
V = room volume

Note that to use this equation correctly, the unit of Q needs to be flow rate per hour and V needs to be a volume in the same unit in which the flow rate is calculated. If for example the volumetric flow rate is given in cubic feet per minute (cfm) and the room volume in cubic feet, Q needs to be multiplied by 60 to get ACH. If instead the unit of Q was liters per second (L/s) and the room volume in cubic meters, it needs to be multiplied by 3.6.

If the air in a room is assumed to be 'well-mixed', then the ACH is a decay constant (λ_{ACH}) with respect to the concentration of a contaminant in the air[6].

This is not an empirical observation, and 'well-mixed' has a somewhat circular definition—a room is well-mixed if the ACH predicts the decay. In the case of a room being well-mixed with respect to the average concentration of a contaminant where the source is in the room, the well-mixed assumption requires only that the air leaving the room has the same average contents as the entire room. If the source of the contaminant was in the air supply, a sufficient definition of well-mixed with respect to the average concentration of contaminant would be that the concentration in the air outlet is the same as the air inlet.

We may also care about whether particular parts of a room are well-mixed, such as the 'breathing zone', which is defined in ASHRAE (American Society of Heating, Refrigerating and Air Conditioning Engineers) Standard 62.1 as the "region within an occupied space between planes 3 and 72 in. above the floor and more than 2 ft (600 mm) from the walls or fixed air-conditioning equipment."[7] Depending on the particular ventilation design of the room, the breathing zone could be well-mixed but other parts of the room not, and vice versa. But the important thing to understand with respect to air mixing is whether or not a certain amount of airflow per hour can be treated as a decay constant, or if not, what correction needs to be applied to estimate the rate of decay.

eACH and CADR

An equivalent air change per hour (eACH) can be defined as anything that has the same effect as an air change per hour (ACH). Far-UVC, for example, can be said to have 1 eACH if it reduces pathogen concentration by the same amount as 1 ACH in a well-mixed room. This simply means that, mathematically, an eACH is an exponential decay constant (λ_{eACH}) where the unit is hr.

Clean air delivery rate (CADR) is equivalent to Q in equation 3.4 that defines ACH:

Equation 3.5

$$\mathrm{eACH} = \frac{\mathrm{CADR}}{V}$$

As for equation 3.4, attention needs to be paid to the units of CADR and volume when making calculations.

There are two important things to note about the difference between ACH and eACH. First, ACH is assumed to be contaminant-invariant (i.e. all contaminants are assumed to be subject to the same air mixing, and thus decay at the same rate). eACH and CADR, however, are not contaminant-invariant.

Consider gaseous versus aerosol contaminants. An in-room mechanical HEPA filter will have an eACH and CADR versus aerosol particles, but in the absence of an additional filter type (such as activated carbon) it will have little or no effect on the gas. And as shall be seen later, in the case of far-UVC the eACH and CADR will be different with respect to different pathogens.

Second, once the 'well-mixed' assumption is relaxed, ACH is no longer equivalent to eACH. The well-mixed assumption is necessary for Q/V to be treated as a decay constant with respect to the average concentration of a contaminant.

A common practice among ventilation practitioners is to use a 'mixing factor' (sometimes referred to as 'K') to discount ACH to estimate an 'effective' ACH (i.e. what is the decay per hour, which is the same thing as this definition of an equivalent ACH).

There is very little in the recent published literature on mixing factors for ventilation, and references to it are more typically found in manuals for industrial ventilation practice or in standards such as ASHRAE 62.1 which describes the concept of Zone Air Distribution Effectiveness (E_z). E_z is the degree to which ACH should be discounted specifically with regards to contaminants in the breathing zone[7].

Reda et al., 2023[8] surveyed the literature and concluded that mixing factors fail to accurately estimate air change rates:

> "Given the difficulty of the topic, such mixing models (K and Ez) are calculated assuming the uniform mixing and reported in a rather subjective manner according to professional experience and the rule of thumb. Meanwhile, the existing data are rough estimates. Due to these limitations, these factors (K and Ez) fail to accurately estimate air change rates and thereby improve building ventilation performance."

Further research into accurate modeling of ventilation would greatly improve our understanding of the relative effectiveness of ventilation and other interventions such as far-UVC.

Key takeaways

- Airborne pathogen viability decays due to many factors including deposition, environmental factors, ventilation, and the use of air cleaning technologies.
- When a contaminant is emitted at a constant rate, its steady-state concentration depends on how quickly it is removed from the air.
- Doubling decay rate cuts steady-state pathogen concentration in half, therefore achieving greater reductions requires greater increases in decay.
- Far-UVC adds to decay, similar to ventilation and filtration, reducing airborne pathogen levels.
- ACH (air changes per hour) measures how often air is replaced in a room; it assumes perfect mixing, and does not account for filtration or inactivation.
- ACH can be treated as a decay constant in well-mixed rooms, but real-world airflow varies, meaning actual effectiveness depends on ventilation design, air distribution, and room occupancy.
- eACH (equivalent ACH) represents the pathogen removal effect of air cleaning technologies like far-UVC and filtration, translating their impact into an air exchange equivalent.
- CADR (clean air delivery rate) quantifies the volume of air effectively cleaned per unit time, allowing comparison across different air purification methods, including filtration and UV.
- Far-UVC's eACH varies by pathogen because different microbes have different UV susceptibilities, affecting how efficiently they are inactivated.

How do we study how well far-UVC reduces transmission of airborne diseases?

Effectiveness

The effectiveness of an intervention at reducing transmission of disease means how many infections it prevents in real-world environments. Or to put it concretely: if far-UVC were installed in a school classroom, or an urgent care waiting room, or a conference center, how many illnesses would be prevented?

This deceptively simple question is very challenging to study. This is not a property of far-UVC in particular, but rather there are general limitations to studying interventions that occur at the level of a place, when the endpoint of the study is how many people get sick.

The challenges of cluster-randomized trials

Suppose a device was invented that stopped 100% of the transmission of respiratory infections in the room in which it was placed. How would we prove, empirically, that such a device worked?

Cluster-randomized trials are the gold standard for assessing the effectiveness of treatments at a group level. In a cluster-randomized trial, researchers randomly assign an intervention like far-UVC to some places, treat other places as a control, and compare the infections of the people who spend time in the places with devices, and the people who spend time in the control places. However, cluster-randomized trials come with a number of challenges.

The first challenge is that we can't easily measure how much a disease was transmitted between individuals in a given place. We can only measure who has an infection or was infected, and know what places they have been. For some populations, the risk of acquiring a respiratory infection is concentrated only in one particular place: for example, residents of a long-term care facility or a military barracks. But these are the exception rather than the rule. Most people have multiple possible places where they can acquire respiratory infections, and no one place constitutes the vast majority of their overall infection risk.

Therefore, even if an environmental intervention is 100% effective, it is essentially impossible to measure a 100% reduction in infections using a cluster-randomized control trial. Even if no-one contracted an infection *in* a far-UVC-treated location, in the vast majority of cases, people who visited that place could get sick in many other places. To put it more technically, if one intervention is tested to prevent transmission in a particular place, the measured effect size that can possibly be observed in a cluster-randomized trial of the people who occupy that space is reduced proportionately to how likely the people are to acquire an infection in that place relative to other places. In many situations, if a cluster-randomized trial detected a 20% reduction in infections among (say) children in a particular classroom, that could represent a far larger reduction in the amount of transmission that is actually occurring inside the classroom.

Furthermore, in some circumstances the intervention itself can displace transmission to other places or times. The latter effect is clearly visible in the study of the effectiveness of upper-room UV against measles in New York State schools published in 1947[9]. The total reduction in infections over the study period in the school that had UV lamps in all classrooms

was not statistically significant, but it can clearly be seen from the data that there was a successful 'flattening of the curve'—a reduction in the rate of transmission—in the school where all classrooms had UV lamps. In the school where half of the classes had UV lamps, the results seem to show infections being partially displaced. One critical factor was that children mixed on school buses, so children who avoided measles in the irradiated classrooms could catch it on the bus.

Another historical example during the 1957–58 influenza pandemic in a TB hospital also demonstrated this displacement of transmission. While the ward equipped with upper-room UV showed dramatically lower infection rates among patients (1.9% versus 18.9% in the control ward), staff who worked in both wards had infection rates similar to the control ward (18%). No airborne pathogen is as infectious as measles[10], and displacement of infections is especially likely to occur with very infectious diseases. This is another reason why effect sizes in cluster-randomized studies can be small even when interventions could be very effective at reducing transmission risk within their spaces. A larger study in more schools would have been necessary in order to establish whether the apparent reduction in relative attack rate among non-bus riders in the irradiated classrooms was evidence of a strong effect on infection risk.

Therefore, the first challenge of cluster-randomized studies is how to design a study that can demonstrate a large reduction in infections, as this is not always possible even if an intervention is effective at reducing transmission in a particular space. Pathogen genomics may partially address the challenge by identifying transmission chains through single nucleotide polymorphism (SNP) differences in whole genome sequences. This could potentially exclude sources of infection that occur in places outside the study, and is worthy of further investigation.

The second challenge is that even in the same kinds of places—hospitals, classrooms, nursing homes, offices—the number of infections between places can have a very high natural variance, and therefore a relatively small effect size signal can be masked by a lot of noise.

The combination of these two challenges means that in a cluster-randomized trial, the study needs to be very large in order to be adequately powered to detect the effect, even if the intervention causes a large decrease in infections. This is illustrated by a recent investigation into the use of N95 respirators compared to medical masks to reduce the burden of influenza among health care staff. Nearly 4000 health care workers were enrolled in the study at 137 outpatient sites, and the study collected data over 4 respiratory illness seasons. Post-trial analysis revealed that a trial twice as large would have been necessary to have a >80% chance of detecting a 25% reduction in laboratory-confirmed influenza infections at $p = 0.05$ (statistical significance)[11].

There are other examples where a cluster-randomized studies of group-level infection prevention interventions need substantial scale in order to be adequately powered. A proposed study investigating the use of far-UVC and Corsi-Rosenthal boxes to reduce the transmission of respiratory virus in Bangladeshi schools estimated a need to enroll 300 classrooms in 60 schools[12]. An ongoing study of HEPA filtration devices in care homes in England enrolled 1196 residents across 90 homes with 2 years of data gathering[13]. Over 350 public schools in Denver are currently enrolled in an ongoing epidemiological study on the effect of HEPA filtration devices on school absence[14,15]. There is also a cluster-randomized trial for far-UVC in Nova Scotia that is expected to report this year. Far-UVC devices were placed in the common areas of long-term care facilities, covering approximately 500 residents across 11 neighbourhoods in three facilities. The intervention was randomized by neighborhood (with test and control present in each facility)[16]. It remains to be seen whether any of these study designs are adequately powered.

A recently published trial attempted to measure the effect of HEPA filtration on infections in a residential care facility[17]. This trial demonstrates why properly powered large-scale studies are crucial for evaluating transmission suppression interventions. The study randomized 135 residents (70 to intervention-first, 65 to control-first) in a crossover design to receive either real or sham HEPA filters in their rooms. While it found a potentially meaningful 27.5% relative reduction in infections (31% in the HEPA group versus 42% in control), the study was unable to definitively prove effectiveness in its primary intention-to-treat analysis because it was only powered to detect an unrealistically large 50% reduction. This led to the misleading conclusion in the paper's key points that "air purifiers with HEPA-14 filters do not reduce the incidence of acute respiratory infections"—when in fact the study was simply underpowered to detect even substantial benefits. Multiple critics were quick to point out that to detect a more realistic 25% risk reduction (30% infection rate versus 40% control), the study would have needed around 350 participants rather than the 135 enrolled[18,19]. Similarly, the DANMASK trial, which investigated the effectiveness of masks at preventing the transmission of COVID-19, similarly powered its analysis to detect only a ≥ 50% reduction in infections[20]. When it found a more plausible but not statistically significant 18% reduction, this generated a widespread misinterpretation that masks had been 'proven ineffective'.

Unfortunately, studies that are underpowered and fail to find statistical significance often promote incorrect conclusions that interventions 'don't work' when in fact they may provide meaningful benefits that the study wasn't designed to detect. Cluster-randomized studies therefore need to be designed very carefully in order to understand what effect size can even be achieved given the infection dynamics of the particular infections, people and spaces, and what sample size is going to be required in order to detect that effect[21]. Furthermore, researchers cannot know for sure in advance if a study is adequately powered to detect a given effect, as it requires making assumptions about the variance and correlations that will be observed across trial clusters. If the question we are interested in is "does this intervention reduce transmission risk in a particular place?", many studies that have been conducted in the past are not even capable of answering this question in principle due to inadequate scale.

Alternative methods of measuring transmission

Enrolling and managing studies that are comparable in scale to Phase III pharmaceutical trials requires significant overhead, but one of the main drivers of expense in cluster-randomized studies is the laboratory testing that is usually required to measure infections among study participants.

The protocol for the proposed Bangladesh study (mentioned above) calls for twice-weekly visits to every classroom and PCR testing of all symptomatic children[12]. The N95 respirators study required PCR testing of all symptomatic participants, two random samples each season while asymptomatic, and serological testing before and after each season for all participants[11]. It

is therefore desirable to gather data that is cheaper and more scalable than laboratory testing, in order to make conducting studies at this scale more practical. However, these alternatives also come with drawbacks.

Wastewater surveillance offers one potential alternative, providing population-level data on infection prevalence at a fraction of the cost of individual testing[22]. By sampling building or community wastewater systems, researchers can monitor viral loads without the need for individual participation. However, wastewater measurements are only loosely correlated with actual infection rates due to variations in viral shedding, environmental degradation, and dilution effects. The relationship between reduced airborne transmission and changes in wastewater viral loads is indirect and influenced by many confounding factors, reducing wastewater surveillance's statistical power to detect intervention effects. It is best used in large catchment areas that smooth over the differences in shedding patterns between individuals.

Absenteeism tracking represents another low-cost approach that leverages existing administrative systems. These data are particularly attractive because they require minimal additional infrastructure or personnel to collect. The main limitation is that absences have low specificity for infections of interest–students and workers miss school or work for many reasons unrelated to the infections being studied. This means that much larger sample sizes are needed to detect a given reduction in actual infection rates compared to direct testing approaches. An additional benefit of measuring absenteeism over other surrogates like wastewater tracking or infection rates is that it can be correlated to other quantified impacts, such as business productivity and costs, or student learning and educational outcomes.

Hospital admission rates can be accessed through existing healthcare reporting systems and provide a standardized metric across different locations. While this data is relatively inexpensive to collect, hospitalizations represent only the most severe cases, which are typically a small fraction of total infections. A technology that reduces overall infections by 50% may only reduce hospitalizations by a much smaller percentage. This substantial dilution of the measurable effect means that studies using hospitalization data as an endpoint typically need to be much larger to demonstrate efficacy compared to those using direct infection testing.

The key challenge with all these surrogate measures of infection transmission is the tradeoff between cost and statistical power. While surrogate measures enable larger and longer studies by reducing per-participant expenses, their indirect relationship with infection rates means that more sites and participants are needed to achieve adequate statistical power. A study powered to detect a 25% reduction in PCR-confirmed infections might need to be many times larger to detect the same reduction using surrogate endpoints. Researchers must carefully weigh these tradeoffs when designing studies and selecting outcome measures.

The need for real-word studies

We need data on the real-world effectiveness of far-UVC in reducing disease transmission among humans. First, we need to understand its true practical effectiveness, not just its theoretical potential, in order to calibrate its role within a layered pandemic defense strategy. For example, studies are needed to determine how airborne pathogen concentrations translate into actual human infections. Second, for far-UVC to have a meaningful impact on pandemic prevention, it would need to be widely adopted in public spaces, which requires building public trust through robust evidence. Third, regulatory bodies and standards organizations need empirical data to develop appropriate guidelines and requirements. Finally, policymakers and building operators need comparative effectiveness data to evaluate far-UVC against other options for reducing airborne transmission in the built environment.

Key takeaways

- Directly studying effectiveness at transmission suppression in real-world environments is challenging. However, cluster-randomized trials remain the gold standard of evidence and there are numerous ongoing and attempted efforts to execute them for interventions to reduce airborne transmission.
- It is difficult to know in advance whether a cluster-randomized trial is adequately powered to detect a given effect, as researchers may not know what effect size is even achievable assuming the intervention works as intended, and power calculations depend on variables which can only be observed in the course of the trial.
- There's a risk that underpowered or poorly designed effectiveness trials are interpreted as showing that an intervention 'doesn't work' when in fact a study may not even have been capable of showing the intended effect.
- The use of alternative endpoints to measure transmission of airborne infection can enable larger and relatively less expensive trials, but this comes at the cost of study power.

Efficacy

Efficacy is the measure of how well far-UVC inactivates pathogens. It is presumed to be linked to effectiveness through reducing the probability that a susceptible person is exposed to an infectious dose. The reported 98.4% reduction in airborne bacteria observed in Eadie et al., 2022[1] is an efficacy study, with the implication being that a large reduction in the quantity of viable airborne pathogen ought to lead to a large reduction in airborne transmission.

Pathogen surrogates

Efficacy studies often use surrogate pathogens, enabling experiments to take place at lower biosecurity levels. Until recently, handling live SARS-CoV-2 required a BSL-3 lab[23], leading researchers to use surrogates such as the common cold coronavirus HCoV-OC43 (BSL-2)[24] or bacteriophages such as MS2 and T1 (BSL-1)[25]. While SARS-CoV-2 has now been reclassified to BSL-2[23], much of our understanding comes from these surrogate studies.

Surrogate pathogens can also have other properties that make them more amenable to study. For example, in order to detect large reductions in pathogen load, we need to be able to culture the pathogen to a high initial concentration. One of the reasons that MS2 bacteriophage is commonly used as a surrogate is that it is easy to culture, relatively robust to environmental conditions, and inexpensive. Ideally, a surrogate's susceptibility to UV should be as similar to the microbe of interest as possible. For example, sometimes it is common to use an enveloped bacteriophage as a surrogate for an enveloped virus, or a non-enveloped bacteriophage for a non-enveloped virus. But the main requirement of a surrogate is that its dose-response to UV be representative of the pathogen.

Efficacy studies can also be conducted in 'real' spaces with naturally occurring pathogens, as well as with non-microbial surrogates such as DNA-tagged aerosols[26]. However, these studies are not straightforward, and the concentration of the pathogen will in many situations not be high enough to detect a signal; 'real' spaces may also have high background variation in pathogen levels, for example associated with changing occupancy or activities of occupants.

How efficacy is measured

Efficacy studies are often quoted as a reduction in the amount of pathogen when exposed to far-UVC compared to non-exposed controls. Sometimes this is quoted as a log reduction—for example the 98.4% reduction in Eadie et al., 2022 would be a 1.8 log reduction in the concentration of pathogen[1].

However, percent reductions and log reductions are not straightforward to interpret. Altering the experimental setup—such as the duration of the experiment—can dramatically change the log reduction observed. Furthermore, the size of the chamber in which the experiment is conducted can also dramatically affect the observed reduction. By themselves, log reductions are not a useful metric for understanding the efficacy of air cleaning technologies. Even worse, it is not uncommon in the air cleaning market for log reductions to be reported that are not compared to adequate controls.

Calculating eACH and CADR provides a more useful metric for translating experimental results to real-world applications for air cleaning, as they relativize the reduction to the duration of the experiment and the size of the chamber in which the experiment is conducted.

Calculating eACH and CADR: decay versus steady state tests

Calculating eACH and CADR from an efficacy experiment requires different methods depending on whether the test uses continuous introduction of the challenge agent or not ('steady-state' versus 'decay' method).

In the decay method, the pathogen is introduced and then is allowed to decay naturally, with far-UVC lamps on and off. The eACH and CADR can then be calculated by comparing the rate of decay with and without the lamps on using equation 3.2.

Alternatively, if a pathogen is continuously introduced at a constant rate, then equation 3.3 can be used to estimate the change in decay that is consistent with the observed change in pathogen concentration. However, in order to use this method to calculate an eACH, the base rate of decay without the intervention must be known. The control arm of the decay test could be used to calculate base decay, or alternatively it can be estimated from ventilation rates and other known sources of decay such as surface deposition. Given the difficulties of translating nominal ventilation rates to effective decay (see above ACH) and the sensitivity of the eACH calculation to base decay, it may be prudent for researchers to conduct a decay test even if they intend to use the steady-state method, in order to more accurately characterize the base decay.

Steady-state and decay tests have different advantages and disadvantages. Both have been used in studies of far-UVC, but the test methods that have been established as consensus standards all employ the decay method (see *Guidance, standards, and regulations* section). In a direct comparison between the decay and steady-state method, one study found higher inactivation with the steady-state method[27]. It's not currently clear which test method is preferable for measuring devices which are intended to be used for practical application.

Efficacy in controlled chamber studies

Data from four studies of efficacy in large chambers is currently in the public domain, three of which[1,28,29] have been published in peer-reviewed journals. The fourth study has been reported at an academic conference, and the talk is publicly available[27].

TABLE 3.1. Summary of publicly available far-UVC efficacy data in room-sized bioaerosol chambers.

Study	Method	Pathogen	eACH	CADR (L/s)	# Far-UVC devices	CADR/device (L/s)
Eadie et al., 2022[1]	Steady-state	*S. aureus*	184	1,636	5	327
Hiwar et al., 2025[29]	Steady-state	*S. aureus*	133 (estimated)	1,190	5	238
Hiwar et al., 2025[29]	Steady-state	*P. aeruginosa*	122 (estimated)	1,091	5	218
Ratliff et al. (DI Water), 2025[28]	Decay	MS2 bacteriophage	1.24	29	2	15
Ratliff et al. (simulated saliva), 2025[28]	Decay	MS2 bacteriophage	2.06	49	2	25
Blatchley Presentation, 2024[27]	Steady-state	T1 bacteriophage	1.79	28	4	7
Blatchley Presentation, 2024[27]	Decay	T1 bacteriophage	1.1	17	4	4

*Studies had differences in fixtures, radiant power, and/or average fluence rate.

CADR for Eadie et al., 2022[1] calculated from reported eACH and chamber volume of 32 m^3. eACH for Ratliff et al.[28] calculated from reported CADR and chamber volume of 3000 ft^3. CADR for Blatchley[27] calculated from reported eACH and room volume of 57.1 m^3.

Across these controlled room-sized chamber studies, far-UVC showed very high eACH and CADR against aerosolized *Staphylococcus aureus* and *Pseudomonas aeruginosa*, but minimal eACH and CADR against two bacteriophages.

Until we know how susceptible the surrogate pathogens are to far-UVC, it is difficult to understand or interpret these studies. For example, it could be the case that *S. aureus* is particularly susceptible to far-UVC, and the bacteriophages particularly resistant. There are many more studies on pathogen susceptibility to far-UVC than efficacy, and this will be covered in the next section. But overall, if the real-world efficacy of far-UVC against pathogens we care about is more accurately represented by Eadie et al., 2022, then it is an extremely promising technology. If it is more accurately represented by the Ratliff and Blatchley studies, it holds far less promise.

Efficacy in real spaces

In addition, there is one far-UVC efficacy study that has been conducted in an occupied room against naturally occurring pathogens—specifically, a mouse cage cleaning room which contains high concentrations of aerosolized murine norovirus (MNV)[30]. Four far-UVC fixtures were placed on the ceiling of the 95 m³ room, and the concentration of aerosolized MNV estimated with lamps on and lamps off. The researchers observed a 99.8% reduction in concentration when the lamps were on.

This result is made even more spectacular when we consider the base level of pathogen decay in this space. The mouse cage cleaning room ventilation rate is nominally 36 air changes per hour based on the air supply. The ACH, calculated at 7 different points in the room using a CO_2 monitor, ranged from 31.5–72.4 ACH, with the latter reading taken near the pathogen air sampler.

Using equation 3.2 and the nominal ACH of 36, this would suggest an additional 14,800 eACH added from the use of far-UVC (assuming a well-mixed space), if the reduction can be interpreted as a change in the average steady-state pathogen concentration. But this calculation is highly sensitive to the assumption around the base decay, and applying a mixing factor could reduce the estimated eACH by as much as a factor of 10. It should also be noted that the seemingly narrow confidence interval on the measured reduction in MNV (95% CI: 98.2–99.9%) translates to a very large difference in the estimated eACH, ranging from 200 to 3000 assuming a mixing factor of 10.

Key takeaways

- Efficacy studies measure how well far-UVC reduces the concentration of pathogens.
- For the purpose of air cleaning, the key efficacy metrics are eACH and CADR.
- Among the three lab-based studies available to date in room-sized chambers, one has shown far-UVC to have very high efficacy against bacteria, but two studies have shown much lower efficacy against bacteriophages.
- There has been one far-UVC efficacy study in a real space using naturally occurring virus (murine norovirus), which also showed very high efficacy.
- In order to understand and interpret these efficacy studies, we need to know the susceptibility of the surrogate pathogens, and how these compare to pathogens of concern.

Susceptibility

Susceptibility is the relationship between UV dose—how much UV a pathogen is exposed to—and efficacy. Quantitatively, this relationship is expressed using a modified version of the exponential decay equation:

Equation 3.6

$$S = e^{-kD}$$

with S = survival fraction
D = UV dose
k = UV susceptibility constant

The unit of k is the inverse of the dose, and therefore usually either m²/J or cm²/mJ, just as in the conventional decay equation the unit of λ is the inverse of time (e.g. hr). The higher the k value for a pathogen, the more susceptible the pathogen is to inactivation from UV.

In a handful of papers in the UV literature, k is calculated in base 10 rather than base e, see for example Ma et al., 2021[31]. A k in base 10 can be converted to a k in base e by using the change of base rule of logarithms, which results in multiplying a k in log 10 by 2.303 (as $e^{2.303} = 10$).

Experimental procedures for measuring k

In order to estimate k, equation 3.6 illustrates the basic requirements. The pathogen is exposed to different radiant exposures and for each, the survival fraction is calculated as a ratio of the pathogen quantity (i.e., PFU/ml, TCID50) at that dose relative to the pathogen quantity in sham irradiated controls. Susceptibility studies are more practical than efficacy studies, and usually involve survival in water rather than in air.

For explanations of typical experimental methodologies for measuring dose and survival fraction as well as techniques for culturing, aerosolizing and controlling irradiation of the pathogen, we recommend reading the methods section of Welch et al., 2018 and Lu et al., 2024[32,33].

k values depend on the pathogen, the UV wavelengths, and the environmental conditions. For example, the effect of increasing relative humidity on a number of pathogens (particularly bacteria, and some viruses) is to lower their susceptibility to conventional UVC. A pathogen population may also not have a single k, and in some cases inactivation of the pathogen is better described using a biexponential dose-response relation of the type:

Equation 3.7

$$S = (1 - f)e^{-k_1 D} + fe^{-k_2 D}$$

with f = resistant fraction of pathogen population
k_1 = susceptibility of sensitive fraction
k_1 = susceptibility of resistant fraction

This is well-illustrated in Welch, 2022, which showed that biexponential decay provided a much better fit to the observations of the survival of HCoV-OC43.

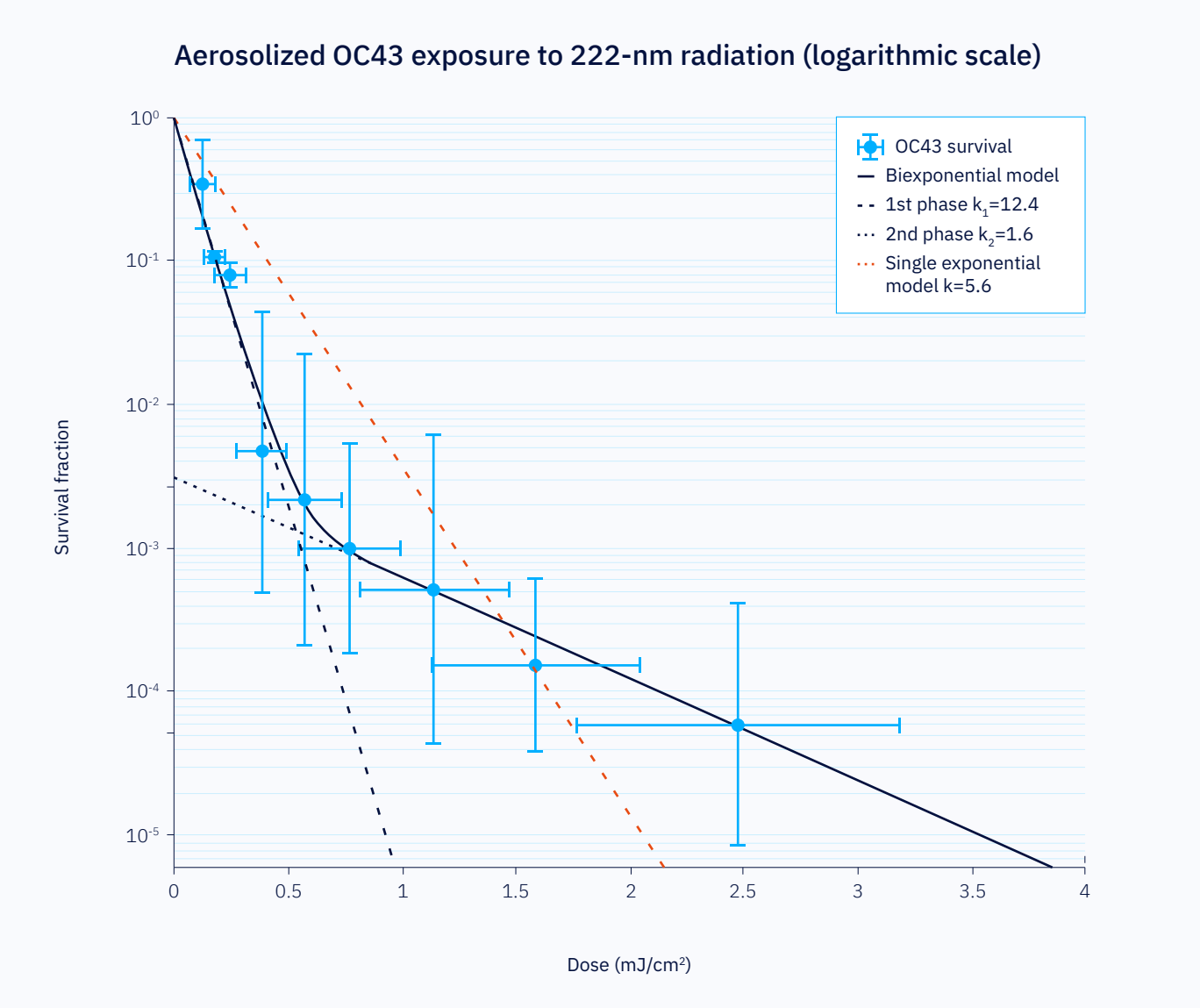

FIGURE 3.1. Excerpted from Welch, 2022[34]. Survival fraction of coronavirus OC43 exposed to 222-nm radiation fitted with the two-phase decay model. The circle markers represent the mean survival values for a given mean exposure dose. The x-error bars show the standard deviation of the doses for the 1600 particles in the simulation, and the y-error bars show the standard deviation of the experimental repeats of survival fraction. The two-phase decay model fit to the data is included on the graph (solid line), as well as lines representing the decay of the first and second stages separately (dashed and dotted lines, respectively). The single exponential model fit to the same data is included for comparison.

In this instance, a single exponential model underpredicts inactivation at doses <1.5 mJ/cm², and overpredicts at higher doses >1.5 mJ/cm².

How much the 'resistant fraction' matters to efficacy depends on how large the resistant fraction is and how much reduction in pathogen load you are trying to achieve. The 0.3% of OC43 that is modelled with the lower k will have relatively little impact unless you were trying to achieve 99%+ reduction in pathogen load. At a 50% reduction, the 0.3% resistant fraction will have a negligible impact.

The existence of multi-phase decay does not appear to be due to properties of the pathogen, i.e. that there are individual virions or bacteria that are more resistant to far-UVC. Rather, it is more likely explained by physical 'self-shielding' from UV which happens when microorganisms are clustered together[35]. It is an open question how much the physical clustering that occurs in controlled studies in small bioaerosol chambers (where pathogens have often been cultured to a very high concentration) is representative of clustering of pathogens in human respiratory aerosols.

Variation by pathogen

k values can vary substantially by pathogen. For example, Lu et al., 2024[33] compare susceptibility for various bacteria and bacteriophages using different types of UV, with the variations in k across pathogen spanning approximately one order of magnitude:

TABLE 3.2. From Lu et al., 2024. Wavelength-specific inactivation rate constants for aerosolized E. coli, *S. epidermidis*, *S. enterica*, MS2, P22, and Phi6. The inactivation rate constants were calculated from the linear fitting lines of survival curves (±95% CI).

UV sources	*E. coli*	*S. epidermidis*	*S. enterica*	MS2	P22	Phi6
222-nm KrCl* excilamp	21.93 ± 0.86	3.05 ± 0.35	4.97 ± 0.38	5.75 ± 0.57	17.14 ± 0.76	20.08 ± 0.49
254-nm LP UV	25.09 ± 2.03	4.25 ± 0.22	5.73 ± 0.48	3.46 ± 0.29	9.71 ± 0.23	6.38 ± 0.77
268-nm UV-LEDs	27.56 ± 1.43	5.92 ± 0.30	8.86 ± 0.37	4.00 ± 0.21	14.56 ± 0.82	9.66 ± 0.66
298-nm UV-LEDs	6.81 ± 0.83	1.79 ± 0.11	2.51 ± 0.22	1.54 ± 0.23	3.22 ± 0.06	3.71 ± 0.24
311-nm UVB	3.85 ± 0.31	1.01 ± 0.21	1.46 ± 0.20	0.77 ± 0.09	1.65 ± 0.08	2.15 ± 0.14
365-nm UVA	1.01 ± 0.17	0.13 ± 0.02	0.65 ± 0.09	0.17 ± 0.02	0.53 ± 0.06	0.84 ± 0.11

In another analysis of published data, susceptibility of 59 animal viruses and 33 bacteria to conventional UVC spans one and a half orders of magnitude, and susceptibility is relatively well-predicted by a genomic model[36].

Relative humidity

Relative humidity (RH) can significantly affect the UV susceptibility of airborne pathogens, though these effects are complex and species-dependent. For a thorough discussion on the effects of RH on germicidal UV, focusing on conventional UV, see Kowalski, 2009, chapter 2.9[37]. Most bacteria show decreased UV susceptibility at high RH, while viruses show mixed results—some experience increased susceptibility with higher RH, others show decreased susceptibility or no effect. The mechanisms behind these RH effects are varied and include DNA conformational changes (particularly around 70% RH)[38], changes in microbe size due to water absorption[39], and changes in natural pathogen stability[40]. The medium in which a pathogen is aerosolized may also impact its response to RH[41].

For example, in studies of *S. marcescens* (a common bacterial surrogate), some researchers found only mass increases with higher RH while others observed increased mean diameter, highlighting the complexity of these effects[37]. With viruses, Harper found that some viruses, such as vaccinia and influenza, showed small increases in die-off at high humidity, while poliovirus peaked in die-off around 50% RH[42]. Natural decay rates for aerosolized viruses in air tend to be low at normal humidities and can generally be neglected when evaluating UV effects.

There is not currently any published data showing the effect of relative humidity on far-UVC inactivation of pathogens.

Inconsistencies in k across studies

There is also significant divergence in k values of the same pathogen and pathogen family across different studies. Coronaviruses are the pathogens that we have the most far-UVC data points for in aerosol. These are summarized in Table 3.3.

TABLE 3.3. Summary of published far-UVC inactivation constants for aerosolized coronaviruses.

Study	Pathogen	Medium	Relative humidity	k (cm^2/mJ)
Buonanno et al., 2020[43]	HCoV-229E	5% MEM 95% DI Water	50–70%	4.1 (95% confidence 2.1–4.8)
Buonanno et al., 2020[43]	HCoV-OC43	5% MEM 95% DI Water	50–70%	5.9 (95% confidence 3.8–7.1)
Welch et al., 2022[34]	HCoV-OC43	5% MEM 95% DI Water	60–70%	5.6 (single exponential model) 12.4 ± 0.4 for most susceptible 99.7% 1.6 ± 0.1 for remainder
Kitagawa Presentation, 2023[44]	SARS-CoV-2	10% MEM 90% PBS	Not given	2.6* ± 0.2 for most susceptible ~90%
Lu et al., 2025[45]	HCoV-OC43		65 ± 3 %	14.26 ± 0.64

*Denotes that k values were calculated in the study in base 10 rather than base e, and have been multiplied by 2.303.
MEM = Modified Eagle's Medium, PBS = phosphate-buffered saline, DI = deionized.

By contrast, the susceptibility estimates for coronavirus inactivation in liquid show much lower variance, and in absolute terms are more comparable to the lower k estimates for aerosol:

TABLE 3.4. Summary of published far-UVC inactivation constants for coronaviruses in liquid.

Study	Pathogen	Medium	k (cm²/mJ)
Robinson et al., 2022[46]	SARS-CoV-2	DMEM	1.48
Kitagawa Presentation, 2023[44]	SARS-CoV-2	10% MEM 90% PBS	1.6* ± 0.1
Kitagawa Presentation, 2023[44]	SARS-CoV-2	1% MEM *99% PBS*	2.4* ± 0.1
Schuit et al., 2022[47]	SARS-CoV-2	Water	3.8 ± 2.8 for most susceptible ~90–99%
Schuit et al., 2022[47]	SARS-CoV-2	Simulated Saliva	1.4 ± 2.0 for most susceptible ~90–99%
Ma et al., 2023[48]	SARS-CoV-2	PBS	1.6* ± 0.05
Ma et al., 2023[48]	HCoV-229E	PBS	2.4* ± 0.1

The magnitude of variation in susceptibility in aerosol experiments is not unique to coronaviruses, nor unique to far-UVC. For example, Table 3.5 summarizes susceptibility studies of the bacteria *Serratia marcescens* (a tuberculosis surrogate) to 254-nm UV at low to moderate humidity. *Denotes that k values were calculated in the study in base 10 rather than base e, and have been multiplied by 2.303. DMEM = Dulbecco's Modified Eagle Medium.

TABLE 3.5. Summary of published far-UVC inactivation constants for aerosolized *S. marcescens.*

Study	Relative humidity	k (cm²/mJ)
Fletcher et al., 2003[49]	Low RH	9.39
Nakamura, 1987[50]	Low RH	1.13
Sharp, 1940[51]	Low RH	4.45
Ko et al., 2000[52]	22–33%	5.75
Lai et al., 2004[53]	36%	22.00
VanOsdell and Foarde, 2002[54]	50%	4.31
Peccia and Hernandez, 2001[38]	50%	4.5
Lai et al., 2004[53]	68%	9.2

Human aerosols

While far-UVC is generally highly effective at inactivating airborne pathogens, its efficacy can be influenced by the surrounding environment. In particular, when pathogens are contained by protein-rich media, some far-UVC radiation will be absorbed by these surrounding proteins before it can reach the pathogen. Respiratory pathogens emitted by infected humans are transported within the airborne particles that are the normal product of the human respiratory system.

At least one attempt has been made to model the optical properties of human respiratory aerosol particles to estimate the potential attenuation of the far-UVC dose that reaches pathogens due to absorption by proteins in the aerosol[55]. Hill et al. modeled how UV wavelengths, including 222 nm and 254 nm, interact with aerosol particles of varying sizes and compositions, including simulated respiratory media. Their findings emphasized that smaller aerosols (<2 μm) allow deeper UV penetration with minimal shielding, while larger droplets (>4 μm) exhibit significant attenuation, especially at 222 nm, due to its shorter penetration depth. The study also highlighted how proteins and other components in respiratory media contribute to shielding effects, particularly in larger particles, reducing the UV dose that reaches pathogens. This modeling approach provides valuable insights into how aerosol composition and size influence UV efficacy in real-world scenarios and underscores the importance of considering physiologically relevant aerosols in far-UVC research.

While not necessarily representative of the particular protein content of human respiratory aerosols, other researchers have attempted to simulate the protein contents of human aerosols in a controlled lab setting. Artificial/simulated saliva is a lab-made mixture of salts, proteins, and other factors designed to mimic human saliva. It is considered a decent proxy for saliva, but it may not be as useful as a proxy for the protein content of human respiratory aerosols.

Aerosols generated when coughing, breathing, or sneezing vary in composition and size depending on the site of generation in the respiratory system. The smaller respiratory aerosols which may be disproportionately responsible for airborne transmission originate from deeper in the respiratory tract[56,57]. The difference in composition plausibly impacts the susceptibility of pathogens within them to UV, for example because of potential protein shielding. Furthermore, the artificial saliva composition can differ between studies, confounding comparisons and extrapolation to real-world situations. Determining the interplay between aerosol composition, size, and impact on pathogens through real-world studies is crucial for understanding the efficacy of far-UVC.

There are currently two peer-reviewed publications examining the effect of medium on far-UVC inactivation in air. The first is Monika et al., 2025[58], in which inactivation of MS2 and Phi6 bacteriophage using both a KrCl* lamp and a low-pressure mercury lamp were compared using SM Buffer and artificial saliva as the medium. Phi6 was measured to be more susceptible to both sources of UV when using artificial saliva, and MS2 was more susceptible for the low-pressure mercury lamp, but no significant difference was measured for the KrCl* lamp. This increased pathogen susceptibility when suspended in protein-rich media is the opposite effect to that predicted by Hill et al.[55]

The second study is Ratliff et al., 2025[28], which compared inactivation of MS2 in simulated saliva and deionized water. In this study the effect of the simulated saliva was directionally the same—the measured clean air delivery rate was higher when using simulated saliva rather than deionized water.

While this is a speculative hypothesis, it is possible that there are other effects on pathogen stability from far-UVC being absorbed by other proteins within aerosols. In what way the micro-environment of aerosols is affected by proteins absorbing far-UVC is unknown, but recent research suggests that SARS-CoV-2 may be sensitive to the pH of the aerosol it is contained in[59]. Two recent papers suggest that the formation of reactive oxygen species (ROS) play a role in far-UVC inactivation of bacteria[60,61], and it is theoretically possible that absorption of far-UVC by the protein contents of human respiratory aerosols could lead to the creation of ROS that microbes contained within the aerosol become exposed to. The possibility that far-UVC may affect the stability of pathogens through some alteration in the micro-environment of human respiratory aerosols should be considered.

An alternative potential explanation for the effect seen in these studies is that the simulated saliva substantially increased the stability of the pathogen in the control arm. In Ratliff's study, higher CADR was found against MS2 in simulated saliva not only when using far-UVC but also and when using a HEPA filter, when there is no known mechanism by which the medium should affect the efficacy of a filter given the particle size distributions were similar[28].

Given all the challenges and variability observed in lab-based efficacy studies, identifying experimental designs to establish the effect of far-UVC against pathogens in actual human respiratory aerosols should be a high priority.

Key takeaways

- Pathogen susceptibility is measured by its dose response to UV, usually called a k or z value.
- k values can vary significantly for different pathogens, in different exposure conditions, and even across different studies for the same pathogen.
- The impact of proteins in human aerosols on pathogen susceptibility to far-UVC has not yet been quantified.

Modeling efficacy and susceptibility

The key question is: does this uncertainty about germicidal susceptibility matter? It is possible that far-UVC, when used within safe exposure limits, could be effective enough anyway even if k constants are lower in real-world (i.e., non-laboratory) conditions. Conversely, it is possible that far-UVC is not effective enough, even against highly susceptible pathogens in ideal conditions, to be practically employed for whole-room disinfection.

A simple dimensional analysis shows how k constants can be used to estimate the eACH and CADRs that may be achievable from the use of far-UVC. It is very important to note that unlike studies that attempt to measure susceptibility and efficacy directly, the results derived in this section have not been shown to be empirically true. Rather, the mathematical definitions of eACH, CADR and k (along with a critical simplifying assumption) allow for translating between experimental measures of efficacy and susceptibility.

The critical assumption that enables this simple analysis is that we can use an average UV fluence rate, and implicitly a uniform dose rate distribution (i.e. all pathogen in a space is dosed at the same rate at all times), to estimate inactivation. The importance of this assumption will be discussed by the end of this section, but up front it should be noted that the use of averages may lead to poor estimates of efficacy from susceptibility.

Estimating eACH from k and assumption of uniform fluence rate

Equations 3.2 and 3.9 are structurally similar. In order to translate between them we require a relationship between UV dose and time in hours. In other words, if we can calculate the dose per hour, then we can translate between UV susceptibility k and decay rate per hour λ.

Dose per unit time is the definition of UV fluence rate (I) (see *Far-UVC primer* section). To translate between the dose D of equation 3.6 and the time t in equation 3.2, we use the equation for UV dose:

Equation 3.8

$$D = \left(\frac{I}{1{,}000}\right) \cdot t$$

with D = UV dose (mJ/cm²)
I = UV fluence rate (μW/cm²)
t = time (seconds)

We can therefore substitute equation 3.8 into equation 3.6. Note that in equation 3.8, time t is in seconds, but to compare to ACH we want time t to be in hours or 3600s.

Equation 3.9

$$S = e^{-k\cdot\left(\frac{I}{1000}\right)\cdot(t\cdot 3600)} = e^{-(k\cdot I\cdot 3.6)\cdot t}$$

Recalling the definitions of ACH (a decay constant λ_{ACH} assuming the room is well mixed) and eACH (anything that has the same effect as an ACH), the estimate for eACH using far-UVC is:

Equation 3.10

$$\mathrm{eACH} = k \cdot I \cdot 3.6$$

If we know k, I, and all the other sources of decay, we can also estimate the ratio of the average steady-state pathogen concentration with and without UV when there is a continuously emitting source, using equation 3.3:

Equation 3.11

$$\frac{C_{\mathrm{ss\_UV}}}{C_{\mathrm{ss\_base}}} = \frac{\lambda_{\mathrm{sum}}}{\lambda_{\mathrm{sum}} + (k \cdot I \cdot 3.6)}$$

with C_{ss_base} = average steady-state concentration without UV
C_{ss_UV} = average steady-state concentration with UV
λ_{sum} = other non-UV sources of decay (hr^{-1})
k = pathogen inactivation constant (cm^2/mJ)
I = average fluence rate ($\mu W/cm^2$)

It is important to reiterate that these equations are only valid assuming a uniform distribution of dose, and the fluence rate being used is the average across the whole room. Faulkner et al., 2024 used room average fluence rates to estimate the efficacy of far-UVC in their study of the energy costs of meeting recommended CDC and ASHRAE 241 air cleaning requirements in a typical office[62].

By contrast, Welch et al., 2025 use equation 3.9 to estimate 'breathing zone efficacy' based on estimates of fluence rates using film dosimetry worn by study participants[63]. While this method can calculate the rate of decay we would expect pathogens to be subject to while in the breathing zone, pathogens circulate around the room after being exhaled and before they are inhaled by anyone. The efficacy of technologies such as upper-room UV depend on this mixing between the breathing zone and the irradiated zone.

If the worn film dosimetry methodology was applied to upper-room UV, where the efficacy is produced by the mixing between air in the breathing zone and air in the unoccupied irradiated zone above, measuring fluence rates in the breathing zone and applying these equations would dramatically underestimate the efficacy of the technology.

When ASHRAE standard 241 refers to clean air being delivered "in the breathing zone," this is therefore not intended to mean the rate of pathogen decay locally in the breathing zone, but the rate of decay experienced by pathogens before they are breathed in, crucially including the decay that occurs while not in the breathing zone.

If instead we wish to estimate efficacy against short-range transmission that occurs entirely in the breathing zone, eACH is not a valid metric, as we are no longer in the framework of room-average concentrations. Instead, we must know the time elapsed between exhale and inhale, so that a dose can be calculated from fluence rate and equation 3.6 used to estimate the survival fraction before the pathogen is inhaled. If the time elapsed between inhale and exhale is a matter of seconds, then there is not much time for the pathogen to accumulate a substantial dose. For this reason, we expect far-UVC to be less effective against short-range transmission than long-range transmission.

Inferring susceptibility from efficacy studies

These equations can also be used to estimate what pathogen susceptibility is consistent with the results of an efficacy study, assuming a uniform dose distribution. However, even among susceptibility and efficacy studies that are reported in the same paper, there is significant inconsistency when attempting to translate between susceptibility and efficacy:

TABLE 3.6. Comparison of inferred susceptibility constants from published efficacy studies, and direct measurements from susceptibility experiments conducted by the same research group.

Study	Pathogen	Reported eACH	Fluence rate (μW/cm²)	Implied k from efficacy (cm²/mJ)	Direct k from susceptibility study (cm²/mJ)
Eadie et al., 2022[1] (High dose)	*S. aureus*	184	2.5	20.6	~0.9
Hiwar et al., 2025[29]	*S. aureus*	133 (estimated)	2.5	14.8	~0.9
Hiwar et al., 2025[29]	*P. aeruginosa*	122 (estimated)	2.5	12.5	n/a
Ratliff (simulated saliva), 2025[28]	MS2 bacteriophage	2.32	~1.3	0.50	n/a
Buonanno et al., 2024[30]	Murine norovirus (MNV)	1,480	0.82	510	2.36
Blatchley presentation, 2025[27]	T1 bacteriophage	1.79	2.16	0.23	0.873

Fluence rate for Eadie et al., 2022 was reported in a presentation at ICFUST 2023[64], and the k is from susceptibility study inferred from data presented in their Figure 3. Fluence rate for Hiwar et al., 2025 matches that from Eadie et al., 2022, and eACH was estimated using the same method reported in Eadie et al., 2022. eACH from Ratliff based on measured 0–90 min CADR for far-UVC fixtures in simulated saliva and chamber size of 3000 ft³. Estimated fluence rate from Ratliff is the average of two independent estimates by Vivian Belenky (1.47) and Holger Claus (1.1) using simulation software. Reported eACH for Buonanno et al., 2024 based on an assumed mixing factor of 10, for a poorly mixed room. Susceptibility for MNV in Buonanno et al., 2024 based on most susceptible 91.5% of population, k for remaining population estimated to be 0.36. Reported eACH from Blatchley presentation based on the steady-state test, rather than decay test.

