Review



# On the Use of Far-UVC Radiation for Disinfection in the Presence of People in Public Indoor Spaces: Literature Review and Critical Analysis

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*Review*

# On the Use of Far-UVC Radiation for Disinfection in the Presence of People in Public Indoor Spaces: Literature Review and Critical Analysis

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**Abstract**

Far-UVC radiation for disinfection in the presence of people in public indoor spaces through unshielded open radiation sources has been promoted for several years, claiming to be a simple solution to reduce infections from airborne pathogens such as bacteria and viruses. This literature review summarizes the existing research on the effectiveness of far-UVC radiation for inactivating pathogens, as well as potential risks to skin and eyes associated with exposure to far-UVC radiation. Further, it discusses radiation protection aspects of using far-UVC radiation in the presence of people, and addresses possible effects of far-UVC radiation on the human environment as well. The literature review shows that despite its antimicrobial and antiviral effectiveness, there is so far no sufficient evidence that far-UVC radiation can be used for disinfection in the presence of people in public indoor spaces without risks for humans and the environment. There are particular concerns about the safety of vulnerable groups such as children, the elderly and people with pre-existing medical conditions. The authors recommend further and extended studies in this field concerning potential risks of far-UVC radiation.

**Keywords:** far-UVC radiation; disinfection; public spaces; vulnerable populations; radiation protection

## 1. Introduction

Ultraviolet (UV) radiation is optical radiation with wavelengths between 100 nm and 400 nm. The wavelength region is further divided into UVA (315 nm to 400 nm), UVB (280 nm to 315 nm) and UVC (100 nm to 280 nm) (CIE, 2020). While around 95% of UVA and 5% of UVB radiation reaches the Earth's surface, solar UVC radiation is completely absorbed in the Earth's atmosphere (IARC, 2012). This means that humans and all life on Earth have evolved in the absence of UVC radiation (O'Hagan and Khazova, 2020). Therefore, the UVC radiation to which humans and the environment can be exposed comes exclusively from artificial sources of optical radiation.

High-energy UVC radiation is capable of killing or inactivating microorganisms and viruses and has been used for many decades to disinfect room air, water or solid surfaces. Although the respective action spectra for the inactivation vary depending on the pathogen, they have in common a pronounced maximum around 260 nm. UVC radiation of this wavelength is most effective at damaging the deoxyribonucleic acid (DNA) and ribonucleic acid (RNA) of microorganisms and viruses, leading to their eradication or inactivation (Voet et al., 1963). Thus, mercury vapour lamps with a primary emission at 254 nm are currently mainly used for disinfection purposes.

Since at these wavelengths not only microbial but also human DNA can be damaged, the UVC disinfection method poses also risks to humans. The risks of UV radiation include e.g., acute damage

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to the eyes, such as inflammation of the cornea (photokeratitis) or conjunctiva (photoconjunctivitis) and damage to the skin, such as erythema, polymorphic light dermatosis, phototoxic reactions with substances and skin cancer (Neale et al., 2023). UV radiation (UVA, UVB and UVC) has been classified as "carcinogenic to humans" by the International Agency for Research on Cancer (IARC) (El Ghissassi et al., 2009, IARC, 2012). That is why every effort has always been made to date to avoid exposing people to UVC radiation from artificial sources, except in the case of necessary medical treatments.

If human exposure cannot be ruled out, for example in a work process, exposure limits (ELs) must be adhered to. In the European Union these are the ELs of the EU Directive 2006/25/EC on the protection of workers against the risks arising from artificial optical radiation (EC, 2006). These ELs are based on the recommendations of the International Commission on Non-Ionizing Radiation Protection (ICNIRP). In its guidelines from 2004 (International Commission on Non-Ionizing Radiation, 2004), the ICNIRP published recommendations for ELs to protect the skin and eyes from harmful effects of incoherent UV radiation.

Research into the use of so-called far-UVC radiation (spectral range between 200 nm and 235 nm (Görlitz et al., 2024), no clear definition internationally) for disinfection has been ongoing for about ten years. Common sources of far-UVC radiation are filtered krypton chloride (KrCl) excimer lamps with a primary emission wavelength of 222 nm and low-residual emission of longer UV wavelengths. Furthermore, UVC light emitting diodes (LEDs) with emission wavelengths of around 233 nm are increasingly being used in medical applications. Particularly since the start of the SARS-CoV-2 pandemic, there have been discussions concerning the use of far-UVC radiation for disinfection even in the presence of people, such as in public indoor spaces through unshielded open sources of radiation, and this method is promoted as a simple solution for reducing infections from airborne pathogens indoors (Blatchley et al., 2023, Arzanani et al., 2025). Due to the very low penetration depth of far-UVC radiation into human tissue in healthy skin only the uppermost non-replicating skin layers will be affected and so it is assumed that no biologically important DNA damage to the skin would occur. Also, increased absorption of far-UVC radiation in the tear film covering the anterior segment of the eyeball would significantly reduce the risk of photokeratitis or photoconjunctivitis. Consequently, it is argued that no acute or long-term health consequences for humans are to be expected.

However, despite the beneficial effect of pathogen inactivation, the evidence remains much too insufficient for far-UVC radiation for disinfection to be used in the presence of people in public indoor spaces without health risks (Pereira et al., 2023, Lerner et al., 2025). In this descriptive literature review, we provide an overview of the existing literature, focusing primarily on original studies, their main findings and methodologies applied. The search was conducted in standard literature databases such as PubMed, BioRxiv, Europe PMC, Google Scholar, Science.gov and Semantic Scholar. The literature is summarized thematically with regard to the effects of far-UVC radiation on microorganisms and viruses, followed by what is known so far about the effects of far-UVC radiation on human skin and eyes. In addition, radiation protection aspects when using far-UVC in the presence of people in public indoor spaces will be discussed. Finally, possible effects of far-UVC on the human environment will be addressed. The review is based on the scientific justification for the recommendation of the German Commission on Radiological Protection (Strahlenschutzkommission, SSK) "Risks of using Far-UVC Radiation for Disinfection in the Presence of People" (SSK, 2023), in whose elaboration the authors participated.

## 2. Eradicating Microorganisms and Inactivating Viruses Using UVC Radiation

The aim of disinfection is to reduce the germ number in relation to microorganisms such as bacteria and fungi, and to reduce the infectivity of viruses. The antimicrobial effect of UVC radiation is well known and is used routinely for disinfecting surfaces in clean rooms or in sterile cabinets and for sterilising medical instruments (see Rudhart et al., 2022). Mercury vapour lamps with a maximum emission in the UVC range at 254 nm (labeled $UVC_{254}$ in this review) are usually used here. The eradication of microorganisms with UVC radiation is based mainly on inducing premutagenic

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damage in the DNA (e.g., cyclobutane pyrimidine dimers, CPDs). As the wavelength decreases, absorption by proteins increases; hence, in the spectral range of far-UVC radiation, protein damage also plays a role in eradication (Hessling et al., 2021). Due to the increased protein absorption and scattering in the biological material with decreasing wavelength, the penetration depth into biological tissue decreases (Zwicker et al., 2022, Zamudio Diaz et al., 2023). Therefore, especially in the case of far-UVC radiation, the exposure of the microorganisms can be attenuated, depending on the examination conditions (e.g., by using different culture media or wound secretions) and thus can deviate significantly from the measured radiant exposure (the product of the irradiance and the irradiation duration). The exposure conditions in different studies must therefore be considered.