In all three studies where k was separately estimated in small chamber studies, there are substantial differences from k inferred from efficacy. The discrepancies also do not uniformly run in one direction. While Ratliff did not conduct a separate susceptibility study, there is one published susceptibility estimate for aerosolized MS2 to far-UVC of 5.75 cm²/mJ, an order of magnitude higher than inferred from Ratliff's efficacy data[28,33]. However, this study found generally high susceptibility for multiple viruses and bacteria. By contrast, Monika et al., 2025 estimated MS2 susceptibility in artificial saliva to be 0.37 cm²/mJ, similar to what would be inferred from Ratliff's data using this simple modeling framework[58]. There are no published inactivation constants for *P. aeruginosa* in aerosol at 222 nm, and the only available aerosol inactivation data at any UV wavelength comes from a 1940 study at 254 nm[51].

As discussed earlier for Buonanno et al., 2024, there may be multiple problems with using the well-mixed room and average pathogen concentration framework for the mouse cage cleaning room study[30]. However, even using the low end of the confidence interval for eACH and using a mixing factor of 10 to obtain an estimated 200 eACH, the implied k would still be around 68 cm²/mJ—substantially higher than measured in the susceptibility study, and substantially higher than any measured susceptibility of any virus in any study we are aware of.

There is currently no explanation for these large discrepancies, and the only thing we can say with confidence is that currently available susceptibility and efficacy data appear to be inconsistent. Combined with the variation in susceptibility measurements across multiple studies, this incongruence is an additional reason that standardization and validation of experiment methodologies needs to be a high priority. It may also prove to be the case that there are more fundamental reasons for why findings in small-chamber experiments do not scale to whole room irradiation, that are yet to be elucidated.

Deriving clean air delivery rates from k and assumption of uniform dose

The assumption of uniform dose allows for an alternative method for estimating the clean air delivery rate of a far-UVC device. It suffers from all the same challenges as deriving eACH but gives as a point of comparison at the level of the device.

Fluence rate (I) is conceptually equivalent to the radiant power of the source (i.e. total photon energy emitted per second), the average distance the photons travel and the volume of the space[65].

Equation 3.12

$$I = \frac{P \cdot L}{V}$$

with P = UV power output (Watts)
L = average photon pathlength (m)
V = volume (m³)

This equation for I can be substituted into equation 3.10:

Equation 3.13

$$\text{eACH} = \frac{P \cdot L}{V} \cdot k \cdot 3.6$$

This equation can then be substituted into the definition of CADR (equation 3.5), which results in the Volume terms cancelling out:

Equation 3.14a

$$\text{CADR} = P \cdot L \cdot k \cdot 3.6$$

Note that in equation 3.14a the unit of CADR is m³/hr and P is Watts. For convenient units, CADR is ideally expressed in L/s, k in cm²/mJ and P in mW, giving equation 3.14b:

Equation 3.14b

$$\text{CADR} = P \cdot L \cdot k \cdot 0.1$$

with CADR = clean air delivery rate (L/s)
P = UV power output (mW)
L = average photon pathlength (m)
K = susceptibility constant (cm²/mJ)

Alternatively, if the desired unit of CADR is cubic feet per minute (cfm), P × L × k is multiplied by 0.212 instead of 0.1.

Comparing far-UVC efficacy to other air cleaning technologies

UV (including far-UVC) and other air cleaning technologies have been compared in modeling studies, as well as in aerosol chamber experiments. One modeling study, using the same framework for modeling efficacy described above, estimated that the UV-based methods in a prototypical office provide pathogen inactivation with lower energy consumption and less thermal discomfort than ventilation[62]. In the lab, Landry et al., 2025[66] found that while HEPA filtration achieved the highest efficacy when tested against a UV-resistant bacteriophage, far-UVC systems likely outperform HEPA in inactivating more UV-susceptible human pathogens such as SARS-CoV-2 and Influenza A(H3N2). There is a need for many more modeling exercises and laboratory-based studies comparing technologies head to head, and the costs and benefits of various air cleaning technologies need to be analyzed holistically to include not just CADR/eACH but also energy consumption, noise and thermal comfort.

In order to illustrate hypothetical CADRs in this framework, we assume 100 mW of far-UVC output from a high-powered undiffused device, and 45 mW from a diffused device (see *Emitters and luminaires* section). If we further assume that ceiling height is a reasonable approximation of average photon pathlength for a ceiling-mounted device, in a space with ceiling height of 2.5 meters we can estimate CADRs using equation 3.14b:

TABLE 3.7. Estimated CADR (L/s)—ray length 2.5 m.

Estimated k	100 mW	45 mW
10	250	112.5
1	25	11.3
0.1	2.5	1.1

For spaces with higher ceilings and therefore a longer photon pathlength, the CADR is higher:

TABLE 3.8. Estimated CADR (L/s)—ray length 4m.

Estimated k	100 mW	45 mW
10	400	180
1	40	18
0.1	4	1.8

These tables illustrate the significance of the current uncertainty about the susceptibility of pathogens contained within human respiratory aerosols. The relative cost-effectiveness of far-UVC relative to other air cleaning technologies will be strongly dependent on differences in the inactivation constant k and also on the average UV ray length in a particular installation.

In terms of energy use, the most efficient commercially available portable air cleaner according to energystar.gov has a clean air delivery rate against smoke of 6.8 (L/s)/Watt, although it is worth noting that this is a small device with a CADR of 58 L/s. Standard ASHRAE 241 *Control of Infectious Aerosols* credits filtration-based portable air cleaners for efficacy against bioaerosols based on a weighted average of their CADR against smoke, dust and pollen with a 30/40/40 weighting (see *Guidelines, standards, and regulations* section). CADRs of portable air cleaners typically do not diverge substantially across these different challenge agents.

Based on a hypothetical 12 W power input, we can compare the CADR/Watt to an efficient HEPA-based portable air cleaner:

TABLE 3.9. Estimated CADR (L/s)/Watt: 2.5 m ray length, 12 W input

Estimated k	100 mW	45 mW
10	20.8	9.4
1	2.1	0.9
0.1	0.21	0.09

This analysis suggests that current far-UVC devices can be more energy-efficient than even the best available portable HEPA filters per unit of disinfection, provided that the pathogen k is above ~3.5 cm²/mJ when ceiling heights are relatively low.

TABLE 3.10. Estimated CADR (cfm)/Watt: 4 m ray length, 12 W input

Estimated k	100 mW	45 mW
10	33.3	15.0
1	3.3	1.5
0.1	0.33	0.15

The longer the photon pathlength, the more effective far-UVC is per unit of energy, and the k value at which far-UVC becomes more energy-efficient than HEPA-based portable air cleaners will also be lower.

MERV 13 filter-based air cleaners

DIY PC-fan-based Corsi-Rosenthal boxes using MERV 13 filters are considerably more energy-efficient and quieter than HEPA-based air cleaners. One typical kit available for purchase has been shown in tests by Intertek to deliver 401 cfm (189 L/s) of clean air against smoke using just 14 W of input power, or 13.5 (L/s)/Watt[67]. That would make it as energy-efficient as a 100 mW output/12 W input far-UVC device for a pathogen with a k of ~6.5 cm²/mJ with a 2.5 m photon pathlength, and more than twice as energy-efficient as the best available HEPA-based portable air cleaner. We take no view as to whether this particular example of a DIY Corsi-Rosenthal box is the best in its class, but rather it highlights the significant differences in potential performance of MERV 13-based air cleaners versus commercially available HEPA-based air cleaners.

In terms of noise, the sound power level of this particular Corsi-Rosenthal box is only 39 dBA, which is an order of magnitude lower than the sound output of HEPA-based portable air cleaners with comparable CADR. For example, one HEPA-based device delivers approximately 400 cfm (189 L/s) on the highest fan setting but with a sound power level of 53 dBA. As decibels are a log scale, with sound doubling every 3 dBA, 53 dBA is approximately 32 times louder than 39 dBA[68].

These noise levels are problematic in many situations. For example, the ANSI/ASA standard for acceptable background noise in classrooms is 35 dBA[69]. Since a device's sound power level, as quoted by manufacturers, is typically measured 1 m away, the portable air cleaners quoted above need to be placed more than 1 meter away from any students in order to meet this standard.

We believe that the promise of portable air cleaners optimized for energy efficiency and sound reduction merits further investigation, although this is outside of the scope of the current document. When deciding whether to prioritize research into far-UVC and whether it is sufficiently promising, we should consider the best possible versions of alternative technologies that could also be worth investing resources into.

Key takeaways

- Far-UVC devices can achieve significant clean air delivery rates (CADR), with higher effectiveness in rooms with greater ceiling heights.
- Far-UVC devices can be more energy-efficient than portable HEPA filters, especially for pathogens with higher inactivation constants (k).
- Portable HEPA filters can have a CADR of up to 600 cfm at their highest settings but require regular filter replacements and produce considerable noise.
- MERV 13 filter-based air cleaners, such as Corsi-Rosenthal boxes, can be quieter and more energy-efficient than HEPA filters but may still fall short of far-UVC's effectiveness under optimal conditions.

The need to model distributions and not averages

All of the foregoing analysis makes two critical simplifying assumptions. The first is that all pathogens in a population are subject to the same average fluence rate. The second is that there is a single k that captures the dose-response relationship to the pathogen.

Relaxing either of these assumptions introduces substantial modeling complexity. However, this complexity may be unavoidable if we want to make accurate predictions.

The effect of non-uniform dose can be illustrated by a simple example. Suppose that instead of a pathogen receiving a uniform dose, half of the pathogen receives 2x of the average dose, and half 0.5x the average dose. The average dose remains unchanged. Or to express it algebraically for a dose D:

$S_{Uniform} = e^{-kD}$

$S_{Non\text{-}uniform} = (e^{-k0.5D} + e^{-k2D})/2$

If we plot $S_{Uniform}$ and $S_{Non\text{-}uniform}$ on a log scale by dose, assuming a k of 1, it can be seen that the more inactivation you wish to achieve, the more misleading the assumption of uniform dose:

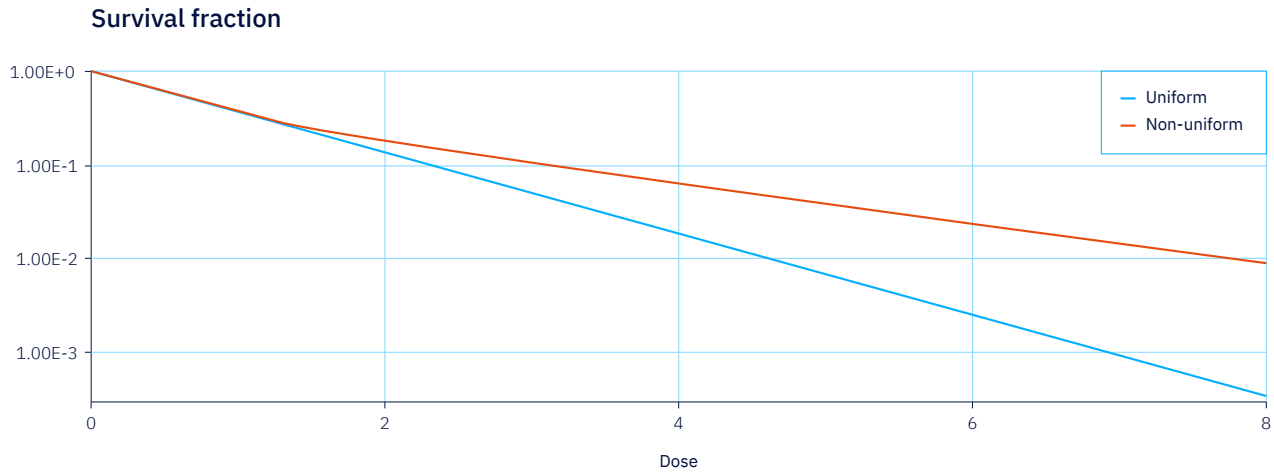

FIGURE 3.2. Survival fraction assuming uniform and non-uniform dose for a k of 1. Analysis by Blueprint Biosecurity.

In this instance, at a dose of 8 mJ/cm², assumption of uniform dose would overpredict inactivation by one and a half orders of magnitude (~2 log versus ~3.5 log). On the other hand, for the first ~log of inactivation it is relatively accurate. Another way of putting this is that at low levels of inactivation the assumption of average dose may in some circumstances suffice, but at higher levels of inactivation what matters is not the average but the tail of the dose distribution.

This same dynamic applies to biexponential decay, assuming we can apply the susceptibility of only the most susceptible fraction. As with uniform dose, this assumption works relatively well for levels of inactivation that are less than the size of the susceptible fraction. But if we take Welch et al., 2022's[34] biexponential function for decay of HCoV-OC43, applying the k to only the 99.7% susceptible fraction would lead us to vastly overestimate reductions at higher doses than if the full biexponential is modeled.

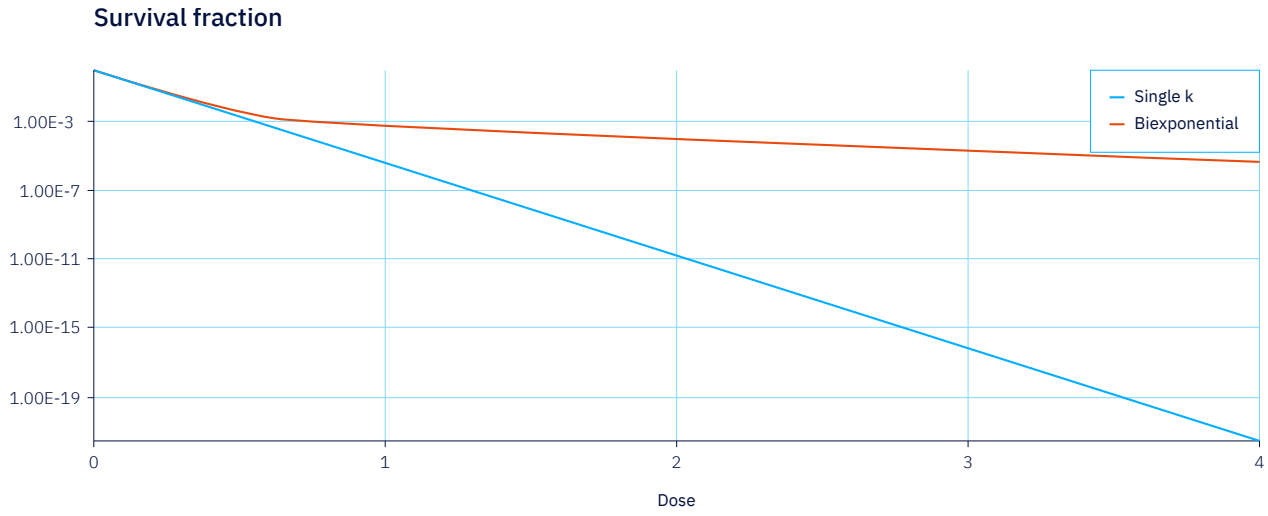

FIGURE 3.3. Biexponential decay versus single k value survival fractions. Analysis by Blueprint Biosecurity.

It is not clear whether we should expect multi-phase decay to occur at room scale with pathogens contained within human respiratory aerosols even if it does occur at the scale of a benchtop aerosol chamber. However, the consequence of relaxing the assumption of uniform dose and single decay constant is that there cannot be a single eACH or CADR for far-UVC, even for a single pathogen. What non-uniform dose and multi-phase decay show us is that the first reductions in pathogen load will come more easily than later reductions, and the marginal eACH or CADR gained from increasing the far-UVC fluence rate would have diminishing marginal returns.

In order to understand the magnitude of this effect, computational fluid dynamics modeling is likely to be required in order to produce realistic estimates of dose distribution in real-world environments. This will allow for both more sophisticated estimates of efficacy based on susceptibility studies, and for translating between efficacy and susceptibility studies and understanding whether or not they are consistent.

A recent innovation is DNA-tagged aerosol tracers, which can be safely aerosolized in a room and used as a surrogate for quantifying pathogen inactivation[26]. If the susceptibility of the tracer is well-characterized under the relevant environmental conditions, this could help us to empirically validate fluence rates and dose distribution in real-world environments.

When we assume these averages—single k, uniform dose—far-UVC seems promising. But we need to move beyond these assumptions in order to establish what is achievable in practical application, and to gain a better understanding of the relationship between far-UVC efficacy and susceptibility.

Key takeaways

- The assumption of uniform dose and a single dose-response relationship allows for simple estimation of eACH and CADR from susceptibility data.
- This simple estimation suggests that at realistic susceptibility, far-UVC may have highly favorable efficacy compared to other air cleaning technologies.
- However, when using these assumptions, published susceptibility and efficacy data are currently inconsistent.
- As well as obtaining more efficacy and susceptibility data using standardized experiment methodologies, more sophisticated computational fluid dynamics modeling is necessary in order to relax the assumption of averages and obtain more realistic estimates of efficacy, and better inference of susceptibility from efficacy studies.
- DNA-tagged aerosol tracers represent a promising approach for validating modeling and quantifying dose and dose distribution in the field.

Challenge studies

Challenge studies—where transmission is deliberately induced in a controlled experimental setting—could advance our understanding of far-UVC in three ways. First, they could explore the dynamics of transmission; second, they could act as a powerful proof of concept; and third, they could assist in the validation of the engineering framework for modeling efficacy.

Animal-to-animal studies provide valuable insights into transmission mechanisms and the environmental factors that influence them.

Human-to-animal challenge studies have played an important role in the development of upper-room UV, and their results will be the basis of the next generation of deployment guidance for ensuring effective installations. Human-to-human challenge studies are a potentially powerful model for proving the concept of interventions like far-UVC, but even if deliberately causing mild human infections to aid research is ethically acceptable, there are a number of practical challenges in human-to-human studies of airborne transmission that have not yet been successfully overcome.

Animal-to-animal challenge studies

Animal-to-animal challenge studies are a valuable tool for examining the dynamics of airborne infection in controlled environments. By studying how pathogens such as influenza virus, SARS-CoV-2, or *M. tuberculosis* transmit between animals such as ferrets, guinea pigs, or hamsters, these studies provide key insights into factors like aerosol particle size, environmental conditions, and infectious dose[70]. Research such as Zhou et al., 2018 and Sutton et al., 2014 demonstrates the utility of these models in studying transmission mechanisms[56,71], while studies like Imai et al., 2012 highlight their role in understanding how viral mutations can enhance transmissibility[72].

Animal-to-animal models can also help us bound expectations for the effectiveness of interventions. For example, Rockey et al., 2024 showed that ventilation alone did not significantly reduce influenza transmission in ferrets, highlighting the role of close-range interactions and fomites in disease spread[73]. Blocking transmission between two animal cages or enclosures using far-UVC would provide useful preliminary data, but falls short of what is needed to justify deploying far-UVC technology in real-world public spaces. The physiological and behavioral differences between animals and humans, combined with the highly controlled nature of these experiments, limit their direct applicability to human environments. These studies can be a useful first step, but stronger evidence is necessary to translate findings into actionable interventions for public health.

Human-to-animal challenge studies

Animal challenge studies, particularly those involving guinea pigs, have been widely used to study airborne transmission of respiratory pathogens and to evaluate interventions such as upper-room or in-duct ultraviolet germicidal irradiation. In these experiments, guinea pigs are exposed to air exhausted from spaces occupied by infected humans, effectively serving as a model for human-to-animal transmission.

Notably, Riley et al., 1962 provided pivotal evidence that tuberculosis is transmitted through airborne droplet nuclei using the human-to-guinea pig model, reshaping public health strategies and solidifying the importance of airborne precautions[74]. This work laid the foundation for the development of interventions like upper-room UV and continues to inform the next generation of deployment guidance for ensuring effective installations. One of the main advantages of this model is its ability to directly link human infectiousness to animal infection outcomes. Additionally, the guinea pig's high sensitivity to pathogens such as *Mycobacterium tuberculosis* makes it particularly useful for tuberculosis research. Several studies have leveraged this model to test the efficacy of airborne infection control measures, including upper-room UV, in real-world settings[75,76]. These studies provide an infection endpoint, allowing researchers to measure the direct impact of interventions on transmission, and have formed the basis for dosing guidelines for upper-room UV[77]. Clark et al., 2015 further emphasized the utility of this model for assessing infectiousness and intervention efficacy under controlled conditions[78]. Beyond tuberculosis, Russell-Lodrigue et al., 2006 and Lowen et al., 2006 demonstrated the utility of guinea pigs as models for studying aerosol transmission of pathogens like *Coxiella burnetii* and influenza viruses, respectively, emphasizing their value for evaluating transmission dynamics and environmental factors such as particle size and temperature[79,80].

However, human-to-animal transmission studies also have limitations. They rely on indirect measures of human infectiousness, as the outcomes are observed in animals rather than humans. Furthermore, the physiological and immunological differences between humans and guinea pigs may limit the generalizability of findings. Despite these limitations, the model remains invaluable for studying complex transmission dynamics and testing interventions under realistic conditions.

This framework highlights the potential for using human-to-animal studies to assess the efficacy of far-UVC in reducing airborne transmission of pathogens in real-world scenarios. Such studies could provide critical data on far-UVC's performance in treating human-generated aerosols.

Human challenge studies

Human challenge trials have historically provided insights into infectious diseases and their transmission dynamics. These studies, where volunteers are deliberately exposed to pathogens, allow researchers to control variables that are impossible to isolate in natural settings, yielding high-resolution data on infection progression, immune responses, and the efficacy of interventions.

One of the most notable examples of the utility of human challenge trials was at the UK Common Cold Unit[81]. This facility was instrumental in the first identification and description of coronaviruses in the 1960s. By exposing volunteers to nasal secretions or throat washes from infected individuals, researchers uncovered critical details about these pathogens, setting the stage for decades of virological research.

Influenza studies have also benefited greatly from this approach. An oft-cited review of influenza human challenge trials is an important resource on the temporality of influenza infections, revealing the timeline of viral replication, symptom onset, and immune response[82]. Norovirus challenge trials have been instrumental in demonstrating the exceptionally low infectious dose (ID50) of the virus, illuminating the dose-response relationship and refining risk assessments for food and water safety[83–88]. More recently, human challenge trials have expanded to include SARS-CoV-2, where participants were inoculated with pre-alpha wild-type SARS-CoV-2 via intranasal drops to provide data on viral emissions into air and environmental persistence[89].

However, while these studies provide valuable data, they primarily rely on direct inoculation (exposure) of participants via methods such as intranasal drops or inhalation through face masks. This limits their applicability to interventions like far-UVC, which aim to interrupt human-to-human transmission. Achieving this requires a shift to studies that more closely mimic real-world conditions of transmission.

Attempts at such designs include Gwaltney et al., 1978, where volunteers were exposed to infected individuals through touch, coughing, and sneezing to investigate routes of transmission[90]. While controversial, the study provides a positive example of how to study human-to-human transmission. More recently, EMIT-2 (currently recruiting) trials have focused on airborne transmission in realistic settings[91]. However, transmission of many infectious diseases spikes and then declines rapidly, making recruitment challenging. It is particularly difficult to recruit participants early in infection, when they may be shedding the most virus, sometimes even before symptom onset. A previous challenge study design using inoculated participants to try and infect other participants failed to achieve consistent human-to-human transmission due to limited viral shedding by the inoculated participants[92].

Despite these difficulties, human-to-human transmission trials represent a potentially powerful tool for testing interventions like far-UVC. If perfected, they could be used to directly evaluate far-UVC's ability to interrupt transmission and allow for direct comparisons against other interventions like ventilation and filtration, or even masking, hand washing, or vaccination. Addressing the challenges of recruitment, participant shedding variability, and study design will be critical to unlocking the full potential of these trials.

Key takeaways

- There is not currently a successful model for human challenge studies of respiratory illness that is relevant to far-UVC.
- Animal study designs are feasible, but may be better characterized as 'efficacy' studies.

Mechanism of action

How far-UVC inactivates pathogens

Far-UVC is believed to operate through a dual mechanism of action. It is absorbed by both the nucleic acids (DNA and RNA) and proteins of pathogens, leading to damage and inability to infect and/or replicate. This dual capability distinguishes far-UVC from traditional UV-C at 254 nm.

The most well-understood mechanism by which far-UVC inactivates pathogens is through DNA and RNA damage. Like 254-nm UV-C, far-UVC photons are absorbed by nucleic acids, leading to the formation of cyclobutane pyrimidine dimers (CPDs) and 6-4 photoproducts (6-4PPs). CPDs are covalent linkages between adjacent pyrimidine bases (thymine/uracil or cytosine, although thymine-thymine dimers are the most important) which prevent proper replication and transcription. This renders microbes non-viable and neutralizes their ability to cause infection. The density of adjacent thymine or uracil nucleotides in a pathogen's genetic sequence is an important predictor of its susceptibility to conventional UVC radiation[36].

In addition to CPDs, far-UVC also induces the formation of 6-4 photoproducts, which are cross-links between adjacent pyrimidine bases but involve different chemical bonds than CPDs. The formation of these photoproducts further disrupts the genetic material of pathogens, adding another layer of damage that impairs repair mechanisms and, as a consequence, the overall function and viability of the pathogen.

Beyond nucleic acid damage, far-UVC's shorter wavelengths allow it to be more readily absorbed by the peptide bonds of proteins, which are abundant in the structural components of pathogens, such as viral capsids and bacterial cell walls. The absorption of far-UVC by these proteins can lead to the disruption of their three-dimensional structure, impairing their function. This disruption is critical because proteins are involved in virtually all biological processes within a cell or virus, including those necessary for the pathogen's survival and ability to infect hosts.

A comparison of assays measuring genome integrity, capsid integrity, and binding integrity showed clear differences between the damage caused by 222-nm far-UVC and conventional UVC, and the importance of this dual mechanism of action[45]:

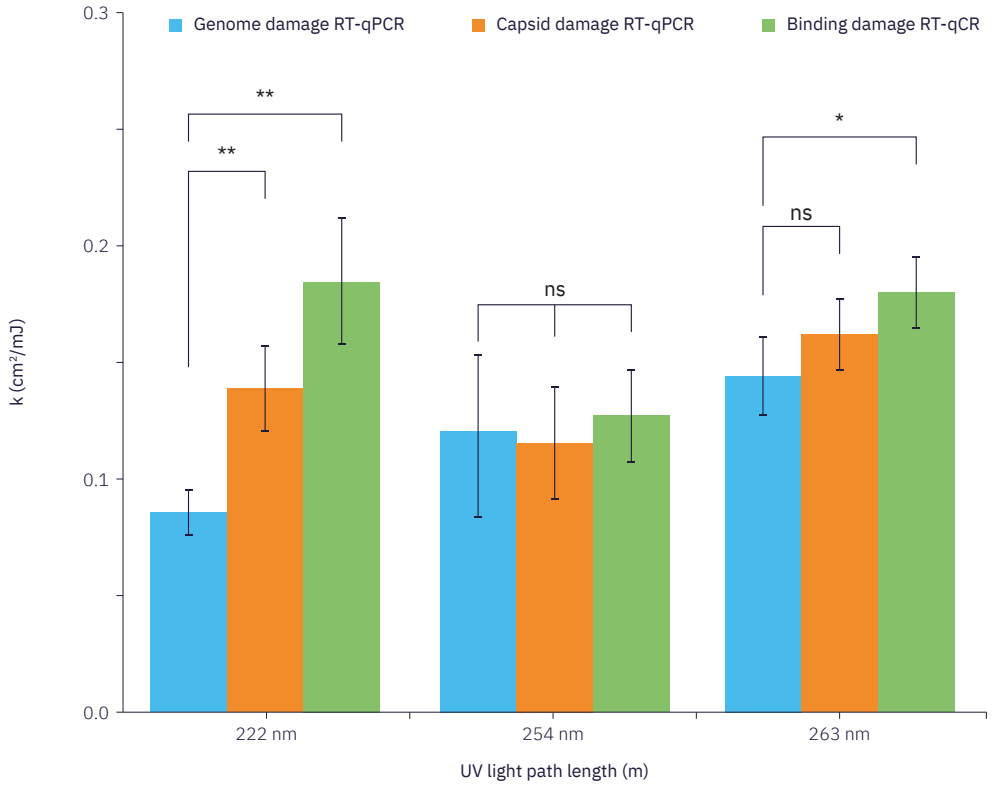

FIGURE 3.4. Excerpted from Lu et al., 2025[45]. UVC damage kinetics of viral RNA measured by standard RT-qPCR (k_{amp}), capsid integrity RT-qPCR ($k_{observed\text{-}capsid}$) and binding damage RT-qPCR ($k_{observed\text{-}binding}$), after 222-nm, 254-nm and 263-nm UVC treatment, respectively. The k values obtained here were calculated from loss of genome copy using the amplicon of nucleocapsid protein genome segment. *p <0.05, **p <0.01, ns represents p >0.05.

As we would expect from the nucleic acid absorption spectrum, 222-nm UV is less efficient than 254-nm at inducing direct genome damage. However, the protein absorption properties of far-UVC are not only qualitatively but also quantitatively important to pathogen susceptibility. For this particular virus (HCoV-OC43) the net effect of both protein and nucleic acid damage was higher susceptibility at 222 nm than 254 nm.

Recent findings suggest that far-UVC may also have an effect on the aerosol droplet itself, which could increase the rate of decay of pathogens within the droplet. Monika et al., 2025 hypothesize that UV could destabilize the protective properties of aerosol droplets, potentially accelerating pathogen decay[58]. While these conclusions remain speculative, they highlight an additional potential mechanism that would enhance far-UVC's germicidal effects against airborne respiratory pathogens.

Acquiring microbial resistance to far-UVC

Unlike antibiotics, which target specific bacterial functions such as protein synthesis or cell wall formation, far-UVC causes widespread and random damage to microbial DNA, RNA, and proteins[32]. The randomness associated with multiple potential pathways for damage makes it harder for pathogens to develop specific resistance mechanisms. Far-UVC disrupts essential processes indiscriminately, reducing the likelihood that a single mutation or adaptation in a pathogen could confer significant survival advantages. However, given there are differences in the susceptibility of different pathogens, the widespread use of far-UVC could place selection pressure on all microbes to evolve the properties of those pathogens that are already less susceptible.

Experimental evidence has shown that certain bacteria, such as extended-spectrum β-lactamase (ESBL)-producing *Escherichia coli*, can develop tolerance after repeated far-UVC exposure[100]. In this study, one bacteria strain exhibited a modest increase in survival after multiple sublethal doses. However, not all bacteria exposed to repetitive far-UVC develop tolerance. For example, another bacterial strain in the same experiment showed no decrease in susceptibility after repeated exposure. In another study, repeated exposure of one strain of *Staphylococcus aureus* to conventional UVC led to a 50% reduction in susceptibility after 7 iterations, but no effect was observed on another strain, nor in *Staphylococcus epidermidis* or *Staphylococcus warneri*[101]. Genomic studies of *E. coli* suggest that the bacteria's increasing tolerance/decreasing susceptibility to conventional UVC may be due to the selection of mutations related to DNA replication and repair[102, 103] or cell membrane structure[104]. Adaptations that occur can come with their own fitness tradeoffs, for example susceptibility to other forms of environmental stress[104].

There is currently no research that we are aware of into susceptibility of viruses to either far-UVC or conventional UVC after repeat exposures. Compared to bacteria, there are fewer ways that viruses can become less susceptible to far-UVC, for example due to their intrinsic lack of repair mechanisms. One study of T7 bacteriophage found reduced susceptibility to 302-nm UVB after serial passage, but it is unclear what (if any) extrapolation should be made to far-UVC[105].

The key question, however, is not whether microbes could evolve under selection pressure to become less susceptible to far-UVC. We should expect this to happen to some degree under sufficient selection pressure. The question is whether the decreased susceptibility would be quantitatively significant. For example, a 50% reduction in susceptibility of *Staphylococcus aureus* from a baseline k of 20 cm^2/mJ to 10 cm^2/mJ would mean that far-UVC would still be likely to remain highly effective. By contrast, a 90% reduction in susceptibility—even from the highest baselines reported in the literature—could result in far-UVC becoming less cost-effective relative to other technologies, or it could mean that the required doses would be higher than acceptable levels for skin and eye safety or ozone generation. If lower susceptibility to far-UVC is observed under realistic conditions of use, it would be prudent to factor this in when determining optimal far-UVC dosing and application guidance.

Despite the potential for reductions in susceptibility under ubiquitous use, far-UVC offers a fundamentally different approach to pathogen control than conventional methods. Its mechanism of random, widespread molecular damage works orthogonally to antibiotics, antiseptics, and traditional cleaning agents, which increasingly face resistance challenges. This independent mechanism of action makes far-UVC a promising complementary tool in a layered approach to infection prevention, particularly for combating pathogens that have developed resistance to chemical and pharmaceutical interventions. Even with potential evolutionary adaptations, far-UVC's physical mode of action would provide a parallel strategy that enhances our overall capacity to control infectious disease transmission.

Key takeaways

- Far-UVC inactivates pathogens through damaging both nucleic acids and proteins.
- There is limited experimental evidence that some bacteria may become less susceptible to far-UVC after repeat exposure, potentially due to selecting for mutations related to DNA replication and repair.
- The impact of any selection pressure that consistent far-UVC exposure would place on viruses is currently unknown. However, viruses are less likely to evolve tolerance to far-UVC than bacteria.

Further reading

- Far-UVC light (222 nm) efficiently and safely inactivates airborne human coronaviruses
- Far-UVC light: A new tool to control the spread of airborne-mediated microbial diseases
- Far-UVC (222 nm) efficiently inactivates an airborne pathogen in a room-sized chamber
- Factors Affecting Reduction of Infectious Aerosols by Far-UVC and Portable HEPA Air Cleaners
- An informal database of UVC Inactivation Constants for both far-UVC and other UV wavelengths has been compiled by Vivian Belenky. This is not a fully exhaustive compendium. Please contact the author at j.vivian.belenky@outlook.com if there is data you believe is missing.

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4. Skin and eye safety

Summary

The acute harmful effects most associated with exposure to UV radiation are erythema (sunburn), photokeratitis (inflammation of the cornea), and DNA damage such as cyclobutane pyrimidine dimers (CPDs) and 6-4 photoproducts (6-4PPs). There is overwhelming evidence that the risks of all of these are significantly reduced for far-UVC wavelengths, as these wavelengths are absorbed by proteins in the outer layers of the skin and eyes, reducing penetration depth.

In addition to erythema, photokeratitis and CPDs/6-4PPs, there will be other biological effects induced by far-UVC, and our understanding of these effects is currently incomplete. Based on currently available data, there is no evidence suggesting that far-UVC has any effects that cause significant short-term or long-term harm within recommended exposure limits. Nevertheless, completing our understanding of the photobiological effects of far-UVC exposure is a clear research priority.

Crucial considerations

- A recent comprehensive journal review concluded that the evidence supports using far-UVC within existing exposure limits, but that further studies are warranted[1].
- As compared to similar doses of UVB or conventional germicidal UVC radiation (i.e., 254 nm), far-UVC exposure to skin and eyes from filtered krypton chloride excimer (KrCl*) lamps, depending on dose, does not result in significant erythema ('sunburn'), photokeratitis ('sunburn in the cornea') or DNA damage. However, many of the recent studies that have been influential in updating our understanding of the effects of far-UVC are from animal models, and there is need for additional human studies.
- Action spectra—relative biological effects of each individual wavelength—are critical to understanding the effects of exposure and avoiding overexposure.
- In situations where deeper layers of skin may be exposed in open wounds (for example, in a hospital's operating theater), the relatively high concentration of protein-rich wound fluids and blood is thought to absorb the far-UVC, protecting underlying tissues.
- While it has not been directly studied, it is believed unlikely that the skin microbiome will be directly affected by far-UVC.
- The effects of protein absorption of far-UVC in the skin, eye, and tear film remain to be elucidated.
- Acute eye overexposure to far-UVC appears to have different effects than conventional germicidal UVC. Overexposure to conventional germicidal UVC causes a delayed onset of photokeratitis, which can be painful and last for many hours. In contrast, overexposure to far-UVC appears to cause a more immediate discomfort that subsides shortly after the exposure.
- There is some research into differences in the effect of far-UVC exposure depending on age, sex, and skin type, but further research is needed and some expert bodies are likely to want this information when updating exposure limits.

Analysis

This section contains many references of different UV doses to skin and eyes. To orient the reader, we provide below the recommended 8-hour exposure limits for different wavelengths of UV, as provided by expert bodies ICNIRP (the International Commission on Non-Ionizing Radiation Protection) and ACGIH (the American Conference of Governmental Industrial Hygienists).

ICNIRP guidelines are intended to be applicable to both working populations and the general public, whereas ACGIH guidance is based on "levels of exposure and conditions under which it is believed that nearly all healthy workers may be repeatedly exposed, day after day, without adverse health effects."[2]

For more information on exposure limits, see *Far-UVC primer* section.

TABLE 4.1. Recommended 8-hour exposure limits for example UVC wavelengths.

Example UV wavelength (nm)	Recommended 8-hour exposure limit (mJ/cm²)		
	ICNIRP	ACGIH (Eye)	ACGIH (Skin)
222 (far-UVC)	23.0	160.7	479.0
230 (far-UVC)	16.0	46.8	158.0
254 (UVC)	6.0	6.0	10.0
270 (UVC)	3.0	3.0	10.0

Note: ICNIRP makes no distinction between skin and eye, and is based on the photokeratitis action spectrum.

Skin safety

Principles of photobiological safety in the skin

The stratum corneum (SC) is the outermost layer of the skin, and is composed of dead, non-dividing cells filled with proteins and lipids[3] (Figure 4.1). These cells form a tough, protective barrier that is regularly shed and replenished. The absence of nuclei in these cells means that there is no risk of the DNA damage and subsequent mutation normally associated with UV exposure, such as the formation of cyclobutane pyrimidine dimers (CPDs) or 6-4 photoproducts (6-4PPs).

Deeper in the epidermis, we find the nucleated but non-dividing stratum granulosum and stratum spinosum. In these strata, a continuous cycle of shedding and replenishment ensures that any damage incurred by far-UVC exposure is both minimal and short-lived. New cells are produced in the lower layers of the epidermis, move to the stratum corneum, and are removed. This process takes around 4 weeks in humans and 8–10 days in mice.

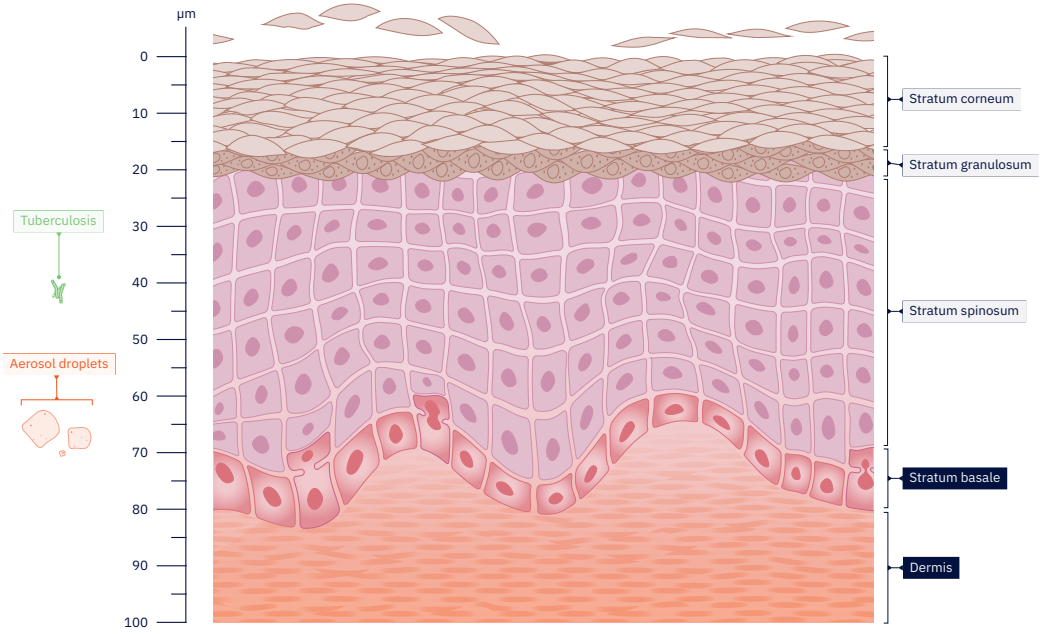


FIGURE 4.1. From Görlitz et al., 2024. The epidermis, with pathogen and aerosol droplets for scale. The outermost layer, the stratum corneum, is made up of dead, flattened cells that form a strong protective barrier. Beneath it lies the stratum granulosum, where cells begin to die and become more flattened as they prepare to move towards the surface. The stratum spinosum contains living cells that are connected by spiny projections, providing structural strength to the skin. The deepest layer, the stratum basale, is where new skin cells are generated through continuous cell division. These cells gradually move upward, maturing through each layer until they reach the stratum corneum, where they form the skin's protective barrier[1].

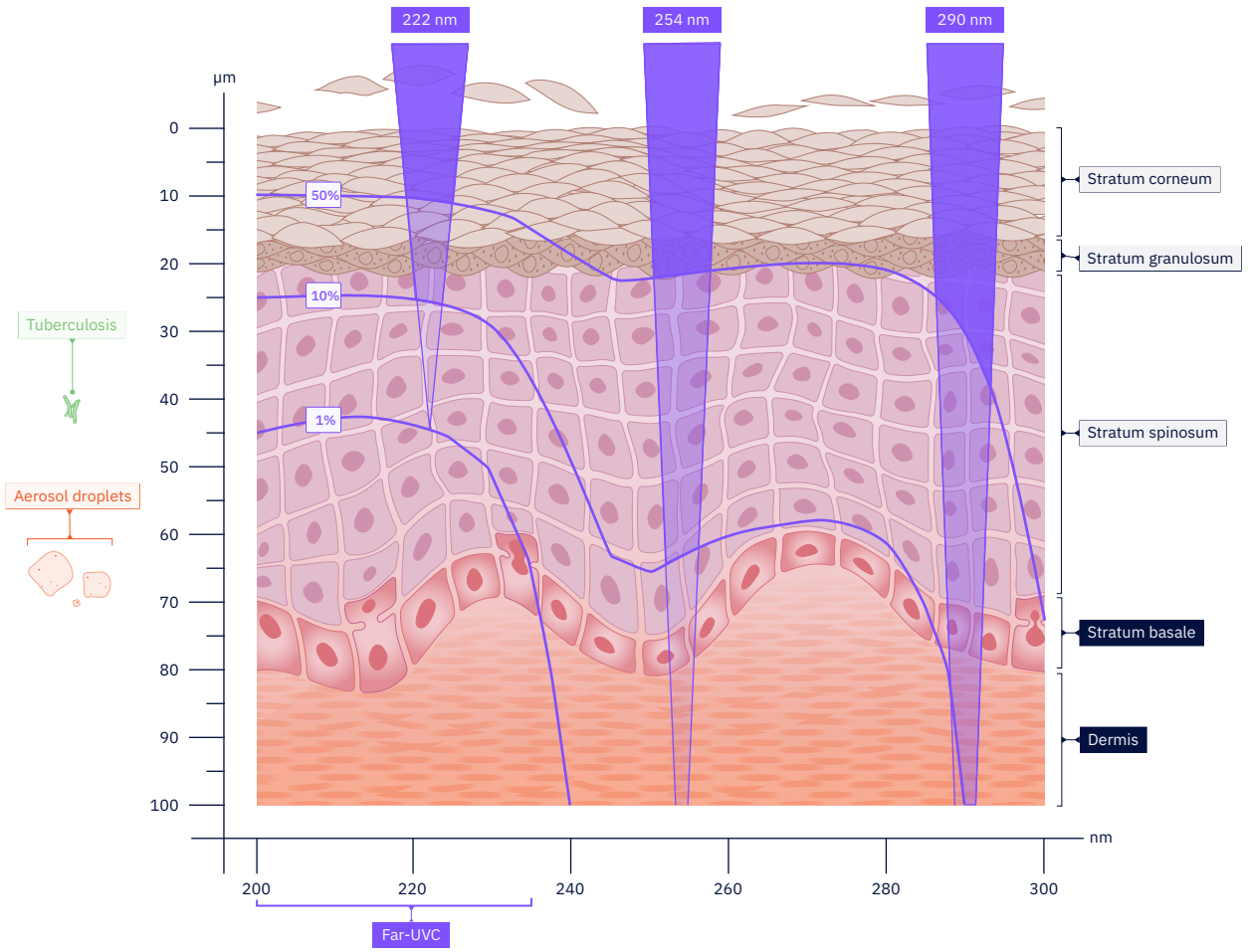


FIGURE 4.2. From Görlitz et al., 2024. Skin penetration depth of UV radiation (200–300 nm) based on a simulation[1,4]. The lines show where 50%, 10%, and 1% of radiation intensity remains after accounting for absorption and scattering.

The depth of UV penetration depends on the wavelength. UV penetration depth into human tissues is substantially driven by protein absorption, which increases significantly at 230–240-nm wavelengths and below. At its simplest level, the reason that far-UVC exposure can be safe for humans while being deadly to pathogens is that pathogens are very small relative to the thickness of the protective protein-rich outer layers of human skin that attenuate far-UVC's penetration to more sensitive tissues.

UVB radiation (280–320 nm) is particularly dangerous, as it can penetrate deeply into the skin while also containing enough energy to cause significant DNA damage. An estimated 1–10% of UVB can reach the basal cell layer, where keratinocytes actively divide and replicate[5]. Melanocytes are also found in the basal cell layer, although these cells do not replicate as frequently as keratinocytes[6]. DNA damage in these dividing cells can lead to mutations that may accumulate over time. DNA is double-stranded and UV damage can lead to breaks in one or both strands, requiring repair. DNA breaks and mutations can lead to skin cancer such as squamous cell carcinoma (see *Evaluating cancer risk* section). This contrasts with far-UVC, in which far less radiation penetrates beyond the outermost, non-dividing layers of the skin. This limited penetration is the key to the safety profile of far-UVC when compared to other forms of UV radiation.

UV radiation that does reach nucleated cells does not always cause lasting DNA damage, as biological organisms have evolved mechanisms to mitigate and repair the damage. Photoreactivation reverses CPDs through enzymatic action requiring blue-light. This can be found in certain members of archaea, bacteria, fungi, viruses, plants, invertebrates, and many vertebrates including aplacental mammals. Excision repair, either base excision repair (BER) or nucleotide excision repair (NER) replaces abnormal or damaged DNA bases. Double-stranded DNA breaks are repaired through homologous recombination (HR) or non-homologous end joining (NHEJ). HR is generally considered an error-free pathway since it requires a complementary DNA strand as a template. NHEJ is more error-prone as it joins broken strands independent of sequence homology[7].

Erythema—colloquially known as 'sunburn'—is the primary acute response experienced in skin as a result of ultraviolet radiation exposure, typically appearing ≥ 24 hours after exposure. Erythema induction has not been observed in any humans exposed to a filtered KrCl* far-UVC lamp, even at a dose 38 times higher than the ACGIH 8-hour exposure limit at 222 or 233 nm[8,9]. The inability of far-UVC to produce erythema, even at high doses[10,11] is due to the protective effect of the outer layer of de-nucleated skin cells. A prior study that did find erythema used an unfiltered krypton chloride lamp, and it is now believed that the erythema was caused by the relatively small but consequential emissions

in the non-far-UVC range[12]. The erythema action spectrum previously did not extend below 250 nm, but new studies are expanding into the far-UVC range[13].

Using 3-D human skin culture models, even at a 222-nm exposure of 500 mJ/cm², only minimal formation of DNA damage immediately below the stratum corneum was observed. No increase in 6-4PPs was detected and only 4% of keratinocytes exhibited CPDs, which is significantly less than what would be expected from sun exposure on a typical day[1,12]. Furthermore, human skin 3-D models irradiated with filtered 222-nm far-UVC had lower CPD yield than those exposed to unfiltered 222 nm or UVB[14,15]. 3-D human skin models exposed to narrowband UV at 5-nm increments between 215 and 255 nm (100 mJ/cm²) revealed no DNA damage in the epidermis (stratum granulosum, stratum spinosum, and stratum basale) between 215–235 nm, while significant DNA damage was observed at higher wavelengths (240–255 nm) in all layers, except the stratum basale at 240 nm[16]. Studies at 233 nm on reconstructed models, ex vivo human skin, and healthy volunteers also support the safety of far-UVC[4,9,17].

Acute exposure to far-UVC did not cause significant numbers of CPDs in animal studies[1,18–23]. Compared to control, only 10,000 mJ/cm² or higher of 222 nm resulted in higher levels of CPDs in the outermost epidermal layer of hairless albino mice or Xpa-knockout mice[24]. When the UVC source was triple filtered, the damage from 10,000 mJ/cm² was greatly reduced, suggesting that the most damage was induced by traces of longer wavelength UVC (>235 nm)[24]. Xpa mice, which have a genetic defect similar to the human DNA repair disease Xeroderma Pigmentosum, are highly susceptible to UV-induced DNA damage and cancer, and would be more likely to show adverse skin damage from far-UVC exposure.

It is uncertain to what extent we can extrapolate findings after acute exposures to those arising from prolonged or chronic irradiation, or extrapolate data from animal experiments to humans[1]. Only a few long-term animal studies have been published, and no long-term human data has been reported[22–25]. One study exposed mice to 222-nm far-UVC for 8 hours a day, five times a week, for 66 weeks and found no induction of skin cancer or other skin abnormalities[25].

Taking the in vitro 3-D skin culture and animal studies together, it is thought that a boundary in the 230–240 nm range exists where there is a sudden and steep decrease in UV penetration depth. This decrease would align with an inability to cause CPD or 6-4PP DNA damage in proliferating cells, with the boundary marking the difference between superficial effects and the more serious effects of the more deeply penetrating UVC wavelengths[1]. More refined action spectra inferred from studies with greater discrimination in the far-UVC range and in the transition region between far-UVC and conventional UVC (for example, through the use of a monochromator to produce emissions of specific wavelengths) are critical for providing accurate 'Lamp Exposure Limits' and preventing overexposure[10,11]. This is discussed in more detail in the *Guidance, standards, and regulations* section.

UV responses in different populations

Epidermal skin thickness depends on multiple factors including age, body location, and demographics. For example, infants aged 3–24 months have a SC 20–30% thinner than adults[26]. The epidermis and SC are thicker on parts of the body exposed to the sun compared to those protected from sunlight[27]. Among normally exposed parts of the body, the palms have the thickest SC while the SC is thinnest on the face and eyelids. The effects of far-UVC exposure on mucosal surfaces, such as the lower lip, is poorly defined. Individuals with defects in the DNA damage/repair responses, or those with polymorphisms that decrease these processes, may be more vulnerable to damaging effects from far-UVC than the general population.

Skin type and melanin concentration may influence the impact of far-UVC, as melanin provides photoprotection by absorbing broadband UV and scavenging radicals. This antioxidant property reduces UVR penetration into keratinocyte nuclei, offering greater protection for individuals with higher melanin concentration. Experiments employing far-UVC are essential to determine any potential impacts, if any, of melanin concentration in exposure to far-UVC. Studies have examined the relationship between UV-induced DNA damage and melanin content/distribution in ex vivo human skin exposed to 222-nm and 233-nm UVC. Generally, lower levels of DNA damage were observed in darker-skinned donors[4]. In contrast, similar studies using reconstructed human epidermis (RHE) models exposed to 233-nm UVC produced opposite results, with tanned RHE showing higher levels of UV-mediated free radicals and DNA damage compared to lighter RHE[28]. Nevertheless, as the damage caused by 233-nm UVC is consistently lower than that produced by UVB at doses considered safe for skin, skin color is not regarded as a significant risk factor for this exposure. In vivo studies on healthy humans using 233 nm are ongoing, and a similar experiment should be conducted at 222 nm using normal exposure levels to ascertain any potential impact of melanin concentration.

Other photobiological effects of UV on skin

In addition to erythema, there are alternative measurable outcomes that may be relevant for far-UVC given the differences in its properties compared to UVA, UVB and conventional UVC radiation. For this reason, it is a priority to conduct observational and mechanistic studies to better understand and identify outcome biomarkers and endpoints. Inclusion of biomarkers of DNA damage other than CPDs and 6-4PPs, such as γh2AX foci for double-strand breaks, is valuable[29]. It's crucial to include this information in larger, longer-term safety studies in diverse populations to create more informative action spectra.

Skin yellowing

Visible yellow discoloration was observed on the forearm skin of one investigator after a dose of at least 6,000 mJ/cm² (which is more than 10x higher than the current 8-hr ACGIH exposure limit at 222 nm). However, this appeared to be localized to the stratum corneum, as evidenced by the fact that skin tape stripping reduced the discoloration immediately. Zamudio Díaz et al., 2023 also observed skin yellowing 1 hour after exposure to 60 mJ/cm² 233 nm, with recovery after 24 hours[9]. The mechanism behind this phenomenon is not completely understood, but UV-induced aldehyde production or protein oxidation is possible[30]. Lipid peroxidation and yellowing are observed in solar skin photoaging, suggesting a potential mechanism[1,8,9,31–34]. The yellowing is not thought to be a byproduct of erythema or to result from DNA damage. No erythema has been reported in human subjects, even after receiving high doses. The effect should also be observed in more than one subject. Understanding the underlying cause of this change in the stratum corneum and creating an action spectrum for that effect should be a priority.

Immune suppression

Naturally occurring *trans*-urocanic acid (t-UCA), highly concentrated in the stratum corneum, acts as an endogenous chromophore. This absorbs UV, leading to conversion to the cis-form (c-UCA), and may have an immunosuppressive effect[35,36]. The action spectrum for c-UCA production is primarily in the UVB range. c-UCA may also contribute to UVA-induced photoaging, following UVA exposure, through UVA sensitization. However, the direct mechanisms are not known[37]. While primarily linked to immune suppression, c-UCA has a multitude of effects, including potential links to skin disorders such as atopic dermatitis and urticaria[37]. It remains to be seen if far-UVC will induce c-UCA formation or any of the downstream impacts at all or at the doses used for disinfection. Furthermore, while UVR generally suppresses acquired immunity, there is some evidence that UVR enhances innate immunity by producing antimicrobial peptides and activating immune cells[38]. Whether this is the case for far-UVC remains an open question.

Langerhans cells are distributed throughout the epidermis and recognize pathogens/molecular damage in the skin. UVB damage to these cells induces inflammatory responses, leading to changes in how the skin processes immune responses as well as immunosuppression[39,40]. While not studied yet for 222 nm, 60 mJ/cm² of 233-nm far-UVC showed no significant difference in langerin, a marker for langerhans cells, compared to controls[9].

Microbiome

The hands, arms, neck, eyes, and face are the skin areas most exposed under typical deployment scenarios. The stratum corneum is one site of the skin microbiome, and the hair follicles deep in the skin are one site of the reservoirs for the microbiome[1,41]. Surface disinfection with antimicrobial agents, such as alcohol sanitizers or far-UVC, will most likely only have a transient impact on the microbiome since they don't penetrate to the follicles. It is expected that proximity, exposure duration, number of repeated exposures, and wavelength all may have an impact on the level of microbiome transient disinfection[1]. Investigating the cause of the skin yellowing in the stratum corneum is also important for understanding any impacts of far-UVC exposure on the skin microbiome. Whether there are microbiome impacts depends on the molecular cause of the yellowing and whether this can be spread between layers of the skin or to microbes.

Wounds and burns

One important question is the safety of far-UVC exposure on breaks in the stratum corneum, such as wounds and burns. Hospital-acquired infections remain a serious public health concern, with ~4% of hospitalized patients experiencing a healthcare-associated infection (HAI)[42]. With the rise of antibiotic resistance and increased HAI, disinfection of wounds and burns is another area where far-UVC could be effective, although evaluation of this potential contribution is outside the scope of this report. The original idea for far-UVC use for reducing surgical site infections focused on using the lamps in surgical suites, with the lamps facing downward from the ceiling, potentially irradiating the surgical wound from above[43]. Far-UVC skin exposure limits are higher because of absorption in the stratum corneum, where the cells have no nuclei. In burns and wounds, on the other hand, the stratum corneum is often disrupted, exposing underlying layers of cells that do contain nuclei and therefore could be damaged by far-UVC. However, wounds are often covered with a protein-rich wound fluid[44] which is likely to absorb significant amounts of far-UVC.

The use of 233-nm LEDs as well as 222-nm KrCl* lamps has been evaluated in vitro (outside the body) and in vivo (in animal bodies) for wound disinfection. Initial studies revealed disinfection of superficial wounds on the backs of mice infected with MRSA, with minimal CPD induction, at 40 mJ/cm² 222-nm far-UVC[45]. We'd expect 233-nm far-UVC to be more effective due to penetrating slightly deeper than 222 nm, at the cost of increased CPDs in the upper epidermis. However, in one study, 233 nm was shown to be more effective at disinfection despite producing minimal to no CPDs at the wound site[4]. For superficial surgical site infections and skin infections, 222-nm disinfection appears to be efficacious, with little to no DNA damage[21,45,46].

Key takeaways

- Cumulatively, current evidence suggests little to no harm to the skin from acute far-UVC exposure at a microbicidal/bactericidal dose.
- The outer layer of the skin is composed of non-dividing cells that are regularly shed and replaced. Far-UVC is absorbed by this outer layer, which means it is likely to be less harmful than e.g. UVA and UVB, which penetrate deeper.
- However, the paucity of human studies, chronic exposure studies, and investigation of alternative damage mechanisms renders an incomplete understanding that calls for further research into these areas.

Eye safety

Principles of photobiological safety in the eye

The tear film is the first part of the eye exposed to UV. Tears are an extremely important feature of the ocular surface anatomy, providing a uniform optical surface to minimize aberrations, washing away cellular and external debris, and containing a variety of macromolecules that both nourish the underlying epithelial cell layers as well as proteins designed to perform antimicrobial functions[1,47,48].

The tear film is composed of three layers. The outermost layer is very thin (40–160 nm) and mainly composed of lipids that help prevent evaporation from the underlying and much thicker (3–5 µm) middle aqueous layer. This aqueous layer contains hundreds of proteins and small macromolecules, and its composition can change depending on external stimuli and emotional state. The innermost layer, whose function is to affix the tears to the outer layer of corneal and conjunctival epithelium is a 0.3 µm mucin layer[1,47,48].

Beneath the tear film, the cornea and surrounding conjunctiva are the outward-facing eye structures which interact with light (see Figure 4.3 below). Experimental measurements indicate that the human cornea absorbs wavelengths below 295 nm. Longer wavelengths of UV radiation, however, may reach underlying structures such as the iris and lens[49]. The outermost layers of the corneal epithelium are thought to provide protection similar to the skin stratum corneum, with surface cells being replenished roughly every 7 days[1,50–52].

Unlike the SC, corneal epithelial cells are nucleated. Therefore, DNA damage, including CPDs and 6-4PPs, can be observed in the outermost layer

of corneal epithelial cells after relatively low levels of far-UVC exposure. The cornea is also heavily innervated, with nerve endings terminating in the corneal epithelium and with a wide range of sensory functions[53]. However, these corneal epithelial cells are post-mitotic (non-dividing) and quickly replaced; therefore any DNA damage in these cells is unlikely to cause concern. In contrast, damage to stem cell progenitors in the limbus (at the edge of the cornea), or to basal corneal epithelial cells, might prove more problematic. Nevertheless, no CPDs were observed in limbal stem cells following far-UVC irradiation from a KrCl* lamp at 1,500 mJ/cm², but CPDs were observed at 2,500 mJ/cm² or higher[1,54].

Most conjunctival stem cells are found in the conjunctival fornix, which is located under the eyelids, suggesting that these cells are mostly protected from UV radiation. It is undetermined whether goblet cells, which lie near the surface of the conjunctival epithelium and produce mucin, are at risk for far-UVC-induced damage.

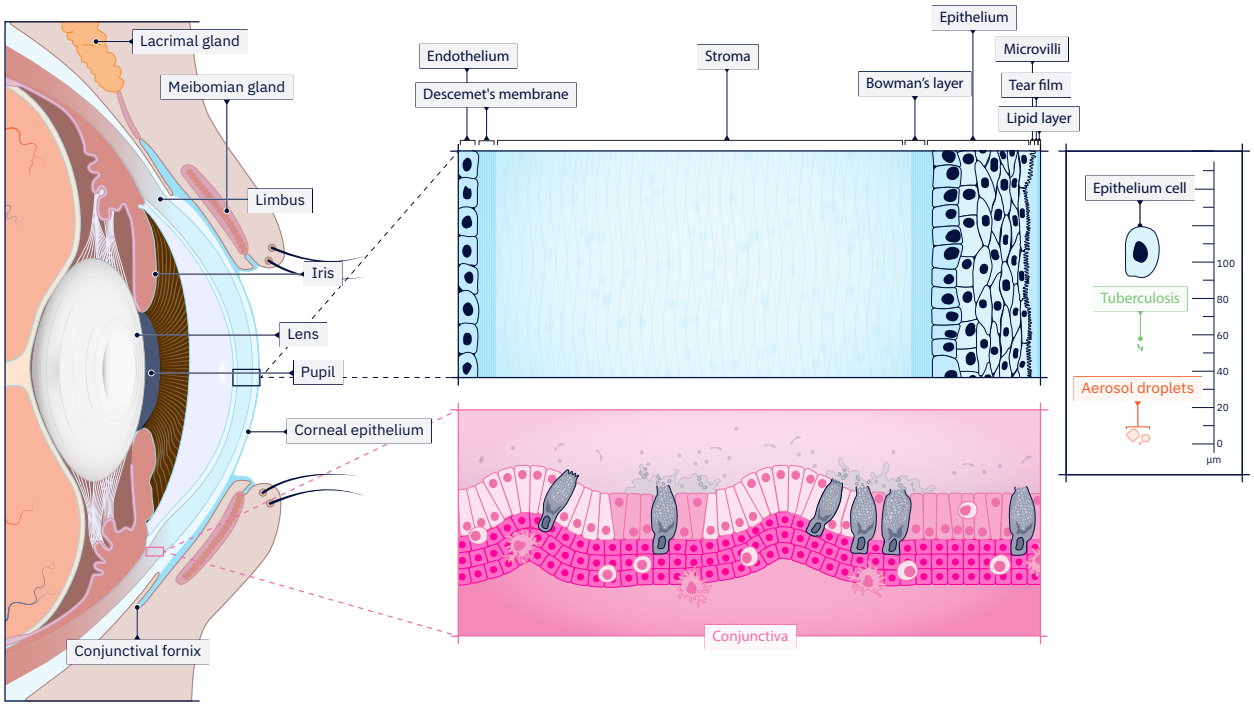


FIGURE 4.3. The corneal epithelium is a dynamic, multilayered tissue essential for maintaining the eye's protective barrier. This epithelium consists of five to six layers of cells, with the deepest layer composed of basal cells[1,53].

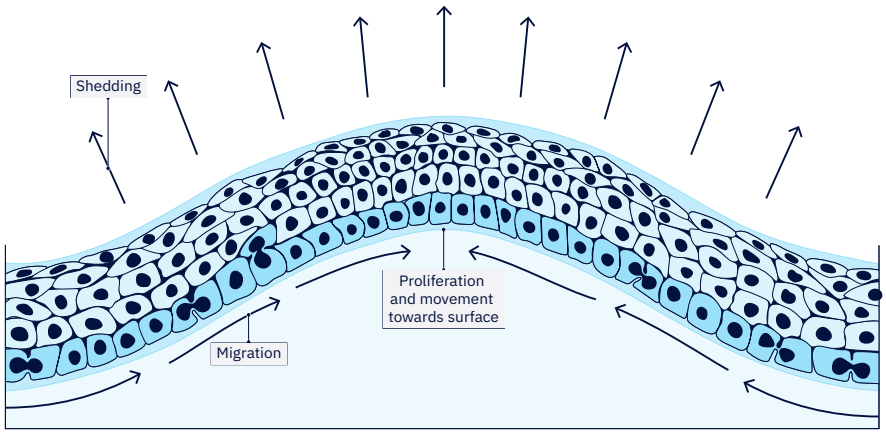
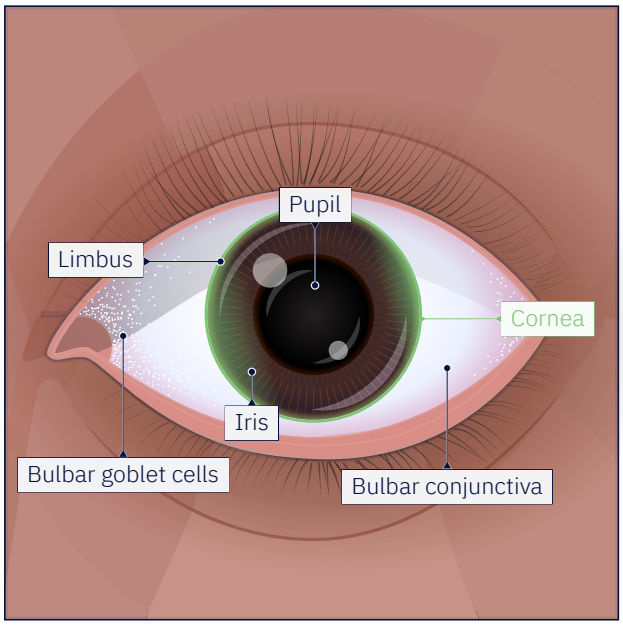
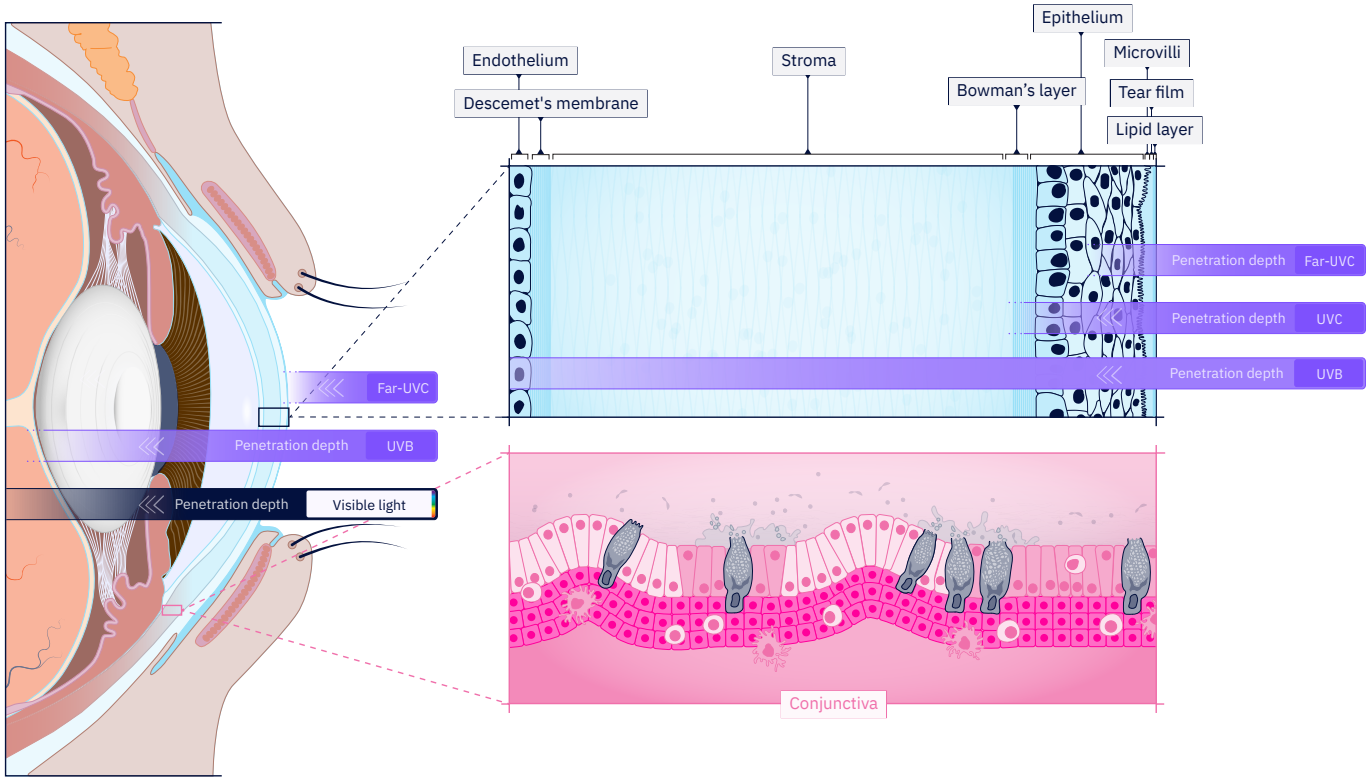


FIGURE 4.4. At the periphery of the cornea, in the region called the limbus, stem cells divide to create transient amplifying cells (TACs). These TACs migrate both centripetally, toward the center of the cornea, and upward through the epithelial layers. As TACs progress, they stop dividing and begin differentiating into wing cells, which make up the intermediate layers. These cells then continue to differentiate into superficial cells, which form the outermost layer. Superficial cells no longer divide and are eventually shed as part of the corneal renewal process. This process typically occurs over a 7–10 day cycle, ensuring that the corneal surface is constantly replenished and its barrier function maintained[55].



← **FIGURE 4.5.** The cornea and bulbar conjunctiva are two parts of the eye exposed to radiation. The cornea is one of the outermost layers of the eye and covers the iris and pupil. The bulbar conjunctiva covers the white part of the eye (sclera) and is where the bulbar goblet cells reside. The bulbar goblet cells lie near the surface of the bulbar conjunctiva and produce mucin, a component of the tear film[56], shown by the green-tinted areas in the illustration.

↓ **FIGURE 4.6.** Far-UVC radiation (200–235 nm) is highly absorbed in the corneal epithelium. Conventional germicidal UVC (around 254 nm) can penetrate deeper, posing higher risks of DNA damage to the replicating cells in the basal layer as well as stromal and endothelial cells[57]. In contrast, because far-UVC is absorbed within the surface layers, it has limited potential to harm the dividing basal cells and deeper tissues[1,58].



Excess exposure to either UVA or UVB is associated with various pathologies of the eye. Both are linked to cataract formation, while UVB is more strongly associated with epithelial metaplasia and overgrowth, called pterygium and pinguicula, and with photokeratitis. UVA can penetrate more deeply into the eye, even reaching the retina where it is associated with macular degeneration, whereas most UVB is absorbed by the cornea and lens. For these reasons, it is important to wear UV-blocking eyewear (that blocks both UVA and UVB) in bright sunlight to reduce excess risk of ocular damage.

The potential for the tear film to attenuate far-UVC absorbance has been the subject of debate. More than 20 peer-reviewed far-UVC papers have claimed that far-UVC is absorbed by the tear film, preventing far-UVC-induced damage to underlying corneal cells and tissue[57,59]. Similar to the stratum corneum of the skin, the tear film contains a relatively high concentration of macromolecules such as proteins, lipids, and sugars, which were thought to attenuate far-UVC penetration to underlying epithelial cells. However, recent studies have shown some CPD formation in the first one or two layers of corneal epithelial cells following far-UVC irradiation in experimental animal studies[54,57,60].

Additionally, a study suggests that the tear film transmits >90% of far-UVC wavelengths between 220–230 nm and that 222-nm doses as low as 3 mJ/cm² lead to reduced viability of cultured human corneal epithelial cells, despite overlay with human tears or tear substitutes of equal protein composition and thickness as in vivo tears[60]. One caveat is that in vitro studies do not always replicate in vivo studies due to the difficulty in replicating an intact tear film containing all three layers. However, physics (Beer's Law) suggests that the tear film will absorb little far-UVC, given its known thickness and known average protein concentration. Some therefore posit that the tear film provides little protection and most far-UVC reaches the outermost epithelial cell layers, where it is absorbed primarily by the proteins in those cells but will also cause some amount of DNA damage. In contrast, the stratum corneum in the skin is de-nucleated, making it impossible for DNA damage to occur in those cells where the majority of far-UVC is absorbed.

Photokeratitis and the causes of eye irritation

Pitts[62,63] reported far-UVC-induced ocular effects in rabbits, primates, and humans, including development of photokeratitis, decreases in visual acuity, and biomicroscopic changes in the cornea. While data from Pitts et al., 1974[62] (using a monochromator with a nominal full-band width of 9.9 nm) is commonly referred to as the photokeratitis action spectrum in humans, implying that the recommended thresholds for human exposure derived from this data are based on avoiding inducing photokeratitis, transient discomfort that presented differently from photokeratitis was observed at the threshold level for wavelengths in the far-UVC region:

> From the above observations, it was felt that the reactions of the cornea to wavebands below 250 nm [were] different from those found with exposure above 250 nm. The signs and symptoms occurred much earlier post-exposure for exposures below 250 nm, and subjective symptoms always returned to normal prior to completion of the experiment. For exposures above 250 nm, the symptoms did not occur until late in the experiment.

For wavelengths above 250 nm, epithelial granules, or small, white, discrete, round spots located in the epithelium were observed. In contrast, wavelengths below 250 nm produced fine, discrete granules, and some subjects reported symptoms including tearing up and a foreign body sensation. Across the studied wavelengths, photophobia (abnormal sensitivity to light) was also variable in presentation and was considered an unreliable indicator of photokeratitis. All subjects who were classified as exhibiting the threshold for photokeratitis demonstrated loss of visual acuity that returned to baseline within six hours. These observations are consistent with those reported in more recent human studies on far-UVC ocular effects.

Research on the acute effects of UV on the eyes usually focuses on the cornea, and photokeratitis specifically refers to inflammation of the cornea. However, the cornea is not the only exposed structure. The conjunctival epithelium is 3–5 cell layers thick. It covers the sclera and lines the inside of the eyelid[56]. Interspersed among conjunctival epithelial cells are goblet cells, whose function is to secrete mucins, an important component of the posterior tear film layer. In addition to goblet cells, other cell types can be found in the conjunctival epithelium, including numerous immunomodulatory cells that migrate through the conjunctiva and stroma. Direct UV irradiation or indirect inflammatory/oxidative stress may impact distribution and function of these cells. Similar to the corneal epithelium, far-UVC radiation may be absorbed by the initial layers of the conjunctival epithelium with unknown adverse health outcomes.

One measure of conjunctival irritation/damage is hyperemia, when the sclera appears red due to blood vessel dilation in the conjunctiva. This is often a response to inflammation (Singh et al., 2021). Sugihara et al., 2024 reported onset of dry eye, conjunctival hyperemia, and mild pain 2–2.5 hours after exposure to far-UVC in 5 participants which disappeared 4.5–11 hours afterwards, employing doses of 22, 50, or 75 mJ/cm² [64]. This observation is similar to that reported by Pitts[62,63] where exposure to 210–230 nm bandwidth radiation resulted in subjective discomfort and conjunctival hyperemia that lessened by 4 hours and disappeared after 6 hours.

While the underlying mechanism is unclear, suggested hypotheses include tear film insufficiency, stimulation or damage to the corneal epithelial nerve plexus, and photo-oxidative production of free radicals in tears[1,65] (Kleinman, personal communication). It is critical that this phenomenon be investigated. Documentation of corneal irritation, the perception or sensation of 'tingling' at the ocular surface, geographic loss of sensitivity (tested via esthesiometry), and other hallmarks of corneal nerve damage, should be evaluated endpoints in future eye studies.

In terms of the consequences of eye irritation, Sliney & Stuck, 2021 noted that an eye sensation of discomfort at higher irradiances may actually reduce the risk of more serious eye exposure because the eyes will automatically close as a reflex to protect them from damage (in the same way that our natural aversion prevents us from looking directly at the sun or bright lights)[65]. Whether or not this is the case depends on the circumstances under which these sensations arise and the underlying biological mechanisms, both of which are not currently well-understood.

Individuals with dry eye or other ocular surface disease may be more vulnerable to far-UVC-induced damage to the corneal and conjunctival epithelium. Given the close proximity of mucin-producing goblet cells to the conjunctival surface, damage to these cells and further loss of tear film adhesion to the ocular surface may exacerbate dry eye symptoms.

Animal studies

Multiple animal studies on the ocular effects of exposure to 207-, 222-, 235-, 254-, and 311-nm UV in rats have been conducted in recent years. Animal studies permit deeper investigation into the effects of UV on the eye, as conducting the same assays in humans would be either impossible or extremely invasive.

Rat eyes exposed to doses of filtered far-UVC (207 and 222 nm), that do induce effects in the case of longer wavelength UV, revealed no significant change in corneal surface integrity, as measured by fluorescein biomicroscopy, histology, and imaging[54,57,60]. Higher exposures (>3,500 mJ/cm²) resulted in measurable damage to the cornea. At supra-threshold doses (1,000 mJ/cm²), both 207 and 222 nm exposure resulted in CPD-positive cells in the most superficial layers of the corneal epithelium and the superior limbus, but not in the inferior limbus[54]. Corneas exposed to 235 nm exhibited CPDs up to the middle layer of the corneal epithelium. Exposure to 254 nm, emitted by a mercury lamp, and 311-nm UVB resulted in CPD-positive cells throughout the corneal epithelium.

More recently, Arden et al., 2024[66] reported no evidence for changes in visual acuity or contrast sensitivity, assessed through optokinetic methodologies, intraocular pressure, or slit lamp detectable corneal damage in a SKH-1 hairless, immuno-competent mouse model chronically exposed to three different intensities of far-UVC over a 66-week period. One limitation of this finding is that exposure was limited to daylight hours when mice were least active.

Studies of actual installations

In a human exposure study, Kousha et al., 2024 simulated an office environment where subjects received a range of doses from 0 to 50 mJ/cm² to the top of the head, five hours per day, over a three-day period[67]. Exposure intensity varied by participant position, with most receiving less than 20 mJ/cm² to the top of the head. Eye dose was not measured. Subjects were queried for self-reported eye discomfort including dry eye, soreness, and fatigue, immediately after and up to one week following exposure. Besides tracking CPDs and other markers of damage, Kousha utilized the Standard Patient Evaluation Eye Dryness (SPEED) and the Ocular Surface Disease Index (OSDI) questionnaires before and after exposure to 222 nm and found no significant change, even with head exposures up to 50 mJ/cm²[67].

Most recently, Sugihara reported no changes in corneal erosion, conjunctival hyperemia, pterygium, or cataract following chronic occupational exposure to 222 nm[68]. Some of these findings are what we would expect, as UV radiation would not be expected to reach the lens (increasing the risk of cataracts) and pterygium is most strongly associated with UVB exposure. Furthermore, the authors reported that the subjects spent less than one hour per day under far-UVC illumination and estimated the daily dose as less than 2.8 mJ/cm². The authors calculated a maximum theoretical far-UVC exposure of 6.4 mJ/cm² as the daily dose theoretically received by a 170-cm tall subject staring at the lamp for eight hours.

The effect of installation design on dose to eyes

In controlled studies and in the recommended photobiological exposure limits, the 'dose to eyes' is intended to represent the actual UV dose that reaches the surface of the eye. However, taking irradiance measurements at eye level without factoring in the particular geometry of the human face can significantly overestimate true eye exposure. For example, in one study using a manikin, only 5.8% of the maximum directly measured dose on the manikin's head, nose and lip was measured on the eye surface[69]. Furthermore, natural anatomical features absent in manikins, such as eyebrows, eyelids, and the top of the head, further shield the eyes from direct exposure to overhead lighting fixtures[69,70].

Taken together, these considerations support the idea that not mounting far-UVC devices on the wall at eye level, and ideally mounting them on the ceiling, likely provides a significant 'safety margin' for eye exposure. The interpretation of photobiological exposure limits incorporated into the UL 8802 standard reflects this fact (see *Guidance, standards, and regulations* section).

Key takeaways

- The outer layers of the eye are mainly composed of non-dividing cells that are regularly shed and replaced. Far-UVC is absorbed by this outer layer, which means that it is likely to be less harmful than e.g. UVA and UVB, which penetrate deeper.
- While the cornea is the usual site for studying far-UVC's effects on the eye, the effects on other ocular structures such as the conjunctiva should be included in future research studies.
- Non-traditional outcome measures are important to develop and assess, as a complete safety profile across different body structures has not been established.
- While initially thought to confer full protection, recent studies suggest that the eye's tear film layer does not significantly attenuate far-UVC radiation.
- The effects of absorption of far-UVC in the outer layer of the human corneal epithelium, especially with respect to anecdotal reports of corneal discomfort after irradiation, are under active investigation.

Further reading

- Assessing the safety of new germicidal far-UVC technologies
- Extreme Exposure to Filtered Far-UVC: A Case Study
- One-year Ocular Safety Observation of Workers and Estimations of Microorganism Inactivation Efficacy in the Room Irradiated with 222-nm Far Ultraviolet-C Lamps

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5. Evaluating cancer risk from different UV wavelengths

Summary

Animal studies commonly used to assess cancer risk from UV exposure have not shown an increase in cancer risk from far-UVC. This observation is consistent with biophysical considerations that limit far-UVC penetration into the epidermis (see *Skin and eye safety* section).

UV radiation as a whole is classed as a carcinogen by expert bodies such as the US National Toxicology Program (NTP) and the International Agency for Research on Cancer (IARC), and far-UVC currently falls under this general classification. The magnitude of cancer risk is not factored into these classifications. Amassing a greater body of evidence may be necessary in order for far-UVC to be formally classified separately from other forms of UV. Further animal studies to evaluate far-UVC cancer risk are warranted, as well as mechanistic investigations into the range of DNA damage types induced by exposure to far-UVC radiation, which may be different from those induced by exposure to longer-wavelength UV.

There are three major types of skin cancer—melanoma skin cancer (MSC), and basal cell and squamous cell carcinomas (termed non-melanoma skin cancers, NMSC). Sun exposure is a risk factor for the more common NMSC form, with squamous cell carcinoma linked to cumulative, long-term chronic lifetime exposure, and basal cell carcinoma more strongly linked to episodic intermittent sunburn.

UVB rays (280–315 nm) found in sunlight are the primary driver of NMSC due to their ability to directly damage DNA, causing the formation of pyrimidine dimers. In contrast, UVA (315–400 nm) induces NMSC by an indirect mechanism involving oxidative stress and free radical production which can lead to DNA damage. Lifetime sunlight UVA exposure also causes age-related photodamage to the skin, as UVA penetrates deeper and can damage the proteins elastin and collagen.

Both UVA and UVB are also implicated in risk for the much rarer but more deadly melanoma, although through different mechanisms. UVB exposure is linked to direct damage to tumor suppressor genes and subsequent carcinogenesis. UVA exposure is associated with immune suppression and altered cell signaling, in addition to indirect DNA damage to tumor suppressor genes, both of which increase the likelihood that nascent melanoma cells will evade the body's cancer detection systems and survive.

The rarity of chronic human exposures to UVC limits our ability to gather statistically relevant epidemiological data. The non-UVC emissions of the most common man-made UVC sources (such as from arc-welding equipment) reduce certainty about which specific wavelengths pose the greatest risk for carcinogenesis. Much of the evidence for the mutagenic potential of UVC radiation is therefore based on its ability to induce DNA damage similar to that of UVB. Animal studies have also demonstrated tumor formation after exposure to low-pressure mercury lamps, which primarily emit 254-nm UVC but also produce ~4% UVB radiation.

Finally, if either conventional UVC or far-UVC were more widely deployed as a germicidal tool, it would be prudent to conduct observational studies assessing its long-term effects across a wide variety of health outcomes.

Crucial considerations

- Non-melanoma skin cancer (NMSC) is well-studied, with known epidemiological and mechanistic links to UVA and UVB exposure.
- A few current mouse studies suggest that far-UVC does not induce cancer formation, even in cancer-prone mouse strains. However, it is important to replicate those findings in other animal models, and under different experimental conditions and designs.
- Epidemiological studies like those conducted for occupational or lifestyle-related UVA/UVB exposure will be warranted if far-UVC becomes widely deployed.
- The risk factors and mechanisms underlying melanoma induction are less clear than those for NMSC. There are few good animal models and no well-defined, standardized, and consistent exposure or outcome measures.
- First-order interactions, such as direct DNA damage, have been the primary consideration when evaluating UVC/far-UVC cancer risk. Second-order interactions and alternative damage mechanisms should be considered and discussed. This might include reactive oxygen species, immunological effects, and potential interactions between UVC/far-UVC and other environmental factors, including sunlight.

Analysis

How does UV radiation exposure cause cancer?

We have long known that excess exposure to high-energy, penetrating ionizing radiation such as x-rays and gamma rays increases cancer risk by causing DNA damage and mutation in critical genes governing cell division[1]. We also know from human epidemiological studies, experimental animal studies, and in vitro studies that solar ultraviolet radiation can also cause DNA damage and mutation in human skin, similarly increasing cancer risk[2]. UV radiation type and corresponding penetration depth (see Figure 4.2 in *Skin and eye safety* section) are two of the most important factors for UV-induced cancer formation. UVA has the lowest energy, penetrates the farthest into biological tissue, is associated with long-term skin photodamage (e.g., wrinkles), and causes oxidative stress, leading to indirect DNA damage[3]. UVB has higher energy than UVA, lower penetration depth, and can directly damage DNA through photochemical mechanisms[4]. UVB exposure is the leading cause of sunburns and is also thought to cause the most skin cancer[5]. UVC has the highest energy, but the shallowest penetration. While solar UVC is completely blocked by the atmosphere, exposure to artificial UVC radiation can result in DNA damage in the upper levels of the epidermis.

UV radiation exposure can lead to direct or indirect damage to the purine and pyrimidine bases in DNA. Cyclobutane pyrimidine dimers (CPDs) and pyrimidine-(6,4)-pyrimidone photoproducts (6-4PPs) are the most common types of DNA base damage resulting from exposure to UVB or conventional UVC wavelengths[6]. Cells contain protein complexes that are designed to recognize and repair the vast majority of different kinds of DNA base damage, including the kinds caused by UV radiation, but sometimes a misrepair occurs during the process, and an incorrect DNA base is inserted, leading to mutation[7]. If the damage occurs in genes that regulate the process of cell division, or conversely, in genes whose role is to promote cell death when cells are irreparably damaged, after many cell divisions and accumulation of additional mutations, this can result in uncontrolled cell growth, which can lead to cancer[8].

Carcinogen classification

The International Agency for Research on Cancer (IARC), part of the World Health Organization (WHO), and the US National Toxicology Program (NTP), both work to identify factors that contribute to cancer. The NTP is formed from parts of several different US government agencies, including the National Institutes of Health (NIH), Centers for Disease Control and Prevention (CDC), and the Food and Drug Administration (FDA)[9].

There is disagreement over the classification of different parts of the UV spectrum as a potential carcinogen, as shown in Table 5.1. Experimental and epidemiological studies indicate that UVA and UVB both have strong causal links to cancer, but only IARC lists these wavelengths as "carcinogenic," whilst NTP is less definitive about the causal link for any particular form of UV radiation, stating that UVA/B considered alone is only "reasonably anticipated to be" carcinogenic. Although no direct epidemiological evidence links UVC and cancer, IARC and NTP place UVC into the same classifications as UVA and UVB. Lastly, the magnitude of cancer risk is not factored into these classifications. An IARC label of "carcinogenic to humans" does not address the likelihood of cancer at low doses: for example this category includes exposures such as ethanol in alcoholic beverages as well as wood dust that, in small amounts, are only weakly linked to cancer.

TABLE 5.1. Classification across the UV spectrum.

	NTP	IARC
Solar radiation	Known to be a human carcinogen	Carcinogenic to humans
Exposure to sunlamps and sunbeds	Known to be a human carcinogen	Carcinogenic to humans
Broad-spectrum UV radiation	Known to be a human carcinogen	
UV radiation (UVA, UVB, UVC)		Carcinogenic to humans
UVA	Reasonably anticipated to be a human carcinogen	
UVB	Reasonably anticipated to be a human carcinogen	
UVC	Reasonably anticipated to be a human carcinogen	

Artificial ultraviolet radiation (UVR) and biological damage

Epidemiological and experimental studies examining the role of ultraviolet radiation emitted from artificial sources have strengthened a causal relationship between UVA or UVB exposure and skin cancer. Tanning beds (sunbeds) emit primarily UVA radiation 10–15 times higher than that found in sunlight, as well as some UVB. Epidemiological data has consistently shown a correlation between the amount of sunbed use and melanoma risk[10–15].

Artificial UVC sources, such as welding torches and germicidal mercury lamps, can also emit non-UVC wavelengths to various degrees. UVB and UVA emissions from these devices may contribute to cancer formation[16]. Welders, especially those using electric arc devices, are exposed to the full UV spectrum, and are also at risk for erythema (sunburn) in unprotected areas[17–20]. Cancer risk from welding UVR is understudied and our understanding is incomplete, but biological considerations suggest that repeated UVR exposure to unprotected areas increases cancer risk, similar to solar UVA and UVB exposure. No studies have demonstrated a relationship between occupational UVC exposure and skin cancer.

Cancer, cancer treatment, and infection risk

Individuals undergoing chemo- or radiotherapeutic cancer treatments are at a higher risk of infection due to immune system suppression caused by the treatment[21]. Surgical interventions, by their very nature, also increase infection risk[22,23].

Non-melanoma skin cancer

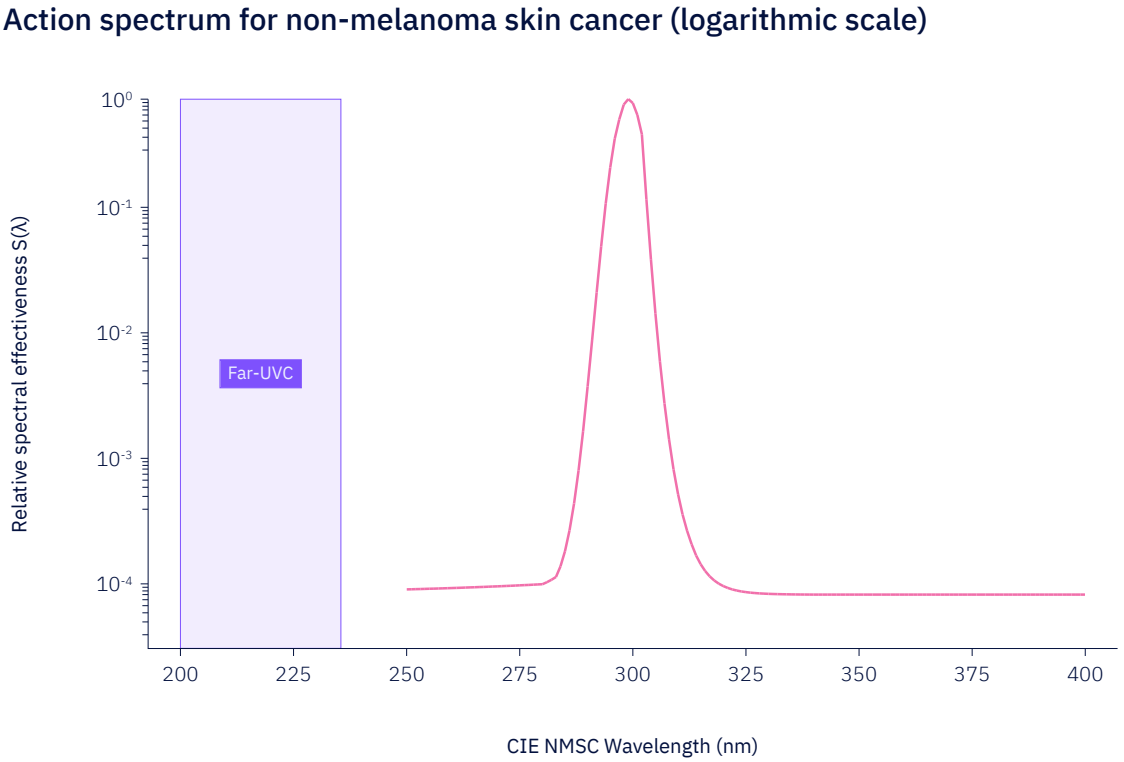

FIGURE 5.1. Redrawn from Görlitz et al., 2024[24]. UV action spectrum from non-melanoma skin cancer, logarithmic scale.

Epidemiological data indicates that cumulative solar UV exposure is highly correlated with non-melanoma skin cancer (NMSC) development. UV exposure is estimated to initiate 90% of NMSCs[25]. NMSCs mainly occur in skin keratinocytes. There are two major forms of NMSC—basal cell carcinoma (BCC) and squamous cell carcinoma (SCC), accounting for ~80% and ~20% of cases respectively[26,27].

An estimated 5.4 million cases of NMSC are diagnosed in the US annually, with 2,000–8,000 deaths each year, primarily from squamous cell skin cancer[28]. In the US, the overall incidence of BCC increased by 145% and SCC by 265% between 1976–1984 and 2000–2010[29,30]. These increases are at least partially caused by dramatic increases in awareness of skin cancer (detection bias), as well as economic incentives in the US healthcare system, since lucrative healthcare screening practices increase the likelihood of skin cancer being detected and diagnosed even if the health risk is relatively minor[31].

Experimental animal studies of the link between UVC exposure and cancer

Historical experimental animal studies reported a causal relationship between UVC exposure and cancer. This conclusion must be tempered, however, by the realization that these experiments primarily used low-pressure mercury discharge lamps, which emit 90–95% at 254 nm (UVC), but also emit measurable levels of UVA, UVB, and visible light[32–36].

One study irradiated mice with 3×10^3 mJ/cm² UVC per week, resulting in 7% of animals displaying tumors after a 52-week exposure[34]. This dose is more than 70 times higher than the maximum permitted human exposure every day. Another study by Sterenborg et al. irradiated hairless albino mice, a cancer-prone model, for 75 min/day, 7 day/week, at 23, 146, or 700 mJ/cm² UVC (30 times the minimum effective dose (MED)) observing tumor formation that scaled with dose[36].

The authors of Sterenborg et al., 1988 argued that "the UVB emitted by the low-pressure mercury lamps was insufficient to account for the induction of tumors at the rate found, as at least 850 days of exposure to the UVB radiation present would be required to induce skin tumors at the rate observed, compared to 161 days with the low-pressure mercury discharge lamp used." A summary of findings by the IARC Working Group stated that "the 4% UVB content of the source, representing a weekly dose of 1,170 J/m², could not be excluded as contributing to the induction of skin tumors."[37] The IARC Working Group stated that "the evidence given to exclude UVB as contributing to the induction of tumors does not obviate the possibility that some interaction between UVC and UVB radiation led to tumor induction."

Melanoma

Melanoma develops in melanocytes. Melanocytes produce the pigment melanin, which absorbs UV radiation and protects deeper layers of the skin from some of the harmful effects of the sun[38]. In the US in 2024, it is estimated that ~100,000 new cases of melanoma occurred, with ~8,300 deaths[39].

While solar UVB exposure is a risk factor for melanoma, the link is more complicated than for NMSCs, with genetics, demographics, and other factors contributing to risk. UV exposure is thought to cause 60–70% of cutaneous malignant melanomas[40,41]. In contrast, acral lentiginous

melanoma is not thought to be caused by exposure to sunlight or UV radiation. This form of melanoma is more common in individuals with darker skin, and often first appears on the palms, soles, under the nails, or in the mouth mucosa[42]. Experimental studies of the relationship between UVC exposure and melanoma are complicated by the lack of a good mouse genetic model, and the variety of other causative factors associated with this disease.

Conventional germicidal UVC lamps

Overexposure to conventional germicidal UVC lamps (254 nm) is quite rare. The few case reports describe acute reactions resolving within a week, including erythema and photokeratitis[43,44], although one study reported long-term effects from acute overexposure[45]. While studies have linked 254-nm UVC exposure with increased CPD formation in skin, very few human studies have assessed cancer risk from germicidal UVC exposure[46–48]. To address this knowledge gap, it is important to perform surveillance and mechanistic studies, similar to those conducted to assess cancer risk from solar and occupational UV exposure. It should be noted that conventional germicidal UV lamps may also emit a small proportion of unfiltered UVB, and this must be taken into account in any future studies.

Sun-sensitizing conditions and UVC

A small percentage of individuals have mutations in genes that govern various aspects of the DNA damage/repair response. For example, Xeroderma Pigmentosum (XP) is a disease that is caused by defects in nine different DNA repair genes involved in repair of UV-induced DNA damage, but additional unidentified genes may play a role. XP is characterized by photosensitivity, rapid severe sunburn, and an increased risk for all types of skin cancer at an early age[49,50]. UVB exposure is most directly associated with skin cancer in XP[51]. The relationship between UVC exposure and cancer in XP, however, is unknown. However, the limited penetration depth of UVC radiation suggests that the risk, if any, may be minimal. To address this knowledge gap, directed research is needed.

Ocular melanoma

Melanoma in the eye (ocular melanoma) most commonly develops in pigmented cells in the middle layer of the eye (uvea). The uvea is composed of three distinct anatomical regions: the iris (the colored layer towards the front of the eye); the choroid (a thin, heavily pigmented middle layer of the eye containing blood vessels and connective tissue); and the ciliary body (a structure surrounding and supporting the iris and lens that transports aqueous liquid from the bloodstream into the eye to maintain intraocular pressure). Melanin granules in melanocytes found in these tissues absorb stray reflections and absorb UVR[52]. In rare cases, melanoma can also occur in other parts of the eye and its external adnexa, including the conjunctiva, eyelid, and orbit[53].

Uveal melanoma is the most common primary intraocular malignancy with an average of five cases per million in the US[54,55]. Around 3,320 new cancers (primarily melanoma) of the eye and the orbit are estimated to have occurred in 2024 with 560 deaths[56].

Impact of ultraviolet radiation on ocular melanoma risk

The eyes spend many hours exposed to environmental UVR. It would be reasonable to suspect that this radiation exposure would increase the risk for melanoma. However, uveal melanoma is currently classified by the WHO as one of the five melanomas triggered by "risk factors other than cumulative damage."[52,57]. In part, this is because the cornea absorbs all UVR shorter than 295 nm[58]. Longer wavelength UVB and UVA pass through the cornea to reach the iris, parts of the uvea, and lens[59]. Nevertheless, the opaque iris serves as an additional barrier to UV penetration, protecting underlying structures from harmful radiation. The adult lens is similarly opaque to UVR, absorbing all wavelengths below 370 nm, and letting through less than 2% of UVA between 370–400 nm[60].