The antimicrobial effect of far-UVC, especially for radiation of filtered KrCl excimer lamps at 222 nm (labeled $UVC_{222}$) and of UVC LEDs at 233 nm ($UVC_{233}$), was shown in several *in vitro* investigations. Narita et al. (2020) showed that though different common environmental and hospital bacterial strains reacted with different sensitivity, no more colony formation could be detected in bacteria after irradiation with a $UVC_{222}$ radiant exposure of 36 mJ $cm^{-2}$. $UVC_{254}$ was similarly effective. Using $UVC_{233}$ LED-irradiation, similar radiant exposure of 27 mJ $cm^{-2}$ to 40 mJ $cm^{-2}$ was needed for a germ reduction by
4-6 orders of magnitude in methicillin-resistant *Staphylococcus aureus* (MRSA) cultured on blood agar plates (Glaab et al., 2021). In this investigation $UVC_{254}$ was more effective inducing the same effect with a radiant exposure of 1.5 mJ $cm^{-2}$. In comparison with bacterial strains, endospores were more resistant to far-UVC radiation, with a $UVC_{222}$ radiant exposure of 96 mJ $cm^{-2}$ needed for inactivation (Narita et al., 2020). While radiant exposure of 96 mJ $cm^{-2}$ at 222 nm resulted in no further endospore colony formation, living endospores were still detected at 254 nm, meaning that endospores exhibited greater sensitivity to $UVC_{222}$ than to $UVC_{254}$ (Narita et al., 2020). Narita et al. (2020) further found that fungal strains also differed in terms of radiation sensitivity. In tests on the yeast *Candida albicans*, no living cells could be detected after irradiation with a radiant exposure of 72 mJ $cm^{-2}$ ($UVC_{222}$ or $UVC_{254}$). Very similar results were shown in *Candida albicans* and *Candida parapsilosis* with nearly no colony forming after irradiation with a radiant exposure of 40 mJ $cm^{-2}$ to 80 mJ $cm^{-2}$ ($UVC_{222}$, $UVC_{233}$ and $UVC_{254}$) (Schleusener et al., 2023). Much higher radiant exposure may be needed to completely eradicate fungal spores. E.g., this effect was only achieved in *Aspergillus niger* spores with $UVC_{222}$ and $UVC_{254}$ radiant exposure of 500 mJ $cm^{-2}$ and 250 mJ $cm^{-2}$, respectively, while *Trichophyton rubrum* was eradicated at a radiant exposure of just 72 mJ $cm^{-2}$ and 36 mJ $cm^{-2}$, respectively. In contrast to $UVC_{254}$, irradiation with $UVC_{222}$ had nearly no effect on the growth of hyphae from *Aspergillus niger* and *Trichophyton rubrum* (Narita et al., 2020).

The sensitivity of virus strains to $UVC_{222}$ radiation differed quite considerably. Whereas feline calicivirus (FCV, non-enveloped virus) was inhibited but not completely eradicated by a radiant exposure of 36 mJ $cm^{-2}$ ($UVC_{222}$ and $UVC_{254}$), no pathogenic viral effect was detected after irradiation of *Influenza* virus A (enveloped virus) with a radiant exposure of just 6 mJ $cm^{-2}$ (Narita et al., 2020). Even lower $UVC_{222}$ radiant exposure of 1.7 mJ $cm^{-2}$ and 1.2 mJ $cm^{-2}$ was needed for inactivation of 99,9% of two human coronavirus species (alpha HCoV-229E and beta HCoV-OC43) (Buonanno et al., 2020, Buonanno et al., 2021b). A comprehensive study including four virus species (two enveloped and two non-enveloped) characterized by different genome compositions (single- and double-stranded RNA and DNA) showed significant inactivation by $UVC_{222}$ being more effective as compared to $UVC_{254}$ (Monika et al., 2025b). Furthermore, it was shown that DNA viruses are more sensitive than RNA viruses. Different genome size, number of reaction sites for UVC-induced photoproduct formation and susceptibility to $UVC_{222}$ damaged proteins of the virus strains are discussed as the reason for the different sensitivity to UVC radiation. In two RNA virus species (non-enveloped single-stranded RNA and enveloped double-stranded RNA), Monika et al. (2025a) observed a higher sensitivity of the enveloped virus to $UVC_{222}$ in comparison to the non-enveloped virus. The use of artificial human saliva as medium for the sample aerosols enhanced this sensitivity to $UVC_{222}$ especially for the enveloped virus. The data indicate that not only DNA damage, but the damage of proteins (e.g., of the virus envelope) are involved in the disinfection mechanism of $UVC_{222}$.

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The authors stated that the higher sensitivity of both virus species to $UVC_{254}$ irradiation in artificial human saliva compared to $UVC_{222}$ may be attributed to viral genome type, virus structural characteristics, and vector media type. Lu et al. (2025) investigated the inactivation mechanisms and effectiveness of different UVC wavelengths against the airborne human coronavirus OC43, which is an enveloped RNA virus. They demonstrated that inactivation with $UVC_{254}$ and $UVC_{263}$ primarily involves genome damage, whereas $UVC_{222}$ damages the genome, proteins (particularly the nucleocapsid (N) and spike (S) proteins, leading to compromised capsid integrity and reduced binding ability to host cells), and lipid components of the viral envelope.

To investigate the bactericidal effect of $UVC_{222}$ and $UVC_{233}$ in relation to the risk of infection in skin wounds, several studies were worked out in situ and in vivo. In a mouse model a germ reduction of two powers of ten was observed after irradiation with a $UVC_{222}$ radiant exposure of 75 mJ $cm^{-2}$ with a significant number of non-inactivated germs, probably attributable to shading in the skin furrows or hair follicles (Narita et al., 2018a, Narita et al., 2018b). Irradiation of infected wounds also reduced the germ count. However, inhibition was lower than in intact skin. The diminished effect is likely due to the absorbing effect of the wound exudate, which has a high protein content. Above a radiant exposure of 150 mJ $cm^{-2}$, reduction in the germ count was observed up to 12 days after irradiation, i.e., during wound healing. $UVC_{254}$ was often somewhat more effective than $UVC_{222}$; however, after irradiation with $UVC_{254}$, in addition to a reduced germ count, DNA damage (measured based on CPD formation) was observed in the cells of the wound bed, an effect that was not achieved after irradiation with $UVC_{222}$. In another setting, MRSA-infected areas of skin in SKH1 mice were studied after irradiation with $UVC_{222}$ and $UVC_{254}$ (Ponnaiya et al., 2018). It should be noted, however, that in this case the bacteria were applied to a film, which prevented penetration of the epidermis prior to irradiation. An incision was made in the infected areas and immediately closed with a suture. A significant reduction in germ count was also observed here on day 2 and day 7 after both forms of irradiation. In a clinical study involving 20 volunteers, a significant germ reduction was observed after irradiation of healthy skin with a $UVC_{222}$ radiant exposure of 500 mJ $cm^{-2}$ (Fukui et al., 2020).

As already described, the inactivating effect of $UVC_{222}$ is inhibited by protein rich media, like e.g., wound exudate. Using longer wavelengths like $UVC_{233}$ might overcome this problem but bear the risk of damage induction in underlying skin cells. Detailed investigations showed that sweat, albumin and wound exudate, which were used as a medium while irradiating the microorganisms, greatly reduce the inactivating effect of $UVC_{222}$ compared to $UVC_{254}$ (Zwicker et al., 2022, Sicher et al., 2024, Matsuura et al., 2022). If, as usual, saline was used as a medium, $UVC_{254}$ was only slightly more effective. The use of $UVC_{233}$ demonstrated that sweat hardly reduced the inactivating effect at all. According to an increase of salts and proteins contained in a matrix, albumin, artificial wound exudate, mucin and artificial saliva decreased the germ reduction to a higher extent. However, irradiation of skin biopsies with $UVC_{233}$ induced DNA damage in cells in the upper epidermal layers to a greater extent than under $UVC_{222}$ (Zwicker et al., 2022, Welch et al., 2026) (see next chapter). A summary of the aforementioned studies on the effect of UVC radiation on microorganisms and viruses is provided in Table 1. Recent publications on the inactivation mechanism of $UVC_{222}$ (Fukushi et al., 2025, Narita et al., 2025) describe a clear production of reactive oxygen species (ROS) and the inhibition of photoreactivation (an important photolyase-dependent DNA repair mechanism in bacteria) after irradiation of bacteria with $UVC_{222}$. Since these effects were not observed after irradiation with $UVC_{254}$, it is indicated that the germicidal effect of $UVC_{222}$ may not only involve CPD induction but also ROS induced protein damage and inhibition of (repair) enzymes.