Studies of far-UVC and cancer

To date, experimental animal studies suggest that far-UVC does not promote skin cancer of any type due to its limited penetration past the stratum corneum (the outer layer of the skin). One recent hairless-mouse study reported no evidence of skin cancer or abnormal skin growths after chronic 222-nm exposures of varying intensity (66 weeks, 5 days/week, 8 hours/day)[47]. In another study, mice with defects in the XP gene family were exposed to far-UVC radiation but did not develop skin cancers, swelling of the ears, or erythema[48]. In contrast, similar mice exposed to UVB developed tumors.

The fact that UVC or far-UVC biophysically cannot penetrate deeply into skin suggests that these UV radiations cannot directly damage melanocytes, which lie within deeper skin layers. Any hypothesis for far-UVC-induced melanoma formation must include (an) indirect mechanism(s) whereby UVR-induced photochemical reactions in the upper epidermis interact with melanocytes located much deeper in the stratum basale. The same rationale applies to individuals with genetic defects in DNA repair.

As far-UVC is an emerging technology, most studies evaluating the impact of chronic far-UVC exposure in human subjects have, to date, involved only a handful of people and lasted no longer than a few months[61]. One exception to this is the 36-month follow up to the Sugihara study on ocular safety[62]. The precautionary principle suggests that skin cancer screening should be included in empirical research of long-term real-world deployments, such as a cluster-randomized clinical trial, in order to provide epidemiological evidence in humans.

Further reading

- Assessing the safety of new germicidal far-UVC technologies

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6. Ozone and indoor air quality

Summary

We know that far-UVC generates ozone through photolysis, and the rate of generation per unit of fluence rate is relatively well quantified. However, how this translates to occupants' exposure to ozone and ozone reaction products is highly sensitive to conditions—particularly outdoor air ventilation rate and what other chemical precursors are present in the space. The potential health impacts of increases in indoor ozone levels are highly uncertain (see *Ozone epidemiology* section), but there is an established consensus among indoor air quality experts that any ozone emissions indoors are undesirable. Ozone can be removed with activated carbon filters and catalysts.

The question is therefore not *whether* far-UVC creates ozone, which is beyond doubt, but how *much* ozone is created by a given amount of far-UVC, how *much* of the ozone (and the products of chemical reactions that consume ozone) are mitigated through ventilation or removed by activated carbon filters or catalysts, and how the potential costs of this should be weighed against the infection prevention benefits of far-UVC.

Crucial considerations

- How much ozone far-UVC generates depends on the particular wavelength. Shorter wavelengths generate more ozone.
- For filtered krypton chloride excimer (KrCl*) lamps that have so far been assessed, experimental measurements and first principles calculations support an estimated ozone generation efficiency of 7–10.5 ppb per hour per μW/cm² of fluence rate.
- The expected increase in steady-state ozone concentrations in a space with 1 air change per hour is low to mid single digits ppb per μW/cm² of average fluence rate. This is a small amount relative to both outdoor ozone and fluctuations that can be observed in background levels of indoor ozone, but relatively large compared to the average levels of indoor ozone, which show a central tendency of ~4–6 ppb. Indoor ozone reacts with other substances, which is also known as 'ozone loss'. The products of this ozone loss may be more significant for health than ozone itself.
- It is possible that there are photochemical interactions that affect indoor air quality other than those caused by ozone generation that we have not yet characterized, and research is ongoing.
- In addition to adequate ventilation, ozone mitigation options include catalysts and activated carbon filters.

Analysis

Quantifying ozone production from far-UVC

Far-UVC photons will cause ozone (O_3) generation through direct photolysis of O_2 molecules in the air (i.e. the photons break down the O_2 molecules). Each photolyzed O_2 molecule leads to the formation of two ozone molecules.

$$O_2 + hv \rightarrow O + O$$
$$O + O_2 + M \rightarrow O_3 + M$$

Where hv is a photon with wavelength <242 nm, and M is any other molecule (such as N_2 or another O_2). When wavelengths are below 200 nm, the O_2 absorption cross section is large enough to promote rapid photolysis under typical tropospheric conditions, and this rate of ozone generation increases rapidly with decreasing wavelength. It is for this reason that this section of the electromagnetic spectrum is referred to as 'vacuum UV'—since it is rapidly absorbed by oxygen, it can only be used in spaces without oxygen (such as a vacuum).

Above 200 nm, the propensity for an O_2 molecule to be photolyzed decreases at a linear rate from 200 nm to 242 nm. However, O_2 absorption at 222 nm—the peak wavelength produced by the most common far-UVC lamps—is strong enough to generate detectable increases in O_3 at fluence rates relevant for far-UVC applications.

a When converting between O_3 in ppb and μg/m³, we use a value of 1 ppb = 1.96 μg/m³. This value will vary slightly based on temperature and air pressure.

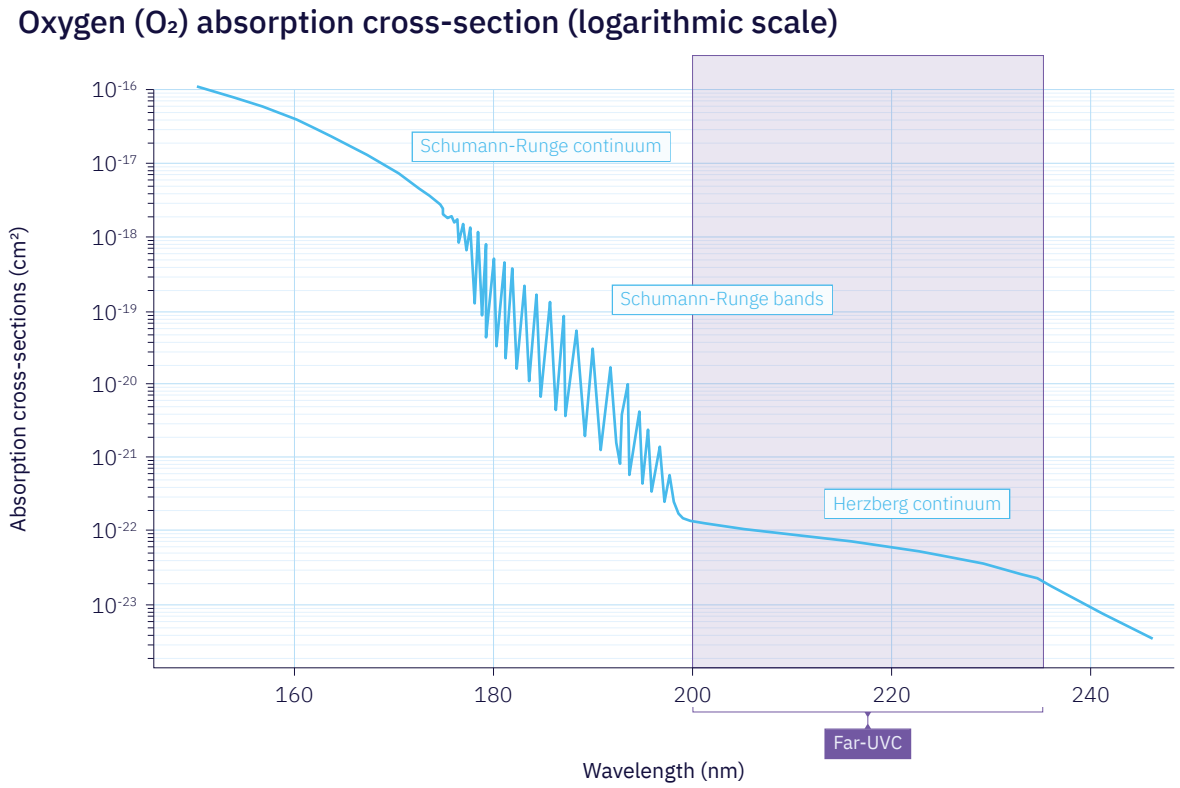

FIGURE 6.1. Excerpted from Watanabe, Inn and Zelikoff, 1953[1]. Absorption cross-section of O_2 (log scale).

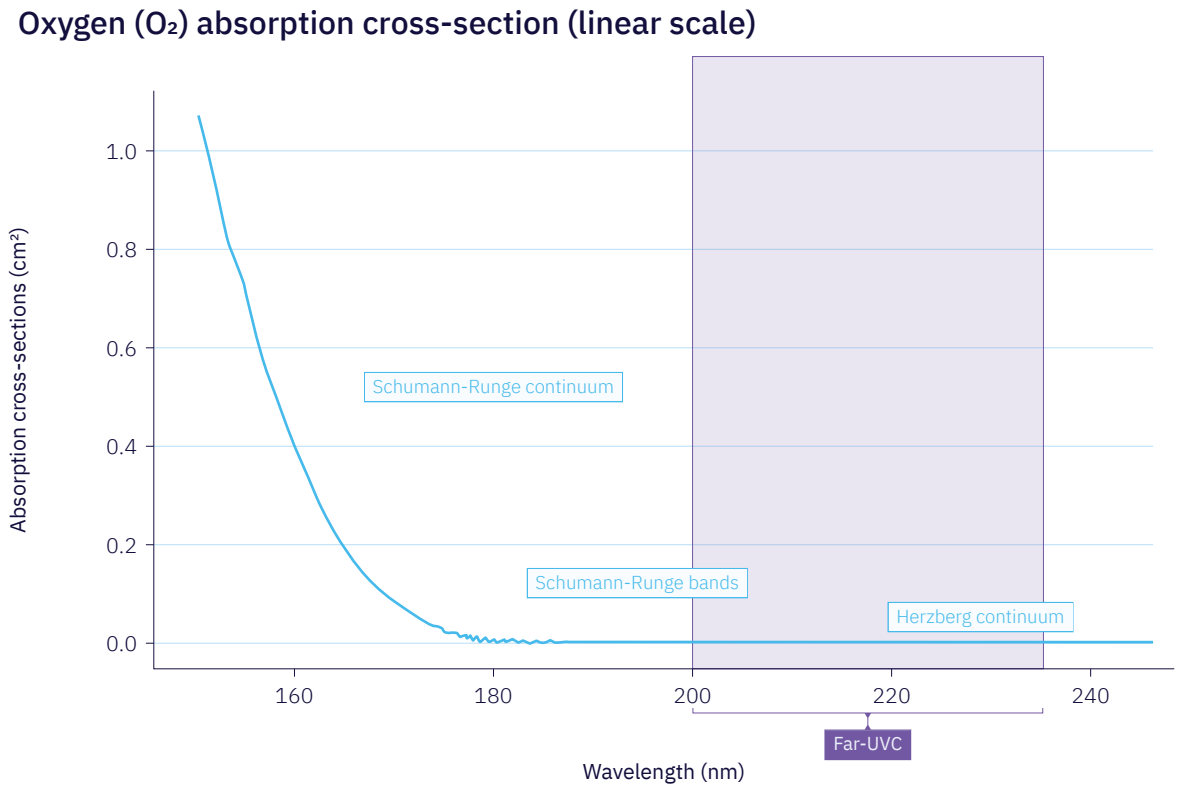

FIGURE 6.2. Excerpted from Watanabe, Inn and Zelikoff, 1953[1]. Absorption cross-section of O_2 (linear scale).

To understand how fast far-UVC produces ozone in a given space, we need to take into account both the intrinsic ozone generation efficiency of the wavelengths emitted from a far-UVC device and the total amount of far-UVC employed in the space.

Equation 6.1

$$P = G * I$$

with P = Ozone production rate (ppb/hr)
G = Ozone generation efficiency (ppb/hr/μW/cm²)
I = Room average fluence rate (μW/cm²)

Note that many manufacturers and studies quote the measured *irradiance* of a far-UVC device at a particular distance from the lamp, which makes sense in the context of photobiological safety. However, the *average fluence rate* in a room is not only a property of the irradiances at any given distance from the lamp but also the average photon pathlength and the room volume (see *Efficacy* section). The ozone production rate from a far-UVC device is therefore dependent on not just the device, but the context in which it is used.

There are currently three studies which have directly measured the ozone generation efficiency of KrCl* lamps in controlled experimental chambers. It is important to note that these lamps all have filters which result in the reduction of emissions <200 nm and >235 nm. In these studies, the ozone generation efficiency ranges from 7–10.5 ppb/hr per μW/cm² of average fluence rate[2–4]. This is a relatively small range given the uncertainties inherent in fluence rate determinations.

The first of these studies (Peng et al., 2023) also estimated the ozone generation efficiency using a first-principles model based on the absorption cross-section of O_2 and the fully characterized emission spectrum of one of the tested emitters. The authors arrived at an identical figure to their experimental finding of 10.5 ppb/hr/(μW/cm²). All further calculations in this document use this figure.

However, it is worth noting that the ozone generation rate of a different model using the same bulb was approximately 25% lower. This is believed to be due to the different emissions spectrum caused by the use of a diffuser[3]. It is important to replicate these ozone generation efficiency calculations using different devices from different manufacturers. This could validate the underlying photochemical model of ozone generation based on O_2 absorption cross-sections and the fully characterized emission spectrum of each device.

Given the underlying photochemical model, we should expect the ozone generation efficiency to be modestly sensitive to the exact emission spectrum of a source within the far-UVC range. For example, an ozone generation efficiency of 6.7 ppb per hour per μW/cm₂ would be expected for a monochromatic device that emitted only 222 nm, but devices emitting different wavelengths would have different efficiencies[5].

In order to translate *ozone production* into expected changes in ozone concentrations, we also need to know the rate at which ozone is removed with ventilation or lost to chemical reactions.

Key takeaways

- Far-UVC generates ozone from photolysis of O_2.
- Current studies show an ozone generation efficiency ranging from 7–10.5 ppb/hr/(μW/cm²), at least for filtered krypton chloride excimer lamps.
- Theoretical models and controlled experimental data are broadly consistent, but further validation with different devices is recommended.

Modeling framework for indoor ozone concentrations

Nazaroff and Weschler, 2022[6] present a framework for understanding the determinants of indoor ozone concentrations. Ozone is brought in from outdoors through natural and mechanical ventilation and also removed through the same means. Once indoors, ozone is lost through chemical reactions with surfaces, human occupants, any NO_x gases present, and with volatile organic compounds (VOCs). The potential importance of the byproducts of these reactions is discussed later in this section.

These factors are modeled as a decay constant k, which is a summary statistic of the average rate at which ozone reacts in an indoor space, and has the unit hr. The greater the k constant, the more 'ozone per hour' reacts away in an indoor space.

The single largest cause of ozone decay in most spaces is from reactions with surfaces. Nazaroff and Weschler, 2022 only identified two studies that have directly quantified the whole-room average ozone decay rate needed for this modeling framework, and both are of residences. Measurements in 43 southern California residences provided a decay per hour of 2.8 ± 1.3 (Lee et al., 1999[7]), and a study of 15 bedrooms in 14 Chinese residences estimates 2.8 ± 1.1 (Yao and Zhao, 2018[8]). While there is an extensive literature on ozone loss to indoor-relevant surfaces, these two studies are the best available empirical data on whole-room average ozone decay rates from reactions with indoor surfaces.

In the absence of indoor sources and where the only source of indoor ozone is outdoors, indoor/outdoor ozone ratios should approximately conform to equation 6.2, taken from Weschler and Nazaroff, 2023[9].

Equation 6.2

$$[O_3]_{\text{in\_outdoor source}} \approx [O_3]_{\text{out}} \cdot \frac{\lambda_{\text{ACH}}}{\lambda_{\text{ACH}} + k_{\text{sum}}}$$

Where λ_{ACH} is the air exchange rate with outdoor air—ACH stands for 'air changes per hour'. k_{sum} represents all the various decay sources for indoor ozone loss, of which surfaces are typically the most important. Therefore, observed indoor/outdoor ozone ratios and realistic assumptions about ventilation rates put a bound on what the values of k_{sum} can plausibly be. Nazaroff and Weschler, 2022 collated indoor/outdoor ozone concentrations from dozens of studies, inferring a central estimate for

the surface loss coefficient of ~2/hr, rather than 2.8/hr as documented in the studies of residences. These estimates are sources from a number of different environments, and may not be representative of the spaces in which germicidal UV would be applied. For calculations in this document, unless otherwise stated, a k_{sum} value for losses to reactions of 2/hr is used.

In a recent study, Sørensen et al., 2024[10] measured k_{sum} in four small offices and found k_{sum} (in their Table (6.1) labeled k_{loss}) ranging from 0.83 to 3.93:

TABLE 6.1. From Sørensen et al., 2024[10]. k_{ACR} is the air change rate equivalent to λ_{ACH}, k_{loss} is equivalent to k_{sum}, and k_{decay} is the total rate of decay equal to k_{ACR} + k_{loss}

Experiment	Season	Office	Occupancy	k_{decay} (hr)	k_{ACR} (hr)	k_{loss} (hr)
#1	summer	A	unoccupied	1.13	0.24	0.89
#2	fall	A	unoccupied	1.54	0.17	1.37
#3	fall	A	1 person	2.08	0.14	1.93
#4	fall	A	2 persons	4.05	0.12	3.93
#5	summer	B	unoccupied	1.17	0.33	0.84
#6	fall	B	unoccupied	1.33	0.27	1.07
#7	fall	B	1 person	1.73	0.34	1.39
#8	fall	B	2 persons	1.89	0.19	1.7
#9	summer	C	unoccupied	1.16	0.3	0.86
#10	fall	C	unoccupied	1.33	0.24	1.08
#11	summer	C	1 person	1.47	0.24	1.23

For sensitivity analysis conducted later, we assume a lower bound of k_{sum} of 0.5.

The effect of adding far-UVC as a source of ozone indoors

One way of thinking about equation 6.2 is that there is a rate at which ozone is added indoors ($[O_3]_{out} \cdot \lambda_{ACH}$) and a rate at which it is removed ($\lambda_{ACH} + k_{sum}$). ($[O_3]_{out} \cdot \lambda_{ACH}$) is analogous to the ozone production rate P from equation 6.1 for an indoor source. Ozone generated from far-UVC (or any other indoor source) is chemically indistinguishable from outdoor ozone and thus is removed in the same way.

Equation 6.3

$$[O_3]_{\text{in\_indoor source}} = \frac{P}{\lambda_{\text{ACH}} + k_{\text{sum}}}$$

Substituting P from equation 6.1, the increase in ozone concentration indoors from introducing far-UVC as an indoor source of ozone can therefore be calculated following equation 6.3.

Equation 6.4

$$[O_3]_{\text{in\_UVC}} = \frac{I \cdot G}{\lambda_{\text{ACH}} + k_{\text{sum}}}$$

Combining this with equation 6.2, the complete equation for indoor ozone concentration in the presence of far-UVC is:

Equation 6.5a

$$[O_3]_{\text{in\_total}} \approx [O_3]_{\text{in\_outdoor source}} + [O_3]_{\text{in\_UVC}}$$

Equation 6.5b

$$[O_3]_{\text{in\_total}} \approx \left([O_3]_{\text{out}} \cdot \frac{\lambda_{\text{ACH}}}{\lambda_{\text{ACH}} + k_{\text{sum}}}\right) + \left(\frac{I \cdot G}{\lambda_{\text{ACH}} + k_{\text{sum}}}\right)$$

Assuming an ozone generation constant G of 10.5 ppb/hr per μW/cm² and holding outdoor ozone levels constant, we can therefore estimate the ozone increases that we would expect to observe under 1 μW/cm² of room average fluence rate.

TABLE 6.2. Estimated change in average ozone concentration from 1 μW/cm² of far-UVC. Sensitivity analysis of expected ozone increase (ppb) at 1 μW/cm² of fluence rate from a KrCl* lamp, at different λ_{ACH} (0.1-10) and k_{sum} (0.5-4).

Ozone increase (ppb)							
λ	0.1	0.5	1	2	3	5	10
k_{sum}							
4	2.6	2.3	2.1	1.8	1.5	1.2	0.8
3.5	2.9	2.6	2.3	1.9	1.6	1.2	0.8
3	3.4	3	2.6	2.1	1.8	1.3	0.8
2.5	4	3.5	3	2.3	1.9	1.4	0.8
2	5	4.2	3.5	2.6	2.1	1.5	0.9
1	9.5	7	5.3	3.5	2.6	1.8	1
0.5	17.5	10.5	7	4.2	3	1.9	1

For comparison, the median indoor ozone concentration from the literature summarised in Nazaroff and Weschler, 2022 is 4-6 ppb[6].

The impact of changing ventilation on indoor ozone levels

Ventilation (λ_{ACH}) rates vary considerably, in commercial environments based on occupancy and the use of central HVAC systems, and in residential environments based on factors such as windows being open or shut, outside climate conditions and the construction of the property. The variation in residential air change rates generally follows a lognormal distribution—see for example the following Table 6.3, taken from Nazaroff, 2021[11], which summarizes the geometric mean (GM) and geometric standard deviation (GSD) across various studies using CO_2 as a tracer gas to estimate ventilation rates in residences.

Using equations 6.2 and 6.4, we can compare the increases in indoor ozone concentrations that we would expect to see under far-UVC irradiation with those we would expect from changes in ventilation.

If we assume an outdoor level of ozone of 30 ppb, we can use the equations to estimate the steady-state indoor ozone concentration under different ventilation and k_{sum} reaction loss assumptions, and therefore estimate the effect on steady-state ozone concentrations of increasing λ (ventilation) by 1. As per Nazaroff and Weschler, 2022, we conservatively assume that ~25% of the outdoor ozone is lost in the mechanical ventilation system before it reaches the indoor space[6] (and hence for the modeling purposes, $[O_3]_{out}$ is 22.5 ppb rather than 30 ppb).

TABLE 6.3. From Nazaroff, 2021[11]. Residential air-change rates utilizing CO_2 as a tracer. References and notes omitted.

Location	N sites	Housing type	GM (hr)	GSD
Guangzhou, China	202	Apartment bedroom	0.29	2
China (F, W, Sp)	273	Apartment	0.29	2
China	294	Bedrooms	0.33	2.2
France	450	Dwelling	0.43	2.3
Denmark	500	Child's bedroom	0.46	2.1
France	57	Bedroom, heating	0.51	2.6
France	58	Bedroom, nonheating	0.51	2.3
Sweden	21	New houses	0.62	1.4
Slovakia	45	Apartment	0.64	1.9
China (Winter)	223	University dorms	0.7	2.3
Sweden	20	Passive houses	0.71	1.3
China (Summer)	59	Apartment	0.86	3.6
China (Summer)	223	University dorms	4.4	3

Variations in ventilation in a single residence over time also follow a lognormal distribution[12].

TABLE 6.4. Estimated increase in ozone concentration of increasing λ_{ACH} by 1. The table shows the expected change in steady state O_3 concentration (in ppb) if λ_{ACH} was increased by 1 above the base level. Base λ_{ACH} ranges from 0.1-10, k_{sum} 0.5-4.

Ozone increase (ppb)							
Base λ	0.1	0.5	1	2	3	5	10
k_{sum}							
4	4.3	3.6	3	2.1	1.6	1	0.4
3.5	4.8	3.9	3.2	2.2	1.6	1	0.4
3	5.3	4.3	3.4	2.3	1.6	0.9	0.4
2.5	6	4.7	3.6	2.3	1.6	0.9	0.3
2	6.9	5.1	3.8	2.3	1.5	0.8	0.3
1	9.7	6	3.8	1.9	1.1	0.5	0.2
0.5	11.7	5.6	3	1.3	0.7	0.3	0.1

If we directly compare these increases to those from adding 1 μW/cm² of far-UVC from a KrCl* lamp illustrated in Table 6.2, in many cases the change in indoor ozone concentration from increasing ventilation is greater than the change from far-UVC:

TABLE 6.5. Difference between increase of λ_{ACH} (ventilation) by 1 and adding 1 μW/cm² of far-UVC. The figures were calculated by subtracting the values of Table 6.2 from those of Table 6.4. A positive number therefore means that there is a greater change in O_3 concentration (in ppb) from increasing λ_{ACH} by 1 than from 1 μW/cm² of far-UVC from a KrCl* lamp.

Difference in ozone concentration (ppb)							
Base λ	0.1	0.5	1	2	3	5	10
k_{sum}							
4	1.7	1.3	0.9	0.4	0.1	-0.2	-0.3
3.5	1.8	1.3	0.8	0.3	-	-0.3	-0.4
3	1.9	1.3	0.8	0.2	-0.1	-0.4	-0.4
2.5	2	1.2	0.6	-0.1	-0.3	-0.5	-0.5
2	1.9	0.9	0.3	-0.4	-0.6	-0.7	-0.6
1	0.2	-1	-1.5	-1.6	-1.5	-1.2	-0.8
0.5	-5.8	-4.9	-4	-2.9	-2.3	-1.6	-0.9

This modeling demonstrates the importance of characterizing background levels of (and variations in) ventilation. This helps us to contextualize the relative increase in ozone concentrations from far-UVC and also to remove any confounding in real-world observations.

The impact of outdoor ozone variations on indoor ozone levels

Background levels of outdoor ozone vary significantly day to day and hour to hour. Outdoor ozone levels follow a diurnal (24-hr) cycle, with ozone levels typically peaking in the afternoon and being lowest in the early morning. The magnitude of the difference can be substantial; in a recent study the average difference between daytime peak and nighttime minimum ozone concentration in Chinese cities in the summertime was 45 ± 13 ppb[13]. The diurnal ozone cycle is also clearly visible in one of the few studies observing real-world ozone formation from the use of far-UVC. Below a figure from Kalliomäki et al., 2023 is reproduced[14], with the indoor ozone concentration clearly following the outdoor ozone.

Background variations in outdoor ozone levels can, in some circumstances, introduce a significant confounding effect when researchers attempt to estimate the impact of far-UVC on indoor ozone concentrations.

Mixing

It should be noted that this modeling framework implicitly utilizes the 'well-mixed room assumption' (see *Efficacy* section). According to this assumption, ventilation rates can be treated as a decay constant with respect to the average concentration of a contaminant, and additionally there is no short-circuiting between the air inlet and outlet that would also lessen the residence time of ozone introduced from outdoors. A well-mixed room is likely a more reasonable assumption for a gaseous contaminant like ozone than for a pathogen, but it would be fruitful for indoor air quality researchers to consider the impact of mixing on this modeling framework.

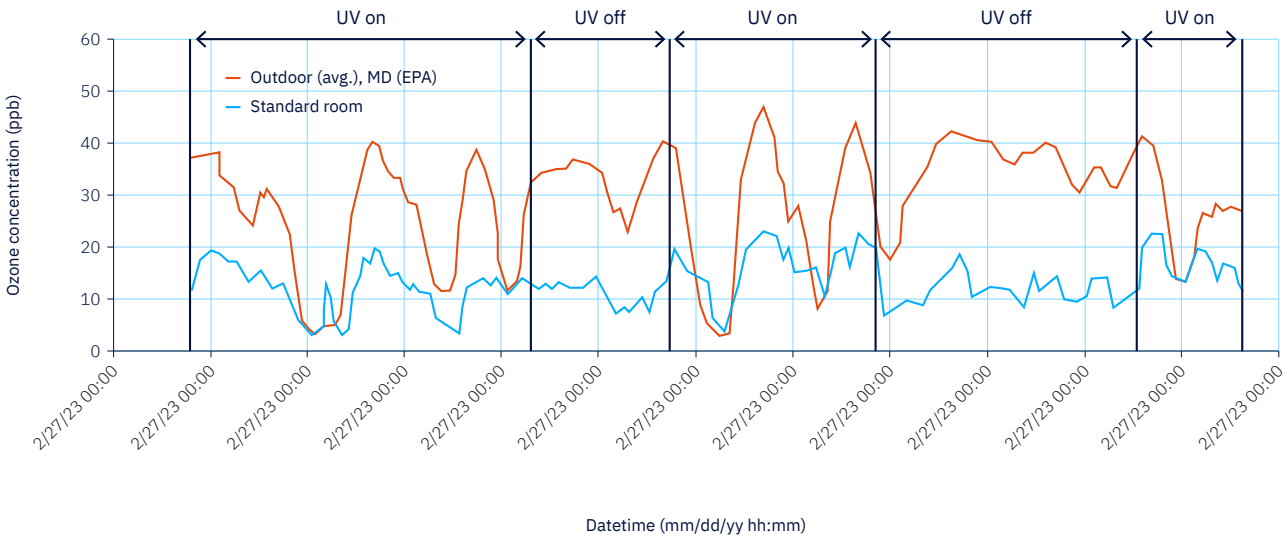

FIGURE 6.3. Excerpted from Kalliomäki et al., 2023[14]. Standard hotel room O_3, CO_2, and UFP measurements from February 27 through March 11, 2023. Periods with far-UVC lamps and in-room HEPA filtration on and off and fan coil unit states (on, off, and auto) are shown. During the auto state, the fan coil unit ran intermittently as controlled by a thermostat and motion sensor. During the filtering periods the fan coil unit had a MERV 13 filter and the HEPA filter unit had a HEPA filter but they had no filter during other times.

Key takeaways

- Ozone decays indoors due to chemical reactions, with surfaces being the most important source of loss in most spaces.
- Background indoor ozone concentrations are a function of outdoor ozone concentrations. They depend on the balance of ventilation (which brings in ozone) and ozone reactive loss (with chemicals on surfaces and in the air).
- Variations in indoor ozone concentrations due to changing ventilation and outdoor ozone levels can be substantial relative to a central tendency of 4–6 ppb in the published literature.
- Increases in ozone from realistic use of far-UVC will therefore often be small relative to observed variations in indoor ozone levels.
- When taking measurements in the field, variations in background ozone levels need to be adjusted for when estimating the impact of far-UVC.

Ozone generation from far-UVC devices in real-world spaces

As of the time of writing, there are five studies (three published, two in preprint) documenting ozone increases from far-UVC devices in 'real' environments, as opposed to experimental chambers (see table 6.6)[5, 10, 14-16]. In all of these studies, the results account for the variation in the background levels of ozone while the experiments were taking place.

For the Link et al., 2024[15] and Peng et al., 2023[5] experiments, where the ozone generation efficiency is reported or can be directly imputed from reported data, they are consistent with each other and within our previous estimate of 7–10.5 ppb/hr/(μW/cm²).

For Kalliomäki et al., 2023[14], an estimated k_{sum} decay rate can be inferred from their report that a 1 ppb increase in outdoor ozone was associated with a 0.33 ppb increase in indoor ozone, and a reported average air changes per hour (ACH) of 1.4. In order for this indoor/outdoor ozone ratio and ACH to be consistent with equation 6.2, a k_{sum} of 2.8 is implied. This k_{sum} estimate combined with an estimated fluence rate of 1.75 and 1.4 ACH implies an ozone generation rate of 13.7 ppb/hr/(μW/cm²). This is a crude estimate using the average values and regression point estimates, and it is possible that there was measurement error, that <200-nm emissions were less well-filtered on this particular lamp, or that there were electrical discharges on the surface of the excimer lamp that acted as a separate source of ozone[17].

For Narouei et al., 2024[16], there isn't data in the paper on k_{sum} or data that allows k_{sum} to be implied. However, we can calculate what the implied ozone generation rate would be with a plausible k_{sum}. At a k_{sum} of 2, the implied ozone generation rates are 30.7 for the 'low power' test (using a single far-UVC fixture with lower output) and 6.2 for the 'high power' test (using four fixtures with higher output). In the 'low power' test, it is worth noting that there is only a 2.7 ppb increase observed in average ozone concentration, in the context of background levels varying between 2 and 20 ppb and employing an ozone monitor with a reported precision of ± 1.5 ppb. In the context of the effect size and the instrument precision, 2–20 ppb background variation in ozone is substantial. We hypothesize that measurement inaccuracy in the low power test is likely to be the source of the substantially larger than expected ozone generation efficiency.

TABLE 6.6. Studies of ozone increases in real-world settings under far-UVC irradiation.

Study	Location	Publication	Fluence rate	Air changes per hour (ACH)	k_{sum}	Background O_3 range	Average O_3 increase	Implied ozone generation efficiency
			$\mu W/cm^2$	hr^{-1}	hr^{-1}	ppb	ppb	ppb/hr/ (mW/cm^2)
Peng et al., 2023[5]	Small sealed office	Peer reviewed journal	1.0[b]	0.62–0.96	0.5–2.3 (avg 0.78)	~1–7	6.5	8.6[c]
Kalliomäki et al., 2023[14]	Hotel room	Pre-print	1.7–1.8	1.2–1.7 (avg 1.4)	Not reported	~5–20	5.7	n/a
Link et al., 2024[15]	Restroom	Peer reviewed journal	3.2	1.1	3.7	~0–25	5.3[d]	8.0
Link et al., 2024[15]	Restroom	Peer reviewed journal	3.2	2.2	2.0	~5–25	6.5[e]	8.5
Narouei et al., 2024[16]	Office	Pre-print	0.29	1.3	Not reported	2–20	2.7	n/a
Narouei et al., 2024[16]	Office	Pre-print	5.19	1.3	Not reported	2–20	9.5	n/a

The fifth study—not included in table 6.6—is Sørensen et al., 2024[10], who conducted multiple experiments in four different offices, including experiments where they changed lamp placement and therefore estimated pathlength. Fluence rates were not calculated, and therefore ozone generation efficiency cannot be directly estimated. However, the expected correlation between increase in ozone production and UV pathlength occurs (for pathlengths of 3 m or less), and the rate of O_3 production of the same lamp corresponds with controlled experimental studies. These observations are therefore consistent with the ozone generation efficiency that was found or can be implied from other studies, as illustrated in Figure 6.4. Figure 6.4 shows an apparent flattening of the O3 production curve at pathlengths >3 m. This merits some discussion, as there is no theoretical reason that this flattening should occur if fluence rate is scaling with estimated pathlength.

Without an independent estimate of fluence rate, we can't rule out that the fluence rate did not fully scale with the estimated pathlength provided in the Sørensen paper, and therefore ozone production didn't scale either. Pathlength was measured by distance of the lamp to the wall, and it is a reasonable hypothesis that a greater proportion of photons terminate at the ceiling and floor as the lamp is moved further away from the wall. If this is the case, the true average photon pathlength was no longer increasing as the horizontal distance was increased beyond 3 m.

The other possibility offered by the authors is that O_3 production is not fully linear but requires a certain minimum UV intensity to occur. No such flattening appears to occur in the experiments in Peng et al., although it is worth noting that the shorter pathlength experiment was in a real office environment and the longer pathlength experiment in a Teflon chamber. Only further studies that vary fluence rate and measure and/or estimate all the relevant variables independently can confirm which explanation is correct.

We encourage researchers conducting studies of far-UVC and ozone generation in real spaces to analyze their results in this modeling framework, and to independently estimate k_{sum}, ventilation rates, and fluence rates. Link et al., 2024 is thus far the only study in real-world spaces to directly provide all the pieces of the puzzle that allow us to calculate ozone generation efficiency.

b This fluence rate is implied by the estimated differences in volume and estimated average photon pathlength in the office compared the Teflon chamber experiments reported in the same study, where the fluence rate was characterized with chemical actinometry. It is not a direct measurement or estimate provided in the paper.

c 8.6 is reported in the paper based on a more dynamic model. A simple model using the central k_{sum} estimate and average ACH of 0.78 would imply an ozone generation efficiency of 10.2.

d These are the average O_3 increases in the Fan Off condition, matching the reporting of the k_{sum} figures. From private correspondence with Michael Link.

e Average O_3 increases from when the fan was Off, matching the reporting of the k_{sum} figures. From private correspondence with Michael Link.

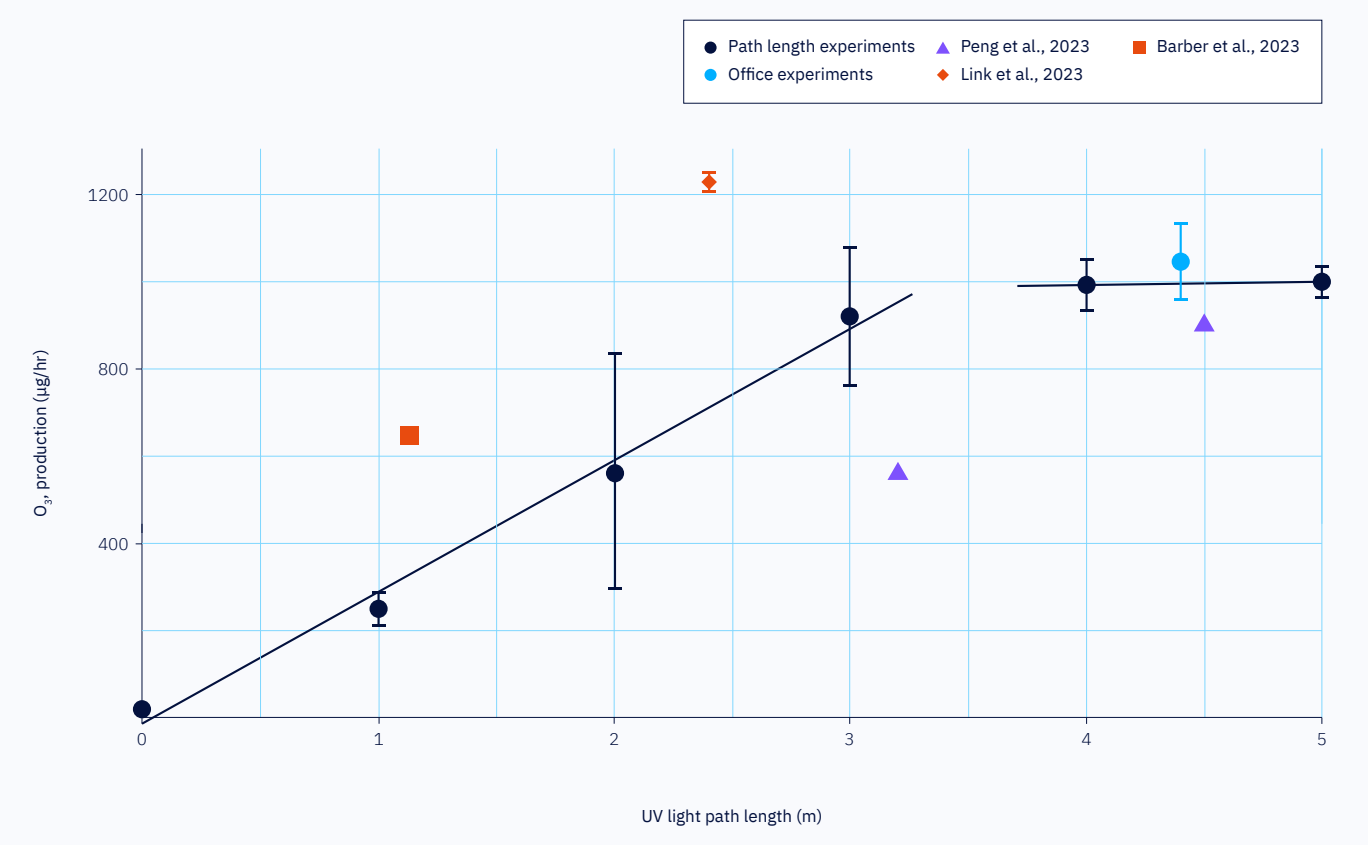

FIGURE 6.4. Excerpted from Sørensen et al., 2024[10]. Correlation between O_3 production (µg/hr) and UV light path length (m) according to the Sørensen et al. study (circles) and the pre-existing literature (triangles, square, and diamond). The equation displayed gives the linear correlation from 0 to 3 m, while the error bars indicate the standard deviations. Pressure corrections have been made for the results from Peng et al. to account for the reduced ambient pressure in Boulder where the experiments were carried out.

Key takeaways

- Studies so far suggest that ozone increases from far-UVC installations in real-world settings are broadly consistent with the theoretical framework discussed above, but more high-quality data is needed.
- Measurements need to have sufficient precision to accurately measure the expected effect. This often requires high-precision, research-grade instrumentation.
- It is critical to separately measure ozone concentration, k_{sum}, ventilation rates and fluence rates in order to establish robust modeling assumptions.

Ozone loss

Focusing on increases in indoor ozone concentrations obscures what may be the most important dynamic. The k_{sum} ozone decay constant represents reactions that destroy ozone but create other products, and the concentrations of these products (and in some cases their chemical precursors) are themselves heavily influenced by ventilation. As discussed in the *Ozone epidemiology* section, these products also have potential negative health effects.

k_{sum} is a summary statistic for the rate of ozone loss to all sources, such as reactions with chemicals present on surfaces, VOCs in the air, and chemicals produced by our own bodies, especially the skin oil constituent squalene[18]. Except in rare cases where ozone is catalytically decomposed (see *Ozone mitigation* subsection below), each ozone reaction leading to this 'loss' of ozone creates two initial chemical products, which can then go on to react with other chemicals present in the indoor environment[9]. These subsequent reactions and their products are often referred to as 'secondary chemistry'.

In contexts where ozone is just coming in from outdoors, with no indoor sources, ozone loss is the same as the difference between the indoor and outdoor ozone level—i.e. if the outdoor ozone level is 25 ppb and the indoor level is 5 ppb, the ozone loss is 20 ppb.

In more general terms, $[O_3]_{loss}$ can be defined as the difference between $[O_3]_{in}$ and what $[O_3]_{in}$ would be if the decay constant $k_{sum} = 0$. Equations 6.2, 6.4 and 6.5 can therefore be substituted and rearranged:

Equation 6.6

$$[\mathrm{O_3}]_{\mathrm{loss\_outdoor\ source}} \approx [\mathrm{O_3}]_{\mathrm{out}} \cdot \frac{k_{\mathrm{sum}}}{k_{\mathrm{sum}} + \lambda_{\mathrm{ACH}}}$$

Equation 6.7

$$[O_3]_{loss\_outdoor\ source} \approx [O_3]_{in\_outdoor\ source} \cdot \frac{k_{sum}}{\lambda_{ACH}}$$

Equation 6.8

$$[O_3]_{loss\_UVC} \approx [O_3]_{in\_UVC} \cdot \frac{k_{sum}}{\lambda_{ACH}}$$

or

$$[O_3]_{loss\_UVC} \approx \frac{I \cdot G \cdot k_{sum}}{\lambda_{ACH} \cdot (k_{sum} + \lambda_{ACH})}$$

Equation 6.9

$$[O_3]_{loss\_total} \approx [O_3]_{loss\_outdoor\ source} + [O_3]_{loss\_UVC}$$

Suppose an increase in $[O_3]_{in}$ has been observed. Has ozone loss increased or decreased? These equations show that it depends on the cause of the increase in $[O_3]_{in}$. If it is due to an increase in $[O_3]_{out}$ (equation 6.6), or due to an indoor source like far-UVC (equation 6.8), then the increase in $[O_3]_{in}$ also means an increase in $[O_3]$loss. However, if there is an increase in $[O_3]_{in}$ due to increasing ventilation (λ_{ACH}) bringing outdoor ozone indoors at a faster rate, then $[O_3]_{loss}$ has actually decreased (equation 6.6).

Comparing equations 6.6 and 6.8 is also instructive on the degree to which varying ventilation affects ozone loss depending on the source of ozone. In the case of ozone loss from outdoor ozone, ozone loss is inversely proportional to (k_{sum} + λ_{ACH}). However in the case of an indoor source of ozone like far-UVC, ozone loss is inversely proportional to λ_{ACH} * (k_{sum} + λ_{ACH})—a much stronger dependence.

At low rates of ventilation, changes in ozone loss from an indoor source can be substantial even from fairly small amounts of ozone generation. For example, suppose we are using a fluence rate of just 0.1 μW/cm² in a typical indoor space with a k_{sum} of 2, at 0.1 ACH—the lower bound of what might be expected in residences. The increase in indoor ozone concentration and ozone loss using an ozone generation constant of 10.5 would be:

Equation 6.4 (example)

$$[O_3]_{in\_UVC} \approx \frac{0.1 \cdot 10.5}{2 + 0.1} = 0.5\ ppb$$

Equation 6.8 (example)

$$[O_3]_{loss\_UVC} \approx 0.5 \cdot \left(\frac{2}{0.1}\right) = 10.0\ ppb$$

Ozone loss from an indoor source is far more sensitive to changes in ventilation than indoor ozone concentrations are, and the potential health effects of the products of ozone loss (see *Ozone epidemiology* section) drive much of the concern about ozone among indoor air quality experts.

TABLE 6.7. Estimated sensitivity of ozone concentrations and ozone loss to ventilation.

Assumptions:

Fluence rate	1 μW/cm²
Ozone generation constant	10.5 pph/hr/(μW/cm²)
k_{sum}	2 hr^{-1}

ACH	Increase in $[O_3]_{in}$ (ppb)	Increase in $[O_3]_{loss}$ (ppb)
0.1	5	100
0.5	4.2	16.8
1	3.5	7
2	2.6	2.6
5	1.5	0.6
10	0.9	0.2
20	0.5	0.2
30	0.3	0

Products of ozone loss

The best-understood product of ozone loss is the creation of secondary organic aerosol (SOA), through the reaction of ozone with VOCs. This does not mean that other products of ozone loss are necessarily less important when it comes to health effects, but rather that we do not currently have a framework for quantifying specific products and relating them to hazard ratios from epidemiological studies.

SOA represents a small fraction (~5%) of the total yield of products from ozone loss. The vast majority of the loss is due to reactions with surfaces, of which approximately half then partition to the gas phase with the balance of products remaining on surfaces[9]. The following discussion is restricted only to what we know about secondary organic aerosol.

The total mass of 'fine' particles—those with a diameter of less than 2.5 microns—is commonly referred to as $PM_{2.5}$. $PM_{2.5}$ in outdoor air has been regulated by the EPA in the United States since 1997, after numerous studies showed $PM_{2.5}$ levels to be more important to health than the previously monitored and regulated measures that included larger particles (PM_{10})(see for example the seminal 'Six Cities Study'[19]). The literature about harms from outdoor particulate matter is significantly more extensive than that of ozone. The current consensus is that there is not a threshold below which $PM_{2.5}$ exposure is benign[20], and that the exposure-response relationship may have a steeper slope at lower $PM_{2.5}$ concentrations[21].

We can estimate the average yield of $PM_{2.5}$ using the ozone loss framework. What is required is a parameter for the quantity of secondary organic aerosol produced for every ppb of ozone loss.

Weschler and Nazaroff, 2023[9] estimate what they term a $Yield_{SOA}$ of 0.03–0.07 µg/m³ for every ppb of ozone loss in an indoor space. Using equation 6.8 we can therefore estimate the concentration of secondary organic aerosol in µg/m³ per µW/cm² of fluence rate, using an ozone generation constant of 10.5 ppb/hr/(µW/cm²), varying k_{sum} and λ_{ACH} and taking the high end of the estimate for $Yield_{SOA}$ (0.07 µg/m³).

TABLE 6.8. Estimated PM yield from 1 µW/cm² of far-UVC from a KrCl* lamp.

Estimated PM yield							
λ	0.1	0.5	1	2	3	5	10
k_{sum}							
4	5.12	0.93	0.42	0.18	0.1	0.05	0.02
3.5	5.1	0.92	0.41	0.17	0.09	0.04	0.01
3	5.08	0.9	0.39	0.16	0.09	0.04	0.01
2.5	5.05	0.88	0.38	0.15	0.08	0.04	0.01
2	5	0.84	0.35	0.13	0.07	0.03	0.01
1	4.77	0.7	0.26	0.09	0.04	0.02	0
0.5	4.38	0.53	0.18	0.05	0.03	0.01	0

In poorly ventilated spaces, it seems reasonable to assume that the generation of secondary organic aerosol is at least ~1 µg/m³ for every ~1 µW/cm² of room average fluence rate from a KrCl* lamp, and could be as high as ~5 µg/m³. At higher levels of ventilation, it will be substantially less. In the case of Link et al., 2024[22], ~3.2 µW/cm² of room average fluence rate produced 1.8 µg/m³ ± 0.7 of SOA, or 0.56 µg/m³ ± 0.2 of SOA per µW/cm² of room average fluence rate. For context, the EPA estimates[23] the mean annual average $PM_{2.5}$ levels outdoors across over 300 sites in the United States to be 8.5 µg/m³.

It should be noted that this estimate implicitly assumes that the creation of ozone alone explains the SOA formation under far-UVC irradiation. Furthermore, in environments with high levels of ozone-reactive VOCs (such as after using household cleaning products containing limonene) the $Yield_{SOA}$ will be greater. Other factors that will affect particle formation and growth include relative humidity and the size and density of pre-existing particles in the air. While 0.03–0.07 µg/m³ for every ppb of ozone loss is a reasonable central estimate to use, we should expect the true variation to potentially be significantly wider than this range.

Weschler and Nazaroff, 2023 assume that the $Yield_{SOA}$ is proportional to ozone loss. However, from first-principles reasoning this will not always be the case. The ozone loss framework implies that ozone loss is inversely proportional to the ventilation rate at any given level of indoor ozone concentration. However, having a fixed $Yield_{SOA}$ implicitly assumes that the concentrations of precursors to SOA formation are constant, and this will not always be the case. For example, some precursors to SOA formation, such as VOCs from cleaning products, will have their own concentrations affected by ventilation. In this case, we'd also expect $Yield_{SOA}$ to depend on ventilation. This creates a 'double effect' where ventilation is reducing the concentrations of both the VOC precursors that are available to be oxidized by ozone and also the products of that oxidation. In this specific case, SOA concentrations from ozone loss ought to be inversely proportional not to ventilation, but approximately to the square of ventilation.