The data described above from *in vitro* and *in vivo* studies proves the germicidal effect of $UVC_{222}$, which is certainly comparable with that of $UVC_{254}$. This is also confirmed in a review by Hessling et al. (2021), which considers the publications on the use of far-UVC radiation for disinfection from the last 100 years. According to Hessling et al., the publications suggest that a $UVC_{222}$ radiant exposure of 100 mJ $cm^{-2}$ reduces all pathogens by several orders of magnitude. It should be noted at this point that this radiant exposure is roughly four times higher than the EL of 22.7 mJ $cm^{-2}$ at 222 nm aimed at protecting the eyes and skin from actinic UV hazards (International Commission on Non-Ionizing

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Radiation, 2004). It is also clear, however, that there are considerable differences in the radiation sensitivity of microorganisms whose inactivation is desired. These differences can depend on the size of the genome but also, e.g., in the case of viruses, on the existence of a viral envelope (enveloped and non-enveloped viruses). The pigmentation of the microorganisms, such as in the *Aspergillus niger* fungus, may also be the reason for reduced sensitivity to UVC radiation. As $UVC_{222}$ is absorbed and dispersed to a considerable extent by proteins and other biological substances, these effects are particularly pronounced when using $UVC_{222}$. The very different radiant exposure required to effectively inactivate microorganisms in the described in vitro and in vivo settings (Hessling et al., 2021, Hessling et al., 2022) is probably due to the same reason.

In summary, the data shows that the inactivating effect of $UVC_{222}$ is in principle comparable to the effect of $UVC_{254}$. When applied, however, it must be considered that $UVC_{222}$ is very strongly absorbed and scattered by proteins and other biological substances. This can severely restrict the inactivating effect and is probably also the reason for a partly very variable radiation sensitivity in the microorganisms that are to be inactivated. There is a possibility, when using far-UVC radiation in public indoor spaces, that the microbiome of the human skin and the eye surface could also be affected. Data on the use of $UVC_{233}$ demonstrates a better penetration depth due to the lower level of absorption by proteins, which is associated with increased DNA damage in epidermal cells beneath the corneal layer compared to $UVC_{222}$. In principle, prior to the routine use of far-UVC radiation, e.g., for disinfection prior to medical interventions, a benefit-risk analysis should also be carried out, including potentially vulnerable individuals in particular. Also, to be considered here is that chemical disinfectants have a good effect in surface disinfection.

**Table 1.** Overview of the studies into the effect of UVC radiation on microorganisms and viruses. The values specified for radiant exposure refer to the conditions described in the references. The irradiance and irradiation duration are usually not stated therein.

| Publication | Study object | Medium in which exposure occurred | UV source / wavelength (filtering, if applicable) | Radiant exposure | Effect |
|---|---|---|---|---|---|
| Narita et al. (2020)† | Bacterial strains *Staphylococcus aureus, Pseudomonas aeruginosa, Echerichia coli, Campylobacter jejuni, Salmonella enterica, Bacillus cereus, Clostridium sporogenes* | 0.5 ml suspension in a 35 mm culture dish | KrCl excimer lamp / 222 nm, filtered | 36 mJ $cm^{-2}$ | No further colony formation in any of the bacterial strains detected; sensitivity of the bacterial strains varied |
| | | | Mercury vapour lamp / 254 nm | | Equally effective as 222 nm; a few colonies were still detectable for *Clostridium sporogenes* only |
| | Endospores of | | KrCl excimer lamp / 222 nm, filtered | 96 mJ $cm^{-2}$ | No further colony formation |

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| | *Bacillus cereus* and *Clostridium sporogenes* | | Mercury vapour lamp / 254 nm | | Living endospores detected |
| | Endospores of *Clostridiodes difficile* | | KrCl excimer lamp / 222 nm, filtered | 50 mJ $cm^{-2}$ | No further colony formation |
| | | | Mercury vapour lamp / 254 nm | 30 mJ $cm^{-2}$ | Reduction in living endospores reached a plateau |
| | Yeast *Candida albicans* | | KrCl excimer lamp / 222 nm, filtered | 72 mJ $cm^{-2}$ | No further living cells |
| | | | Mercury vapour lamp / 254 nm | | |
| | Fungal spores *Aspergillus niger* | | KrCl excimer lamp / 222 nm, filtered | 500 mJ $cm^{-2}$ | Complete eradication |
| | | | Mercury vapour lamp / 254 nm | 250 mJ $cm^{-2}$ | Complete eradication |
| | Fungal spores *Trichophyton rubrum* | | KrCl excimer lamp / 222 nm, filtered | 72 mJ $cm^{-2}$ | Complete eradication |
| | | | Mercury vapour lamp / 254 nm | 36 mJ $cm^{-2}$ | Complete eradication |
| | Fungal hyphae *Aspergillus niger* | | KrCl excimer lamp / 222 nm, filtered | 1000 mJ $cm^{-2}$ | Very small effect on the growth of *Aspergillus niger* |
| | | | Mercury vapour lamp / 254 nm | 250 mJ $cm^{-2}$ | Growth severely restricted |
| | Fungal hyphae *Trichophyton rubrum* | | KrCl excimer lamp / 222 nm, filtered | 250 mJ $cm^{-2}$ | Very small effect on the growth of *Trichophyton rubrum* |
| | | | Mercury vapour lamp / 254 nm | 72 mJ $cm^{-2}$ | Growth severely restricted |
| | Virus strain Feline calicivirus | 0.2 ml suspension in | KrCl excimer lamp / 222 nm, filtered | 36 mJ $cm^{-2}$ | Inhibited but not completely eradicated |

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| | (non-enveloped virus) | a 35 mm culture dish | Mercury vapour lamp / 254 nm | | Equally effective as 222 nm |
| | Virus strain *Influenza* virus A (enveloped virus) | | KrCl excimer lamp / 222 nm, filtered | 6 mJ $cm^{-2}$ | No further cytopathogenic effect detectable |
| | | | Mercury vapour lamp / 254 nm | | Equally effective as 222 nm |
| Glaab et al. (2021) | Methicillin-resistant *Staphylococcus aureus* (MRSA) | Blood agar plates | LED array / 233 nm, full width at half maximum (FWHM) 12 nm, filtered above 240 nm | 27 mJ $cm^{-2}$ to 40 mJ $cm^{-2}$ | Higher bacteria concentrations nearly completely inactivated (reduction by 4-6 orders of magnitude) |
| | | | Mercury vapour lamp / 254 nm | 1.5 mJ $cm^{-2}$ | The same effect as $UVC_{233}$ (reduction by 6 orders of magnitude) |
| Schleusener et al. (2023) | *Candida albicans* | Kimmig agar plates | KrCl excimer lamp / 222 nm, filtered | ≥ 20 mJ $cm^{-2}$ | Strong reduction of colony growth |
| | | | LED array / 233 nm, FWHM 12 nm, filtered above 240 nm | 40 mJ $cm^{-2}$ | Colony growth reduced by more than 98% |
| | | | Mercury vapour lamp / 254 nm | 80 mJ $cm^{-2}$ | Complete eradication |
| | *Candida parapsilosis* | | KrCl excimer lamp / 222 nm, filtered | ≥ 20 mJ $cm^{-2}$ | Strong reduction in colony growth |
| | | | LED array / 233 nm, FWHM 12 nm, filtered above 240 nm | 40 mJ $cm^{-2}$ | Colony growth reduced by more than 98% |
| | | | Mercury vapour lamp / 254 nm | 40 mJ $cm^{-2}$ | Complete eradication |
| Buonanno et al. (2020), | Human alpha coronavirus 229E | Aerosol | | 1.7 mJ $cm^{-2}$ | 99.9% virus inactivation |

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| Buonanno et al. (2021b) | | | KrCl excimer lamp / 222 nm, filtered | (irradiance 100 μW cm$^{-2}$) | |
| | Human beta coronavirus OC43 | | | 1.2 mJ cm$^{-2}$ (irradiance 100 μW cm$^{-2}$) | 99.9% virus inactivation |
| Monika et al. (2025b) | Bacteriophage MS2, a single-stranded RNA non-enveloped virus | Virus droplets | KrCl excimer lamp / 222 nm, filtered | Up to 12 mJ cm$^{-2}$ (irradiance 0.0889 mW cm$^{-2}$) | Sensitivity measured as inactivation rate constant k. A greater k value indicates a higher sensitivity of the tested virus to the applied UVC radiation. k = (1.343 ± 0.070) cm$^2$ mJ$^{-1}$ |
| | | | Mercury vapour lamp / 254 nm | Up to 16 mJ cm$^{-2}$ (irradiance 0.387 mW cm$^{-2}$) | k = (0.811 ± 0.031) cm$^2$ mJ$^{-1}$ |
| | Bacteriophage Phi6, a segmented double-stranded RNA enveloped virus | | KrCl excimer lamp / 222 nm, filtered | Up to 8 mJ cm$^{-2}$ (irradiance 0.0889 mW cm$^{-2}$) | k = (1.604 ± 0.075) cm$^2$ mJ$^{-1}$ |
| | | | Mercury vapour lamp / 254 nm | Up to 40 mJ cm$^{-2}$ (irradiance 0.387 mW cm$^{-2}$) | k = (0.207 ± 0.013) cm$^2$ mJ$^{-1}$ |
| | Bacteriophage M13, a single-stranded DNA filamentous non-enveloped virus | | KrCl excimer lamp / 222 nm, filtered | Up to 8 mJ cm$^{-2}$ (irradiance 0.0889 mW cm$^{-2}$ | k = (2.061 ± 0.087) cm$^2$ mJ$^{-1}$ |

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| | | | Mercury vapour lamp / 254 nm | Up to 10 mJ cm$^{-2}$ (irradiance 0.387 mW cm$^{-2}$) | k = (1.494 ± 0.061) cm$^2$ mJ$^{-1}$ |
| | Bacteriophage T4, a double-stranded DNA non-enveloped and tailed virus | | KrCl excimer lamp / 222 nm, filtered | Up to 4 mJ cm$^{-2}$ (irradiance 0.0889 mW cm$^{-2}$ | k = (3.672 ± 0.313) cm$^2$ mJ$^{-1}$ |
| | | | Mercury vapour lamp / 254 nm | Up to 4,3 mJ cm$^{-2}$ (irradiance 0.387 mW cm$^{-2}$) | k = (2.946 ± 0.214) cm$^2$ mJ$^{-1}$ |
| Narita et al. (2018a), Narita et al. (2018b) | In vivo study in BALB/c mice with MRSA infection on healthy skin | Mouse skin, in vivo | KrCl excimer lamp / 222 nm, filtered | 75 mJ cm$^{-2}$ | Germ reduction by 2 log colony-forming units (CFU), but 2.5 log CFU remained on the skin |
| | In vivo study in BALB/c mice with MRSA infection in wounds | | KrCl excimer lamp / 222 nm, filtered | 75 mJ cm$^{-2}$ | Germ reduction by 0.9 log CFU |
| | | | KrCl excimer lamp / 222 nm, filtered | 1500 mJ cm$^{-2}$ | Germ reduction of 1.6 log CFU |
| | | | Mercury vapour lamp / 254 nm | | Slightly more effective than 222 nm, but also DNA damage in the cells of the wound bed |
| Ponnaiya et al. (2018)† | MRSA-infected areas of skin in SKH1 mice | Mouse skin, in vivo | KrCl excimer lamp / 222 nm, filtered | 40 mJ cm$^{-2}$ and 300 mJ cm$^{-2}$ | Significant germ count reduction on day 2 and day 7 post irradiation without skin damage |
| | | | Mercury vapour lamp / 254 nm | | Significant germ reduction on day 2 |

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| | | | | | and day 7 post irradiation |
| Fukui et al. (2020)† | Study subjects, one area on the back | Human skin, in vivo | KrCl excimer lamp / 222 nm, filtered | 500 mJ $cm^{-2}$ | The mean colony count dropped from 7.21 ± 7.48 before irradiation to 0.05 ± 0.23 five minutes post irradiation and 0.79 ± 2.53 thirty minutes post irradiation; significantly increased CPD values in irradiated skin biopsies |
| Zwicker et al. (2022) | Methicillin-resistant *Staphylococcus aureus* DSM 11822 | Sodium chloride solution | KrCl excimer lamp / 222 nm, filtered above 230 nm | 20 mJ $cm^{-2}$ (irradiance 3.34 mW $cm^{-2}$) | Log10 reduction of 4.4 |
| | | | | 40 mJ $cm^{-2}$ (irradiance 3.34 mW $cm^{-2}$) | Slight increase in bacterial reduction |
| | | Artificial sweat pH 8.4, albumin 0.3%, artificial wound exudate, mucin 0.5% | | 20 mJ $cm^{-2}$ (irradiance 3.34 mW $cm^{-2}$) | Log10 reduction between 0.64 and 1.59 |
| | | | | 40 mJ $cm^{-2}$ (irradiance 3.34 mW $cm^{-2}$) | Slight increase in bacterial reduction |
| | | Sodium chloride solution | LED array / 233 nm, FWHM 12 nm, filtered above 240 nm | 40 mJ $cm^{-2}$ (irradiance 0.041 mW $cm^{-2}$) | Log10 reduction of 5.68 ± 0.35 |
| | | Artificial sweat pH 8.4 | | | Log10 reduction of 5.7 ± 0.03 |
| | | Albumin 0.3% | | | Log10 reduction of 2.50 ± 0.73 |

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| | | Artificial wound exudate | | | Log10 reduction of 2.29 ± 0.81 |
| | | Mucin 0.5% | | | Log10 reduction of 1.59 ± 0.35 |
| | | Artificial saliva | | | Log10 reduction of 1.13 ± 0.42 |
| | | Sodium chloride solution, artificial sweat pH 8.4, albumin 0.3%, artificial wound exudate | Mercury vapour lamp / 254 nm | 40 mJ $cm^{-2}$ (irradiance 0.29 mW $cm^{-2}$) | Strong reduction of viable microorganisms, log10 reduction between 5.84 and 6.37 |
| | | Mucin 0.5% | | | Log10 reduction of 1.45 |
| Sicher et al. (2024) | *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Staphylococcus haemolyticus*, *Staphylococcus lugdunensis*, *Klebsiella pneumonia*, *Pseudomonas aeruginosa* | Sodium chloride solution | LED array / 233 nm, FWHM 12 nm | 40 mJ $cm^{-2}$ (irradiance 44 µW $cm^{-2}$) | Reduction by more than 5 log for most of the bacteria |
| | | Artificial sweat, albumin solution 0,3%, artificial wound exudate | | | Reduction by more than 3 log for most of the bacteria |
| | | Mucin solution, artificial saliva | | | Reduction less than 2 log |

† The authors declare a conflict of interest.

## 3. Potential Effects of Far-UVC Radiation on the Eyes and the Skin

The depth of penetration into the skin and eye is of particular importance in terms of the health-relevant effects of UV radiation (see Figures 1 and 2). The potential effects of far-UVC radiation on the eyes and skin are discussed in the following four subsections.