Reality is of course messier—ozone-initiated surface chemistry can also contribute to SOA formation, and ventilation could accelerate the diffusion of VOCs from areas where they might have a high concentration (e.g. a bathroom) to the rest of a building[f]. However, it is a reasonable hypothesis that the ozone loss framework may underestimate $Yield_{SOA}$ at low rates of ventilation and/or overestimate it at high rates of ventilation.

Does ozone loss alone explain secondary organic aerosol generation under far-UVC?

Ozone (O_3) is not the only potential oxidant created by far-UVC. O_3 can also be further photolyzed by far-UVC photons to form highly reactive OH radicals.

$$O_3 + hv \rightarrow O(^1D) + O_2$$
$$O(^1D) + H_2O \rightarrow OH + OH$$

Note that only a fraction of excited oxygen $O(^1D)$ will react with water as opposed to oxygen (to re-form ozone) or nitrogen (to form NO), with the fraction depending on relative humidity. At 40% relative humidity and 20°C, approximately 6.5% of $O(^1D)$ will react with H_2O (Jesse Kroll, private correspondence).

OH can also be formed through reactions of O_3 with alkenes already present in the environment.

$$\text{Alkene} + O_3 \rightarrow OH + \text{other products}$$

Barber et al., 2023[3] found that under exceptionally high fluence rates in a small chamber (45 µW/cm²) OH radicals being generated under far-UVC irradiation will affect the aerosol content separately from what would be expected from ozonolysis reactions alone. Separately, two other studies using lower fluence rates in larger chambers found that modeling based on ozonolysis alone was sufficient to account for the vast majority of secondary organic aerosol due to reactions with the VOC limonene[24,25]. Similarly, in the real-world bathroom study of Link et al., 2024[15], the authors found no need to posit any additional source of SOA other than ozone-initiated chemistry.

f Thanks to Charlie Weschler for adding this nuance in private correspondence.

OH generation due to photolysis of O_3 will be far more sensitive to fluence rate than OH production from alkenes. The rate of production of OH from reaction of O_3 with alkenes depends on O_3 and alkene concentrations, and therefore fluence rate only directly affects O_3 concentrations—it does not affect the yield of OH from a given quantity of O_3. However, in the case of OH production through photolysis the fluence rate affects both the rate of O_3 production and the yield of OH from a given quantity of O_3. In the simplified case where there was no background O_3 (i.e. only O_3 produced by far-UVC), we would therefore expect OH production to be proportional not to fluence rate, but rather to fluence rate squared.

New particle formation

Goss and Kroll 2024[25] found new particle formation from far-UVC in a small chamber at high fluence rates (45 μW/cm²) that does not appear to be explicable by ozonolysis or OH oxidation of easily measurable concentrations of VOCs. This new particle formation was not observed at fluence rates relevant to real-world applications. Link et al., 2024 also found new particle formation[15], although without an ozone-only control it is not possible to distinguish between particle formation from ozone-initiated pathways and any other as-yet unelucidated particle formation pathways. It should be noted that in the experiment in an office space reported in Narouei et al., 2024, with an estimated fluence rate of 5.19 μW/cm², no significant new particle formation or change in the distribution of particle size was observed[16].

Variation in SOA formation

We should expect the SOA products of ozone reactions to vary across indoor spaces by orders of magnitude more than the variation in the indoor/outdoor ozone ratio. To take just one example, limonene is a chemical in a class known as monoterpenes. Monoterpenes can typically be found in plant extracts and essential oils, and they are used to provide fragrances to consumer products. Limonene, which is found in the peel of citrus fruits and is cheap to manufacture, provides the fresh lemon or citrus smell commonly used in cleaning products. Limonene is an example of an ozone-reactive VOC that will react efficiently with ozone to form SOA.

Jenks et al., 2024[24] cite typical limonene concentrations in indoor spaces of 4–12 ppb. In the new proposed ASTM standard for evaluating the performance of portable air cleaners, the 'typical' concentration of limonene used to test the air cleaners is 5 ppb[26]. In an extensive literature review of concentrations of chemicals in residences, the difference between the 25th and 95th percentile of limonene concentrations was a factor of 100[27]. In places like spas, limonene levels can spike dramatically after the use of scented oils, reaching up to 3,000 ppb[28]. High limonene emissions from using scented cleaning products, in combination with indoor ozone, can create air quality issues similar to those found in polluted urban areas[29].

Formaldehyde

Formaldehyde is another product of ozone loss. It is a major indoor pollutant of concern with well-known health effects under high exposure (see for example Hauptmann et al., 2009[30]), and is formed by many oxidation reactions, including between limonene and O_3. In highly controlled experimental settings, an increase in formaldehyde has been observed under far-UVC irradiation in the presence of normal background levels of limonene[26], although the magnitude (low single digit ppb in a sealed test chamber) is small relative to ambient levels of formaldehyde in urban and indoor air, and to recommended exposure limits[31].

Key takeaways

- Ozone reacts with surfaces, VOCs, and human skin oils, leading to the creation of secondary chemical products.
- Secondary products may be of greater relevance to health than ozone itself.
- Increased ventilation generally reduces the concentration of these secondary products despite increasing steady-state ozone levels.
- Ozone appears to be the primary driver of secondary organic aerosol (SOA) formation under far-UVC, though other mechanisms including OH radicals may also contribute.
- SOA production can vary depending on the indoor environment and the presence of specific VOCs such as limonene.
- Variations in limonene concentrations will also drive differences in other potential chemistry, such as the formation of other VOCs like formaldehyde. It is not currently known whether any of these non-SOA byproducts are quantitatively significant.
- Other potential photochemical interactions that could affect indoor air quality

Thus far, we have discussed products that are downstream of ozone chemistry. However, we must also ask whether far-UVC creates other significant photochemical interactions that are not mediated by ozone chemistry. Sørensen et al., 2024[10] detected increases in acetic acid and butyl fragments that are not explained either by O_3 or OH oxidation chemistry. They hypothesized that these increases were caused by either direct photolysis of another compound or by far-UVC accelerating off-gassing of surfaces that was already occurring. While these increases were small relative to exposure limits for acetic acid and butanol respectively, the study illustrates the possibility that far-UVC could involve relevant photochemistry that is separate from ozone chemistry.

Our knowledge about the propensity of far-UVC to interact with chemicals in the air is based almost entirely on stratospheric chemistry, as no far-UVC is naturally found below the ozone layer. The absorption cross-sections that we do have are therefore primarily based on molecules that can be found in the stratosphere. There has been no cause to investigate the propensity of far-UVC to interact with the much wider range of molecules found in the built environment.

Not only are the unknowns large in terms of predicting the chemical consequences of far-UVC, there are also significant unknowns in understanding what if any toxicological significance there could be of any changes that do take place. EPA's ECOTOX database[32] contains over 13,000 chemical species. In the vast majority of these cases, we only have indicative evidence of the toxicological significance of the chemical. Between the uncertainty around what chemical species could potentially be created and the uncertainty around whether their creation is significant to health in the relevant quantities, there are 'unknown unknowns'.

However, if chemical changes observed in the air under far-UVC irradiation are adequately explained by well-known ozone chemistry, then the potential effects of far-UVC are bounded by the epidemiological

literature on harms from outdoor ozone exposure (see *Ozone epidemiology* section). The first priority should be taking careful, non-targeted measurements with sensitive equipment in diverse environments, along the lines of Link et al., 2024[15], and looking for chemistry that can't be explained by ozone generation and secondary chemistry. It should be noted that there is an inherent tradeoff in mechanistic studies between using realistic fluence rates (and therefore potentially getting more generalizable results) and using higher fluence rates that allow for sufficient signal to detect small effects.

The second priority is to consider what chemical factors are most likely to be ubiquitously present in occupied spaces utilizing far-UVC, of which the most obvious is the chemical impacts of the occupants themselves. The propensity of the skin oil constituent squalene to react with ozone[18] and the secondary chemistry engendered creates a 'human oxidation field' that surrounds each of us[33]. These occupant-specific factors are likely significant mediators of how increased ozone levels change the chemical species we are exposed to. It would be prudent to establish that far-UVC does not materially alter the dynamics of this air chemistry that is highly local to the human body, beyond the effect of ozone generation.

Key takeaways

- Current understanding of far-UVC interactions is based on stratospheric chemistry, leaving many unknowns about indoor impacts.
- Sensitive, non-targeted measurements in diverse environments are needed to detect unexpected chemical changes under far-UVC exposure.

Current consensus on indoor ozone emissions

The consensus that generating ozone indoors ought to be avoided wherever possible predates the emergence of far-UVC. For example, the following position statement was first adopted by the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) in 2015, and was reaffirmed in 2024[34]:

> Certain air cleaners produce ozone by design to achieve air-cleaning effects and the removal of contaminants. Additionally, ozone can be produced as a by-product of air-cleaning processes. Any air-cleaning device that uses electricity during [the] air cleaning process has the potential to generate ozone. In practice, ozone generation is associated with air cleaners that use high-voltage coronas or pin ionizers (e.g., some precipitators or ionizers), UV light of a sufficiently small wavelength (some photocatalytic oxidizers and UV-C air cleaners), and by some plasma air cleaners. Packaged air cleaners employing different air-cleaning technologies may use or produce ozone; examples include ozone generators or ionizers.
>
> Ozone is harmful for health and exposure to ozone creates risk for a variety of symptoms and diseases associated with the respiratory tract (Koren et al. 1989; Touloumi et al. 1997; Bell et al. 2004). Many products of ozone homogeneous and heterogeneous reaction processes also create risks for health, including formaldehyde, unsaturated aldehydes (produced during the reaction of ozone with ketones and alcohols), and ultrafine particles (secondary organic aerosols) (Weschler 2006).
>
> Ozone emission is thus undesirable. However, there is no consensus on the safe level of ozone. For example, ASHRAE's Environmental Health Committee (2011b) issued an emerging issue brief suggesting "safe ozone levels would be lower than 10 ppb" and that "the introduction of ozone to indoor spaces should be reduced to as low as reasonably achievable (ALARA) levels." Still, even widely used guidelines are not entirely consistent with all available epidemiological literature on the effects of ozone, and there is relatively little known about the long-term effects of exposure to low concentrations of ozone.
>
> The current state of the science regarding the health effects of ozone strongly suggests that the use of air cleaners that emit ozone by design should not be permitted; the same information and advice is given by the US EPA, among others (EPA 2013). There is more uncertainty about recommendations for air cleaners that do not use ozone by design for air cleaning but produce ozone unintentionally, as a by-product of their operation. There are devices that emit ozone but at the same time reduce concentrations of other harmful contaminants. The state of the science does not allow making highly certain trade-offs between increased exposure to ozone and the ozone reaction by-products and reduced exposure to other contaminants.
>
> In the absence of robust information regarding safe levels of ozone, the precautionary principle should be used. Any ozone emission (beyond a trivial amount that any electrical device can emit) should be seen as a negative and use of an ozone-emitting air cleaner, even though the ozone is an unintentional by-product of operation, may represent a net negative impact on indoor air quality and thus should be used with caution. If possible, non-ozone-emitting alternatives should be used.
>
> At the beginning of the coronavirus pandemic, the Ventilation in Buildings guidance issued by the CDC (FAQ 8)[35] recommended that air cleaning technologies employed should have UL 2998 certification, a 'Zero Ozone Emissions' test that is discussed later in the section on *Guidance, standards, and regulations*.

Given the uncertain but potentially meaningful effect sizes in the epidemiological literature on ozone exposure (discussed in the next section on *Ozone epidemiology*), and that prior to far-UVC there has been no compelling reason to use ozone-generating devices indoors, it is entirely reasonable that this consensus has emerged. The potential of far-UVC to have benefits that could outweigh these risks requires the careful tabulation of costs and benefits in a manner that has not previously been required.

Ozone mitigation

Commercial off-the-shelf technology for removing ozone from the air already exists, although it is not widely employed outside of commercial aviation[36]. The technology works by causing chemical reactions with ozone that create harmless products, as opposed to the (potentially) harmful products of other oxidation reactions that would otherwise occur.

As discussed below, these technologies likely require filtration of other contaminants in order to maintain their efficiency at removing ozone, and therefore it is possible that their use could synergize with the use of

mechanical filtration (e.g. HEPA) in a combined air cleaning solution. If ozone mitigation proves to be necessary for far-UVC to reach its potential, this area requires more research and development.

Given the potential harms of indoor ozone exposure even in the absence of far-UVC (see *Ozone epidemiology* section), the widespread deployment of ozone removal technologies could potentially be justified on their own terms. However, such an analysis is outside the scope of this document.

Activated carbon

Activated carbon consumes ozone through oxidation reactions resulting in either carbon dioxide (CO_2) or carbon monoxide (CO) products[37]. While both CO_2 and CO can pose health risks, they only do so in quantities vastly higher than those plausibly produced by these reactions. This is an instructive case for the dictum "the dose makes the poison"; taking the National Ambient Air Quality Standard for protection of public health as an example[38], the maximum recommended average concentration over 8 hours for CO is 9 parts per million, whereas for ozone it is 70 parts per billion. Similarly, ambient levels of outdoor CO_2 are typically in the range of 300–500 ppm. Transforming ozone into CO and/or CO_2 is therefore not a cause for concern.

We expect that readily-available activated carbon-based commercial devices will be effective at removing ozone, and these are currently being tested under challenge with far-UVC devices in research labs. The main drawbacks of activated carbon filters are the potential bulk of an adequately sized filter and that, like any filter, they will need periodic replacement.

Activated carbon filters can also remove other products of concern, such as VOCs[39]. If we were to discover that there are other photochemical interactions that produce products of concern, then using activated carbon filters alongside far-UVC could mitigate their impact as well. However, this feature has a drawback—while it is possible for there to be minimal degradation in the performance of activated carbon filters in removing ozone over several years[40], high VOC loads will reduce the filters' ozone removal efficiency over time[41].

Performance will also degrade if the filter accumulates dust and particulate matter, necessitating the additional use of other forms of filtration (e.g. HEPA or MERV 13) to protect the performance of the carbon filter. Performance can also be affected by temperature and relative humidity, and there is a tradeoff between putting more airflow through the filter and the amount of ozone removed every time air passes through the filter (i.e. the single pass efficiency of ozone removal declines with the rate of airflow)[42].

Catalysts

The other primary method of ozone removal is through catalytic decomposition using a metal oxide catalyst. Ozone adsorbs to the catalyst to initially form a peroxide or superoxide intermediate species, which then desorbs as O_2[43]. This is not to be confused with photocatalytic air cleaners, which can potentially create a number of unintended byproducts[44]. Catalysts will also remove some VOCs and may be particularly effective at removing formaldehyde, which can undergo oxidation and decomposition into carbon dioxide and water[45].

Catalysts can be either 'passive' or 'active'. In the case of an active catalyst, air is actively moved with a fan over a catalytic surface, much as air is actively moved through a filter. Passive catalysts, on the other hand, use exposed building materials such as clay-based paint that will catalytically decompose ozone as the air circulates naturally around a room[46–49]. Active catalyst devices can likely achieve removal rates comparable to activated carbon filters, whereas the potential for passive catalysts requires more investigation and will be affected by airflow patterns in the room as well as how much of the interior surface can be coated with the catalyst.

Catalyst performance can degrade over time for a number of reasons, all which ultimately come down to a reduction in the surface area that is available for catalytic reactions to take place. For example, a report[50] on degradation in performance of catalysts used in commercial aviation demonstrated that contaminants leaking into the air stream such as lubricating oils, hydraulic fluids, and/or de-icing agents reduced the available surface area and degraded catalyst performance. In the use case of indoor spaces, the most likely source of soiling will be dust and particulate matter, which could be mitigated for an active catalyst by pre-filtering the air to maintain performance. However, unless the filter contained sufficient activated carbon, this would not prevent semi-volatile organic compounds (SVOCs) from potentially soiling the catalyst. In the case of passive catalysts, cleaning the catalytic surface may be necessary to maintain performance. Studies of long-term catalyst performance in real world environments are warranted.

Key takeaways

- Ozone can be effectively removed from indoor air using activated carbon filters and metal oxide catalysts, but this adds additional costs and requires energy to move the air through the filter or catalyst.
- Mitigating indoor ozone from outdoor sources may have public health benefits independent of mitigating ozone from indoor sources like far-UVC specifically (see *Ozone epidemiology* section).
- Further development of these technologies is needed to ensure their effectiveness in real-world applications.

Further reading

- Model Evaluation of Secondary Chemistry due to Disinfection of Indoor Air with Germicidal Ultraviolet Lamps
- Indoor Air Quality Implications of Germicidal 222 nm Light
- Ozone Generation from a Germicidal Ultraviolet Lamp with Peak Emission at 222 nm
- Significant Production of Ozone from Germicidal UV Lights at 222 nm
- Ozone Loss: A Surrogate for the Indoor Concentration of Ozone-Derived Products
- Ozone and ultra-fine particle concentrations in a hotel quarantine facility during 222 nm far-UVC air disinfection

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7. Ozone epidemiology

Summary

While far-UVC devices generate a small quantity of ozone relative to prevailing levels of outdoor ozone (see *Ozone and indoor air quality* section), this does not necessarily mean it is insignificant to human health.

We have far fewer studies on the potential harms of ozone exposure relative to other air pollutants, such as fine particulate matter. However, the evidence we do have suggests that ozone has an exposure-response relationship with all-cause mortality. This needs to be taken into account when using far-UVC, with a particular focus on the potential risks of the products of secondary chemistry in low ventilation environments.

The magnitude of potential harms from indoor ozone generation is uncertain, but potentially large enough that it should be explicitly factored into cost-benefit calculations. However, the potential harms are not so large that they cannot be significantly outweighed by benefits if far-UVC is highly effective at preventing the spread of respiratory disease.

Crucial considerations

- Indoor ozone and products of ozone loss are correlated with outdoor ozone levels.
- Exposure to any of outdoor ozone, indoor ozone, and the products of ozone loss may contribute to the health associations found in the outdoor ozone epidemiological literature.
- Thresholds below which ozone is benign, if they exist, may be different in different contexts depending on the causes of the association between ozone and adverse health outcomes. It is not necessarily the case that a 'threshold effect' based on outdoor ozone epidemiology means that such a threshold can naively be applied indoors.
- Depending on these relative contributions, small long-term increases in exposure to ozone indoors **could** be associated with an increase in all-cause mortality.
- These effects may be higher in some populations, such as infants or the elderly.
- If the health risk associations found for outdoor ozone are caused by the products of ozone loss, then the potential harms of indoor ozone generation will be more severe in poorly ventilated spaces.
- On the other hand, if the health risk associations for outdoor ozone are caused by direct ozone exposure, then increasing ventilation could exacerbate the problem by bringing in ozone from outside and further increasing indoor ozone concentration.
- Technology that mitigates ozone formation directly ought to reduce potential health risks no matter the causal mechanism.

Analysis

Epidemiology of outdoor ozone

Long-term epidemiological studies estimate an 'exposure-response relationship' arising from correlations between outdoor ozone levels, mortality, and other health impacts such as respiratory and cardiovascular disease. In work related to far-UVC, the most commonly cited paper has been Turner et al., 2016[1], which estimated a hazard ratio (HR) for all-cause mortality of 1.02 for every 10 parts per billion (ppb) increase in average outdoor ozone levels in the United States. The data came from nearly 669,046 participants in a long-term cancer cohort study from 1982–2004.

There is also evidence of shorter-term increases in mortality due to increases in ozone exposure. In a study of 95 US urban communities, Bell et al., 2004[2] found a 0.52% increase in daily mortality following a 10 ppb increase in the previous week's ozone level.

Two more recent studies on ozone mortality risk focus on specific subpopulations that could be at higher risk. Xue et al.[3] investigated the exposure response of outdoor ozone levels to mortality in children under 5 in low- and middle-income countries, and estimated a HR for all-cause mortality of 1.064 for every 10 ppb, over three times higher than the estimate of Turner et al. However, this was only observed for outdoor concentrations above 50 ppb,

which will be discussed further in the subsection on threshold effects. Zhang et al.[4] estimated a HR of 1.097 for every 10 ppb of ozone in a study population of over-65s in China, where the average age was 87. This is five times higher than Turner et al.'s estimate, and with no threshold effect reported.

The negative health effects are observed across a wide variety of vascular and respiratory diseases:

TABLE 7.1. From Turner et al., 2016[1]. All-cause and cause-specific mortality hazard ratios in relation to each 10-unit increase in air pollutant concentrations, 1982–2004 follow-up in American Cancer Society prevention study II cohort, United States (n = 669,046).

Cause of death	ICD-9 Codes; ICD-10 Codes	Deaths (n)	HBM O_3	Regional $PM_{2.5}$	Near-source $PM_{2.5}$	LUR NO_2
All-cause mortality	All	237,201	1.02 (1.01–1.04)	1.04 (1.02–1.06)	1.26 (1.19–1.34)	1.01 (1.00–1.03)
Diseases of the circulatory system (plus diabetes) (48)	390–459; I00–I99, E10–E14	105,039	1.03 (1.01–1.05)	1.07 (1.04–1.10)	1.41 (1.29–1.54)	1.03 (1.01–1.05)
Cardiovascular	410–440; I20–I25, I30–I51, I60–I69, I70	84,132	1.03 (1.01–1.05)	1.07 (1.04–1.10)	1.35 (1.23–1.49)	1.03 (1.01–1.06)
Ischemic heart disease	410–414; I20–I25	45,644	0.98 (0.95–1.00)	1.10 (1.07–1.13)	1.40 (1.23–1.60)	1.05 (1.02–1.08)
Dysrhythmias, heart failure, cardiac arrest	420–429; I30–I51	18,314	1.15 (1.07–1.23)	1.05 (0.99–1.10)	1.09 (0.94–1.27)	0.99 (0.95–1.04)
Cerebrovascular disease	430–438; I60–I69	17,085	1.03 (1.00–1.07)	1.07 (1.02–1.11)	1.22 (1.05–1.42)	1.02 (0.98–1.07)
Diabetes	250; E10–E14	4,890	1.16 (1.07–1.26)	1.04 (0.96–1.12)	1.25 (0.97–1.62)	1.09 (1.00–1.19)
Diseases of the respiratory system	460–519; J00–J98	20,484	1.12 (1.07–1.18)	1.15 (1.09–1.21)	1.31 (1.15–1.50)	1.07 (1.02–1.13)
Pneumonia and influenza	480–487; J10–J18	6,599	1.10 (1.03–1.18)	1.24 (1.12–1.37)	1.24 (0.94–1.64)	1.07 (1.00–1.14)
COPD and allied conditions	490–496; J19–J46	9,967	1.14 (1.08–1.20)	1.06 (1.00–1.12)	1.17 (0.97–1.42)	1.04 (0.99–1.10)
Lung cancer	162; C33–C34	16,342	0.96 (0.91–1.00)	1.13 (1.07–1.21)	1.31 (1.05–1.63)	0.94 (0.90–0.99)

While the 1.03 HR for diseases of the circulatory system is lower than the 1.12 HR for diseases of the respiratory system, the absolute mortality due to circulatory disease is approximately 30% higher than respiratory disease due to the substantially higher underlying level of risk. Approximately ¼ of the estimated impact on deaths from respiratory illness are accounted for by pneumonia and influenza, demonstrating that infectious disease and air quality are not separate phenomena, but interact with each other.

Asthma

A recent meta-analysis by Zheng et al.[5] estimated a hazard ratio of 1.008 for the effect of a 10 μg/m³ increase in *either* peak 8-hour outdoor ozone *or* average 24-hour outdoor ozone on emergency room visits or hospitalizations for asthma. Assuming 1 ppb of ozone is equal to 1.96 μg/m³, the hazard ratio for these outcomes is therefore 1.016 per 10 ppb.

Key takeaways

- Studies suggest that higher outdoor ozone levels are associated with increased mortality from cardiovascular and respiratory diseases.
- Children under 5 and the elderly may experience higher risks from ozone.

Translating outdoor ozone epidemiology indoors

Recalling the equation for indoor ozone concentrations from Weschler and Nazaroff[6] (see the *Ozone and indoor air quality* section, equation 6.2):

$$[\mathrm{O_3}]_{\mathrm{in}} \approx [\mathrm{O_3}]_{\mathrm{out}} \cdot \left(\frac{\lambda_{\mathrm{ACH}}}{\lambda_{\mathrm{ACH}} + k_{\mathrm{sum}}} \right)$$

And the equation for 'ozone loss' from chemical reactions (equation 6.6), which creates byproducts:

$$[\mathrm{O_3}]_{\mathrm{loss}} \approx [\mathrm{O_3}]_{\mathrm{out}} \cdot \left(\frac{k_{\mathrm{sum}}}{\lambda_{\mathrm{ACH}} + k_{\mathrm{sum}}} \right)$$

If we assume that λ_{ACH} (ventilation rate) and k_{sum} (ozone decay due to chemical reactions) are independent of outdoor ozone levels, then any change in outdoor ozone will correlate to changes in indoor ozone, ozone loss and the products of ozone loss.

Therefore, even if we knew that the hazard ratio was caused by variations in outdoor ozone (rather than them just being correlated), it would not be possible using epidemiological studies to determine whether outdoor ozone directly causes these health effects or whether it does so through its impact on indoor ozone or ozone loss, and therefore what the exposure-response relationship is for each factor independently.

There are several possibilities for where the balance of the harm falls, including:

- It might be that there is a concentration threshold below which the antioxidant capacity of our respiratory system can handle breathing in ozone without harmful long-term effects. If this is the case, we would expect the impact of exposure outdoors (where concentrations are higher) to be greater than that of indoor ozone (where concentrations are lower).

- It might be that there is no such threshold. If this is the case, because we spend much more of our time indoors, even though the absolute level of ozone indoors is lower than outdoors, indoor ozone makes up the majority of our exposure and therefore the harm observed.

- It might be that the secondary products of ozone loss are much worse for human health than ozone itself, and therefore the harm is associated with ozone loss.

Whatever the health effects are from (for example) our estimated ~1–5 μg/m³ of secondary organic aerosol for every ~1 μW/cm² of fluence rate in low ventilation conditions (see *Ozone and indoor air quality* section), or from other ozone loss products, *if these effects are caused only by ozone-initiated chemistry, they are already contained within the hazard ratio from outdoor ozone epidemiology*. The only effects of far-UVC on indoor air quality that would not be 'contained' within this hazard ratio are products that cannot be explained through ozone-initiated chemistry. For this reason, it is important to establish whether modeling ozone-initiated chemistry is sufficient to explain real-world observations of chemical changes in occupied spaces—if chemical changes are all accounted for by ozone, this permits us to use outside ozone epidemiology to at least bound the potential harms, even with the difficulties of causal decomposition.

Insofar as we have separate toxicology data on the exposure-response relationship for secondary organic aerosol (SOA)—one of the products of ozone loss—it is entirely in the context of outdoor air pollution. As SOA is a type of $PM_{2.5}$, epidemiological studies on the health effects of outdoor $PM_{2.5}$ are often used to estimate the health effects of SOA production. Outdoor $PM_{2.5}$ will also be present indoors through infiltration and ventilation, and therefore these epidemiological studies factor in both exposure to $PM_{2.5}$ outdoors and indoors, as they do with ozone.

But even supposing we had correctly decomposed the epidemiological exposure-response relationship for $PM_{2.5}$, there are many reasons for skepticism that this would translate to the exposure-response relationship of SOA produced from ozone loss. There is evidence that smaller particles, often referred to as Ultra Fine Particles or $PM_{0.1}$, could be more harmful than $PM_{2.5}$ (see for example Peters et al.[7]). Toxicology studies in mice suggest that the relative harm of toxic substances is proportional not to mass but surface area[8], and the surface area to mass ratio will increase exponentially with smaller particle size. The particle size distribution of outdoor $PM_{2.5}$ and particulate matter produced under far-UVC irradiation indoors will likely be different.

The implicit assumption of outdoor $PM_{2.5}$ epidemiology is that the particular chemical composition of the particles does not matter. This assumption is less likely to hold when translating to a different chemical context. For example, hydrophobic and hygroscopic particles deposit at different rates in lungs[9] and there are also differences in the oxidative potential of different aerosols[10,11]. The specific chemical composition of outdoor $PM_{2.5}$ may generally be similar enough over time that it makes sense to assume no effect from the particular chemical composition of the aerosol mass nor any effect from size distribution. However, it is not clear that this is valid for indoor-sourced PM.

While in the United States today outdoor $PM_{2.5}$ is now primarily constituted by secondary organic aerosol, especially in the summertime[12], in many of the studies used to assess the harms of PM exposure the secondary organic aerosol will constitute a minority of the total composition (since this was true in the past). For example, since the time of the seminal Six Cities study on air pollution and mortality[13], sulphur dioxide and nitrogen oxide emissions in the United States have fallen by around 90%[14].

Furthermore, we should not assume that the harm of ozone loss comes primarily from inhaling aerosol or gas phase products into our respiratory system. While the hazard ratio from outdoor ozone for mortality due to diseases of the circulatory system is lower than for the respiratory system, the absolute mortality from circulatory diseases is higher due to having a higher base rate[1]. Roughly half of the ozone loss to surface reactions remain on surfaces[6], and the potential harms of those reaction products,

which could for example be ingested after contact with surfaces, is also not known. For gas phase products, dermal absorption may also be an important pathway to products entering the circulatory system[15].

The data in Turner et al., 2016 suggests that either SOA from ozone loss does not have the same harms as outdoor $PM_{2.5}$, or SOA from ozone is not a material cause of the association between outdoor ozone and all-cause mortality. This is because while the ozone HR is four times *higher* for respiratory disease (1.12 per 10 ppb) relative to diseases of the circulatory system (1.03 per 10 ppb), it is the inverse for near-source $PM_{2.5}$—the HR of 1.17 per 10 µg/m³ for respiratory disease is 60% *lower* than the HR of 1.41 for diseases of the circulatory system[1]. If the ozone hazard ratio was actually caused by SOA from ozone loss, and SOA from ozone loss had the same effect on health as $PM_{2.5}$, then we would expect the causes of mortality to be similar.

One potential product of ozone loss on which there is some epidemiological data is formaldehyde. Formation of formaldehyde under far-UVC illumination has been observed in artificial experimental conditions[16]. The epidemiological data is inconsistent on whether formaldehyde constitutes a risk for leukaemia or other forms of cancer[17–20]. However, cancer risk is not one of the mortality causes with which ozone exposure is associated in the epidemiological literature, suggesting that whatever the cause of the association with mortality is, it is not through the most concerning potential risks of formaldehyde exposure.

If ozone loss did prove to account for a significant proportion of the harm of outdoor ozone, then this would be particularly challenging to characterize. As noted above, the exact quantity and chemical composition of the products of ozone loss could vary dramatically in different indoor spaces, far more than the variation in indoor ozone concentrations.

The challenging nature of this research means that it is vital to make assumptions clear and explain calculations and methodologies in detail when making an estimate of the potential health effects of indoor ozone generation.

Until we can decompose the cause of the association between ozone and mortality in outdoor ozone epidemiology, we cannot even be confident whether using ventilation to mitigate ozone production from far-UVC lamps is beneficial. If it is the products of ozone loss that cause the harm, then moderate levels of ventilation ought to meaningfully reduce potential unintended harms from the use of far-UVC, given the strong dependence of ozone loss on ventilation (see *Ozone and indoor air quality* section). If instead it is the presence of the ozone itself that causes the harm, then additional ventilation can further increase the indoor ozone concentration by bringing more ozone in from outdoors. If it is ozone itself that causes the harm, it would also raise the question of whether in general the use of ventilation or far-UVC causes the greatest reduction in pathogens for the lowest increase in ozone exposure, particularly if outdoor ozone levels are elevated.

Key takeaways

- The relationship between the health effects of outdoor ozone exposure, indoor ozone exposure and exposure to the products of ozone loss is unclear.
- More ventilation could either reduce or increase health risks, depending on whether ozone or its byproducts are the main concern.

Is there a threshold below which ozone is benign?

If there was a threshold below which there was limited or no evidence of harm from ozone, then this could function as a *de facto* exposure limit for indoor air quality under far-UVC irradiation. So long as ozone concentration did not exceed this threshold , then we could be confident that there was no significant harm to human health from far-UVC-generated ozone. On the other hand, it's possible that there is no threshold below which ozone is benign, and that the harms increase linearly with exposure at all concentrations.

Threshold effects from outdoor ozone epidemiology

There is mixed evidence on threshold effects for ozone exposure. As noted above, Xue et al.[3] found a substantial hazard ratio for all-cause mortality in children under 5—1.064 for every 10 ppb increase—but this was only for levels above 50 ppb, suggesting a threshold effect. By contrast, Zhang et al.[4] found a linear exposure-response (i.e. no threshold) among older adults in China.

Turner et al.[1] tested extensively for potential thresholds:

> Threshold models, defined by setting the O_3 [ozone] concentration to 0 below the threshold and the concentration minus the threshold value otherwise, were examined at 1-ppb increments across the entire exposure range. Potential modification of O_3 associations by age at enrollment, sex, education, BMI, cigarette-smoking status, passive smoking, prior cardiovascular disease (high blood pressure, heart disease, stroke, or diabetes) or respiratory disease (asthma, emphysema, or chronic bronchitis) at enrollment, and temperature was assessed using multiplicative interaction terms. Two-sided P values based on the likelihood ratio statistic were calculated to assess their significance.

The authors found only limited evidence of a threshold:

> There was some evidence that a threshold model improved model fit for respiratory mortality at 35 ppb (P = 0.002) compared with a linear model using year-round but not summertime O_3 (HR per 10 ppb using threshold O_3 indicator at 35 ppb for respiratory mortality, 1.17; 95% CI, 1.11–1.22)... Results were somewhat suggestive of a threshold for circulatory mortality at 35 ppb (P = 0.07).

It is worth noting both that the assumptions behind statistical significance do not hold if the test is being repeated multiple times, and that even if the threshold remained statistically significant after adjusting for multiple comparisons, respiratory mortality accounts for slightly less than half of the total risk. For mortality due to circulatory disease (the majority of deaths), the evidence of a threshold was much weaker than for respiratory disease.

As for the effects of outdoor ozone on asthma emergency room visits and hospital visits, only one paper estimated a threshold (Zu et al.[21]) and it was at 40 ppb.

There is precedent in other living organisms for threshold effects of ozone exposure. Ozone is also known to affect crop yields[22], with different crops being sensitive to ambient ozone concentrations at different thresholds. We of course cannot directly apply these plant thresholds to humans, but this demonstrates the point that organisms plausibly have resistance or repair mechanisms that protect against lower levels of oxidant exposure. Modeling estimates suggest that increased anthropogenic emissions of nitrogen oxides (NO_x) and other precursors meant that tropospheric ozone was up to 40% lower in 1850 than it is today[23]. Even though outdoor ozone concentrations in the past were likely lower than those seen today, both humans and plants may have evolved under ozone concentrations that were higher than, or comparable to, those in modern indoor environments.

At best, the evidence for a threshold is mixed. If there is such a threshold, it may be somewhere between 35 ppb and 50 ppb of *outdoor ozone*.

Translating outdoor ozone thresholds indoors

Suppose for the sake of argument that there was a threshold effect at 35 ppb. What would this mean for increases in indoor ozone under far-UVC irradiation? It depends on the decomposition of ozone harms into $[O_3]_{out}$ (ozone from outside), $[O_3]_{in}$ (ozone generated inside) and $[O_3]_{loss}$ (ozone reacting and changing into other products).

The literature summary in Nazaroff and Weschler[24] finds median indoor ozone levels in residences of 6 ppb (Interquartile Range (IQR) 2–11 ppb) with outdoor ozone levels of 22 ppb (IQR 16–35 ppb) and a median indoor/outdoor ratio of 25% (IQR 11–37%). If a 35-ppb outdoor threshold was supported by the epidemiological literature, then the only thing we could say with confidence would be that indoor ozone levels and/or ozone loss *consistent with outdoor ozone being 35 ppb* are benign. This is anywhere around 4–10 ppb of indoor ozone based on the IQR. It is certainly *possible* that the entire harm comes from $[O_3]_{out}$ but this cannot be deduced one way or the other from the epidemiological evidence. Therefore, we cannot rule out that single-digit increases starting from single-digit ppb concentrations of indoor ozone—such as could be expected from typical uses of far-UVC—could be harmful, even if there is a threshold effect in outdoor ozone epidemiology that is much higher than typical indoor levels.

Key takeaways

- Some evidence suggests a possible threshold level of outdoor ozone below which there is no harm, but the data is mixed, and the threshold may vary between different populations and settings.
- Even if there's a threshold effect in outdoor ozone epidemiological studies, it's uncertain whether and how this applies to indoor ozone levels.

We should be very uncertain about the harm of ozone exposure

We should place large uncertainty bounds around the health effects of the unintended air chemistry of far-UVC. None of the estimates quoted above are based on studies from which we can directly infer causality, and a number of the assumptions that go into calculating proportional hazard ratios are unlikely to hold[25]. But even leaving aside causation, there is significant uncertainty about the scale of the association.

For example, Turner et al., 2016 earlier used information from participants in a large (n=669,046) prospect cancer study that began in 1982. However, this was not the first time that that dataset was used to estimate ozone harms. A previous analysis[26] of that same dataset found a higher hazard ratio for $PM_{2.5}$ harms (1.08 for all-cause mortality in a two-pollutant model, versus 1.04 for Regional $PM_{2.5}$ in Turner et al.[1]). However, ozone was actually negatively associated with all-cause mortality (HR 0.989, confidence interval 0.981–0.996), although this hides a positive association with respiratory illness (1.04) and negative with cardiovascular disease (0.983).

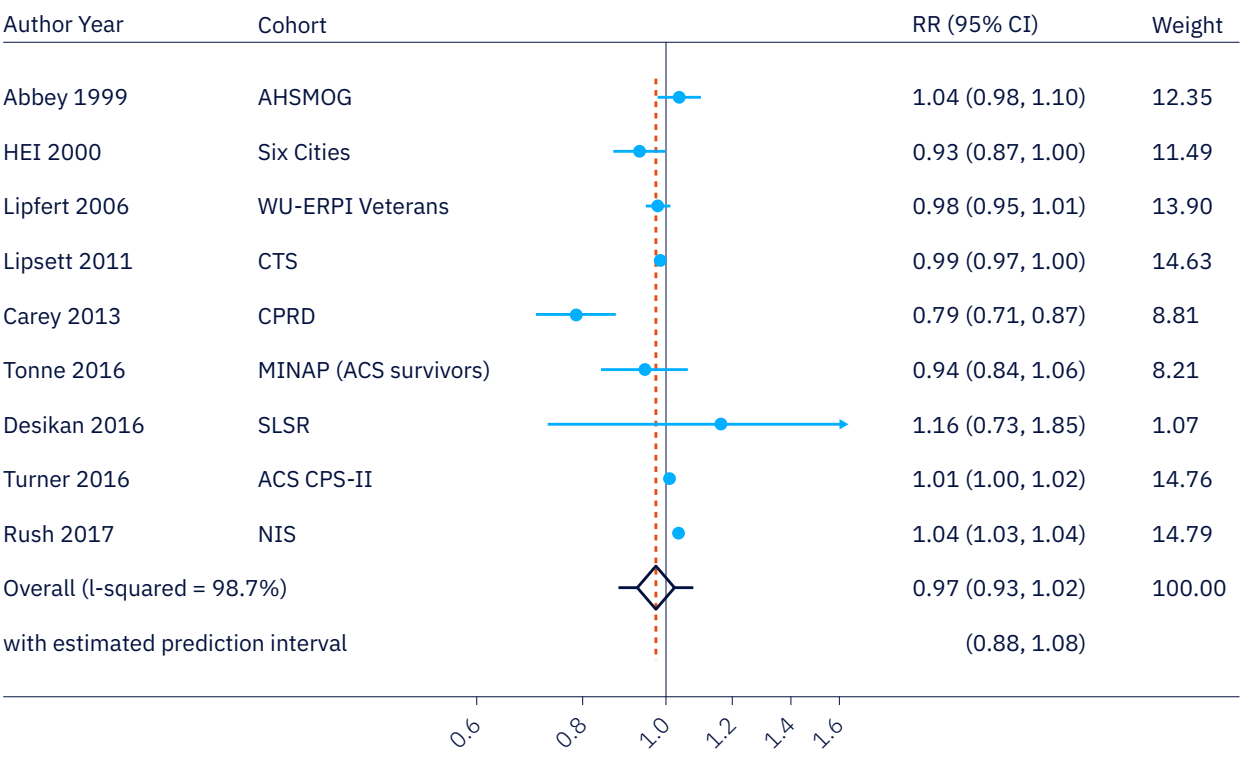
The Turner et al. analysis is superior, using new and more sophisticated estimates of ambient ozone, $PM_{2.5}$, and NO_2 concentrations to more accurately estimate individual exposure, as well as 50% more participants and nearly double the number of deaths. It is quite plausible that long-term exposure could be important, with the risk associated with that exposure therefore manifesting later in life. However, this is an enormous update in hazard ratio over a short time period between studies using similar methodologies, and our confidence that this latest estimate is definitively correct should be tempered appropriately.

Ozone epidemiology is an intrinsically challenging subject to study, in no small part due to large numbers of potential confounding variables. Ambient ozone levels are not randomly distributed across geographic regions—they vary due to a combination of emissions sources, atmospheric chemistry, weather patterns, and altitude[1]. As a result, health outcomes associated with ozone exposure can be influenced by a range of factors, including socioeconomic status, baseline air quality, and local climate conditions that affect both outdoor and indoor exposure levels. These hidden correlations can make it challenging to determine the precise health risks of ozone exposure, as well as the extent to which behaviors and environmental conditions may amplify or mitigate its effects.

There are also other earlier studies that show different results to Turner et al. One meta-analysis[27] identified fifteen studies going back to 1999, of which nine were included. This analysis (which put only a 14% weight on the Turner et al. study) found no association between *average* ozone levels and all-cause mortality, though it found a similar association to Turner et al. between mortality and *peak* ozone exposure. These associations are reproduced below in Figure 7.1. (NB: the values in the figure are expressed as hazard ratios per 10 μg/m³, as opposed to per 10 ppb. To convert between these, a factor 1.96 μg/m³ per pbb is used).

Mortality estimates from meta-analysis

O_3 annual exposure and all-cause mortality. Cochran's Q: Chi-square = 98.7, df = 8, P < 0.001. tau^2 = 0.004.



O_3 peak exposure and all-cause mortality. Cochran's Q: Chi-square = 78.48, df = 6, P < 0.001. tau^2 = 0.0002.

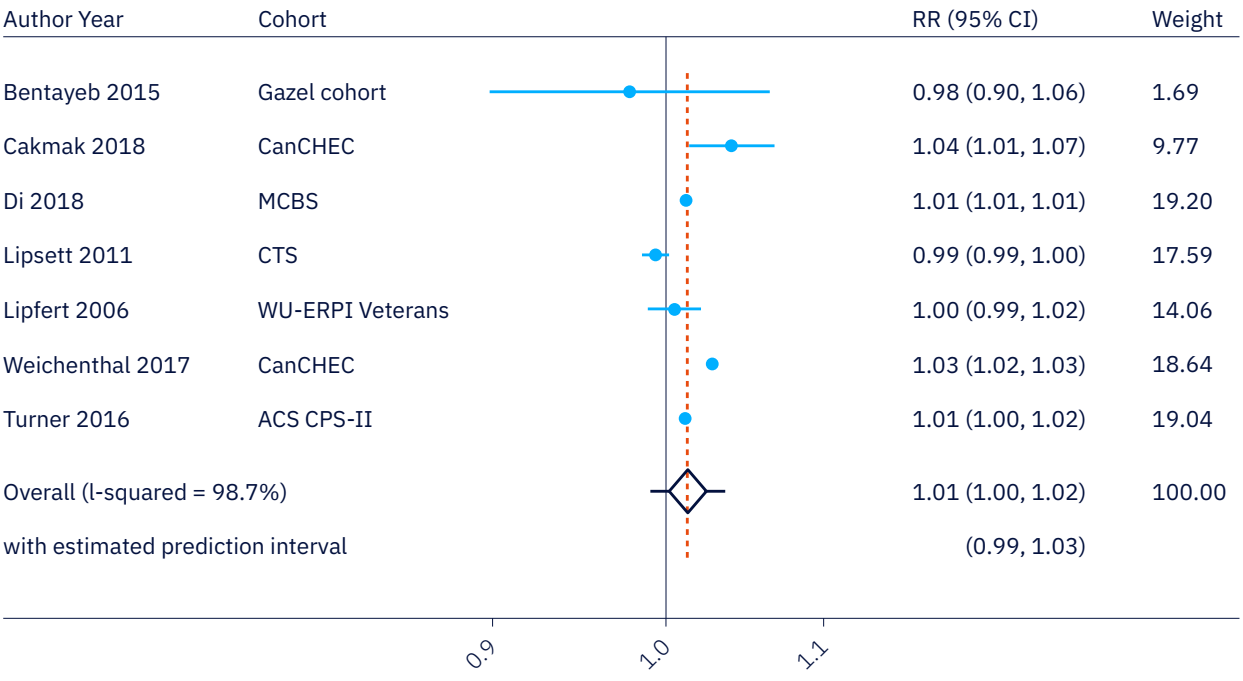


FIGURE 7.1. Excerpted from Huangfu and Atkinson, 2020.[27]

This could also represent some evidence of a threshold effect—if threshold effects were material then we would potentially expect peak ozone to have stronger effects than average concentrations.

The importance of NO_x emissions in increasing tropospheric ozone levels means that variations in local ozone will be related to variations in local NO_x. Vehicles are the most important source of NO_x emissions in urban areas, and therefore locally NO_x levels will vary spatially with proximity to roads and temporally with traffic conditions[28]. Local spatiotemporal variations in NO_x and therefore ozone levels mean that regional ozone monitors and models will in many cases not represent the conditions to which individuals are actually exposed. Our understanding from talking to experts is that this problem is likely to be more substantial for measuring the health impacts of ozone than for outdoor $PM_{2.5}$, where the certainty about the health effects is much higher.

With all of these caveats in place, we nevertheless think that it is responsible to attempt to bound the potential harms of ozone generation from far-UVC were it to be ubiquitously deployed, using the best available estimates from the epidemiological literature.

Key takeaways

- The health effects of ozone, especially indoors, are highly uncertain due to many confounding factors.
- Different studies offer varying estimates of ozone's health risks, with some earlier studies showing no clear association between ozone and mortality.

Bounding the potential harm of far-UVC ozone generation

Far-UVC could substantially reduce the burden of respiratory illness and potentially prevent future pandemics. Given this context, it is important to estimate some bounds for the plausible extent of harm from indoor-generated ozone under far-UVC irradiation, so that we can compare these harms against the potential benefits of deploying far-UVC.

This section is not a full cost-benefit analysis, but intended to gesture towards the importance of conducting such an analysis, and to highlight these calculations' sensitivity to critical assumptions.

Note that the following should be taken as possible orders of magnitude estimates only in a hypothetical scenario where far-UVC was deployed ubiquitously.

Pivotal assumptions include whether there are threshold effects for ozone exposure, how to attribute the harm of outdoor ozone epidemiology to particular causes, and whether exposure to ozone and ozone loss is *ubiquitously* and *permanently* increased.

The best case scenario for potential harms from indoor-generated ozone under far-UVC irradiation would be if the harm comes from ozone itself rather than products from ozone loss, and if there is a threshold effect, and if most indoor spaces (even under far-UVC irradiation) are below that threshold. In the vast majority of situations with normal levels of outdoor ozone, realistic far-UVC installations are not going to increase indoor ozone levels above 35–50 ppb.

Assuming instead that there is not such a threshold but that the cause of harm is still direct exposure to ozone itself, one estimate of harm would be to estimate the total quantity of individual exposure to ozone both outdoors and indoors, and decompose the epidemiological harm linearly with total indoor and outdoor exposure. This is the methodology of Xiang et al.[29], which attempted to model the hypothetical benefits of reducing indoor ozone exposures in urban China.

For this quantitative estimate of hypothetical harm under ubiquitous deployment, we will estimate the proportion of total ozone exposure that occurs indoors, and then rebase that hazard ratio to how indoor ozone changes with outdoor ozone (i.e. a 10 ppb increase in outdoor ozone causes a less than 10 ppb increase in indoor ozone).

We estimate the proportion of total ozone exposure from time spent indoors to be:

Equation 7.1

$$\mathrm{Exp}_{\mathrm{in}} = \frac{C_{\mathrm{in}} \cdot T_{\mathrm{in}}}{(C_{\mathrm{in}} \cdot T_{\mathrm{in}}) + (C_{\mathrm{out}} \cdot T_{\mathrm{out}})}$$

Where $C_{in/out}$ = Ozone concentration indoors/outdoors
$T_{in/out}$ = Time spent indoors/outdoors

For C_{in}, we use the median indoor/outdoor ozone ratio found in the Nazaroff and Weschler literature review (25%[24], and C_{out} is normalized to 1). For T_{in}, a commonly used estimate for high-income countries is that people spend 90% of their time indoors, and 70% in their homes (see for example Klepeis et al., 2001[30]).

Plugging these parameters into the above equation yields the following estimate of the proportion of total ozone exposure accounted for by indoor ozone:

$$\mathrm{Exp}_{\mathrm{in}} = \frac{\sim 25\% \cdot \sim 90\%}{(\sim 25\% \cdot \sim 90\%) + (1 \cdot \sim 10\%)} \approx \sim 69\%$$

On these assumptions, ~69% of the hazard ratio would therefore be accounted for by indoor ozone exposure. To calculate the final hazard ratio for an increase of 1 ppb indoor ozone exposure, we would then need to rebase the hazard ratio to indoor ozone concentration, rather than the outdoor ozone concentration.