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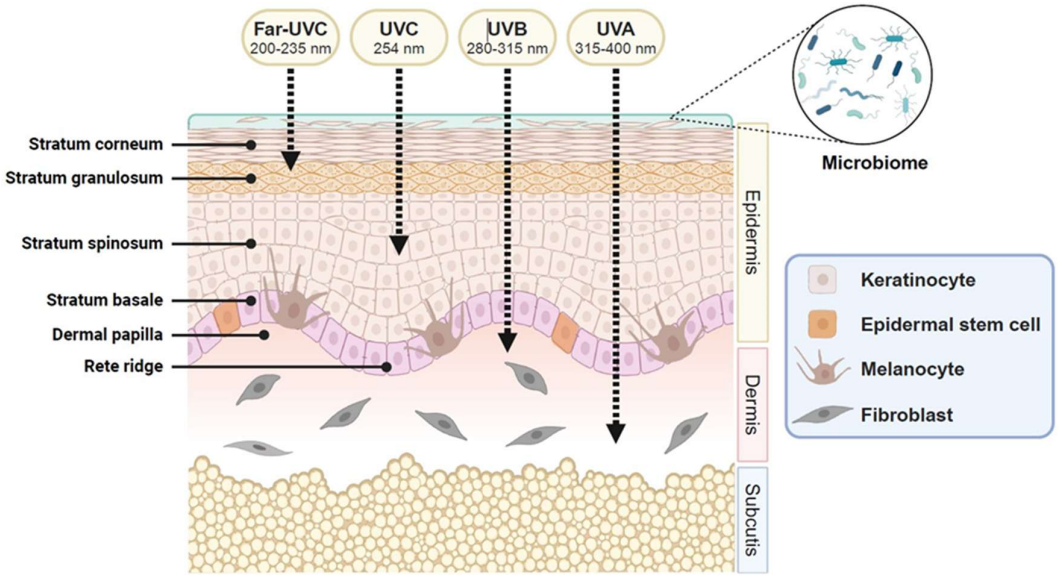


**Figure 1.** Diagram of the penetration depths of optical radiation in the human skin at different UV wavelengths (created with BioRender).

It should also be noted that the outermost protective barrier of the skin and eye surface is formed by their microbiota, which are composed of specific resident microorganisms that appear in a microbiome (Masoudi, 2022, Wang et al., 2022, Azzimonti et al., 2023). The protective barriers provide protection against potentially damaging environmental factors and are exposed directly, on the other hand, e.g., to UV radiation. In response to such exposure the microbiota can also change (Burns et al., 2019, Patra et al., 2019).

### *3.1. Evidence from Cell Studies*

Shortwave high-energy UVC radiation has a lower penetration depth than UVB radiation but greater potential to induce premutagenic lesions such as cyclobutane pyrimidine dimers (CPDs) and pyrimidine-pyrimidone (6-4) photoproducts (6-4PPs). The UV action spectrum of the DNA photoproduct CPD is e.g., similar to the action spectrum for erythema, the action spectrum of melanogenesis in human skin, and non-melanoma skin cancer (CIE, 2006, Young et al., 1998, Parrish et al., 1982). In DNA, the maximum energy absorption (100%) is within the UVC spectral range at 260 nm, whereas in the UVB spectral range 20% of the radiation energy is absorbed at 290 nm and only 3% at 300 nm (Cadet et al., 2015). The UV-induced DNA damage is the basis for the inactivating effect on microorganisms. Furthermore, skin carcinogenesis is primarily attributed to UV-induced DNA damage as premutagenic lesions.

At the cellular level, far-UVC radiation is significantly attenuated by absorption in the cytoplasm before it reaches the cell nucleus (Green et al., 1987). In a study by Ong et al. (2022), monolayer cell cultures (keratinocytes and retinal cells) were exposed to UVC radiation of different wavelengths (222 nm, 254 nm and 277 nm) and identical irradiance. After 60 minutes of irradiation with $UVC_{254}$ and $UVC_{277}$, no cell growth was noted and apoptosis markers as well as the induction of CPDs were detected. Cellular effects were also observed after irradiation with $UVC_{222}$: Cell growth was still significant but decreased compared to the non-irradiated control. No apoptosis markers and no CPDs were detected, however, a number of γH2AX-positive cells was observed, suggesting genotoxic stress. The data shows rather convincingly that $UVC_{222}$ can also penetrate cells and trigger a cellular radiation response if, as is the case here, there is no protective effect from a corneal layer (skin) (de Feraudy et al., 2010, Stope, 2021). It is known that, depending on the radiant exposure, UV radiation leads to the formation of reactive oxygen species (ROS) in cells which, for their part, can cause oxidative damage to proteins, DNA and lipids (Bose et al., 2020, de Jager et al., 2017, Volatier et al.,

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2022). In skin biopsies and skin models ROS formation after irradiation with far-UVC was shown (Tavares et al., 2023, Zwicker et al., 2022). Nishikawa et al. (2024) investigated the cytotoxic effect of $UVC_{222}$ and $UVC_{254}$ in a colon carcinoma cell line. They were able to show that after exposure to a cytotoxic equidose (30 mJ $cm^{-2}$), more CPDs were induced after $UVC_{254}$ than after $UVC_{222}$. At the same time, damage to the cell membrane was observed, which only occurred after irradiation with $UVC_{222}$. The authors conclude that the cytotoxic effect of far-UVC radiation is based not only on DNA damage but also on damage to the membrane.

In a study by Valanciute et al. (2025), primary human tracheal tissue and cells exposed to a continuous wave of $UVC_{222}$ and pulsed $UVC_{206}$ and $UVC_{222}$ at radiant exposures of 5 mJ $cm^{-2}$, 25 mJ $cm^{-2}$ and 50 mJ $cm^{-2}$, exhibited increased γH2AX signalling as a marker of DNA damage. The continuous wave and pulsed $UVC_{222}$ caused the formation of 6-4PPs. Significant decrease in cell viability 12- and 48-hours post-irradiation was observed, with no recovery, indicating continuous cell death beyond initial exposure.

Liu et al. (2025) conducted a systematic review and meta-analysis of studies evaluating the disinfection efficacy of $UVC_{222}$ and $UVC_{254}$, as well as studies exploring the safety of $UVC_{222}$ and $UVC_{254}$ for mammalian cells. The results show that the disinfection efficacy of $UVC_{222}$ is 1.382 times greater than that of $UVC_{254}$, and that the proportion of normal cells producing CPDs is 21.1% lower for $UVC_{222}$ than for $UVC_{254}$ at the same radiant exposure. The authors stated limitations concerning the great heterogeneity of the reviewed study data because they did not assess potential sources of the heterogeneity (like speed of reaction, duration of exposure distance from the radiation source, environmental conditions, etc.). They furthermore state that their study lacks standardized testing of conditions for comparing the efficacy of different UV wavelengths.

### *3.2. Skin*

The skin is the largest organ of the human body and due to its structure (see Figure 1) acts as a physical barrier, protecting the body against potentially damaging environmental factors. However, the skin itself (as an organ) can also be affected by these harmful influences (e.g., UV radiation). The thickness of the skin and its individual layers varies considerably and depends, among other factors, on the localization. The epidermis (50 µm -150 µm), as the uppermost layer of the skin, is a multilayered squamous epithelium consisting primarily of keratinocytes. Starting from the basal cell layer, keratinocytes differentiate to form the prickle cell layer (stratum spinosum), the granular layer (stratum granulosum) and, as non-nucleated cells, the corneal layer (stratum corneum, 8 µm–20 µm), being ultimately shed as scaly skin. In this way, the epidermis is renewed approximately every four weeks. It is assumed that the original cells for skin cancer are localised in the basal cell layer and in the hair follicles (Blanpain, 2013, Gaggioli and Sahai, 2007, Garcia et al., 2011). UV radiation of longer wavelengths, such as UVB and UVA radiation, penetrates the skin more deeply (37% of UV radiation at 300 nm and 340 nm reaches a depth of 15 µm and 54 µm, respectively) than UVC radiation (Finlayson et al., 2022). Measurements have shown that 82% of the energy of $UVC_{254}$ radiation is absorbed by 20 µm of the corneal layer (Bruls et al., 1984); however, DNA damage occurs in epidermal cells at this wavelength (Starzonek et al., 2019). In the far-UVC spectral range, the corneal layer is hardly penetrated at all because the radiation energy is heavily absorbed by proteins, especially peptide bonds (Preiss and Setlow, 1956, Zamudio Diaz et al., 2023).

Several experimental in vitro and in vivo studies have been conducted on the effect of far-UVC radiation on human skin (e.g., Sicher et al., 2024, Tavares et al., 2023, Zwicker et al., 2022, Buonanno et al., 2017, Buonanno et al., 2020, Buonanno et al., 2021b, Fukui et al., 2020, Hickerson et al., 2021) and the skin of hairless mice and porcine skin (e.g., Yamano et al., 2020, Forbes et al., 2021, Glaab et al., 2021, Gutierrez-Bayona et al., 2025, Buonanno et al., 2016). Although in some studies much higher radiant exposure was used than would be permissible for human exposure, the vast majority of the studies revealed no measurable negative effects and no evidence of the induction of skin cancer (Welch et al., 2023, Blatchley et al., 2023).

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In a study of Woods et al. (2015), signs of skin redness and CPDs were found in two of four healthy subjects with skin phototypes I and II. An unfiltered UVC lamp with a peak wavelength of 222 nm was used here in the radiant exposure range below the threshold for the bacteriostatic effect. The UV emission portion in the wavelength range above 250 nm was 3%. Any damage to the cells in the basal cell layer is attributed by the authors to the longwave UVC emission components when basal cells lying above the tips of the dermal papillae (see Figure 1) were not shielded sufficiently by the tissue above. This study was repeated with similar and higher radiant exposure by Fukui et al. (2020) using optically filtered radiation from excimer lamps ($UVC_{222}$) with no UVC emissions above 230 nm. No signs of erythema could be observed here. However, a small but significant increase in CPDs was found, compared to non-irradiated skin. Eadie et al. (2021) also found no cutaneous effect from exposing human skin type II on the forearm to radiation with a cumulative $UVC_{222}$ radiant exposure of 1500 mJ $cm^{-2}$. Only a radiant exposure of 6000 mJ $cm^{-2}$ resulted in a weak and transient yellowish discolouration of the exposed top layer of the epidermis. In this case, no erythema was noted at exposures up to 18000 mJ $cm^{-2}$. CPDs were not investigated. Using a human skin model (EpiDerm), Buonanno et al. (2021a) found no significant increase in premutagens (CPDs) or other DNA lesions after applying 125 mJ $cm^{-2}$ of filtered far-UVC radiation; only at an exposure of 500 mJ $cm^{-2}$ a slight increase in CPDs was observed. Yamano et al. (2020) applied a $UVC_{222}$ radiant exposure of 500 mJ $cm^{-2}$ to two genotypes of hairless mice that are highly susceptible to carcinogenesis. Weak evidence of increased premutagenic lesions was found in these mice, but only in the uppermost cells of the epidermis. This result has been verified by in vivo studies in human skin (Hickerson et al., 2021). Zwicker et al. (2022) demonstrated that irradiation of excised human skin with a $UVC_{233}$ radiant exposure of 40 mJ $cm^{-2}$ resulted in CPD formation directly beneath the stratum corneum. No more CPDs could be detected 24 hours after irradiation. Attention was drawn to the fact that acute damage to the superficial layers of the epidermis plays no role in the delayed effects, as these cells die within a few days. Welch et al. (2026) exposed human skin biopsies of two donors undergoing plastic surgery to $UVC_{222}$, $UVC_{233}$ and $UVC_{254}$ radiation. After a radiant exposure of 100 mJ $cm^{-2}$ $UVC_{233}$ and $UVC_{254}$, 8% and 45% of the epidermal cells were positive for CPDs, respectively. Localisation of the damaged cells within the epidermis was not shown. No CPDs were observed in the samples exposed to100 mJ $cm^{-2}$ $UVC_{222}$. In a recent study by Tavares et al. (2023), examination of reconstructed human skin (RHS) after a single $UVC_{222}$ radiant exposure of 500 mJ $cm^{-2}$ and 1500 mJ $cm^{-2}$ revealed no significant induction of CPDs or 6-4PP photoproducts. However, thickening of the epidermis was observed 48 hours after irradiation with the highest radiant exposure. When examining the proteome, a pathway analysis showed no changes for most pathways compared to the untreated control. There were only indications of a possible inhibition of skin regeneration processes after irradiation with $UVC_{222}$. However, in the RHS specimens irradiated with $UVC_{222}$, increased expression of matrix metalloproteinases MMP-1 and MMP-9, which are associated with UV-induced skin ageing, was noted. In parallel, expression of the inhibitor of these metalloproteinases (TIMP-1) was found to be increased. Together with induction of ROS, which was also detected, radiation effects induced by $UVC_{222}$ were then observed in RHS. In mice subjected to chronic irradiation, there were again no radiation-induced structural histological changes after 40 days of daily exposure to eight hours of radiation ($UVC_{222}$, 25 mJ $cm^{-2}$). In the study of matrix metalloproteinases, only the expression of MMP-9 was found to be slightly increased. Neither acute nor chronic irradiation with $UVC_{222}$ resulted in DNA damage.

The results of the first study for estimating the risk of skin cancer and non-cancerous skin lesions from long-term exposure (15.2 months) to $UVC_{222}$ revealed no signs of an increased risk of skin cancer in hairless mice (Welch et al., 2023). The 96 animals kept in groups were exposed to eight hours of inhomogeneous irradiances daily, five days per week ($UVC_{222}$ radiant exposure of 55 mJ $cm^{-2}$ to 400 mJ $cm^{-2}$), for 66 weeks. A film dosimeter (OrthoChromic Film OC-1) was used in the study to measure the radiant exposure. The authors also found no evidence of an increase in non-cancerous skin lesions from far-UVC radiation.

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The study of Zamudio Diaz et al. (2025a) aimed at evaluating the skin safety of $UVC_{233}$ for potential applications in antisepsis and public area decontamination. A biocidal dose of $UVC_{233}$ induced superficial DNA damage, showing lower damage than that induced by suberythemal UVB exposure, a recognized safe radiant exposure for human skin. Older and dark-skinned participants exhibited higher residual DNA damage 24 hour-post-exposure compared to younger and lighter-skinned individuals, though nearly complete repair was observed after seven days. Repeated exposures, up to cumulative radiant exposures of 120 mJ $cm^{-2}$ and 240 mJ $cm^{-2}$, did not induce significant changes on skin pigmentation, antioxidant activity, or immune response. However, DNA damage accumulation suggested limitations in repair mechanisms over 24 hours. According to the authors, these results confirm the safety of a single exposure to $UVC_{233}$, but highlight potential risks with repeated applications, emphasizing the need for careful radiant exposure regulation and further *in vivo* studies, including well-established treatments at $UVC_{222}$.

Since effects in the skin associated with exposure to far-UVC radiation are largely dependent on the stratum corneum thickness (SCT), consideration must be given to the fact that the SCT does not differ significantly between individuals with normal and sensitive skin (Richters et al., 2017) and is generally not dependent on sex, lineage/ancestry and age, whereby the skin of elderly subjects (average age 50 years) was compared in this case with skin of younger subjects (average age 29 years) (Tagami, 2017). There are no statistically significant differences in SCT between individuals with skin cancer and people who are healthy (Lock-Andersen et al., 1997). In infants, however, the stratum corneum is 30% thinner and the epidermis 20% thinner than in adults. The corneocytes are 20% and the cells of the granular layer 10% smaller than those in adults, suggesting faster cell turnover in infants. The density and size distribution of the papillae in the skin also vary. A pronounced direct structural relationship between the morphology of the stratum corneum and the dermal papillae was observed only in the skin of infants (Stamatas et al., 2010). So far, no robust data is available concerning the effects of regular or prolonged exposure to far-UVC radiation, e.g., of injured or damaged skin or in vulnerable populations such as children (BfS, 2022).

A summary of the studies into the effect of UVC radiation on the skin is provided in Table 2.