Equation 7.2

$$\mathrm{HR}_{\mathrm{in}} = \frac{\mathrm{Exp}_{\mathrm{in}} \cdot \mathrm{HR}_{\mathrm{out}}}{C_{\mathrm{in}}/C_{\mathrm{out}}}$$

Taking an all-cause mortality increase of 2% per 10 ppb outdoors per Turner et al.[1], and assuming 69% of that is caused by (and linear in response to) indoor ozone exposure:

$$\mathrm{HR}_{\mathrm{in}} = \frac{\sim 69\% \cdot 2\%}{\sim 0.25} \approx\sim 5.5\% \text{ per 10 ppb}$$

Therefore, on these assumptions, a permanent and ubiquitous increase in indoor ozone of 1 ppb would increase all-cause mortality by ~0.55%.

It is extremely important when it comes to responsible use of far-UVC that this number is substantially lower if ozone concentrations were only increased in *nonresidential* indoor public spaces. Taking the same data but only using the ~20% of time on average that people spend indoors in places that are not their home:

Equation 7.3

$$\mathrm{Exp}_{\mathrm{public}} = \frac{C_{\mathrm{public}} \cdot T_{\mathrm{public}}}{(C_{\mathrm{public}} \cdot T_{\mathrm{public}}) + (C_{\mathrm{home}} \cdot T_{\mathrm{home}}) + (C_{\mathrm{out}} \cdot T_{\mathrm{out}})}$$

$$\mathrm{Exp}_{\mathrm{public}} = \frac{\sim 25\% \cdot \sim 20\%}{(\sim 25\% \cdot \sim 20\%) + (\sim 25\% \cdot \sim 70\%) + (1 \cdot \sim 10\%)} \approx\sim 15\%$$

Rebasing the hazard ratio to indoor ozone concentrations rather than outdoors:

$$\mathrm{HR}_{\mathrm{public}} = \frac{\sim 15\% \cdot 2\%}{\sim 0.25} =\sim 1.2\% \text{ per 10 ppb}$$

To put this hazard ratio into context, it would represent ~4,000 excess deaths per annum in the United States for every 1 ppb increase in ozone across *all* nonresidential indoor public spaces, assuming ~3.27 million baseline deaths per annum.

It is important to put this into context. This is an estimate for if far-UVC was deployed in every square foot of indoor public space in the United States without any attempt to mitigate ozone production and/or ensure adequate ventilation. CDC estimates up to 50,000 deaths per annum from flu, and it is likely that endemic COVID-19 is adding another 50,000 on top of this[31]. This is before we estimate the value of preventing the transmission of the next pandemic pathogen.

Furthermore, if we target far-UVC deployment in spaces with the highest infection risk, then we can potentially achieve a proportionally greater effect on reducing infectious disease while limiting exposure to ozone. Lamps may not need to be switched on at full power at all times, further limiting ozone exposure.

These simple but flawed estimates of potential mortality risk from small changes in chronic ozone exposure suggest that we should take this concern seriously when deploying far-UVC. It should be factored into cost-benefit analyses, with appropriate uncertainty and caveats. But if far-UVC proves to be highly effective at suppressing the transmission of respiratory pathogens, such that we could meaningfully reduce the burden of pandemic and endemic respiratory disease, these estimates suggest that there could be a substantial net benefit in terms of lives saved.

Asthma emergency room visits or hospital admissions

Zheng et al.[5] estimate a 1.6% increase in emergency room visits or hospitalizations for asthma per 10 ppb of outdoor ozone. If we apply the same linear exposure logic to increases in indoor ozone:

$$\mathrm{HR}_{\mathrm{in}} = \frac{\sim 69\% \cdot 1.6\%}{\sim 0.25} \approx\sim 4.4\% \text{ increase per 10 ppb}$$

Or for nonresidential spaces only:

$$\mathrm{HR}_{\mathrm{public}} = \frac{\sim 15\% \cdot 1.6\%}{\sim 0.25} \approx\sim 1.0\% \text{ increase per 10 ppb}$$

In terms of scale, according to Nurmagambetov et al.[32], the average annual cost of hospitalizations and emergency room visits per person with treated asthma in the United States was $629, with inpatient admissions constituting the majority of the cost. The study estimates that there are 15.4 million patients with treated asthma in the US, and multiplying these together produces an estimate of $9.7 billion in healthcare costs for treated asthma. Therefore, we would estimate every 1 ppb ozone increase in *all* indoor public spaces, on this framework, to translate to approximately ~$97 million in asthma-related healthcare costs per annum.

What if the harm is due to ozone loss?

Another possibility is that the harms detected in epidemiological studies are not due to ozone exposure but to exposure to the products of ozone chemistry indoors. There is some suggestive evidence that this may be the case. Reanalysis of data from two studies (one of healthy adults, one of children with asthma) of biomarkers of cardiorespiratory pathology found more evidence of these biomarkers being associated with ozone loss rather than ozone exposure[33]. If this were generally the case, how would we estimate the hazard ratio?

All exposure to ozone loss is indoors, and taking a typical indoor/outdoor ozone ratio of 25%, or a typical ozone loss ratio of 75%, we can rebase the outdoor ozone hazard ratios on the level of ozone loss:

$$\mathrm{HR}_{\mathrm{loss}} = \frac{\mathrm{HR}_{\mathrm{out}}}{[\mathrm{O}_3]_{\mathrm{loss}}/[\mathrm{O}_3]_{\mathrm{out}}} = \frac{2\%}{\sim 75\%} \approx\sim 2.67\% \text{ per 10 ppb}$$

In scenarios with 1 ACH and k_{sum} (decay rate) of 2, this produces estimates for the total mortality increase of increasing indoor ozone by 1 ppb similar to those produced above using the linear ozone exposure method. However, recalling the analysis in the *Ozone and indoor air quality* section, ozone loss is far more sensitive to ventilation than indoor ozone concentrations are. If it is the case that the hazard ratio from outdoor ozone epidemiology is driven substantially by exposure to the products of ozone loss, this greatly magnifies the importance of ensuring adequate ventilation. But it also means that with adequate ventilation, the harm from far-UVC could be lower than a linear ozone exposure model would predict.

Key takeaways

- The potential harms from far-UVC-generated ozone are large enough that they should be considered in cost-benefit calculations, but small enough that they may be significantly outweighed by benefits if far-UVC is highly effective at preventing the spread of respiratory disease.
- Different causes of the harm imply different scales of harm in different circumstances, as well as different harm mitigation strategies.
- Estimates of potential harms are necessary for cost-benefit analysis, but there is no single correct way to make an estimate using the epidemiological data.

Further reading

- Long-Term Ozone Exposure and Mortality in a Large Prospective Study

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8. Emitters and luminaires

Summary

Nature makes it easier to produce certain UVC wavelengths than others. The development of 222-nm krypton chloride excimer (KrCl*) emitters enabled a flurry of far-UVC research, and they are used nearly exclusively in commercial applications today.

KrCl*-based luminaires are currently expensive. To achieve average fluence rates likely to be highly effective for air disinfection (see *Efficacy* section), upfront costs today are on the order of thousands of dollars for every thousand square feet of indoor space protected. This is prohibitive for many applications.

However, there is scope to significantly reduce the effective annual cost per milliwatt of far-UVC output from KrCl* sources, without any fundamental technological breakthroughs. Basic improvements in product engineering could make KrCl* sources far more cost-effective. Key developments would include a high-transmittance, long-lasting optical diffuser, dynamic regulation of optical output, and extension of lifetime. However, these improvements will only come about with additional private investments, which will only be made if there is promise of a substantial market.

Semiconductor-based emitters like light-emitting diodes (LED) and second harmonic generation (SHG) emitters offer potential advantages over KrCl* sources. These technologies can target different far-UVC wavelengths and be produced at chip-scale, enabling a wider variety of luminaire form factors. SHG emitters in particular have the potential to produce very clean emission spectra, which would not require filtering. However, they are early in development, and the economics of semiconductor manufacturing are especially sensitive to scale.

Crucial considerations

- Krypton chloride excimer (KrCl*) sources are currently the leading choice for far-UVC whole-room disinfection, and will likely be for the next 5–10 years. KrCl* emitters primarily produce far-UVC at a wavelength of 222 nm, but have some emissions outside the far-UVC range. High-quality filters minimize these emissions while maintaining high output at 222 nm.
- KrCl* luminaires could be significantly more cost-effective without fundamental technological breakthroughs. However, the required investment in product engineering needs to be financially justified by the prospect of a substantial market.
- Increasing the output of diffused luminaires is an important avenue of cost reduction for the safe use of far-UVC in spaces with typical (8–9 ft) ceilings.
- Semiconductor-based technologies have some important benefits and are promising, but have technological challenges they need to overcome before they are able to replace KrCl* sources for whole-room disinfection.
- The improvements necessary for widespread deployment will likely occur if there is a market to sustain the industry and attract investment.

Note on terminology

Elsewhere in the Blueprint, we have referred to far-UVC 'lamps' in the colloquial sense, as in a floor lamp or a ceiling lamp. However, in illumination engineering, *lamp* exclusively refers to the light-producing component (i.e. the 'bulb') of the final assembled *luminaire* or *lighting fixture*. In this section of the text, *emitter* refers to the underlying radiative element (e.g. a krypton chloride excimer bulb, an LED chip), and *luminaire* refers to the final assembled lighting product, which includes optics, housing, power supply, etc. To improve readability, where a generic term is preferable to specifying *emitter* or *luminaire*, we use *source*.

Analysis

What are the important factors for a good far-UVC source? Why are they important?

Upfront cost

Cost is one of the main factors that affects how widely far-UVC sources will be adopted. High costs limit deployment in public spaces where far-UVC could offer significant public health benefits. Driving down the cost of production through economies of scale, investments in product engineering, and new innovations will be crucial for cost-effective applications.

Power output

The key metric for a far-UVC source is its total far-UVC output, typically in milliwatts (mW).

Lifetime & reliability

Though an appropriately powerful far-UVC source is desirable, it is not beneficial if that source is short-lived and requires frequent replacement. Ideally, a far-UVC source should not need replacing for many years.

Spectral purity

Spectral purity refers to the ability of a far-UVC source to emit at the target wavelength with minimal unwanted emissions at other wavelengths. High spectral purity is critical for safety, as even small emissions in the 240–300 nm range can penetrate deeper into human tissues with potentially harmful effects (see *Skin and eye safety* section). Conversely, more ozone is produced as wavelengths decrease, particularly below 200 nm (see *Ozone and indoor air quality* section). High-quality filters are often needed to achieve spectral purity by blocking non-target wavelengths.

Radiant intensity distribution

Alongside its spectral content, a far-UVC source's maximum safe and effective power will depend on its radiant intensity distribution. Engineering a more uniform fluence rate helps prevent hotspots, which can either exceed safe exposure limits or force intensity reductions that leave surrounding areas underdosed. This can be done by effectively diffusing a high-power far-UVC source, or distributing a greater number of low-power far-UVC sources throughout a space. More uniform distribution may also increase far-UVC's efficacy against pathogens, especially in spaces with low air mixing or high degrees of shadowing, but this requires further study (see *Efficacy* section).

Operational flexibility

Operational flexibility refers to the ability to adjust the far-UVC source based on changing needs or environmental conditions. For example, some spaces might require higher UV doses during peak infection seasons, or lower intensities when occupants are closer to the source. This requires that sources be readily dimmable without loss of other critical functions, such as reduction of effective lifetime.

Form factor

The physical size, shape, and flexibility of a far-UVC source (its form factor) significantly influence where and how it can be deployed. Another element of form factor is the inherent directionality of semiconductor-based emitters, which in principle allows for their radiation output to be used more efficiently.

Wavelength selectability

The most widely used far-UVC wavelength is 222 nm, because that is the peak emission of KrCl* emitters. However, there is no specific reason to think that this is the ideal far-UVC wavelength when considering tradeoffs between efficacy, photobiological safety, and ozone generation.

KrCl* excimer lamps

Krypton chloride excimer (KrCl*) sources, which primarily emit far-UVC at a wavelength of 222 nm, are the most commonly employed sources in commercial applications today. The majority of far-UVC efficacy and safety research published in recent years also uses KrCl* sources.

Excimer (short for **exci**ted d**imer**) emitters are a type of gas-discharge lamp. A glass tube is filled with a gas, or mixture of gasses (in this case, krypton and chlorine); a current is passed through the gas, exciting it; finally, excited particles return to the ground state, emitting a photon of the wavelength proportional to the energy[1].

FIGURE 8.1. Examples of non-excimer gas-discharge lamps: left, a compact fluorescent lamp (image: Creative Commons CFL bulbs by Ervins Strauhmanis, licensed under CC BY 2.0); right, a sodium vapor streetlight (image: Creative Commons Old sodium vapor lamp by Robert Ashworth, licensed under CC BY 2.0).

Excimers require specific gas compositions (either a noble gas, or a mixture of a noble gas and a halogen) at fairly low pressures[2], and are driven by a pulsed high-voltage waveform. While there are other ways to achieve excimer luminescence, the most common and successful type of excimer emitter involves the **dielectric barrier discharge (DBD)**[3], where the dielectric barrier is most commonly the glass envelope of the bulb.

This approach protects the electrodes from corrosive chlorine, and enables long lifetimes[4].

Finally, KrCl* sources (like all far-UVC sources) require high-quality quartz glass to maximize the transmittance of high-energy far-UVC photons.

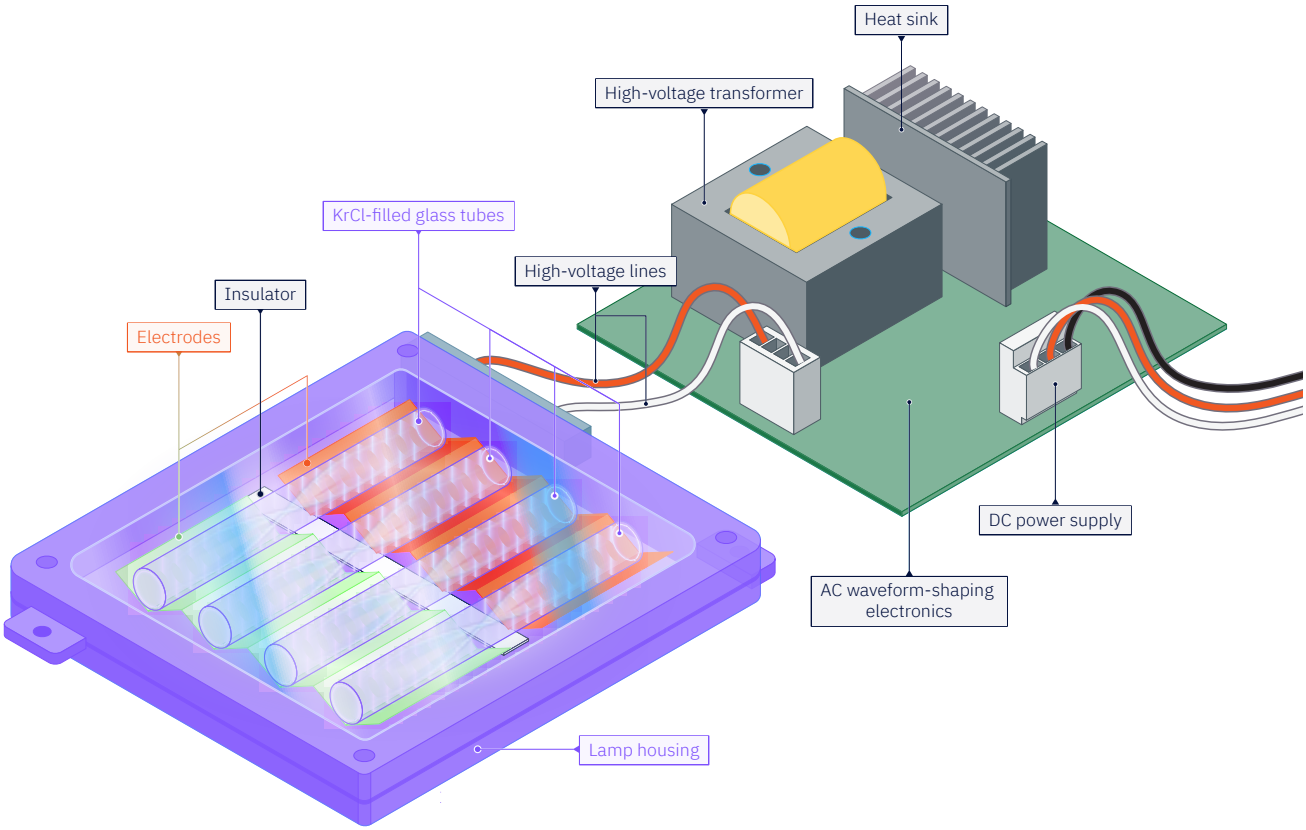

FIGURE 8.2. Diagram of a KrCl* emitter and ballast. Figure by J. Vivian Belenky.

The excimer reaction and KrCl* spectra

Excimer source emission spectra are determined by the reactions taking place inside the plasma. The primary excimer reaction that proceeds under low pressure in KrCl* emitters is the 'harpoon' reaction: $Kr^* + Cl_2 \rightarrow \mathbf{KrCl^*} + Cl$. The *KrCl** dimer rapidly returns to ground state and emits a 222-nm photon, with alternative decay transitions forming a small peak at 200 nm and a broader spectrum out to 240 nm[5].

However, other reactions compete with the primary excimer reaction. At higher chlorine pressures, chlorine may destroy KrCl* dimers before they can decay; it also forms Cl_2^* excimers, which decay to emit 259-nm photons. At higher pressures, $KrCl_2^*$ excimers at 325 nm may also form. Finally, the visible purple glow of a KrCl* source is accounted for by atomic krypton discharges. The contributions of each of these factors to the ultimate spectrum emitted by a KrCl* source is seen below:

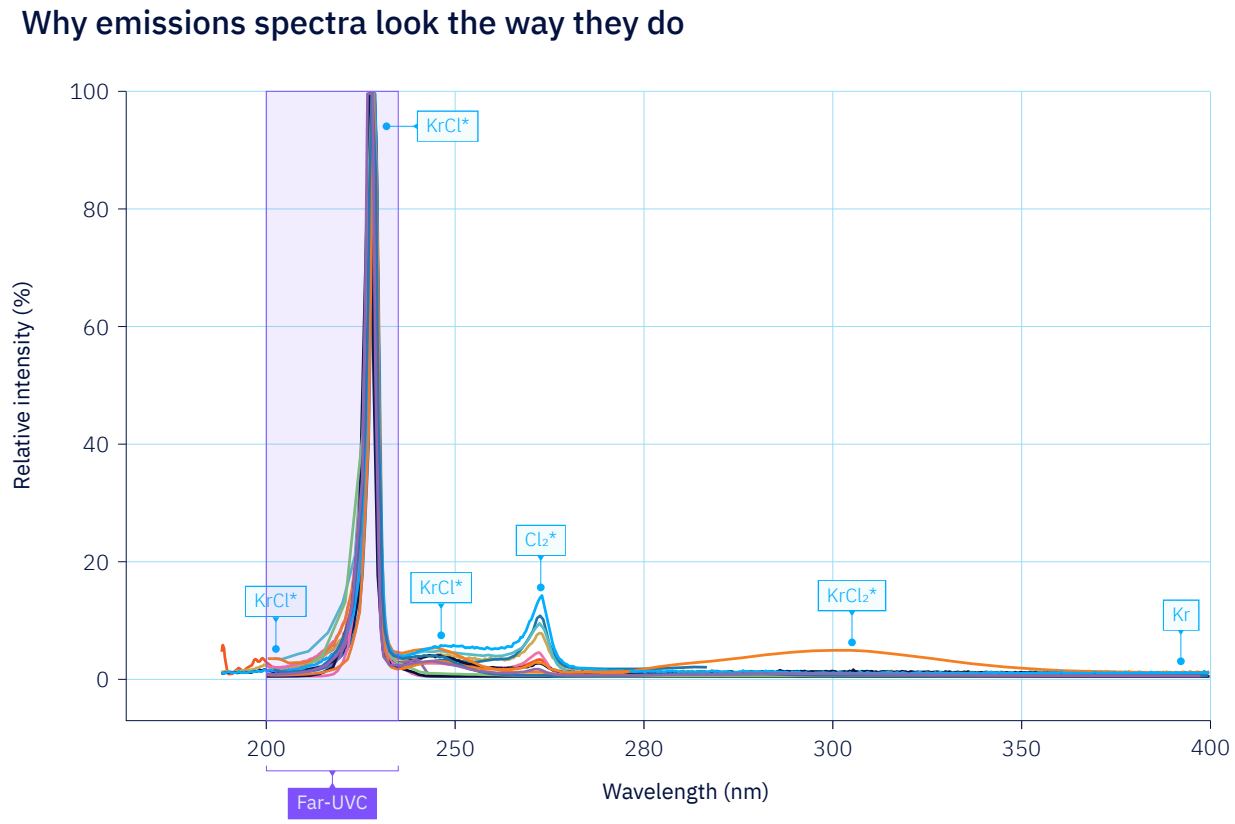

FIGURE 8.3. The shape of the KrCl* emission spectrum is driven by specific reactions[6–8]. Figure by J. Vivian Belenky.

Far-UVC optics and luminaire components

Besides the far-UVC source itself, important components of a far-UVC luminaire are filters, diffusers, and reflectors.

Filters are part of almost all commercially available KrCl* luminaires, and all are interference (dichroic) filters. Interference filters are manufactured by depositing thin alternating layers of HfO_2 and SiO_2 onto the quartz via a process called sputtering. The more layers, the more selective the filter–but also the more expensive, since sputtering is a time-consuming process requiring highly specialized machinery. Because of this, the cost and performance of commercially available filtered KrCl* luminaires vary substantially.

In addition to cost and selectivity, interference filters also exhibit angle-dependent performance, meaning that their effectiveness at blocking non-222-nm emissions can vary depending on the incident angle. At off-axis angles, the filter's cutoff wavelength may shift, potentially allowing more out-of-band emissions to pass through.

The primary difference between filters is the amount of out-of-band emissions they allow through, but also their total output at 222 nm. Although a filter may be designed to only subtract >240-nm wavelengths, in practice, filtered sources' output at 222 nm is meaningfully reduced as well.

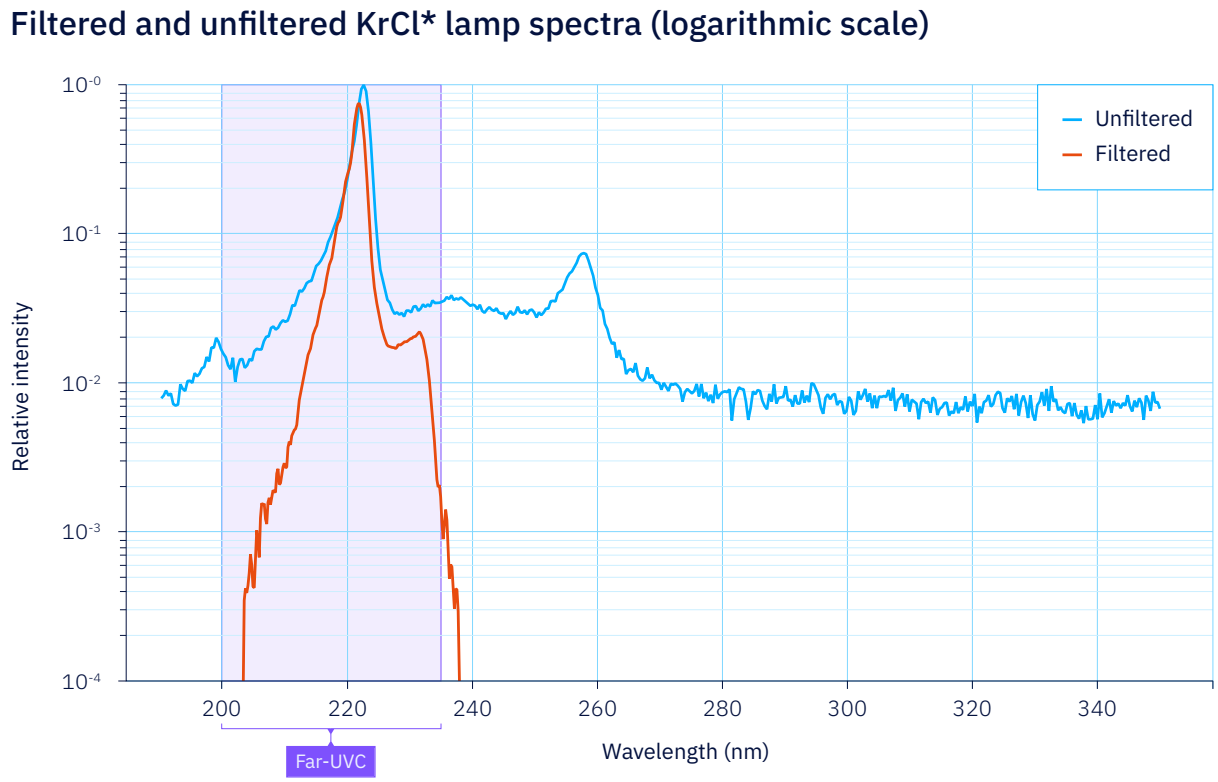

FIGURE 8.4. Comparison of a filtered and unfiltered KrCl* spectrum, logarithmic scale. Data from OSLUV Assays[9].

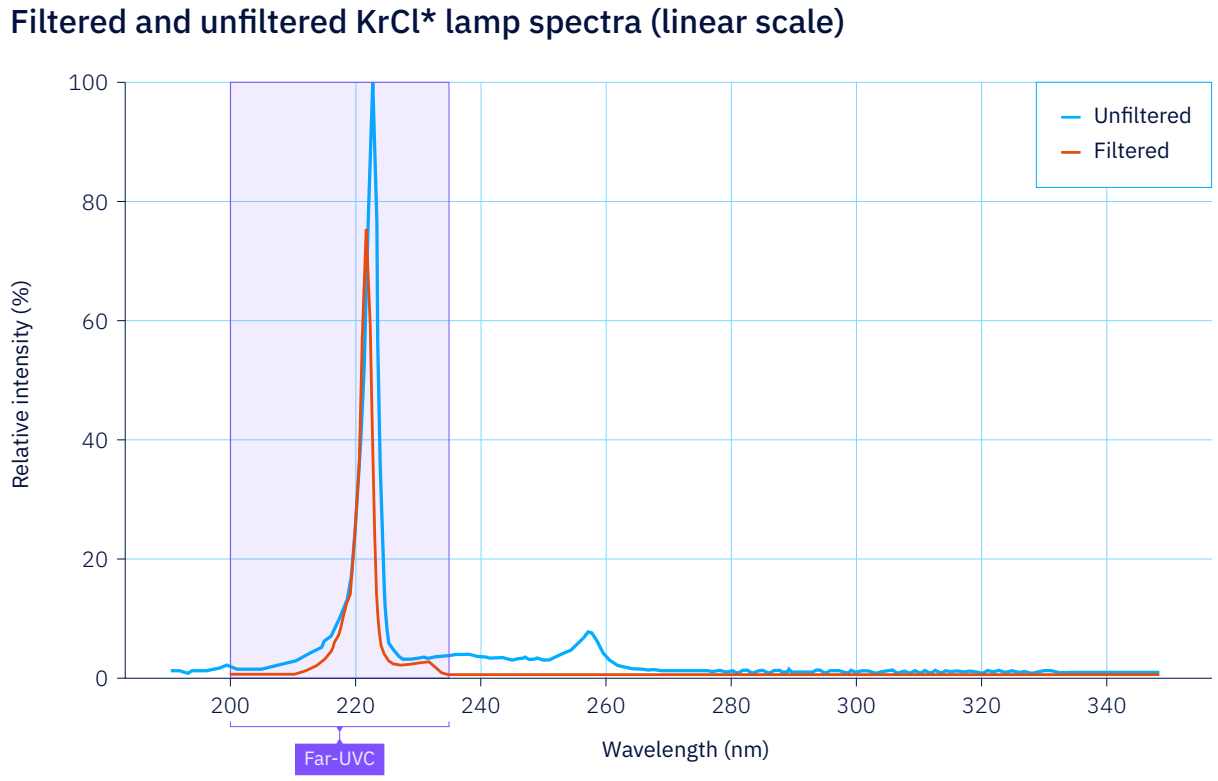

FIGURE 8.5. Comparison of a filtered and unfiltered KrCl* spectrum, linear scale. Data from OSLUV Assays[9].

Diffusers change the angular distribution of radiation to be more dispersed. This may be desirable in some circumstances (such as spaces with low ceilings) because a well-diffused source of the same total output will much more easily comply with UV exposure limits.

In many applications, a more uniform dose distribution can be beneficial for both safety and efficacy. If the dose is uneven, this can create 'hotspots'—areas where the UV intensity is higher than the surrounding regions. This requires engineers to design a system with a lower average fluence rate (and thus lower efficacy) to ensure compliance with safety thresholds across the entire space. Conversely, 'dead zones'—areas where UV intensity is lower than needed for effective pathogen inactivation–should be minimized to ensure adequate disinfection. Well-designed diffusion and fixture placement help balance UV intensity across a space, improving overall performance while maintaining safety limits.

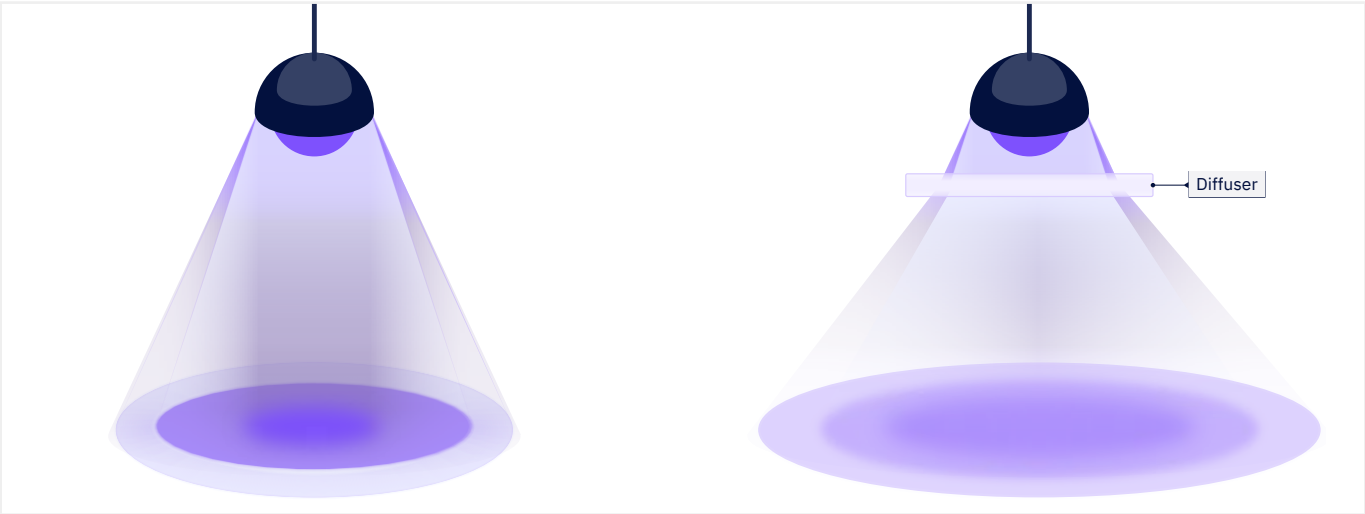

FIGURE 8.6. A diagram showing the effect of a diffuser. In practice, diffusers are integrated into the luminaire.

Unfortunately, the PTFE (Teflon) diffusers commonly used also significantly absorb far-UVC, reducing the total output of commercially available diffused luminaires by 60–75%.

Despite this downside, diffused luminaires are generally more suitable for ceiling-mounted installations in lower-ceilinged spaces. For example, using the UL 8802 photobiological safety standard based on ACGIH Skin TLVs (see *Guidance, standards, and regulations* section), one commercially available undiffused high-powered luminaire[10] can only be safely mounted flush in a ceiling of at least 2.7 meters (9 feet), whereas the same source with a diffuser could be mounted in a ceiling as low as 2.3 meters (7.5 feet).

Finally, **reflectors** line the inside of the fixture and redirect radiation to increase effective output[11]. PTFE/Teflon is often used for this, along with polished aluminum.

Determinants of KrCl* performance

In general, the goal for a high-performance KrCl* source is to output as many 222-nm photons and as few other photons for as long as possible. These goals are sometimes in conflict with one another.

For example, the primary determinant of KrCl* lifetime is the amount of chlorine remaining in the glass. The chlorine depletes over time as it reacts with defects in the glass envelope. Higher chlorine pressure increases the emitter's lifetime, but reduces the efficiency of the KrCl excimer reaction[12], as well as increasing 259-nm emissions, requiring more filtering.

On the other hand, higher-quality filters remove more >240 nm output, but also reduce total output. They also take longer to make, use more materials, and are more expensive.

As another example, optimizing the driving voltage of the luminaire electronics results in both improved radiative efficiency and extended lifetime[13]. However, this requires careful radio-frequency (RF) engineering, and due to the required high voltages, it may result in a luminaire that gives off substantial electromagnetic interference that can put products out of compliance with regulated product standards (see *Guidance, standards, and regulations* section).

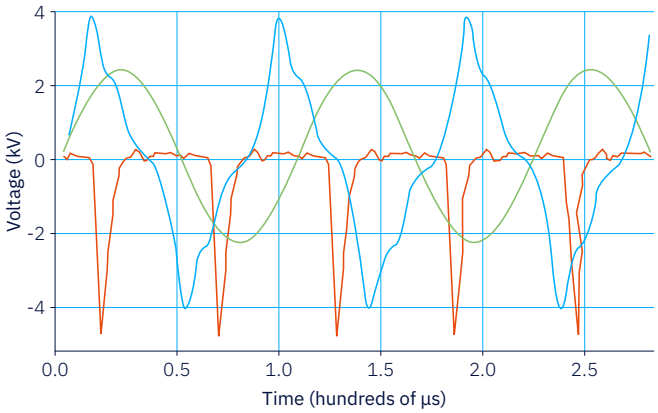

FIGURE 8.7. Several different possible excimer driving waveforms. Sinusoids are easiest to generate, but pulsed operation results in best performance. Figure by J. Vivian Belenky.

In other cases, these goals are not in tension. Higher-quality glass has fewer defects, transmits more 222 nm photons, and reacts less with chlorine, resulting in improved output and performance—though it is more expensive. Similarly, lifetime can be extended by reducing the reactivity of chlorine by operating the system at a lower temperature, or by passivating (coating a material so that it is less reactive) the interior of the glass tube[14].

Though intrinsic efficiencies for KrCl* emitters are predicted to be as high as 15–20%[3], and observed in the lab as high as 12.5%[15], the best commercially available emitters achieve a radiative efficiency of around 4%. This is primarily due to the lifetime-output tradeoff, and commercial sources at 15–20% efficiencies are relatively unlikely. Losses in the driving electronics and absorption in the emissive glass and filter bring commercial sources' actual efficiency down to around ~1%, and with diffusers to ~0.25%. As a result, a state-of-the-art 11-Watt luminaire produces ~110 mW of far-UVC, or ~40 mW if diffused[16,17].

What should researchers know about KrCl* sources?

KrCl* sources have variable outputs depending on how long they have been switched on and how old they are.

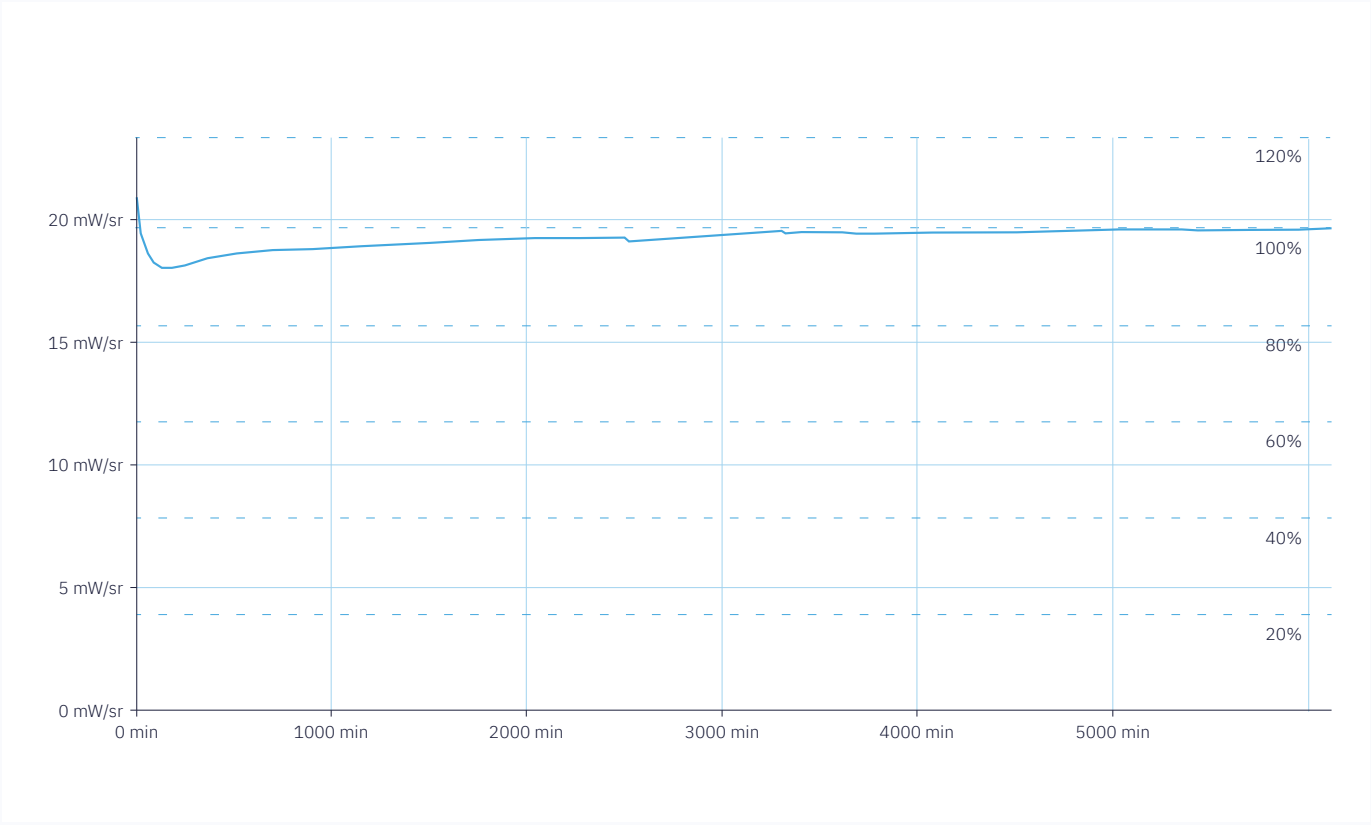

FIGURE 8.8. A fresh KrCl* source will have significant varying power output when first switched on before slowly approaching an equilibrium. Source: the OSLUV project[7].

In order to control this behavior, fresh sources used for research should be 'burned in', meaning left on, for at least 100 hours. Even after this burn-in, there will still be a characteristic 'warm up' behavior, where sources take some time to reach approximate thermal equilibrium after they are switched on. However, this is not as extreme after burn-in, and their output reaches a value close to equilibrium sooner[7].

Manufacturers do not generally report the variability in output across units of the same model, but data collected by the OSLUV Project suggests that this variability can be at least 10–20%[7].

So, when performing research that depends on accurate measurement of far-UVC irradiance at a point:

- Always measure the source output at the point of interest at the time the experiment will take place.
- Burn in a fresh source for at least 100 hours before characterizing.
- Once a source is burned in, it should still be warmed up for 1–2 hours before performing experiments with it.

How much do KrCl* lamps cost?

Currently, a filtered KrCl* luminaire can be purchased for anywhere between $450 and $2,500[18–21]. However, to estimate true lifetime cost, there are many other considerations than the sticker price.

Estimating lifetime cost of safe and effective installations

Because pathogen inactivation is dependent on the dose of far-UVC rather than the number of fixtures, we believe that the most useful metric is the annualized cost per mW of far-UVC output.

The total lifetime cost of a far-UVC device can be broken down into:

Equation 8.1a

$$\text{Luminaire cost per annum} = \text{amortised upfront cost} + \text{annual operating cost}$$

Equation 8.1b

$$\text{Amortised upfront cost} = \frac{\text{Device cost} + \text{Installation cost}}{\text{Luminaire lifetime}}$$

Equation 8.1c

$$\text{Annual operating cost} = (\text{Cost of electricity} \cdot \text{Luminaire energy consumption} \cdot \text{Luminaire usage per annum})$$

Or to obtain an annualized cost per mW we divide luminaire cost per annum by UV output:

Equation 8.2

$$\text{Cost per mW per year} = \frac{\text{Luminaire cost per annum}}{\text{UV power output}}$$

With such an estimate, we can further estimate roughly how much it would cost per annum to produce a given room average fluence rate at different costs per mW. For a ceiling-mounted fixture, this is fairly straightforward to estimate. To a first approximation, the average photon pathlength of a far-UVC fixture mounted on the ceiling, facing down, is the ceiling height. Recalling equation 3.12 from the *Efficacy* section:

$$I = \frac{P \cdot L}{V}$$

with P = UV power output (W)
L = average photon pathlength (m)
V = volume (m^3)

If we define L as approximated by ceiling height H, and volume as:

$$V = H \cdot A$$

with H = ceiling height (m)
A = floor area (m^2)

Substituting H for L and V for H×A in equation 3.12, the H terms cancel out and fluence rate I is equal to:

$$I = \frac{P}{A}$$

Therefore, for every m^2 of floorspace, approximately 10 mW of ceiling-mounted far-UVC output would be required to create 1 µw/cm^2 of fluence rate. A US classroom is typically around 900 ft^2 or 84 m^2, so to produce 1 µW/cm^2 of far-UVC fluence rate in such a space would likely require approximately ~840 mW of ceiling-mounted far-UVC output. This simplified estimate of far-UVC output requirements is 5–10% higher than simulations using the Illuminate software[22]. In the analysis below, we use the more conservative estimate.

Simulations of an 84 m^2 classroom and a ceiling height of 2.75 m using Illuminate suggest that corner mounting rather than ceiling mounting would reduce the installed power required to produce the same room average fluence rate by as much as 30–40%. However, this does increase the risk of eye overexposure relative to a ceiling-mounted fixture. To be conservative both for the purpose of reducing eye exposure but also for calculating cost, we assume ceiling mounting.

Installation cost

For a device to be certified to the voluntary standard ANSI/CAN/UL 8802, it must be permanently mounted (see *Guidance, standards, and regulations* section). We conservatively assume for the purpose of this analysis that installation by a qualified electrician will cost $200 per luminaire.

Lifetime and energy costs

Though energy-inefficient on an input/output basis, a typical far-UVC luminaire consumes only 11 W. At the US average energy cost of 18 cents per kilowatt-hour, it would cost $4.12 to run a single luminaire for a year, assuming usage for eight hours a day, five days a week. As we shall see below, this makes energy a relatively minor component of the overall cost.

KrCl* manufacturers typically claim that their luminaires have a lifetime of up to 10,000 hours, which would mean that replacement would only be necessary every 5 years assuming this same usage (or approximately once every 1 year if run 24 hours a day / 365 days a year). Unfortunately, most far-UVC manufacturers do not publish data necessary to validate these claims, but it does allow for at least an approximate comparison of the range of annualized costs of a mW of far-UVC. Validation of lifetime claims through independent testing is warranted.

Annualized lifetime cost estimate

In the below table, we present four example filtered luminaires for which we have third-party characterization data collected on a comparable basis. We calculate the indicative cost per annum to produce 1 µW/cm² of far-UVC fluence rate in a typical US classroom, assuming 5 year luminaire lifetime, and luminaire operation of 8 hours a day, 5 days a week. Luminaire power outputs are based on testing by the OSLUV Project[9]. These luminaires do not necessarily represent the most cost-effective filtered sources currently available, but rather highlight the significant differences across commercially available products, even if we assume equal lifetime.

TABLE 8.1. Annualized cost estimates for example fixture installations.

Fixture type	Device cost	Install cost	Total mW	Lifetime (years)	$/mW/year	Energy cost/mW/year	Total cost/year per µW/cm² in a typical classroom
Undiffused	$1,700	$200	107.5	5	$3.53	$0.04	$3,003
Diffused	$800	$200	22.8	5	$8.77	$0.18	$7,436
Diffused	$1,600	$200	51.2	5	$7.03	$0.08	$5,973
Diffused	$2,500	$200	32.2	5	$16.77	$0.13	$14,196

Note: all total outputs listed are shown after a few hundred hours of 'burn-in', when KrCl output stabilizes[23]. The table assumes that 840 mW of output is needed to produce 1 µW/cm² of far-UVC fluence rate.*

A naive reading of this table would suggest that undiffused sources are more cost-effective. However, diffusion is often necessary in order to comply with consensus standards in lower ceiling height spaces (see *Far-UVC optics and luminaire components* above).

The output lost with the use of a diffuser raises the question of whether it would be more cost-effective to comply with consensus standards by dimming an undiffused fixture. Dimming is not a feature of luminaires that are currently sold, but it is in principle doable. Based on simulations conducted in Illuminate for an undiffused device[10], the output would have to be dimmed by approximately 65% in order to be mountable face down at a ceiling height of 2.3 m and comply with UL 8802 using the ACGIH Skin exposure limit. By contrast, a diffused fixture can typically be mounted at this height and comply with the standard with significant headroom to increase power output while remaining in compliance.

Furthermore, diffusers can reduce 'hotspots' and improve the distribution of dose in a space, which we believe is beneficial to safety, and likely beneficial to efficacy. We therefore think that the priority for reduction in effective cost per mW for ceiling-mounted installations in typical 8–9 ft drop-ceiling spaces should be improvement in the diffuser to increase total power output.

As can be seen from the table 8.1, at current fixture cost, producing 1 µW/cm² of fluence rate in a typical-sized classroom is expensive. Even if the required fluence rate for effective disinfection was in the range of ~0.2 µW/cm²—a reasonable best-case scenario given the available data (see *Efficacy* section)—costs for ceiling-mounted installations would still be in the range of $600–$2,800 per year for a typical classroom-sized space. This is likely to be cost-prohibitive for applications outside of high-risk spaces such as healthcare settings. We must therefore ask what are the reasonable prospects for future cost reduction.

Prospects for KrCl* cost reduction

One manufacturer projected in 2024 that they would achieve a device cost of $1/mW by 2028, an order of magnitude improvement[24]. There are a number of avenues through which this degree of cost reduction can potentially be achieved in the next 3 years.

The far-UVC market is currently subscale

Components that presently cost $200-300 can, we believe, readily cost $50/unit. But the emitter market is currently very small, and gross margins for both component and fixture manufacturers are likely higher than we would expect in a thriving competitive market.

Alternative applications

Some of this cost-reduction via economies of scale may be achieved by finding additional market niches for far-UVC technology besides whole-room disinfection. Some promising work has been done on allergen reduction[25], water disinfection[26–28], and mold control[29–32], but we do not evaluate these applications in this Blueprint.

Technology improvement

There is room for cost reduction through improvements to product design. Some of this is what might be termed 'catch-up growth'. There are some techniques for improving KrCl* radiative output and lifetime that were freely published as far back as the 1990s, but few manufacturers use these. A larger market would incentivize the adoption of these methods in more commercial products.

For example, the main consumable in KrCl* emitters is chlorine gas. Manufacturers could improve their lifespan by optimizing gas mixtures or using replaceable chlorine gas vials, which may lower costs.

Even if it proves not to be cost-effective to replace chlorine gas, for most commercially available fixtures, the emitter as a whole is not replaceable either. This means that the entire luminaire must be replaced at the end of lifetime, and reduces the overall cost-effectiveness. Emitters that are

easily replaceable by users could significantly reduce effective lifetime cost per mW.

Luminaire costs could also be reduced by developing technologies that lower component costs. For example, the dichroic filter is the single most expensive component of a filtered KrCl* source. Absorption filters, which might be substantially cheaper if not quite as performant as dichroic filters, are under investigation[8], but none are yet commercially exploited.

While manufacturers typically claim up to 10,000 hours of lifetime, based on discussions with industry experts we believe up to 30,000 could be achievable.

Table 8.2. Possible future cost estimates for KrCl* luminaire installations.

Fixture type	Device cost	Install cost	Total mW	Lifetime (years)	$/mW/year	Energy cost/mW/year	Total cost/year per µW/cm² in a typical classroom (840 mW)
Possible future	$110	$200	110	15	$0.19	$0.04	$191

Sensors

Accurate, low-cost sensors for monitoring far-UVC intensity could improve safety and efficiency. Current sensors are expensive, but advances in semiconductor-based detectors may provide cheaper, real-time monitoring solutions, ensuring consistent output and regulatory compliance. While not a primary cost driver, better sensors could enhance system reliability and reduce overexposure risks, and potentially reduce the need for dimming or duty cycling for standards compliance. A dimmed or duty-cycling luminaire has the same input costs but lower output, so if sensors can be used to remove the need for dimming or duty cycling, this will effectively reduce cost per utilized mW, potentially enough to make up for the increased cost of adding the sensor.

There are a number of commercially available luminaires that employ sensor technology such as LIDAR[33] and mmWave[34] . However, none of these specifically employ sensors to facilitate UL 8802 compliance while significantly increasing the output of luminaires that can be safely installed at a given ceiling height.