**Table 2.** Studies into the effect of UVC radiation on the skin.

| Publication | Study object | UV source / wavelength (filtering, if applicable) | Radiant exposure | Effect | | |
|---|---|---|---|---|---|---|
| | | | | DNA damage | | Other endpoints |
| | | | | CPDs | 6-4PPs | |
| Ong et al. (2022) | Human cell cultures (HEK-A keratinocytes and ARPE-19 retinal cells) | KrCl excimer lamp / 222 nm, filtered | Irradiance 73 µW $cm^{-2}$, irradiation duration 60 min | None | | Cells retained their ability to grow<br>No apoptosis markers detected, despite a number of γH2AX-positive cells suggesting DNA damage<br>Tumour suppression down-regulated one to three days after exposure. |

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| | | Mercury vapour lamp / 254 nm | | CPD formation | | Apoptotic cell death induced by DNA damage |
| | | LED array / 277 nm | | CPD formation | | Apoptotic cell death induced by DNA damage |
| Glaab et al. (2021) | Porcine skin | LED array / 233 nm, FWHM 12 nm, filtered above 240 nm | 40 mJ $cm^{-2}$ (irradiance 46 µW $cm^{-2}$, irradiation duration 14.5 min) | (3.7 ± 1.5)% of epidermal cells with CPDs to the total amount of cells in the microscopic images | (2.3 ± 0.8)% of epidermal cells with 6-4PPs to the total amount of cells in the microscopic images | |
| | | Mercury vapour lamp / 254 nm | 40 mJ $cm^{-2}$ (irradiance 50 µW $cm^{-2}$) | (58 ± 11)% of epidermal cells with CPDs to the total amount of cells in the microscopic images | (69 ± 12)% of epidermal cells with 6-4PPs to the total amount of cells in the microscopic images | |
| Nishikawa et al. (2024) | Colon cancer cell line DLD-1 cultured in a monolayer | KrCl excimer lamp / 222 nm, filtered | 30 mJ $cm^{-2}$ (irradiance 0.974 mW $cm^{-2}$) | (3.37 ± 0.02) ng/µg DNA (measured using ELISA) | | Cell death induced by the damage to the cell membrane, shown by the propidium iodide (PI) uptake. |
| | | Mercury vapour lamp / 254 nm | 30 mJ $cm^{-2}$ (irradiance 0.614 W $cm^{-2}$) | (13.1 ± 1.62) ng/µg DNA (measured using ELISA) | | Cell death induced by the DNA damage. |
| Valanciute et al. (2025) | Human lung cells in vitro | Continuous wave $UVC_{222}$, filtered (KrCl excimer lamp) Pulsed laser $UVC_{222}$ | 5 mJ $cm^{-2}$ 25 mJ $cm^{-2}$ | | | Phosphorylation of γH2AX at Ser139, a marker of DNA double strand breaks. |

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| | | | | | | |
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| | | Pulsed laser $UVC_{206}$ | | | | |
| | Human tracheal tissue | Continuous wave $UVC_{222}$, filtered (KrCl excimer lamp) Pulsed laser $UVC_{222}$ Pulsed laser $UVC_{206}$ | 5 mJ $cm^{-2}$ 25 mJ $cm^{-2}$ 50 mJ $cm^{-2}$ | | Continuous wave and pulsed $UVC_{222}$ induced 6-4PPs formation in the upper epithelial cells of the trachea. | Significantly higher number of γH2AX (Ser139)-positive cells compared to non-irradiated tissue. |
| | Human lung cells in vitro | Continuous wave $UVC_{222}$, filtered (KrCl excimer lamp) Pulsed laser $UVC_{222}$ Pulsed laser $UVC_{206}$ | 5 mJ $cm^{-2}$ 25 mJ $cm^{-2}$ 50 mJ $cm^{-2}$ | | | Significant decrease in cell viability 12- and 48-hours post-irradiation, with no recovery, indicating continuous cell death beyond initial exposure. |
| Fukui et al. (2020)† | Test subjects, one area on the back | KrCl excimer lamp / 222 nm, filtered above 230 nm | 500 mJ $cm^{-2}$ | Small, but significant increase in CPDs | | No signs of erythema |
| Eadie et al. (2021) | In vivo, skin type II on the Fitzpatrick scale, inner forearms of one subject (self-tests) | KrCl excimer lamp / 222 nm, filtered | 1500 mJ $cm^{-2}$ (irradiance 6.1 mW $cm^{-2}$) | | | No visible changes in the skin |
| | | | 6000 mJ $cm^{-2}$ (irradiance 6.1 mW $cm^{-2}$) | | | Yellowing of the skin, reversed within 24 h |
| | | | Up to 18000 mJ $cm^{-2}$ (irradiance 6.1 mW $cm^{-2}$) | | | No erythema |
| Buonanno et al. (2021a)† | 3D human skin model EpiDerm-FT | KrCl excimer lamp / 222 nm, filtered | 23 mJ $cm^{-2}$ to 150 mJ $cm^{-2}$ (irradiance | No significant increase in CPDs | No significant increase in 6-4PPs | |

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| | | | | | | |
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| | | | 0.59 mW cm$^{-2}$) | | | |
| | | | 500 mJ cm$^{-2}$ (irradiance 0.59 mW cm$^{-2}$) | Small but statistically significant increase in CPDs in the uppermost epidermal layer | No significant increase in 6-4PPs | |
| | | KrCl excimer lamp / 222 nm, unfiltered | 23 mJ cm$^{-2}$ to 500 mJ cm$^{-2}$ (irradiance 0.85 mW cm$^{-2}$) | CPD formation | Formation of 6-4PPs | |
| Yamano et al. (2020)† | Two genotypes of hairless albino mice (wild type and photosensitive *Xpa* knockout type) | KrCl excimer lamp / 222 nm, filtered | Wild type: 500 mJ cm$^{-2}$, irradiation three times a week for 10 weeks; *Xpa* knockout type: 50 mJ cm$^{-2}$ and 100 mJ cm$^{-2}$, irradiation twice a week for 10 weeks (irradiance 1 mW cm$^{-2}$) | | | No skin tumours or corneal damage after 15 weeks of observation |
| | | | Both genotypes 500 mJ cm$^{-2}$ | CPD formation in the outermost layer of the epidermis | | No erythema or ear swelling |
| Hickerson et al. (2021) | Ex vivo human skin model | KrCl excimer lamp / 222 nm, filtered | 6100 mJ cm$^{-2}$ (irradiation duration 1000 s) | Minimal CPD formation, limited to the | | |

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| | | | | upper layer of the epidermis | | |
| | | Narrowband UVB lamp / maximum emission at 311 nm | 515 mJ cm$^{-2}$ (irradiation duration 188 s) | CPD formation throughout the epidermis | | |
| | In vivo, forearms of two subjects (study authors) | KrCl excimer lamp / 222 nm, filtered | 6100 mJ cm$^{-2}$ | Minimal CPD formation, limited to the upper layer of the epidermis and few CPD-positive cells in the subjects with thicker stratum corneum | | |
| Zwicker et al. (2022) | Excised human skin / reconstructed equivalents of human skin | KrCl excimer lamp / 222 nm, filtered above 230 nm | 40 mJ cm$^{-2}$ (irradiance 3.34 mW cm$^{-2}$) | (0.5 ± 0.5)% CPD-positive keratinocytes | | |
| | | KrCl excimer lamp / 222 nm, filtered above 230 nm | 80 mJ cm$^{-2}$ (irradiance 3.34 mW cm$^{-2}$) | Not observed | Not observed | |
| | | LED array / 233 nm, FWHM 12 nm, filtered above 240 nm | 20 mJ cm$^{-2}$ to 60 mJ cm$^{-2}$ (irradiance 0.041 mW cm$^{-2}$) | Negligible (on the surface directly beneath the stratum corneum) | | |