Possible future costs

Taking all the above together, we can re-evaluate the cost of installing far-UVC in a typical classroom using a possible future scenario of approximately $1/mW, with a 30,000 hour/15 year lifetime.

If fluence rates needed to be effective at preventing airborne transmission are in the order of ~0.2 µW/cm², the cost of installing far-UVC in a classroom could feasibly be less than a few dollars a year per student.

Key takeaways

- Luminaire lifetime is at least as important as power output for determining effective cost.
- Diffused sources are less cost-effective on a per mW basis, but may be the only option at lower ceiling heights.
- Far-UVC luminaires are currently expensive.
- Our best guess is that in the future, KrCl* luminaires could be cheap enough that wide-scale commercial use will be possible, particularly if fluence rates required for effective disinfection are low.

How might far-UVC be generated in the future?

Though KrCl* is the dominant source of far-UVC today, alternative technologies are under development in both academia and commercial startups. Alternative approaches could potentially allow for:

- Different (ideally smaller) form factors, enabling a wider variety of fixture designs
- Lower voltages, requiring less complex electronics to manage electromagnetic interference (EMI)
- Ability to target different wavelengths, i.e. other than 222 nm

Krypton bromine excimer emitter

As well as KrCl*, there are alternative excimer-based emitter designs, and these can generate different far-UVC wavelengths. Krypton bromine excimer (KrBr*) sources, emitting at 207 nm, were the first far-UVC sources investigated for germicidal effectiveness[35]. However, the KrBr* reaction has a lower intrinsic efficiency, and filtering the out-of-band emissions was more difficult.[2,36] This meant that KrBr* sources ultimately proved impractical, and none are commercially available today.

Photoluminescent emitters

Non-far-UVC excimer sources can be used to create far-UVC emissions through photoluminescence (PL). PL is a process whereby luminescent material re-emits radiation after absorbing photons (as opposed to electrons) from an external source. For example, UVC phosphors can be excited using a 172-nm xenon excimer (Xe_2*) source, creating broad-spectrum UVC emissions peaking at 230 nm[37]. Alternatively, magnesium zinc oxide can be excited by a 146-nm krypton excimer (Kr_2*) source, producing UVC emissions peaking at 202 nm[38]. A number of potential approaches are feasible, and it could be possible to filter out unwanted emissions outside the far-UVC range. However, to our knowledge, no PL device has been demonstrated to be suitable for whole-room disinfection of occupied spaces.

Semiconductor-based approaches

There has been substantial interest in developing far-UVC emitters based on semiconductor technology, primarily predicated on the notion that in traditional lighting, visible light LEDs have proved highly scalable and energy-efficient. However, it should be noted that the widespread adoption of visible light LEDs benefited from the sheer size of the existing general lighting market, which supports the fixed costs of semiconductor

manufacturing. Whether the cost efficiency gains possible through semiconductor technology will be realized for far-UVC generation will depend in large part on whether the market for far-UVC expands sufficiently, as well as whether developers can solve challenges unique to producing higher energy far-UVC photons.

Cathodoluminescent emitters

Cathodoluminescence (CL) is a phenomenon where photons are generated by electrons exciting a luminescent material. Many familiar technologies use cathodoluminescence, including cathode ray tube televisions (CRT TVs) and scanning electron microscopes. CL emitters allow the usage of electroluminescent materials that are otherwise unsuitable for LEDs, like magnesium zinc oxide[39,40] (peak emission 229 nm), hexagonal boron nitride (peak emission 215 nm)[41], or a UV-emitting phosphor[42] (peak emission 230 nm). Like KrCl* and LEDs, published spectra of CL sources in the far-UVC contain unwanted wavelengths, which likely cannot be changed with any method beyond optical filtration.

Traditional CL devices that pump electrons through a vacuum (like CRT TVs) are not semiconductor-based. Their fundamental radiative efficiency is limited; they require high voltages and vacuum-sealing; chip-scale miniaturization is possible, but challenging[43].

Purely solid-state emitters that employ CL to produce far-UVC are feasible using impact ionization[44,45] and emitters employing zinc oxide doped with manganese to produce visible light already exist[38]. Despite utilizing a different underlying technology, these emitters would have a number of similarities to LEDs, and require solving similar engineering challenges, such as the need for cooling in order to achieve sufficient output and efficiency. While not purely solid-state, a semiconductor-based CL device primarily emitting at 215 nm has been designed[46] and is commercially available[47].

Expert forecasts[48] predict that CL devices will be the first semiconductor-based emitter to meet current KrCl*-tier performance for whole-room disinfection. CL emitters may also find niches outside of the applications that KrCl* sources are currently serving.

UVC LEDs

The blue (and consequently, white) LED was a transformational invention for the general lighting industry, one that secured a Nobel prize for its inventors[49–51]. As such, there has been much excitement over the prospect of UVC LEDs, which are built on a similar material platform of gallium nitride (GaN). Some early indicators do suggest that conventional UVC LEDs are on a comparable trajectory to blue LEDs[52].

Commercial 270-nm LEDs are closing in on wall-plug efficiencies of 10%, and firms are aiming to reach 20% within the next year. Whether these promises will bear out for common UVC wavelengths 260–280 nm remains to be seen, but regardless, this kind of forecasting has questionable relevance to far-UVC LEDs. UVC LED performance is strongly dependent on wavelength, and plummets exponentially for shorter UVC wavelengths[53,54]. The trend is also true of lifetime[55].

Shorter-wavelength aluminum gallium nitride (AlGaN) LEDs are more challenging because they require a higher relative abundance of aluminum. To achieve far-UVC LEDs, the aluminum fraction would have to be close to 90%, very close to pure aluminum nitride (AlN). Many challenges emerge at such large aluminum fractions—the materials grow with more defects, are more opaque to short wavelengths, are more difficult to dope, and are more difficult to make good electrical contacts with, along with many other issues[53].

Some of these issues are also relevant to 260–280-nm UVC LEDs, and far-UVC LEDs will benefit from advancements in that industry. Others, however, are relatively unique to far-UVC LEDs. For some of these issues, fundamental research is required, such as understanding the role of point defects in radiative efficiency. For others, various device design approaches are being used to address them, and some are seeing early commercial application. Even relatively simple innovations in fixture design can materially improve LED performance, for example through efficient passive cooling[56].

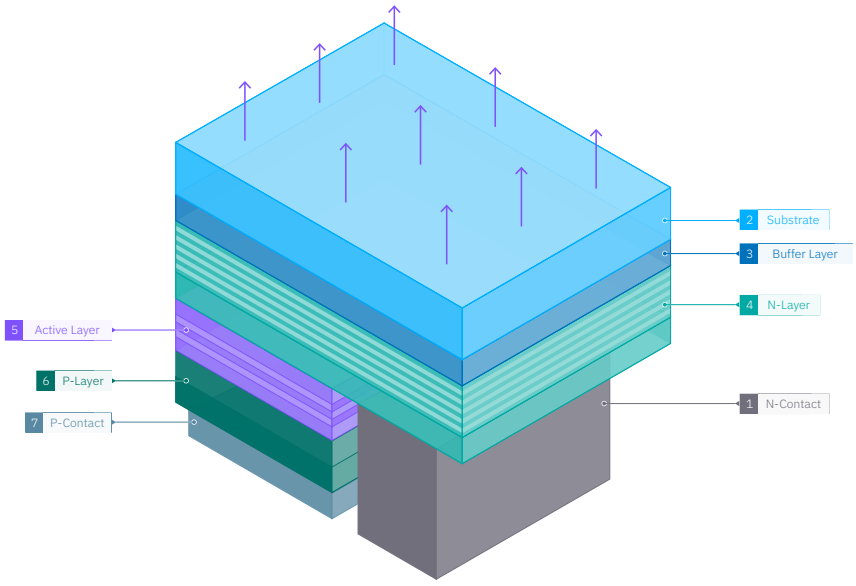

FIGURE 8.9. Excerpted from Kneissl et al., 2011[57]. A diagram of the components of a far-UVC LED. This is only one LED design, and there are multiple viable variations on the arrangement of these components.

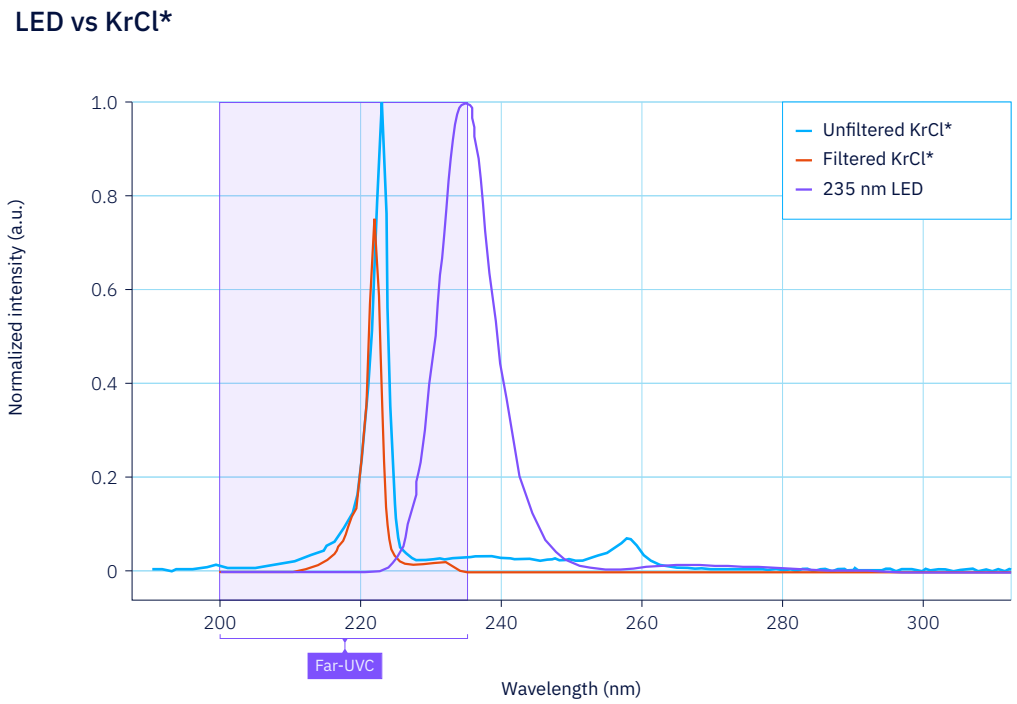

FIGURE 8.10. Representative spectra of LEDs and KrCl* emitters. Source: the OSLUV Project[7] and Silanna UV[58].

An additional consideration is that LEDs are not monochromatic. Current UVC LEDs have fairly wide peaks, and long, lower-energy tails. Though they are likely to improve with time, filtering will likely still be necessary to use them in applications where KrCl* is used currently.

Despite these challenges, expert forecasts anticipate seeing far-UVC LEDs that are competitive with KrCl* sources for whole-room disinfection on the ~10 year timescale[48]. This timeline is likely lower for applications where cost per mW is less of a consideration. Whether this prediction bears out, AlGaN devices for UV radiation are well-established and garner substantial academic interest, substantially more than other approaches.

Second harmonic generation emitters

The final technology of note is second harmonic generation (SHG). SHG is an established approach for laser generation when no suitable diode material exists at the desired wavelength–green laser pointers are the classic example[59].

The basic principle of SHG is simple. Start with a laser at twice the desired wavelength–the 'pump' wavelength. Couple the pump laser to a crystal with the appropriate properties. Output half the number of photons at twice the energy and half the wavelength. For far-UVC, it is straightforward to start with the ubiquitous 450-nm blue laser diode, and frequency double it to achieve monochromatic 225-nm emission.

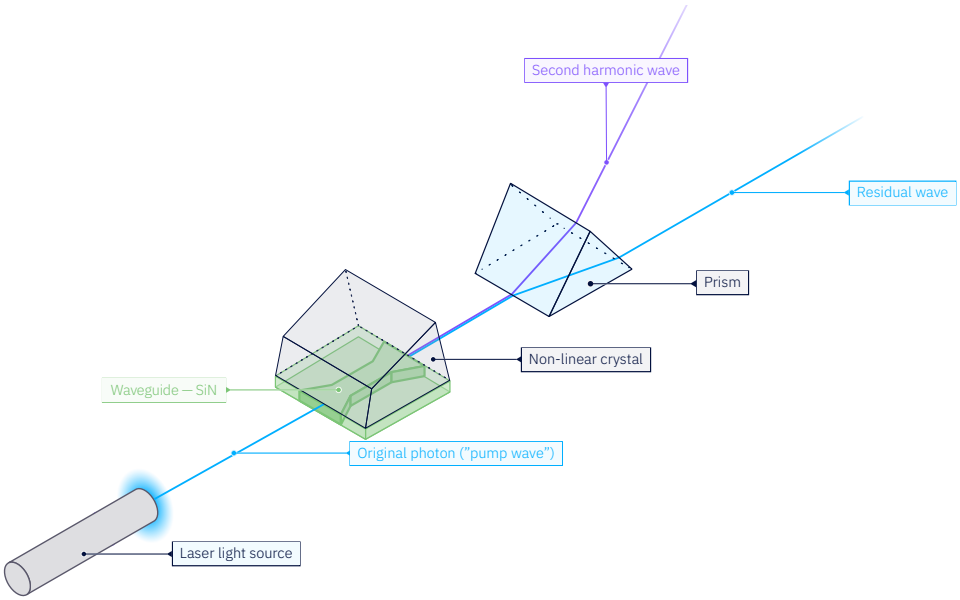

FIGURE 8.11. Diagram of the second-harmonic generation process. Source: Jkwchui from WikiCommons, licensed under CC BY-SA 3.0[60]

Advantages of SHG emitters

This approach has many desirable properties:

- **Spectral purity**: the generated radiation is highly monochromatic, with the peak wavelength an order of magnitude narrower than that achievable by KrCl*. Blue light will likely remain in the spectrum, but this is far less dangerous and easier to remove than UVC or UVB.
- **Wavelength targeting**: by changing the pump wavelength, the output wavelength changes as well.
- **Pump source availability**: blue lasers are a well-developed technology, with wall-plug efficiencies around 40% and reported lifetimes of 20,000–30,000 hours[61].
- **Efficiency**: SHG can theoretically achieve 100% conversion efficiency by integrating a resonant cavity into the device, though in reality this is rarely approached[62,63].
- **Scalable production**: with photonic integration, SHG emitters could be mass-produced at chip scale[64].

Challenges and limitations of SHG devices

As usual, there are challenges and limitations to SHG. Among them is the choice of nonlinear crystal, a material that efficiently halves the wavelength of incoming photons by second harmonic interactions. In addition to being a good nonlinear converter, the material must be transparent at the target wavelength. For far-UVC, there are two primary options: beta barium borate (BBO) and aluminum nitride (AlN).

BBO is generally the material of choice, with conversion efficiencies reported around 15–35%[65–68]. However, complex bulk optics are required to achieve such numbers, and without them efficiencies typically plummet to 0.05%[69], though work with silicon nitride (SiN) waveguides has pushed conversion efficiency as high as 4%. BBO is also sensitive to humidity, potentially making it challenging for applications outside of research[70].

AlN is another option for UV SHG[71–73]. Drawbacks include the relatively lower nonlinear coefficient, and the low transparency in the far-UVC range. However, AlN is easier to grow and more widely used in other semiconductor applications, potentially enabling easier scaling and cost advantages.

More broadly, SHG output will need to be shaped and diffused for general applications, and as a laser source may be subject to different safety standards[74]. As discussed, suitable materials for this application in the far-UVC are limited, and this requirement will likely reduce total power output even without the need for a filter. Cost-effective diffusion of SHG-produced far-UVC has not yet been demonstrated, but in principle should be physically possible using known materials.

These issues combine to form a picture quite far from the theoretical ideal performance, and claims about cost-effectiveness at large-scale production remain hypothetical. Nevertheless, this is a technology to watch.

Further reading

- Solid-State Far-UVC Emission Roadmapping Workshop Report
- Illuminate
- RP Photonics Encyclopedia- Frequency Doubling
- NALMCO Germicidal UV Training and Certification
- The 2020 UV emitter roadmap

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9. Guidance, standards, and regulations

Summary

A wide variety of organizations are relevant to far-UVC governance. These organizations set guidance, standards, and regulations pertaining to UV safety, ozone exposure, air cleaning efficacy and applications, GUV devices, and industries like healthcare. Within the United States, very few regulations directly pertain to far-UVC. In other jurisdictions, such as the European Union, consensus standards on UV safety have been incorporated into regulation of consumer products.

Even without legal force, voluntary standards can shape markets. Consumers who purchase portable air cleaners are informed about their efficacy through clean air delivery rates advertised by manufacturers and produced under test standards such as ANSI/AHAM AC-1 *Method for Measuring Performance of Portable Household Electric Room Air Cleaners*. Installation of Upper Room UV in hospitals is guided by NIOSH 2009-105 *Environmental Control for Tuberculosis: Basic Upper-Room Ultraviolet Germicidal Irradiation Guidelines for Healthcare Settings*. For many buyers, compliance with product standards such as ANSI/CAN/UL 8802: *Ultraviolet (UV) Germicidal Equipment and Systems* can be a *de facto* procurement requirement, even if it is not always *de jure*.

Far-UVC consensus standards need to be iterated to ensure that the guidance and best practices they encode reflect the most recent scientific understanding, and the particular properties of far-UVC lamps. The standards development process is typically slow, as is the cycle to update any regulations based on those standards. In some cases, further research needs to be done in order to rationally update or create a new standard. In other cases, improvements should be made now based on what we already know. Standards are based on a balance of interests, which means evolution is to be expected even in the absence of significant changes in scientific understanding.

We expect that if far-UVC is widely used in public spaces, it will be regulated. Consensus standards and recommendations from expert bodies will form the substantive content of those future regulations, as they do today. If we can create effective standards and guidance in advance of the desire of governments and the public for legal regulations, this will help ensure that far-UVC, and the market for air cleaning technologies as a whole, can grow and thrive.

Crucial considerations

- Guidelines, standards and regulation of far-UVC are a complex patchwork that differ by jurisdiction and in the degree of voluntary versus mandatory compliance.
- Standards often apply UV exposure limits in a more conservative way than the expert guidance documents they reference, by managing peak irradiances rather than time-weighted average exposure of occupants.
- In some circumstances, standards and guidelines are incorporated into legal regulation.
- Some standards, such as methods of testing for far-UVC efficacy, cannot be created until certain open research questions are resolved.
- There is a particular need to develop new standards related to ozone generation.

Definitions

Guidance is simply a recommendation from an expert body. This can come from a wide variety of organizations including public health agencies, professional associations and other expert bodies. Guidance is produced in a wide variety of ways for different reasons. For our purposes, anything that recommends but is not a consensus standard (see below) or does not have the force of law, we term 'guidance'. Guidance can be highly technical, or it can provide practical advice to practitioners and the wider public. For example, guidance includes both the recommendations on UV exposure limits from ICNIRP and ACGIH (see *Far-UVC primer* section) and also the public-facing consumer advice on the use of portable air cleaners provided by the US Environmental Protection Agency (EPA)[1], even though these are very different resources.

A **consensus standard** is a technical document that has gone through a rigorous development process, incorporating the expertise and interests of different parties. These usually take years to finalize. Different standards bodies have different procedures, but they all include provisions for

openness, transparency, balance of interests and clear processes for review, voting and appeal. Standards are voluntary: people may choose to comply, but they do not themselves create any legal requirements.

Organizations that have produced or accredited standards relevant to far-UVC include:

- American National Standards Institute (ANSI)
- ASTM International (formerly American Society for Testing and Materials) (ASTM)
- International Organization for Standardization (ISO)
- International Electrotechnical Commission (IEC)
- UL (formerly Underwriters Laboratory)
- American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE)
- Illuminating Engineering Society (IES)
- Association of Home Appliance Manufacturers (AHAM)
- International Commission on Illumination (CIE)

These organizations are a mixture of standards development and standards accrediting bodies. For example, ANSI primarily accredits standards created by other bodies (such as UL, ASHRAE, AHAM, and IES) while ISO develops a wide range of standards in-house as well as recognizing standards developed by other bodies such as CIE.

Standards that have been accredited by ANSI generally have the prefix 'ANSI' before the name of the body who developed the standard. ANSI standards (among others) are part of a mutual recognition agreement with Canada, and therefore the prefix ANSI/CAN can commonly be found. Standards accredited by European Standards Organizations have the prefix 'EN'. In this document, for the standards that are accredited we generally use the relevant prefixes except where it makes sense to omit for brevity.

A **regulation** is simply anything that has the force of law. As we shall see, it is not uncommon for consensus standards to be incorporated into legislation. The most ubiquitous mechanism by which far-UVC may be regulated in the future is via building codes, which are in most cases standards or model codes voted into law by the 'authority having jurisdiction'. This authority may be (in the United States) the municipal government, the county authority, the state, the federal government in the case of federal buildings, or the relevant branch of the military on military bases. In all cases, the state codes supersede the local codes, but do not affect federal or military authorities. As such, many standards that are facially 'voluntary' are actually quasi-national, with nationwide authorities voting on the adoption of the new standard into building codes within a few years of a new version of that standard being released.

Analysis

Efficacy

Guidance

There is relatively little guidance on minimum recommended efficacy for GUV devices specifically, and none for far-UVC. The US Centers for Disease Control and Prevention (CDC) recommends 5 or more air changes per hour (ACH) (or equivalent) for infection control in the workplace[2], but there is no guidance on how to calculate the equivalent air changes per hour (eACH) from the use of a particular GUV system. For upper-room UV, the CDC points to the 2009 NIOSH guidance for tuberculosis control[3], but this guidance is not relevant to whole-room far-UVC systems.

Currently under development is ASHRAE GPC 37 *Guidelines for the Application of Upper-Air (Upper Room) Ultraviolet Germicidal (UV-C) Devices to Control the Transmission of Airborne Pathogens*[4], which we expect to supplant the 2009 NIOSH tuberculosis guidance for the use of upper-room UV systems. These guidelines cover both safety and efficacy.

For the use of portable air cleaners to achieve 5 eACH, the CDC points at guidance from the EPA[1], which if followed by the user would add approximately 5 eACH under a well-mixed room assumption (see *Efficacy* section for the relationship between eACH and CADR):

Portable air cleaner sizing for particle removal						
Room area (square feet)	100	200	300	400	500	600
Minimum CADR (cfm)	65	130	195	260	325	390

Note this chart is for estimation purposes. The CADRs are calculated based on a 8-foot ceiling. If you have higher ceilings, you may want to select a portable air cleaner with higher CADR.

FIGURE 9.1. Taken from *Guide to Air Cleaners in the Home*, 2nd Edition, EPA Indoor Environments Division 2018[1].

Some test standards that have been developed for measuring the clean air delivery rate (CADR) of air cleaning technologies against bioaerosols are also applicable to GUV devices. In principle a consumer could use the CADRs from these test methods and apply them in the same way. However, as we shall see below, this comes with some additional caveats for GUV technologies.

Standards

Test methods for air cleaners

There are six test standards that provide testing methods for in-room air cleaners against which far-UVC devices could be assessed. In addition, we are also aware of a German draft standard still under review, DIN/TS 67506:2022-02 *Disinfection of room air with UV radiation - UV-C secondary air unit*[5].

The six standards are:

- **ANSI/ASHRAE 185.3** *Method of Testing Commercial and Industrial In-Room Air-Cleaning Devices and Systems for Microorganism Bioaerosol Removal or Inactivation in a Test Chamber* (2024)[6]
- **ANSI/AHAM AC-5** *Method for Assessing the Reduction Rate of Key Bioaerosols by Portable Air Cleaners Using an Aerobiology Test Chamber* (2023)[7]
- **ISO 16000-36** *Standard method for assessing the reduction rate of culturable airborne bacteria by air purifiers using a test chamber* (2018)[8]
- **ASHRAE 241** *Control of Infectious Aerosols; Normative Appendix A* (2023)[9]
- **ASTM E3273-21** *Standard Practice to Assess Microbial Decontamination of Indoor Air using an Aerobiology Chamber*[10]
- **GB/T 18801-2022** *Air cleaner*[11]

In addition to these test methods for in-room air cleaners, there are also test methods that apply to in-duct devices (e.g. ANSI/ASHRAE 185.1) and surfaces (e.g. ANSI/ASHRAE 185.4), which we do not cover in this report.

The first three standards are ANSI- or ISO-accredited. ASHRAE 241 was developed as part of an accelerated process in response to the COVID-19 pandemic, and has not yet been accredited by ANSI. ASHRAE 241 also permits that, where filtration devices have been tested by ANSI/AHAM AC-1 *Method for Measuring Performance of Portable Household Electric Room Air Cleaners*[12] for the removal of dust, pollen and smoke, manufacturers can use these results to infer their expected performance against bioaerosols. ASHRAE 241 also permits the use of other consensus standards to determine effectiveness, including ANSI/AHAM AC-5 and ANSI/ASHRAE 185.3. The latter standard is (at time of writing, April 2025) currently undergoing revision.

These six standards share many features in common, but also some differences summarized in the table below.

TABLE 9.1. Comparison between testing standards for bioaerosol removal or inactivation by in-room air cleaners.

	ANSI/ASHRAE 185.3	ANSI/AHAM AC-5	ISO 16000-36	ASHRAE 241	GB/T 18801-2022	ASTM E3273-21
Chamber volume	>27 m³	30±1.5 m³	>8 m³ , typically 15–30 m³	>22.7 m³	3 m³, 10 m³, 30 m³ or 81 m³	~24 m³
Chamber dimensions	Height 2.4–3.1 m Width 85–100% of length	Height 2.5±0.1 m Width 85–100% of length	n/a	n/a	Specified in QB/T 5364-2019	n/a
Environmental conditions	23±3 °C 50±10% RH	20±3 °C 50±10% RH	23±2 °C 50±5% RH	23±3 °C 50±10% RH	23±3 °C 50±10% RH	Must be monitored and recorded.
Chamber ventilation	Sealed	<0.05 ACH	Airtight	<0.05 ACH	No external airflow	Sealed
Air mixing	Well-mixed conditions with fans, referencing AIHA *Mathematical Models for estimating Exposure to Chemicals* or ASTM E741.	High volume ceiling fan during contaminant generation. Recirculating fan capable of 50 to 250 cfm.	Ensure homogenous distribution with fans across at least three sampling points. 'Homogenous' not defined.	Sufficient mixing as defined in ASTM DD6670 Section 8.4.	Use of stirring fan.	30 CFM muffin fan placed on the floor underneath the nebulizer.
Preferred test bacteria	*S. aureus, S. epidermidis, B. atrophaeus, B. subtilis* (gram+) *E. coli, K. pneumoniae, P. aeruginosa, S. marcescens* (gram-)	*S. epidermidis* (gram+) *A. baumanii* (gram-) *G.stearothermophilus (spore-forming)*	*S. aureus* (gram+) *M. luteus* (gram-)	n/a	*S. albus* 8032, or other appropriate non- pathogenic.	*S. aureus* (gram+) *A. baumannii, K. pneumoniae, P. aeruginosa* (gram-) or other appropriate.
Preferred test viruses	MS2, Phi 6, Phi X174	MS2	n/a	MS2	Phi-X174, MS2, H1N1, H3N2	n/a
Medium	Not specified but required to be reported.	Water and PBS, concentrations not specified but required to be reported.	Distilled or de-ionized water with specified dilution.	Not specified, but required to be reported.	De-ionized water.	Specified dilution of mucin, yeast extract & PBS.
Test method	Decay	Decay	Decay	Decay	Decay	Decay
Reported metric	CADR	CADR	Percent reduction over time t	CADR	Percent reduction over time t	Percent reduction over time t

Note: see Efficacy section for definition of the decay method.

In the case of GUV technologies, it is not currently clear whether these test methods will tend to produce pathogen inactivation rates at the high or low end of the wide distribution that is found in the published academic literature (see *Efficacy* section). One device manufacturer has made their test reports under standard GB/T 18801-2022 publicly available[13]. Given that the source of variation across experiments is not currently known, it is quite possible that orders of magnitude variation could be seen in different facilities that perform the same consensus standard test. Until the source of this variation is known, it is not possible to make specific recommendations about how test methods should be improved.

One clear issue with these test methods when employing GUV devices is that the average photon pathlength—and therefore the average UV fluence rate—will depend on the chamber geometry and placement of the device. In contrast, the clean air delivery rate of an air purifier ought not to be affected to the same degree. While there is usually a requirement to use the device in the chamber in accordance with the manufacturer's instructions, some GUV devices could not be safely installed at all in accordance with relevant consensus standards in some of these chamber geometries, and there may be multiple options for safe installation that change the photon pathlength.

This intrinsic property of GUV devices—that the air cleaning that they will deliver in real-world spaces is not just a property of the device but the way the device is used in the space—needs to be accounted for in the interaction between test methodologies and the way they are employed in guidance and standards for real-world application.

ASHRAE 241

ASHRAE 241 *Control of Infectious Aerosols* is the only current standard of relevance for the application of far-UVC that covers efficacy. It is a new standard, published in 2023 after an accelerated development process commissioned by the White House, and has not yet gone through the ANSI accreditation process. ASHRAE 241 is currently under 'continuous maintenance', which means that changes to the standard can be proposed at any time, in contrast with the usual ASHRAE policy which is to revise standards every five years. Once ASHRAE 241 is ANSI-accredited, it will likely move to the five-year review cycle.

ASHRAE 241 aims to provide a technology-neutral framework for increasing the amount of air cleaning in indoor public spaces, on top of minimum ventilation requirements. It proposes the concept of an Infection Risk Management Mode (IRMM), and the required equivalent clean airflow for infection risk management (ECA_i) in different spaces.

In order to provide guidance, ASHRAE 241 employs an infection risk model that is explained in Appendix D of the standard, but the broad intention is that (under the assumptions of the model) the application of the IRMM requirements would equalize the infection risk to the same low level in different spaces. This resulting amount of air cleaning required in different spaces, over and above the requirements of ANSI/ASHRAE 62.1 *Ventilation and Acceptable Indoor Air Quality,* varies substantially. For example, in some spaces the ASHRAE 241 IRMM requirement translates to a substantially lower requirement than the CDC 5 eACH guideline, and in other spaces it is far in excess of the CDC guidance:

TABLE 9.2. Comparison between ASHRAE 62.1 minimum ventilation requirements and ASHRAE 241 Infection Risk Management Mode requirements.

	Occupant density	Minimum ventilation requirements		Min ACH (3.5 m ceiling)	Total ECA_i	Additional eACH (3.5 m ceiling)
	ASHRAE 62.1				ASHRAE 241	
	Persons/100 m^2	L/s/person	L/s/m^2	hr^{-1}	L/s/person	hr^{-1}
Office	5	2.5	0.3	0.4	15	0.3
Elementary classroom	25	5	0.6	1.9	20	3.2
Gym (aerobics room)	40	10	0.3	4.4	40	12.0
Restaurant dining space	70	3.8	0.9	3.7	30	17.9
Lecture hall	150	2.5	0.3	4.2	25	34.4

Note: occupant density standard assumptions are provided in ASHRAE 62.1. ACH and eACH requirements calculated by Blueprint Biosecurity based on 3.5 m ceiling height and the parameters listed in the table.

For an elementary school classroom with a 3.5 m/11 ft ceiling height, under standard occupancy assumptions, the CDC 5 eACH guidance is almost identical to the ASHRAE 241 standard. However, in the case of a densely packed college lecture hall at that same ceiling height, the minimum ventilation requirements per ASHRAE 241 would be 4.2 ACH, and the ASHRAE 241 IRMM requirements nearly 40 eACH. Under standard occupancy assumptions, to comply with IRMM a restaurant would require equivalent clean airflow comparable to a modern hospital operating theater as per ANSI/ASHRAE/ASHE 170 *Ventilation of Health Care Facilities.*

ASHRAE 241 does permit facilities to meet IRMM requirements by restricting occupancy, and therefore the total ECA_i requirement which is calculated on a per person basis. In the absence of a technological breakthrough, there is no other way IRMM requirements could feasibly be met in many densely occupied indoor public spaces.

One challenge with ASHRAE 241, and for any framework for infection control that is intended to be both technology-neutral and pathogen-agnostic, is the selection of a test organism for the purposes of calculating device efficacy. As noted in the *Efficacy* section, the susceptibility of pathogens to far-UVC varies significantly, raising the question of how to 'credit' efficacy for the purposes of a standard that is intended to be broadly applicable against all airborne infection risks. ASHRAE 241 requires the use of MS2 bacteriophage in efficacy testing, which is less susceptible than pathogens that cause endemic respiratory illnesses such as SARS-CoV-2 and influenza. This means that ASHRAE 241 is conservative with regards to permitting the use of UV technologies to meet its requirements, assuming that the intention of implementing IRMM would be to prevent transmission of these typical respiratory illnesses.

Regulations

Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA)

The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) empowers EPA to regulate the registration, distribution, sale, and use of pesticides in the United States under Sections 2(q) and Section 7[14]. Under FIFRA, anything that prevents, destroys, mitigates, or repels a pest (including any microorganism) is considered a pesticide—including UV which inactivates microorganisms. Therefore, all germicidal UV devices are pesticides regulated under FIFRA.

FIFRA imposes a requirement that a pesticide device be registered with EPA if the device is sold with a substance (typically, chemical in nature—e.g. silver, zinc, copper). If it works solely by physical means (i.e., just the UV radiation), then it does not have to be registered. However, under 40 C.F.R. Part 167, pesticide devices have to be produced in an EPA-registered location[15].

Unlike chemical pesticides, EPA does not do premarket review of UV devices[16]. EPA does, however, regulate misbranding and false or misleading claims. In the surface disinfection market, EPA has specifically defined terms in its guidelines:

- 'Sanitization'—99.9% reduction in specific tested microorganisms (see more in OCSPP 810.2500)[17]
- 'Disinfection'—99.999% reduction in specific tested microorganisms (see more in OCSPP 810.2200)[18]
- 'Sterilization'—99.9999% reduction in specific tested microorganisms (see more in OCSPP 810.2100)[19]

If a manufacturer uses these terms but the device doesn't meet their definitions as supported by acceptable testing data, that can be considered false or misleading. The EPA currently does not have a comparable set of guidelines or specified acceptable testing procedures for air disinfection.

Other relevant terms that can potentially be subject to misleading claims regulation under EPA jurisdiction include[20]:

- Germicidal
- Elimination
- Safe
- Purify

Importers of all FIFRA-regulated devices have to comply with the above labeling/branding definitions and this is checked by EPA regional offices in collaboration with US Customs and Border Protection (CBP)[16]. Each UV device is considered its own product by the EPA, requiring its own data to support any claims. Therefore a device manufacturer cannot rely on testing done by other manufacturers using the same bulb, in the same way that every device that uses a HEPA filter has to undergo its own testing.

While in principle a manufacturer of a UV device can supply data from the various test methods cited above to back up claims, the EPA does not currently specify what tests are required for air disinfection, and does not have to take any action unless there is a consumer complaint. We expect that when a standard air cleaning test method is agreed upon for UV devices, the EPA may well specify what tests they expect when evaluating a marketing claim.

Other regulations

ASHRAE 241 has yet to be adopted into any regulations. However, the Government of New Brunswick in Canada has announced the intention to achieve ASHRAE 241 compliance in new public buildings[21]. In November 2024 there was a failed ballot initiative in the city of Berkeley[22] to apply ASHRAE 241 to all city-owned and -leased facilities. However, the initiative also would have banned the use of all GUV technologies to meet the standard, through mandating that "the City shall not use any air filtration or air disinfection technologies that emit ozone, volatile organic compounds, oxidation byproducts, or excessive decibels, and shall not install any ultraviolet light disinfection technology in such a manner that the light will come into contact with human skin." This would have prohibited the use of both far-UVC and upper-room UV systems, as some UV comes into contact with human skin when upper-room UV systems are used, and there is also some oxidation chemistry under conventional UV irradiation[23].

Besides ventilation-related aspects of building codes, a relevant standard for far-UVC may be the International Energy Conservation Code (IECC), developed by the International Code Council (ICC). It is updated on a 3-year cycle. Section 405 of the IECC pertains to lighting fixtures[24], and stipulates the minimum acceptable lighting power density (LPD, units W/ft^2) for a light fixture. With the advent of the white LED, minimum LPDs have increased, and no currently available far-UVC fixture would be compliant. For this reason, it is critical that GUV fixtures not be classified as 'light fixtures' in this context.

Photobiological safety

Guidance

An explanation of the UV exposure limits recommended by the American Conference of Governmental Industrial Hygienists (ACGIH)[25] and the International Commission on Non-Ionizing Radiation (ICNIRP)[26] is provided in the *Far-UVC primer* section of this report. It is within the gift of any standards organization, legislature, or public health agency to apply guidelines from either ICNIRP or ACGIH, or indeed come up with their own. Here, we go into more detail on the differences between these two bodies.

ACGIH is a non-profit organization. It is substantially funded by subscriptions and sales of its guidance documents. Despite having 'American' in its name, ACGIH Threshold Limit Values (TLVs) are widely used in industrial hygiene and occupational and environmental health and safety practice across the globe. This means that TLVs are not specifically for use in America, but rather generally applicable occupational exposure limits. That is, they are intended to be exposure limits for healthy working adults—not children or photosensitive individuals. This does not necessarily mean that ACGIH TLVs are inappropriate for populations other than healthy working adults, only that these populations are not required to be considered as part of the process of recommending exposure limits. Until the recent update in ACGIH exposure limits, ACGIH and ICNIRP were in alignment on the spectral weightings for UV exposure limits.

Like ACGIH, ICNIRP is an advisory body, and does not issue standards or regulations. Unlike ACGIH, ICNIRP is funded by donations primarily from government agencies. Most importantly, ICNIRP assesses health risks for broad groups of people, not just in occupational contexts. There are also differences between their processes for development and revision of guidelines. Due to its structure and bylaws ICNIRP is a slower moving organization. The fact that there is currently a substantial difference between ACGIH and ICNIRP exposure limits for far-UVC is a reflection of the different nature of the institutions, and it is possible that despite being aligned for many years they will not come fully back into alignment.

What ACGIH TLVs and ICNIRP exposure limits have in common is that exposure limits are based on a time-weighted average (TWA) over an 8-hour period. Thus they are intended to reflect the fact that in real-world situations, individuals are highly unlikely to be exposed at a constant rate over an 8-hour time period. They recommend that the dose should not be cumulatively exceeded during that time period, accounting for the variation in exposure during the time period. As we will see below, different standards have incorporated those exposure limits in different ways, and do not necessarily apply them as a time-weighted average.

Standards

ANSI/IES RP 27.1-22

The Illuminating Engineering Society (IES) publishes standard ANSI/IES RP 27.1-22: *Photobiological Hazards From UV Lamps*[27]. RP 27.1-22 describes the process for assessing photobiological safety of different devices, and contains recommended practices for taking irradiance measurements in an installation using a radiometer.

Different UV devices are assigned different Risk Groups. The Risk Groups are determined based on spectrally weighted irradiance measurements taken a certain distance from the device, with the distance depending on the device. For 'Open room germicidal products for occupied spaces', the irradiance measurement for determining risk groups must be taken 1.0 m from the source. For the spectral weightings, RP 27.1.22 references ACGIH spectral weightings S(λ):

TABLE 9.3. Emissions limits for Risk Groups per ANSI/IES RP 27.1-22.

Risk group	S(λ)-weighted irradiance 1 m from source	Absolute irradiance after applying ACGIH eye S(λ) at 222 nm	Absolute irradiance after applying ACGIH skin S(λ) at 222 nm
RG-3	>3 μW/cm²	>~161 μW/cm²	>~470 μW/cm²
RG-2	0.3–3 μW/cm²	~16.1–161 μW/cm²	~47–470 μW/cm²
RG-1	0.1–0.3 μW/cm²	~1.6–16.1 μW/cm²	~4.7–47 μW/cm²
RG-0	<0.1 μW/cm²	<~1.6 μW/cm²	<~4.7 μW/cm²

Based on these emissions limits, we would expect commercially available far-UVC devices to be RG-1 or RG-0, especially when applying the skin TLVs. Importantly, Section 8.2 of RP 27.1-22 specifically states that "whole room luminaires should be mounted on the ceiling and directed downwards."

In addition to irradiance measurements being taken to determine the risk group of the device, the standard also specifies acceptance testing for whole-room GUV systems to ensure compliance with ACGIH TLVs. Irradiance measurements for assessing skin safety should be performed with a radiometer facing the ceiling, while eye measurements should be performed with the radiometer facing the wall, with an 80-degree field-of-view cone equipped to the detector in order to best approximate the human eye. This is based on the assumption that whole-room systems are mounted on the ceiling and directed down.

When determining whether irradiance measurements taken according to the recommended procedures conform with the ACGIH TLVs, RP 27.1-22 clearly specifies that time-weighted averages (TWAs) should be used in conjunction with the spectral weightings to determine the maximum allowable irradiance at particular points in the room. Appendix B provides a high-level example of how such a TWA could be quite different at different heights depending on the amount of time (for example) that occupants spend standing rather than sitting. However, the standard does not itself provide specific guidance for how time-weighted averaging should be applied by an installer of a GUV system, although it does reference the use of time in motion studies or dose badges as ways of quantifying occupant exposure.

The standard is not specific as to whether the skin or eye S(λ) values should be used to determine the Risk Group, although in the Annex it does suggest that for a ceiling-mounted lamp facing down, the skin TLV may be appropriate, as the eye has some natural protection against radiation from above due to the structure of the eye and face. Of course, this argument only applies if the room occupants spend most of their time

standing or sitting, as opposed to reclining or looking up. This argument will therefore apply in most indoor public spaces, but not necessarily all.

NALMCO (interNational Association of Lighting Management Companies) is a training and technician certification that recently launched three levels of certification for GUV practitioners, and it references ANSI/IES RP-27.1-22 in its materials[28]. A number of the experts who assisted NALMCO in developing the training are members of some of the standard committees referenced in this report.

ISO 15858

ISO 15858:2016: *UV-C Devices - Safety information - Permissible human exposure*[29] also specifies procedures for taking irradiance measurements in installed systems to ensure compliance with exposure limits. Currently it only refers to conventional 254-nm UVC, and it recommends taking irradiance measurements at eye level (between 1.83 and 2.13 m) of an installed system, with the detector facing the device. It specifies the conditions under which measurements need to be taken, which include installation and specific maintenance events such as lamp replacement or movement/adjustment of the system. It currently refers to the ACGIH TLV, and as there is no diversion between ACGIH and ICNIRP thresholds at 254 nm this makes no difference.

A new version ISO/DIS 15858 is currently undergoing development[30]. The updated standard references all UVC wavelengths, including far-UVC. Rather than specifying that measurements should be taken facing the device, as the current standard does, the draft standard recommends walking around with a radiometer and identifying spots in the room where occupants are standing or seated for extended periods of time, and where the irradiance readings appear to be higher. At these 'hot spots', horizontal (i.e. facing the ceiling) and vertical (i.e. facing the walls) irradiance measurements should be taken at a height of 1.8 m. The spectral weightings provided in the standard are taken from the current ICNIRP guidance.

This standard is importantly different to ANSI/IES RP 27.1-22 and the ACGIH/ICNIRP recommended exposure limits in that it does not use time-weighted averages. In a nutshell, rather than considering realistic average exposures, ISO 15858 requires that a hypothetical person who remains in the worst-case-scenario position in the occupied zone of the room for 8 hours still must not be overexposed.

Regulations

ICNIRP guidelines are incorporated into European law for occupational exposures through EU Directive 2006/25/EC[31]. The EU has a formal relationship with ICNIRP, and it is our expectation that EU occupational exposure limits for UV will likely continue to be drawn from ICNIRP rather than ACGIH. However, even if ICNIRP updates their guidance, this would not automatically be incorporated into the EU directive and then by the various national regulators. There will therefore likely be a multi-year process between a change in ICNIRP guidance and a change in occupational exposure limits in EU countries. When a new directive is issued there is a specified timeframe for changes to be incorporated into national legislation. In the case of EU Directive 2006/25/EC, the required timeline was within 4 years.

To our knowledge, ISO 15858 has not been incorporated into regulation.

In the US, the Occupational Health and Safety Administration (OSHA) has the regulatory authority to set occupational exposure limits. These are termed Permissible Exposure Limits (PELs), and are generally found in Standard 29 CFR Part 1910 Subpart Z[32]. However, OSHA has not adopted PELs for UV, and refers to the ACGIH limits on a more informal basis[33].

Products

Standards

IEC/EN 62471

The International Electrotechnical Commission (IEC) publishes IEC/EN 62471: *Photobiological safety of lamps and lamp systems.* It is in many respects identical to ANSI/IES RP-27.1-22 with respect to ultraviolet lamps. It recommends essentially the same procedures for determining Risk Groups, and has the same recommended practices for upper-room UV systems. It also states that open germicidal systems for occupied spaces are intended for use by "professional, competent persons", and the same language is employed by RP 27.1.22.

However, despite these similarities, there are critical differences between the standards for whole-room far-UVC systems. This is due to the fact IEC/EN 62471 references ICNIRP spectral weightings, not those of ACGIH, which places far-UVC devices into different Risk Groups:

TABLE 9.4. Emissions limits for Risk Groups per IEC/EN 62471.

Risk Group	S (λ)-weighted irradiance 1 m from source	Absolute irradiance after applying ICNIRP S(λ) at 222 nm
RG-3	>3 μW/cm²	>~23 μW/cm²
RG-2	0.3–3 μW/cm²	~2.3–23 μW/cm²
RG-1	0.1–0.3 μW/cm²	~0.75–2.3 μW/cm²
RG-0	<0.1 μW/cm²	<~0.75 μW/cm²

Under this classification, some high-powered undiffused lamps will be RG-2 rather than RG-1, and diffused lamps RG-1 rather than RG-0.

When a far-UVC device is categorized as RG-2 by IEC/EN 62471, this creates a number of requirements. In particular, RG-2 devices are required to have proximity sensors to prevent the lamp turning on if an individual is detected within the "area of primary emission". This likely makes RG-2 devices impractical for use for whole-room disinfection of occupied spaces under this standard.

By contrast, the requirements for RG-1 devices are mainly restricted to labeling. This means that unlike ANSI/IES RP 27.1-22 or ISO 15858, IEC/EN 62471 does not specify acceptance testing procedures for taking irradiance measurements of an installed whole-room system using RG-1 or RG-0 devices to ensure compliance with UV exposure limits.

ANSI/CAN/UL 8802

UL (formerly Underwriters Laboratories) publishes standards ANSI/UL 8802: *Ultraviolet (UV) Germicidal Equipment and Systems*[34] as well as ANSI/UL 8803: *Portable UV Germicidal Equipment With Uncontained UV Sources*[35]. These standards apply to all UV systems with emissions 200–400 nm, and not just far-UVC devices. Like IEC/EC 62471, these are product standards that do not require measurements to be taken in the field, but rather in a testing lab.

In order for a device to be UL 8802-compliant it must be permanently mounted in a fixed location, and not have the orientation of the lamp adjustable by the use of ordinary tools. This is to ensure that the UV exposure of the occupants of the room remains consistent with its installation instructions, under which it is tested. Lamps intended for use in dwellings, or that are not permanently mounted, or that can have their orientation adjusted by ordinary tools, can only be certified to the stricter requirements of UL 8803 (see below).

UL 8802 references ANSI/IES RP 27.1-22 and the ICNIRP/ACGIH recommended exposure limits. Although these standards specify time-weighted average exposure limits, UL 8802 does not base its recommendations on the time-weighted average exposure, but rather on a peak irradiance reading under test conditions that are the worst-case scenario consistent with minimum ceiling height and other installation instructions.

For UL 8802 certification a device must be assigned Risk Group 0 according to either IEC/EN 62471 or ANSI/IES RP 27.1-22. However, while UL 8802 uses the same emissions limits as those standards to assign the Risk Group (see Table 9.3 and Table 9.4), the place at which the irradiance readings are taken to determine the Risk Group is not the same, nor is it the same as in IEC/EN 62471. Therefore, devices do not necessarily belong to the same Risk Group per UL 8802 as they do in the other standards.

For devices marked by the manufacturer with a minimum mounting height of at least 2.1 m, UL 8802 mandates comparing the Risk Group emission limits to the highest irradiance measurement in the room that can be found under test conditions, using a radiometer pointed in any direction on the vertical plane 1.9 m above the floor in the most severe possible conditions given the minimum ceiling height and installation instructions supplied with the device. This is 10 cm higher than the height specified in ANSI/IES RP 27.1-22, and the height specified for upper-room UV systems in the appendix to IEC/EN 62471. By referencing both standards, UL 8802 effectively allows either the ACGIH or the ICNIRP spectral weightings to be used to calculate whether the absolute irradiance at this point is within the spectrally weighted emission limit.

When using ANSI/IES RP 27.1-22 to assess compliance with UL 8802 under these test conditions, there is some ambiguity in the standard regarding whether the ACGIH Skin or Eye spectral weightings should be applied. Our understanding from speaking with experts is that when the standard was initially released, the testing laboratories that were certifying devices to UL 8802 were using the Eye spectral weightings. However, now the consensus is that if a device's installation instructions specify that it must be permanently mounted on the ceiling facing down, then the spectral weightings for Skin are used, in accordance with the suggestion made in the Annex to ANSI/IES RP 27.1-22 for applying the TLVs to whole-room fixtures. However, for all other installation instructions, the relevant spectral weighting is the Eye.

When using IEC/EN 62471 to comply with UL 8802, there is no such ambiguity, as there is only a single spectral weighting in the ICNIRP guidelines referred to in that particular standard. For determining Risk Groups, UL 8802 specifies that the irradiance reading be taken at a height of 1.9 m at whatever point in the room produces the highest irradiance reading, whereas IEC/EN 62471 is based on the irradiance 1 m from the lamp for whole-room germicidal devices. This means that UL 8802 is effectively stricter than IEC/EN 62471 for devices with a minimum ceiling height <2.9 m, and vice versa for higher mountings when applying ICNIRP spectral weightings to the emission limits. For devices not marked with a minimum mounting height or where it is less than 2.1 m, the irradiance reading must be taken 20 cm from the device.

It is possible in principle to meet UL 8802 with higher-output devices than might otherwise be possible through the use of sensors that modulate the device output if it detects a person or object being exposed, although this creates further requirements, such as compliance with UL/CSA/IEC 60730 *Automatic Electrical Controls*[36], to ensure that the mechanism is sufficiently reliable. If it does not meet these further requirements, any sensors, motion detectors etc. have to be disabled for the photobiological safety testing.

UL 8802 requires the manufacturer to provide instructions to ensure that the installation does not produce ozone concentrations in excess of an unspecified 'safe value', taking into account the number of lamps used, the ventilation system and other unspecified relevant factors.

ANSI/CAN/UL 8803

UL 8803 is in many respects similar to UL 8802, but it is for lamps intended for use in dwellings, or that are not permanently mounted, or that can have their orientation adjusted by ordinary tools. It also references the use of IEC/EN 62471 or ANSI/IES RP 27.1-22 for calculating Risk Group. Like UL 8802, the device must be classified as Risk Group 0 according to the procedures. However, UL 8803 mandates a different process for making the irradiance measurement and hence determining the Risk Group. Unlike UL 8802, there is no ambiguity about the spectral weightings when using ANSI/IES RP 27.1-22 to determine the Risk Group—the ACGIH Eye spectral weightings are used.

For a product to comply with UL 8803, it must have a motion detector, a specified activation cycle and an operating time limit. The standard specifies an 'exempt perimeter', and the irradiance measurement for the purposes of determining the Risk Group is based on the size of the exempt perimeter. The motion detector must cover the entire exempt perimeter, meaning that the point at which the irradiance readings for determining the Risk Group are taken is determined by the motion detector employed and the device software.

UL 8803-compliant devices have a maximum operating cycle of 60 minutes, unless their irradiance is further tested at a 20-cm distance from the lamp. UL 8803 places a number of requirements on the activation cycle that must occur between operating cycles. These require manual intervention from the user and a delay in order to resume operation.

Given these limitations, our expectation is that UL 8803-compliant devices are unlikely to be suitable for whole-room GUV systems. However, they may have other applications not within the scope of this report.

With regards to ozone, UL 8803 also requires devices emitting with wavelengths less than 250 nm to comply with UL 867 *Electrostatic Air Cleaners* (see below on *Ozone* standards).

Lamp exposure limits

Researchers have proposed that non-monochromatic sources should be labeled with 'Lamp exposure limits' (H_{LEL}) that provide users with the information on the spectrally weighted emissions. While the exposure limits at 222 nm are commonly quoted as the exposure limits for far-UVC devices, Eadie et al., 2024[37] found that the spectrally weighted emissions of different KrCl* lamps can meaningfully diverge depending on the use of filters. The difference in the spectrally weighted exposure limit across these KrCl* lamps varied by a factor of 4 under the ACGIH spectral weightings:

TABLE 9.5. Lamp Exposure Limits from Eadie et al., 2024[37]. The measured irradiance of 14 commercially available KrCl* far-UVC sources at a distance of 20 cm.

		Filtered										Unfiltered			
Lamp number		**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**
Irradiance (mW/cm^{-2})		0.30	0.08	0.16	0.23	0.09	0.02	0.08	0.01	0.15	0.17	0.38	2.01	0.17	0.02
	H_{222}	Lamp exposure limit (H_{LEL})													
ICNIRP	23	23	23	23	23	23	23	23	23	23	22	20	19	19	18
ACGIH (Eye & Skin)	161	154	154	156	147	151	147	127	132	116	108	58	56	47	43
ACGIH (Skin – eye protected)	479	460	443	442	439	419	411	361	361	305	288	145	136	113	100

Note: LEL is determined using the technique described by Buonanno et al.[38] and presented for the three guideline S(λ) curves from ICNIRP and ACGIH. The exposure limit values at 222 nm (H_{222}) for each organization are displayed for comparison.

For all lamps measured, the spectrally weighted lamp exposure limit (H_{LEL}) was lower than the ACGIH TLV at 222 nm. This is to be expected, given that these lamps emit some wavelengths in the conventional UVC range. However, for some filtered lamps this difference was as little as 5%, whereas for at least one unfiltered lamp the spectrally weighted exposure limit was 75% lower than the nominal TLV at 222 nm. This difference is less significant when using the ICNIRP spectral weightings, but at least one unfiltered lamp had a 22% lower H_{LEL} than the nominal ICNIRP guidelines at 222 nm.

Eadie et al. also highlighted that different radiometers do not give the same answer, as radiometers are calibrated (according to standards such as ISO/IEC 17025 *Testing and calibration laboratories*)[39] to an intended source, and that this will therefore produce a different reading depending on whether the intended source is a filtered or unfiltered KrCl* lamp. Knowing the lamp exposure limit would allow practitioners to make corrections based on the H_{LEL} of the lamp and the source to which the radiometer was calibrated.

As it is not possible for anyone without possession of a spectroradiometer (and knowledge of how to use it) to measure the full emissions spectrum of a UV device, there is considerable sense in there being a consensus standard that specifies the procedures for testing labs to do this, and then calculating a lamp exposure limit based on the ACGIH and/or ICNIRP spectrums. Such a standard would very likely be referenced in future iterations of the other photobiological safety standards, as knowledge of a lamp's spectrally weighted emissions is necessary for ensuring compliance.

Regulations

IEC/EN 62471 covers a broad range of products, not just lamps generating ultraviolet radiation, and it is incorporated into regulation in a number of jurisdictions. A lamp—including a far-UVC lamp—must have a CE mark in order to be sold in the EU, and lamps must comply with IEC/EN 62471 to obtain a CE mark. As of 2023, the UK has its own UKCA mark for lamps which also requires IEC/EN 62471 compliance, but it continues to recognize the CE mark as well.

While it is not uncommon for UL standards to be incorporated into state and federal regulations in the US, outside of ozone regulations in California (see below) far-UVC-relevant UL standards remain voluntary only.

While not a photobiological safety requirement, in the US the Federal Communications Commission (FCC) has a mandate to enforce regulations on any products emitting radio frequencies, and according to the law such devices have to be certified to FCC standards in order to be commercially sold[40]. While the UV emissions of far-UVC lamps are not radio frequencies, like almost all electronic devices KrCl* lamps do create radio frequency emissions as a byproduct of operation and thus require FCC certification. Similar requirements exist across a number of product standards mandated in the European Union.

Ozone

Guidance

A number of bodies issue guidance about occupational ozone exposure that apply indoors. NIOSH recommends an occupational exposure limit of 100 ppb as a time-weighted average over 8 hours, with 5000 ppb (5 ppm) considered immediately dangerous to life and health[41]. ACGIH 8-hour TLVs for ozone are 100 ppb for light work, 80 ppb for moderate work, and 50 ppb for heavy work, or 200 ppb over 2 hours[42].

The WHO makes recommendations that apply to outdoor ozone, specifically that the average of daily maximum 8-hour mean ozone concentration in the six consecutive months with the highest six-month running-average ozone concentration not exceed 30 ppb, or exceed 50 ppb on more than 3–4 days per year[43].

ASHRAE has taken an institutional position on ozone-emitting air cleaners[44]:

> The current state of the science regarding the health effects of ozone strongly suggests that the use of air cleaners that emit ozone by design should not be permitted; the same information and advice is given by the U.S. EPA, among others (EPA 2013). There is more uncertainty about recommendations for air cleaners that do not use ozone by design for air cleaning but produce ozone unintentionally, as a by-product of their operation. There are devices that emit ozone but at the same time reduce concentrations of other harmful contaminants. The state of the science does not allow making highly certain trade-offs between increased exposure to ozone and the ozone reaction by-products and reduced exposure to other contaminants. In the absence of robust information regarding safe levels of ozone, the precautionary principle should be used. Any ozone emission (beyond a trivial amount that any electrical device can emit) should be seen as a negative and use of an ozone-emitting air cleaner, even though the ozone is an unintentional by-product of operation, may represent a net negative impact on indoor air quality and thus should be used with caution. If possible, non-ozone-emitting alternatives should be used.

See *Ozone and indoor air quality* section for more details. Reflecting this position, a number of ASHRAE standards have incorporated product standards related to ozone generation (see below).

Standards

UL 867 and UL 2998

The relevant UL standards for ozone are UL 867 - *Electrostatic Air Cleaners*, and UL 2998 - *Zero Ozone Emissions Validations* [45,46]. UL 867 describes the test method for measuring ozone–the size of the chamber, the ventilation rate inside the chamber, the properties of the equipment, and the sampling rate to be used. To meet UL 867, the measurement as per the procedure should not exceed 50 ppb. To meet UL 2998, the same test is referenced, but 5 ppb must not be exceeded.

The first edition of UL 867 - *Electrostatic Air Cleaners* was published in 1980, and until recently it did not refer to ultraviolet radiation at all. It was only in August 2023 that the fifth edition was revised to state that the standard also applied to ultraviolet lamps. UL 2998 *Environmental Claim Validation Procedure (ECVP) for Zero Ozone Emissions from Air Cleaners* is a standard for claiming that any device is 'zero ozone'. This adopted the testing ozone methodology in Section 40 of UL 867. While the threshold for UL 2998 is 5 ppb, it is called Zero Ozone Emission Validation. We believe that this is because it would be difficult to conduct a similar test with a lower threshold while maintaining a strong enough signal-to-noise ratio.

The test method—which was originally designed for electrostatic air cleaners—mandates a 950–1100 cubic foot inert chamber, with a minimum side dimension of 8 feet and a maximum height of 10 feet. Air exchanges with the chamber are set such that the total observed ozone decay rate is 1.33. This is a much lower value than is observed in many real-world environments (see *Ozone and indoor air quality* section).

The test method requires the ozone monitor to be placed in the peak location in the test chamber. However, in order to determine the peak location, the standard mandates that the ozone monitor be placed two inches from the air discharge grille—a feature of an electrostatic air cleaner, but not of a UV lamp. It is our understanding based on testing that has been conducted on existing far-UVC lamps that the monitor is placed two inches away from the lamp surface.

This practice does not make sense for evaluating the ozone emissions of a far-UVC lamp. If the ozone monitor is placed in such a way that it blocks the UV photons emitted from the lamps, then the average photon pathlength will be very short and therefore the room average fluence rate very low (see *Efficacy* section). On the other hand, due to the inverse square law, the fluence rate local to the location of the ozone monitor will be very high. Even if the ozone monitor were not so placed, the location of the lamp and the way it is oriented in the room can dramatically affect the average photon pathlength and therefore the average fluence rate in the chamber.

An additional complication is that high precision ozone detection instruments—such as the 2B Tech 211-G[47]—create their own airflow in order for sufficient air to pass through the device in order to detect ozone. In the case of the 211-G, the nominal flow rate is 2 L/min with 1.2 L/min required for the device to function. Thus, the monitor is not just sampling the local ozone concentration close to the lamp, it will also be sampling the air around it. For an electrostatic air cleaner that is producing its own airflow with the ozone monitor placed in the airstream, this feature of ozone monitors may have relatively minimal impact. However, this may not be the case when testing lamps.

At least two far-UVC devices have passed UL 2998 under these conditions[48-50]. However, our understanding from speaking to experts is that higher-output devices generally pass UL 867 but not UL 2998.

As the test procedure is at best ambiguous for evaluating the ozone emissions of far-UVC devices, it is not clear what the result of the test actually means for how much ozone a device will actually produce in an installation. Furthermore, the standard makes no mention of how many devices can be used in a space, thus creating an incentive to create low-power devices that pass the test and then simply using more devices to create the required fluence rate to be efficacious.

Creating a new test standard that unambiguously measures the ozone production of far-UVC devices is essential. Other standards can then use this test standard in order to set a limit on how many such devices can be used in a given space in order to limit ozone generation to an acceptable level.

ASHRAE

Several relevant ASHRAE standards reference UL 2998. Most importantly, far-UVC devices must have UL 2998 certification to comply with ASHRAE 241. Furthermore, ASHRAE 62.1 states that "Air-cleaning devices shall be listed and labeled in accordance with UL 2998." However, for the purposes of ASHRAE 62.1 UV devices are not currently considered to be 'air-cleaning devices'. For reasons given above, it is vital that new consensus standards for measuring and controlling ozone generation from far-UVC devices be created. These can then be referenced by ASHRAE and other relevant standards bodies.

ASTM WK81750

A new test standard for assessing the generation and removal of chemicals and particles by air cleaners is currently under development by ASTM International[51]. The new test method involves placing the air cleaning device in a sealed chamber and 'challenging' the air cleaner with concentrations of various contaminants that are typical of indoor environments. The testers then quantify the rate at which the air cleaner either reduces or produces the contaminants, including ozone.

The draft protocols of this standard have been used to evaluate a number of air cleaning technologies, including far-UVC lamps[52]. The test procedure removes a number of the ambiguities present in UL 867 and 2998, and is capable of a much lower limit of detection. Unlike UL 867 and 2998, the test standard does not set a requirement that a device must pass; it is currently just a test method for quantifying effects, just as the test methods on efficacy discussed above provide quantification but not requirements a device must meet in order to be used.

However, a test method like this can provide the raw information that future product and application standards can use. As discussed in the *Ozone and indoor air quality* and *Ozone epidemiology* sections, the potential effects of ozone in real-world spaces will be sensitive to the total far-UVC fluence rate and environmental factors such as ventilation. Therefore the number of lamps used, the configuration of lamps in the space, and ventilation must be considered when creating relevant standards and not just the raw measurements of ozone creation under test conditions.

Regulations

In the US, OSHA requires that occupational exposure to ozone should not exceed 100 ppb as a time-weighted average over 8 hours, or 300 ppb over a short period[53]. The EPA has the power to enforce the National Ambient Air Quality Standards (NAAQS) specified in the Clean Air Act through various mechanisms. The current standard for ozone is that the annual fourth-highest daily maximum 8-hour concentration, averaged over 3 years, should not exceed 70 ppb[54], higher than comparable guidance given by the WHO (see above). Other countries typically have comparable regulations for both occupational exposure and ambient outdoor ozone concentrations.

The California Air Resources Board (CARB) currently enforces a regulation that all air cleaning products for sale in California comply with UL 867[55]. There was a bill proposed in the California legislature in 2024 that would have extended this to compliance with UL 2998, although it was later withdrawn[56], partially due to the impact this would have on the nascent far-UVC industry. Our expectation is that despite this bill being withdrawn, California will continue to tighten regulations related to ozone in the coming years and that far-UVC devices will be subject to future regulation.

Healthcare in the United States

Healthcare in the United States operates within a complex web of guidance, standards, and regulations that govern infection control, air quality, and disinfection practices. This system is not always straightforward—some guidelines are advisory, some standards are enforceable through accreditation, and some regulations carry legal consequences. However, these categories are often intertwined, with one authority relying on another to establish best practices or define compliance expectations.

At the top of the regulatory hierarchy, the Centers for Medicare & Medicaid Services (CMS) sets legally binding Conditions of Participation for hospitals and long-term care (LTC) facilities receiving federal funding. CMS does not create its own infection control guidelines but instead requires hospitals to comply with standards set by accrediting organizations such as The Joint Commission, while LTC facilities are primarily regulated through state health department surveys rather than independent accreditation bodies. The Joint Commission, in turn, draws from established best practices set by organizations like the CDC, the Facilities Guidelines Institute (FGI), and the Association for the Healthcare Environment. To verify compliance, CMS requires periodic surveys, which are typically carried out by state health departments. However, hospitals can choose to be surveyed by an accrediting organization like The Joint Commission instead, in which case the state does not conduct its own inspection unless there is a complaint. CMS also retains the authority to directly survey facilities when necessary. In addition to federal oversight, states have their own licensing requirements for healthcare facilities and can revoke a facility's license if serious deficiencies are found during a survey. This step is typically reserved for extreme cases where immediate corrective action is required.

Beyond hospitals and LTC facilities, infection control guidance and regulatory oversight extend to a wide range of healthcare settings, including dialysis centers, ambulatory surgical centers, behavioral health

facilities, federally qualified health centers, and dental offices. These facilities often fall under a mix of CMS requirements, state health department oversight, and accreditation from organizations like The Joint Commission or the Accreditation Association for Ambulatory Health Care (AAAHC). However, they frequently have different air quality standards and infection control expectations than hospitals, sometimes with more limited ventilation infrastructure. In these cases, far-UVC and other air disinfection strategies could play a role in supplementing existing infection control measures, particularly in settings with high patient throughput or aerosol-generating procedures.

This layering of influence means that while some infection control measures may not be explicitly required by law, hospitals that fail to follow them risk losing accreditation, failing compliance audits, or being held liable for lapses in patient safety. For example, the CDC's Environmental Infection Control Guidelines are technically non-binding, but because The Joint Commission and CMS expect hospitals to adhere to them, they become functionally mandatory for facilities that wish to remain in compliance. Similarly, FGI does not directly regulate hospitals, but its design standards for airborne infection isolation rooms (AIIRs) are embedded into building codes and accreditation expectations, making them effectively required.

Guidance

While these guidelines do not have the force of law, they are widely referenced by healthcare facilities, regulatory bodies, and accrediting organizations. Three guidance documents that address GUV are the CDC *Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Settings* (2005)[57], the CDC *Guidelines for Environmental Infection Control in Health-Care Facilities* (2003, updated 2019)[58], and the NIOSH *Environmental Control for Tuberculosis: Basic Upper-Room Ultraviolet Germicidal Irradiation Guidelines for Healthcare Settings* (2009)[59]. These documents were all last updated before significant research on far-UVC emerged, and as such they focus on conventional 254-nm GUV systems.

CDC Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Settings (2005)

The CDC TB transmission guidelines outline a hierarchical framework for TB infection control, prioritizing administrative controls, environmental controls, and personal protective equipment (PPE). Environmental controls include ventilation, HEPA filtration, and GUV as supplementary strategies.

These guidelines introduced the concept of Airborne Infection Isolation (AII) rooms, requiring negative pressure ventilation and high-efficiency air filtration for TB patients. The CDC acknowledges upper-room 254-nm GUV as a validated environmental control method, particularly in settings where achieving high ventilation rates is impractical. The guidance suggests that GUV should be prioritized in settings such as tuberculosis isolation areas, where high ventilation rates are difficult to maintain and where exposure risks for healthcare workers and patients are elevated. However, this guidance does not provide specific dose-response data for GUV effectiveness across different pathogens, nor does it outline an explicit framework for evaluating or integrating emerging technologies like far-UVC.

CDC Guidelines for Environmental Infection Control in Health-Care Facilities (2003, updated 2019)

These guidelines provide broad recommendations for preventing the transmission of infectious agents in healthcare settings. While the document covers ventilation and surface disinfection, it also discusses GUV as an air disinfection method in high-risk areas. It recognizes both upper-room GUV and duct-mounted GUV systems as effective but supplementary air-cleaning technologies.

The 2019 update reaffirmed that GUV can be used for airborne infection control, particularly in areas where ventilation capacity is constrained. However, the document does not provide detailed technical specifications for implementing GUV.

NIOSH Environmental Control for Tuberculosis: Basic Upper-Room Ultraviolet Germicidal Irradiation Guidelines for Healthcare Settings (2009)

This guidance is the primary federal document outlining best practices for upper-room 254-nm GUV, specifically for tuberculosis (TB) control in healthcare settings. The document focuses on settings where TB transmission risk is highest, such as waiting rooms, emergency departments, and isolation areas. It emphasizes that upper-room GUV should supplement, rather than replace, ventilation and air exchange strategies. To ensure effectiveness, the guidance recommends achieving 30–50 μW/cm² fluence rates in the upper-room zone and maintaining sufficient air mixing to bring airborne pathogens into the irradiated space.

The document identifies several key factors that affect GUV effectiveness, including air circulation, humidity levels, and fixture placement. It notes that well-designed GUV systems can inactivate airborne pathogens effectively in spaces with ventilation rates up to 6 ACH, provided that air mixing is sufficient to distribute irradiated air evenly. Without adequate mixing, the efficiency of GUV is significantly reduced, as untreated air may remain in occupied spaces for longer periods. The guidance underscores the importance of fixture placement, recommending that UVGI lamps be installed to ensure maximal exposure of airborne pathogens in the upper portion of a room while minimizing direct exposure to occupants. It also notes that GUV is most effective when combined with mechanical or natural air mixing strategies, such as ceiling fans or HVAC-driven airflow, to ensure that airborne pathogens reach the irradiated zone. Additionally, the document warns that high humidity levels above 60% can reduce GUV efficacy by increasing microbial resistance to UV exposure. These considerations are critical for designing an effective air disinfection strategy that integrates with existing ventilation and infection control measures.

Together, these three guidance documents establish GUV as an acceptable infection control tool, but they do not account for far-UVC due to their last updates occurring before interest in the technology grew. For far-UVC to be formally integrated into infection control policies, these guidance documents would need to be revised or new guidance issued to establish best practices, safety guidelines, and efficacy benchmarks for 222-nm UV systems. One of Blueprint Biosecurity's main goals in our far-UVC program is accelerating the research that these trusted bodies need to update or issue new guidance.

Another guideline not specific to GUV but with some relevance is the Guideline for Disinfection and Sterilization in Healthcare Facilities[60] (2008). This guideline provides recommendations on the appropriate use of disinfection and sterilization methods in healthcare environments for surface and equipment disinfection rather than airborne infection control. While it primarily focuses on chemical disinfectants and sterilization techniques, it does mention ultraviolet disinfection as a potential method for environmental decontamination. Because far-UVC systems are also being researched for their surface disinfection potential, this standard may be relevant for future far-UVC deployment in healthcare settings.

Standards

Facilities Guidelines Institute

The Facilities Guidelines Institute (FGI) establishes design and construction standards for healthcare facilities, including requirements for airborne infection isolation (AII) rooms[61]. These standards are referenced by accrediting bodies such as The Joint Commission and enforced by state health departments. FGI standards define minimum air change rates, pressure differentials, and filtration requirements for spaces where airborne pathogens are a concern. Current FGI guidelines do not include GUV as a primary control strategy for airborne infection isolation. If far-UVC were to be considered as an alternative or supplement to ventilation in these spaces, FGI standards would need to be updated to specify performance benchmarks, required fluence rates, and installation best practices.

The Joint Commission

The Joint Commission (TJC) is the largest and most influential healthcare accrediting body in the United States, overseeing more than 22,000 healthcare organizations, including hospitals, long-term care facilities, behavioral health centers, and ambulatory care centers[62]. The Centers for Medicare & Medicaid Services (CMS) grants The Joint Commission 'deemed status', meaning that hospitals accredited by TJC automatically meet CMS Conditions of Participation and do not require separate federal inspections. However, hospitals that fail to meet TJC standards risk losing accreditation and, by extension, federal funding eligibility.

TJC does not create its own infection control guidelines from scratch but instead draws from authoritative sources such as the CDC, FGI, ASHRAE, and state and federal regulations. It translates these guidelines into enforceable accreditation requirements, ensuring that healthcare facilities meet nationally recognized standards for infection prevention. The most recent updates to the TJC infection control standard requires hospitals to develop and implement policies for preventing airborne and droplet transmission, particularly for novel or high-risk pathogens, and establish protocols for emergency response to emerging infectious diseases[63].

While TJC does not currently reference germicidal UV (GUV) or far-UVC in its infection control standards, it plays a critical role in determining how new technologies are integrated into healthcare settings. If the CDC, CMS, or ASHRAE were to formally endorse far-UVC as an effective infection control measure, it is likely that TJC would incorporate these recommendations into its accreditation criteria, influencing hospital adoption on a national scale.

ASHRAE 170

ASHRAE Standard 170[64] is the primary ventilation standard for healthcare facilities in the United States, defining minimum requirements for airflow rates, filtration, pressure relationships, temperature, and humidity control in hospitals, outpatient facilities, and nursing homes. It establishes specific ACH requirements for different healthcare spaces, including operating rooms, AIIRs, and patient rooms, ensuring that air is properly circulated and contaminants are diluted or removed. ASHRAE 170 is widely adopted into state and federal regulations and is directly referenced in FGI Guidelines for Design and Construction of Health Care Facilities, meaning that it plays a central role in infection control policies.

While ASHRAE 170 does not explicitly reference GUV or far-UVC, it directly impacts how these technologies could be implemented in healthcare settings. Because ASHRAE 170 focuses on ventilation as the primary method of airborne infection control, hospitals are required to meet strict air exchange and filtration requirements before considering supplemental disinfection technologies. However, ASHRAE 241 has opened the door for integrating equivalent clean air measures like filtration, UVGI, and far-UVC into infection control strategies. If ASHRAE 170 were updated to allow far-UVC or upper-room GUV to supplement or partially replace certain ventilation requirements, it could accelerate adoption by formalizing performance benchmarks and installation guidelines for healthcare settings. This would provide hospitals and regulatory bodies with a clear framework for integrating these technologies while ensuring compliance with existing infection control standards.

None of the above healthcare standards currently allow for GUV to be used in place of ventilation. As such, GUV use in healthcare settings outside of TB is almost exclusively an optional add-on, not something required or incentivized by existing standards.

Regulations

FDA

The Food and Drug Administration (FDA) regulates UV devices intended for medical use, such as disinfecting medical instruments or preventing disease in humans. If a UV device is marketed for use in hospitals or healthcare settings, or on patients, it is considered a medical device under the Federal Food, Drug, and Cosmetic Act (FDCA) and requires FDA clearance or approval before marketing.

The distinction between an EPA-regulated and an FDA-regulated UV device depends largely on the claims made[65]. A UV device marketed to kill airborne bacteria in a home, office, or public space remains under EPA jurisdiction, as it is targeting microbes in the environment. However, if the same device is marketed as reducing hospital-acquired infections, protecting immunocompromised patients, or preventing disease transmission, it can become an FDA-regulated medical device. Similarly, UV systems intended to disinfect surgical tools or patient care areas in hospitals fall under FDA oversight. In 2023, the FDA introduced a new category for 'whole-room microbial reduction devices'[66] in unoccupied healthcare spaces, clarifying that certain UV disinfection systems for hospitals may now require FDA clearance in addition to EPA regulation[67]. We are aware of one such device that has achieved FDA approval for UV disinfection of surfaces in unoccupied rooms.

For manufacturers, the regulatory pathway they choose affects compliance costs, time to market, and product claims. EPA regulation under FIFRA is likely faster and less expensive—devices do not require premarket approval, but manufacturers must follow labeling requirements

and avoid unverified medical claims. FDA regulation is significantly more complex and costly, requiring premarket clearance[68], clinical or performance testing, and adherence to FDA's Quality System Regulations[69]. While FDA clearance offers greater credibility, it may add significant delays. Manufacturers seeking to sell UV disinfection devices for healthcare settings must carefully assess whether their claims will trigger FDA medical device requirements, as failing to comply could result in regulatory action or product recalls.

For healthcare facilities, EPA versus FDA jurisdiction may impact procurement, compliance, and liability. Hospitals using EPA-regulated UV devices (such as upper-room GUV fixtures) must independently verify their effectiveness, as EPA does not test or certify device efficacy before sale. In contrast, FDA-cleared UV devices must meet safety and performance standards, offering greater assurance of effectiveness and regulatory compliance. If a hospital purchases an FDA-cleared far-UVC system, it must be used according to FDA-approved indications, and may be subject to routine maintenance and calibration requirements. Conversely, an EPA-regulated far-UVC system can be installed more freely but must adhere to occupational safety guidelines and FIFRA labeling requirements.

Centers for Medicare and Medicaid Services (CMS)

The Centers for Medicare and Medicaid Services (CMS) has significant power over the healthcare sector, largely through its Conditions of Participation (CoPs) for Medicare and Medicaid reimbursement[70]. Hospitals and healthcare facilities must meet CMS infection prevention requirements to remain eligible for federal funding, making CMS one of the most powerful regulatory forces in the healthcare system. While CMS does not directly approve or regulate specific disinfection technologies like far-UVC, its policies influence which infection control measures hospitals prioritize by determining what counts as a Hospital-Acquired Infection (HAI) and what infection prevention strategies are reimbursable.

One of the most important CMS policies related to infection control is its Hospital-Acquired Condition (HAC) Reduction Program[71], which penalizes hospitals with high rates of preventable infections. Under this program, hospitals that perform poorly on infection metrics—including central line-associated bloodstream infections, catheter-associated urinary tract infections, and surgical site infections—receive reduced Medicare reimbursements. This creates a strong financial incentive for hospitals to adopt infection control measures that demonstrably reduce HAIs. Notably, the HAC Reduction Program does not currently include pathogens such as SARS-CoV-2, influenza, or measles, which diminishes a significant incentive for hospitals to implement measures specifically targeting airborne transmission.

CMS have implemented measures that incentivize preventive actions against respiratory infections in LTC facilities and nursing homes, emphasizing the importance of airborne infection control in these settings[72]. LTC facilities will be required to electronically report data on COVID-19, influenza, and respiratory syncytial virus (RSV) as well as vaccination status and hospitalizations of residents due to those pathogens.

Another important consideration is CMS's reliance on accrediting organizations like The Joint Commission to enforce compliance with infection prevention standards. Hospitals can meet CMS infection control requirements either through direct CMS surveys or via accreditation from an approved organization like The Joint Commission. If The Joint Commission or FGI were to adjust their standards to facilitate the adoption of far-UVC, hospitals seeking accreditation could implement it as part of their compliance strategy, making CMS reimbursement more feasible.

Role of other federal agencies

Several federal agencies—including the Department of Veterans Affairs (VA), Department of Defense (DoD), General Services Administration (GSA), and the United States Department of Agriculture (USDA)—are potential large-scale buyers of far-UVC technology. These agencies manage hospitals, military bases, federal office buildings, and food safety facilities, all of which could benefit from enhanced airborne infection control. Because of their size and purchasing power, their decisions to invest in far-UVC could meaningfully shift the market by driving economies of scale and increasing industry competition. These agencies may also seek input from expert agencies like the CDC before adopting new disinfection technologies, meaning government-wide coordination could be key to accelerating far-UVC adoption.

The Department of Energy (DOE) has a particularly relevant role in advancing far-UVC technology, both through scientific research and energy efficiency programs. DOE's Solid-State Lighting Program[73] has historically supported the development of UV-C LEDs and other novel lighting technologies, which could extend to far-UVC sources in the future. Additionally, DOE plays a role in building technology innovation, particularly through its work with the GSA Green Proving Ground program[74], which tests and evaluates emerging energy-efficient building technologies. If far-UVC were positioned as an energy-efficient alternative or complement to ventilation-based infection control, DOE and GSA could play a role in demonstrating its viability in federal buildings, providing critical real-world data for further adoption.

Beyond these agencies, national laboratories under DOE, such as Lawrence Berkeley National Laboratory, Pacific Northwest National Laboratory, or the National Renewable Energy Laboratory, have expertise in indoor air quality, photonics, and lighting technologies. Their further involvement in testing far-UVC systems for safety, efficacy, and energy efficiency could help establish scientific credibility and create the foundation for future standards.

Further reading

- ASHRAE 241
- How Much Ventilation Is Enough?
- Illuminate

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10. UVC impacts on materials

Summary

The built environment contains a myriad of materials that may be exposed to UVC during whole room irradiation. The majority of UV materials degradation research has focused on the effects of the UVA and UVB components of sunlight, as the atmosphere filters out UVC. As such, the impact of far-UVC on materials and resulting second-order effects are largely unknown. While there is research evaluating the effects of conventional (254 nm) UVC irradiation on materials, the protocols for characterizing degradation, including irradiation dose and time, are not standardized, and often the degradation mechanism is complex and material-specific.

Crucial considerations

- Chromophores absorb light and emit light at specific wavelengths. These chromophores can degrade into radicals that form volatile carbonyl products (ketones, aldehydes, carbon dioxide, and carbon monoxide).
- Commercially-formulated polymers often contain additives, impurities, or residual catalysts (i.e., they are not pure polymers), which makes understanding their degradation mechanisms and side products more challenging.
- Wood and paper are vulnerable to UVC radiation due to the degradation of lignin, a complex organic polymer. The complexities of lignin structure result in a variety of secondary products from UVC irradiation, which leads to uncertainty about the secondary effects of UVC degradation.
- Material reflectance can cause greater fluence rates and skin and eye exposures than expected. Metal materials have the highest reflectance of 222-nm far-UVC radiation, though this can be reduced with a protective coating or proper lamp placement.
- Many studies only assess color change when testing far-UVC degradation, while other studies include surface roughness, water contact angle, structural changes, and strength changes. Testing should be comprehensive, but also relevant to the material and the environment in which it will exist.
- We should also assess whether far-UVC causes any off-gassing of harmful compounds when interacting with common materials[1].

Analysis

The impact of UVC on the vast variety of material types and classes, especially those in the built environment, is profoundly understudied. Common experience shows us that ultraviolet radiation, including conventional UVC, can degrade and discolor materials like plastics, fabrics, and wood. While conventional UVC research can be used as a guide, the paucity of data concerning the impact of far-UVC on different material classes suggests a preliminary goal of prioritizing which materials should be tested. A summary of the known impact on materials of UVC and far-UVC, where applicable, is below.

Wood

Wood rapidly absorbs UV in the first ~80 µm of its surface[2]. Lignin, one of the major constituents of wood, is the primary absorber. Upon UV exposure, it breaks down into water-soluble products and generates other species that may absorb radiation[3].

Lignin is mainly degraded due to its capacity to absorb radiation of short wavelengths, with an absorbance peak at 280 nm. Wood's potential absorbance in the far-UVC range depends on the chromophores present[4,5].

Photoinduced degradation of lignin results in the discoloration, often yellowing, observed on the surface of wood[6]. In normal weathering, water assists in removing loosened fibers and particles released during irradiation and also causes leached lignin fragments to move to the surface, leading to discoloration and a rough texture. While this has not yet been directly shown for far-UVC, it is expected that the same effect will occur following photodegradation[7]. However, it is not expected that bare wood will usually be present in the initial deployment built environment. It is expected that most wood present will be covered with a finish and will not be exposed to natural weathering. The impact of far-UVC exposure on these wood finishes is unclear.

Paper

Paper is known to undergo discoloration during and after exposure to UV radiation. However, there are limited research studies that have investigated the degradation mechanism. One study investigated bond, rice, kraft, and amate papers exposed to 254-nm radiation for up to 480 hours. While no chemical decomposition was observed, color loss was observed, especially in the colored papers[8]. Paper is composed of a mixture of cellulose, hemicellulose, lignin, and different lignin compounds. As discussed in the *Wood* section, lignin is degraded by UVC and this leads to a discoloration, often yellowing, of the surface. With far-UVC, there is a strong chance that paper will discolor, but the time and dose required is unclear. Furthermore, it is unclear whether there is a difference between the mechanisms of degradation under 254-nm irradiation and far-UVC irradiation.

Metals

There is likely no effect of UVC and far-UVC on metal surfaces, as metals have available free electrons which can absorb photon energy without undergoing energy transitions or bond dissociation. In one study, aluminum and stainless steel were exposed to 254 nm at a dose equivalent to 16 years of exposure. This showed only discoloration in the stainless steel after the equivalent of 8 years of daily exposure at 36.8 mJ/cm^2 [9]. Generally, metals do not undergo a color change with UVC exposure unless the metal is oxidized, and the authors attributed the stainless steel discoloration to surface oxidation. Neither material showed any changes in structural integrity. While specific testing of far-UVC on commonly used materials in the built environment would provide conclusive evidence for or against the possibility of degradation or discoloration, the lack of structural changes at 254 nm suggests that metals are less likely to be affected than some other commonly used materials.

Metals will reflect far-UVC, but irradiance decreases with distance. Operating rooms, and other areas where a large percentage of the surface area is covered in metal, may pose a risk due to increased reflectance. Therefore, initial measurements should be taken to ensure any reflectance does not increase potential harm or increase exposure beyond daily limits.

Ceramics and glass

Ceramics and glass have tightly-bonded electrons, so they are generally unaffected by UV radiation[10]. There are no literature studies on the degradation effects of UVC on ceramic materials, further indicating that they are believed to be largely unaffected by UVC radiation. However, ceramic and glass materials may have synthetic coatings which may be susceptible to UVC degradation.

Textiles

Various textiles are part of a built environment, from fabric on furniture to the clothes that individuals are wearing. Textiles can be made from natural materials, like wool or cotton, synthetic materials, or a combination. Synthetic fabrics and textiles are expected to have the same degradation mechanisms as those of polymers, described elsewhere.

In addition to the composition, the dyes used to achieve different colors must also be considered when investigating any potential UVC degradation mechanisms.

Accelerated UVC testing was conducted on various fabrics including SILVERTEX, acrylic, cotton, various colors of polyester, and leather. The fabrics, especially green cotton, lost their color intensity with UVC irradiation, turning either more yellow or white depending on the original fabric color. In particular, the cellulose in cotton degraded and turned yellow. No change in tensile strength was observed[9]. Honeywell's evaluation of fabrics used in airplanes also only observed color changes with exposure to 254 nm with a dose corresponding to 10.8 years of daily use. No significant changes in mechanical properties were observed[11].

One study evaluated 222-nm exposure, corresponding to ~6.2 years of daily exposure, on the velour fabric of public bus seats. There was not a quantitative difference in the color of the velour fabric following exposure[12]. Exposure to shorter-wavelength UVC may have less of an effect on the color of the fabric. However, the other studies that observed a reduction in color intensity used different textile compositions, which would impact the degradation mechanism of the fabric.

Polymers

Numerous studies have investigated the degradation mechanisms of polymers over the past decade. Although most of these reports are for UVA (315–399 nm) and UVB (280–314 nm) radiation, some studies explore the effects of UV-C radiation. The American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE) performed a literature study of the different degradation mechanisms of various polymers and established a testing procedure for HVAC materials[10]. This section states some results from ASHRAE's findings and shares insights from other material degradation testing literature.

Pure polymers

For a polymer to absorb UVC, chromophores must be present in the polymer's structure or polymer matrix[10]. In addition to the structure of the polymer, the UV reactivities of the polymers are influenced by the presence of additives and impurities, which may have UV-absorbing properties, formed during polymerization, processing, and/or storage. However, from an initial search in the literature, there are no investigations on how the location of chromophores in a polymer matrix influences the degradation of the polymer. In the future, knowing the effects of chromophore location could be important for understanding the susceptibility of a polymer to degrade with UVC irradiation.

In theory, since polyethylene (PE) and polypropylene (PP) do not have any chromophoric groups in their polymer chain, they do not absorb UV above 220 nm[10]. Therefore, they should be resistant to UVC. Teska and colleagues determined that PP and ultrahigh-weight PE had the least damage when compared to other plastics used in healthcare that were exposed to UVC[13]. PE and PP may still be susceptible to UVA and UVB degradation depending on any leftover initiator residue and/or chromophoric groups formed during polymerization of the polymer[14,15].

Polymers with additives

Polymers are often not pure and have additives that can prevent or enhance UV degradation. Inorganic fillers can form a protective coating on the surface or can act as an energy sink for the UV energy absorbed by the polymer[10]. Exposure of a polymer to UVC can volatilize its surface and reveal the layer of inorganic filler beneath. The inorganic filler can offer protection to the interior of the polymer material[10].

Protective coatings such as epoxy resins often incorporate additives to stabilize the polymer from various environmental factors including light, heat, water, and oxygen[16]. Often these additives are metal powders, such as aluminum, due to their excellent UV reflective characteristics. The addition of spherical aluminum powder to epoxy resin provided some resistance to UV degradation 100 days after exposure, but the analysis did not include the wavelength or dose, making it difficult to understand the mechanism.

For the Drungilas study in the table below, samples were exposed to 222 nm for 50, 100, or 150 hours, corresponding to 96.7, 193.4, and 290 J/cm². Data below is from samples irradiated for 150 hours. Irradiation for 150 hours, equivalent to 290 J/cm² corresponds to ~6.2 years of daily disinfection[12].

For the Harris study in the table below, 36,000 doses over 25 years of in-service was assumed, with each dose being 3 seconds. This would require a minimum 108,000 mJ/cm² per sample. Actual exposure times were calculated based on the measured irradiance of each lamp[17]. The table only reports color changes. No adverse changes in mechanical properties of thermoplastics or textiles were reported. Furthermore, no adverse impacts on flammability properties were observed when evaluated in accordance with the FAA 60 second, vertical Bunsen burner test[17].

TABLE 10.1. Material studies using 222 nm.

Material	Dose J/cm² (hours)	Irradiance µW/cm²	Wavelength (nm)	Findings
Drungilas et al., 2023[12]				
Fiber-reinforced composite (FRC)	96.714 (50) 193.428 (100) 290.142 (150)	537.3	222	Observable color change. Decreased modulus of elasticity. Decreased plasticity. Initiation of micro-crack formation.
PVC	96.714 (50) 193.428 (100) 290.142 (150)	537.3	222	Observable color change. Increased modulus of elasticity. Decreased plasticity.
Velour	96.714 (50) 193.428 (100) 290.142 (150)	537.3	222	No measurable difference in color.
Powder coating paint	96.714 (50) 193.428 (100) 290.142 (150	537.3	222	No measurable difference in color.

TABLE 10.1: Material studies using 222 nm.

Material	Dose J/cm² (hours)	Irradiance µW/cm²	Wavelength (nm)	Findings
Harris et al., 2021—Boeing[17]				
Carpets (100% wool, 100% nylon)	108 (30)	1,000	222	Slight change in color.
Polycarbonate	108 (30)	1,000	222	Moderate yellowing. Noticeable color shifts may occur by 6 months.
Woven drapery (100% wool or 100% fire-retardant polyester)	108 (30)	1,000	222	Insignificant change to slight change in color.
Polyurethane paint	108 (30)	1,000	222	Insignificant change in color.
Decorative laminates	108 (30)	1,000	222	Moderate yellowing. Noticeable color shifts may occur by 24 months.
Polycarbonate with polysiloxane hard coat	108 (30)	1,000	222	No change in color.
Thermoplastic polyurethane	108 (30)	1,000	222	Severe yellowing. Noticeable color shifts may occur by 12 months.
Polyamide (PA12 Nylon)	108 (30)	1,000	222	Slight yellowing. Noticeable color shifts not expected before 5 years.
Vinyl floor mats	108 (30)	1,000	222	Insignificant color change.
Silicone rubber and foam	108 (30)	1,000	222	Slight to moderate yellowing. Noticeable color shifts may occur by 24 months.
Polycarbonate copolymer	108 (30)	1,000	222	Moderate yellowing. Noticeable color shifts may occur by 8 months.
Acrylonitrile Butadiene-Styrene/PVC (ABS/PVC)	108 (30)	1,000	222	Severe yellowing. Noticeable color shifts may occur by 6 months.
Polypropylene	108 (30)	1,000	222	Insignificant color change.
Leathers, genuine and artificial	108 (30)	1,000	222	Slight yellowing or fading.
Woven seat fabrics (100% wool, 92% wool/ 8% nylon blend)	108 (30)	1,000	222	Insignificant color change. Fading.
PVC/Acrylic blend (PVC/PMMA)	108 (30)	1,000	222	Severe yellowing. Noticeable color shifts may occur by 2 months.
Polycarbonate copolymer (Kydex FST)	108 (30)	1,000	222	Moderate yellowing.
Polyphenylsulphone (PPSU)	108 (30)	1,000	222	Severe yellowing. Noticeable color shifts may occur by 3 months.
Polyetherimide (PEI) copolymer	108 (30)	1,000	222	Moderate yellowing. Noticeable color shifts may occur by 24 months.
Polyether-ketoneketone/ polyvinyl fluoride (PEKK/PVF)	108 (30)	1,000	222	Insignificant color change.
Unsaturated polyester resin	108 (30)	1,000	222	Insignificant color change.

Further reading

- Evaluating the impact of 222 nm far-UVC radiation on the aesthetic and mechanical properties of materials used in public bus interiors
- Compatibility of Aircraft Interior Surfaces with 222 nm Far-UV Light Exposure

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Appendix B: impacts on non-human life

Summary

This section discusses the potential effects of far-UVC on other organisms that can be present in indoor public spaces. Most mammals are thought to have the same protections as humans (or more), with fur and lower height reducing their effective exposure to far-UVC from a ceiling-mounted lamp, and therefore this section primarily discusses arthropods and plants.

Initial analysis suggests that arthropods (such as insects and arachnids) will be protected from far-UVC exposure by their chitin- and protein-rich exoskeleton. The vast difference in chitin concentration and exoskeleton thickness across the arthropoda phylum makes it difficult to study each species individually. Instead, studies should evaluate far-UVC absorption/penetration into exoskeletons of various chitin concentrations. The impact of far-UVC on the insect eye is also unclear. Studies suggest that far-UVC in high intensities could have a negative effect on plants, but more research is needed. Similar to arthropods, the absorption/penetration of far-UVC into leaves, flowers, and other plant structures of varying thickness and color should be investigated.

Analysis

Arthropods

Arthropods are (among other criteria) characterized by their cuticle, or exoskeleton—a multi-layered structure secreted by the epidermis, comprising chitin and proteins[1]. Chitin is a fibrous substance composed of polysaccharides, and is one main component of the exoskeleton. Chitin concentration in arthropod exoskeletons can vary between 2% and 45%, with the remaining matter mostly being protein, phenolics, and minerals in some cases[1].

While we have a good understanding of the absorption of far-UVC by the human stratum corneum, we don't have a good understanding of the absorption of the arthropod cuticle, such as comparing a 1 µm-thick layer of stratum corneum in humans versus a 1 µm-thick layer of cuticle in arthropods. Cuticle thickness varies dramatically from just hundreds of nanometers in tiny ants to multiple millimeters in large crabs. Based on studies of ants, we can assume that cuticle thickness roughly correlates with body size. A 'typical' ant has a thorax cuticle thickness of over 10 µm, with large ant species reaching up to 100 µm, though the cuticle will be thinner over joints or more delicate areas[2]. Assuming arthropod cuticles have similar far-UVC absorption properties to the human stratum corneum, most larger insects (bees, flies, beetles) and spiders should experience similar significant attenuation of far-UVC in the cuticle, but this remains to be tested.

Effects on arthropod eyes are harder to assess. Arthropods have compound eyes comprising individual ommatidia capped by a transparent, chitinous corneal lens. The lens is typically of a few µm thick (depending on the species), but chitin content appears low (~20%), and the UV optical properties are poorly understood[3].

Mosquitos' cuticle thickness has been researched in the context of insecticide resistance and was found to be quite thin (1.5–3 µm in mosquito legs)[4]. Mosquitos' procuticles are less sclerotized than those of other arthropods, meaning that they have less crosslinking of structural proteins and chitin to increase stiffness and toughness. One therefore might expect significant amounts of far-UVC penetration into the underlying epidermis and nerves. As mosquitos are a known vector of disease, research on the impact of far-UVC on mosquitos can also help inform other disease vector mitigation strategies.

Plants

So far, the few studies on the effects of far-UVC on plants have found high sensitivity. One study of irradiated Arabidopsis seedlings observed leaf curling and bleaching 2–4 days after exposure to either 100 mJ/cm² from a KrCl* lamp with imperfect filtering, or 1 J/cm² from an unfiltered low-pressure mercury lamp. Plant homeostasis was severely impacted, as the guard cells that help regulate moisture and gas levels were damaged[5]. Generally, the outer layers of plants, including a wax cuticle and single-layered epidermis, have evolved to be thin and transparent to allow maximum light absorption. Furthermore, the composition and thickness of the waxy cuticle varies between species, making it difficult to draw broad comparisons. Two caveats include the lamp used and the dose intensity. The KrCl* lamp used was not perfectly filtered and had some emission above 235 nm. The plants received the far-UVC doses in a short timespan and had high far-UVC intensities during their seedling stage.

Houses and offices are two common places where far-UVC disinfection and house plants intersect. Houses and offices will have different duty cycles for disinfection depending on the occupancy and how often disinfection occurs. Furthermore, the location of the plants, and their

distance from the far-UVC lamps, will impact the effective doses the plants receive. The position of the plants in the office matters—plants are most likely positioned lower to the ground and off-axis from the lamps, suggesting that they will receive only a single-digit mJ/cm² of 222-nm far-UVC per day. However, environments with heavier duty cycles and more uniform distribution may lead to higher exposure and potential plant damage/discoloration. Furthermore, different parts of plants (petals, stems, leaves, etc.) may have different susceptibilities.

It seems as if far-UVC can harm plants, especially early in their life, so it may be wise to keep it out of greenhouses. Otherwise, it is relatively easy for individuals to make sure that their house or office plants are not getting too much irradiation. They can avoid placing them high up in the irradiation zone, or put them in a place that is shaded from the lamp.

We don't think far-UVC's impact on arthropods is a high priority for research, but any impact on plants indoors may limit enthusiasm for deployment. Simple experiments, such as including common house or office plants in real-world deployment studies, could reduce uncertainty or hesitation about deployment.

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