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| | | LED array / 233 nm, FWHM 12 nm, filtered above 240 nm | 80 mJ $cm^{-2}$ (irradiance 0.041 mW $cm^{-2}$) | (18.3 ± 3.0)% CPD-positive keratinocytes (on the surface directly beneath the stratum corneum) | | |
| | | Mercury vapour lamp / 254 nm | 40 mJ $cm^{-2}$ (irradiance 0.54 mW $cm^{-2}$) | (44.2 ± 3.7)% CPD-positive keratinocytes (as far as the basal cells) | (21.5 ± 1.9)% | |
| | | UVB lamp with a small UVA component, 280 nm–400 nm | 3 mJ $cm^{-2}$ (irradiance 0.041 mW $cm^{-2}$) | 94% CPD-positive keratinocytes (as far as the basal cells) | | |
| Welch et al. (2026) | Human skin biopsies of two donors | KrCl excimer lamp / 222 nm, filtered | 100 mJ $cm^{-2}$ | Not observed | | No γH2AX-positive cells |
| | | LED array / 233 nm, FWHM 7.4 nm | | 8% of the epidermal cells positive for CPDs | | No γH2AX-positive cells |
| | | Mercury vapour lamp / 254 nm | | 45% of the epidermal cells positive for CPDs | | (4.3 ± 0.9)% γH2AX-positive cells |
| Tavares et al. (2023) | Reconstructed human skin (RHS) in vitro | KrCl excimer lamp / 222 nm, filtered | 1500 mJ $cm^{-2}$ | No significant induction of CPDs | No significant induction of 6-4PPs | Detachment of stratum corneum and thickening of epidermis 48 hours post irradiation<br>Induction of ROS<br>Possible inhibition of skin regeneration processes |

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| | | | | | | Increased expression of matrix metalloproteinases MMP-1 and MMP-9<br>Intensified expression of the inhibitor of these metalloproteinases (TIMP-1) |
| | | Mercury vapour lamp / 254 nm | | | | Detachment of stratum corneum and granular layer 48 hours post irradiation<br>Increased expression of matrix metalloproteinases MMP-1 and MMP-9<br>Strong induction of ROS |
| | In vivo HRS/J mouse model | KrCl excimer lamp / 222 nm, filtered | 1500 mJ $cm^{-2}$ | No significant induction of CPDs | No significant induction of 6-4PPs | Mild skin damage<br>No histological changes<br>Slightly increased expression of MMP-9 |
| | | Mercury vapour lamp / 254 nm | | Induction of CPDs | Induction of 6-4PPs | |
| | | KrCl excimer lamp / 222 nm, filtered | 25 mJ $cm^{-2}$ per day, irradiation duration 40 days | | | No sunburn or desquamation of the skin on the back<br>Expression of MMP-9, though less effective than at 254 nm<br>Strong induction of ROS |
| | | Mercury vapour lamp / 254 nm | 6 mJ $cm^{-2}$ per day, irradiation | | | No sunburn or desquamation of the skin on the back |

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| | | | | | | |
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| | | | duration 40 days | | | Increased expression of MMP-9<br>Induction of ROS |
| Welch et al. (2023) | Hairless SKH-1 albino mice | KrCl excimer lamp / 222 nm, filtered | Mean radiant exposure of 55 mJ $cm^{-2}$ to 130 mJ $cm^{-2}$ and 400 mJ $cm^{-2}$ (inhomogeneous), irradiation duration 8 hours daily, 5 days per week for 66 weeks (15.2 months) | | | No skin cancer and no non-carcinogenic skin lesions |
| Zamudio Diaz et al. (2025a) | Human skin in vivo in the lumbar and hip regions; healthy subjects of varying skin types and ages | LED array / 233 nm, FWHM 12 nm, filtered above 240 nm | Single radiant exposure of 60 mJ $cm^{-2}$ | At 24 h post-irradiation, DNA damage remained minimal and superficial, with CPD lesions detected in 1.4% of cells in the younger and 4.6% in the older cohort.<br>The younger group exhibited complete repair of the damage 7 days after irradiation. In subjects | No detectable 6-4PPs | |

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| | | | | over 50 years old, however, not all damage was repaired, highlighting a reduced efficiency in DNA repair mechanisms. However, the remaining damage did not exceed 2% CPD positive cells. | | |
| | | | Repeated radiant exposure over four consecutive days, with 24-h intervals between treatments (daily radiant exposure of 60 mJ $cm^{-2}$, up to cumulative radiant exposure of 240 mJ $cm^{-2}$ by day four, or daily radiant exposure of 30 mJ $cm^{-2}$, up to cumulative | Progressive CPD accumulation, suggesting limitations in repair mechanisms over 24 hours. | | No significant changes of skin pigmentation, antioxidant activity, or immune response. |

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| | | | radiant exposure of 120 mJ $cm^{-2}$ by day four). | | | |
| Buonanno et al. (2016)† | Hairless mice | KrBr excimer lamp / 207 nm, filtered | 157 mJ $cm^{-2}$ (irradiation duration 7 h) | CPDs not significantly increased | 6-4PP dimers not significantly increased | Epidermal thickness—no difference compared to the skin of unexposed mice<br>Skin tissue differentiation—no statistical difference compared to unexposed skin |
| | | Mercury vapour lamp / 254 nm | | 35-fold increase in CPDs | 26-fold increase in 6-4PP dimers | 2.8-fold increase in average epidermal thickness<br>Differentiation of keratinocytes – 3-fold increase in K6A synthesis |
| Buonanno et al. (2017)† | Hairless mice | KrCl excimer lamp / 222 nm, filtered | 157 mJ $cm^{-2}$ (irradiation duration 7 h) | CPDs not significantly increased | 6-4PP dimers not significantly increased | Epidermal thickness—no statistical difference compared to unexposed mice<br>Differentiation of keratinocytes—no statistical difference compared to unexposed skin |
| | | Mercury lamp / 254 nm | 157 mJ $cm^{-2}$ (irradiation duration 7 h) | CPDs increased | 6-4PP dimers increased | Average epidermal thickness increased<br>Differentiation of keratinocytes—3-fold increase in K6A synthesis |
| | In vitro 3D model of human skin, EpiDerm-FT | KrCl excimer lamp / 222 nm, filtered | Up to 150 mJ $cm^{-2}$ | CPDs not significantly increased | 6-4PP dimers not significantly increased | |

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| | | | | | | |
|---|---|---|---|---|---|---|
| | | Mercury lamp / 254 nm | | CPDs increased | 6-4PP dimers increased | |

† The authors declare a conflict of interest.

*3.3. Eye*

In the eye, the tear fluid forms the outer barrier on the epithelium of the cornea, protecting the eye from harmful environmental influences (Masoudi, 2022, Peter et al., 2023, Robciuc et al., 2014). The precorneal tear film is a complex mixture of electrolytes, proteins, lipids, metabolites and specific resident microorganisms. Tear factors promote wound healing, suppress inflammation, scavenge free radicals and defend against microbial infection (Pflugfelder and Stern, 2020). The tear film is roughly divided into three layers: the outer lipid, middle aqueous, and inner mucin layer. The proteins in the middle aqueous layer are responsible for immune and inflammatory reactions and wound healing. Far-UVC radiation to the eye is only absorbed to a small extent in the tear fluid. It should be noted in this context that under certain circumstances about 80% of far-UVC radiation can penetrate the tear fluid, although the underlying literature does not provide any insights into the thickness of the tear film that was examined (Michalos et al., 1994).

Like the stratum corneum of the skin, the superficial wing cells act as a protective shield for the underlying corneal epithelium. The corneal tissue with the highest absorption for UV radiation is the Bowman membrane, followed by the endothelium and, due to its layer thickness, the stroma (Peris-Martinez et al., 2021) (see Figure 2). Across the entire thickness of the corneal stroma, 70% to 75% of UV radiation is absorbed at wavelengths under 310 nm (Kolozsvari et al., 2002). The stem cells of the corneal epithelium that are susceptible to cancer can be found in the basal cell layer of the corneal limbus and are shielded by at least three cell layers (Lavker et al., 2004). Almost all UVC radiation is absorbed in the corneal epithelium. The absorption rates are attributed to high levels of tryptophan residues in the proteins of the stroma (Mitchell and Cenedella, 1995) and to high levels of ascorbic acid in the epithelium (Ringvold, 1997, Ringvold, 1998, Brubaker et al., 2000). Whereas both the UVB radiation and the UVA radiation are completely absorbed by the lens of the eye at wavelengths below 365 nm, 1% to 2% of the UVA radiation above 365 nm does reach the retina (Glickman, 2011, Krutmann et al., 2014, Mainster and Turner, 2010).

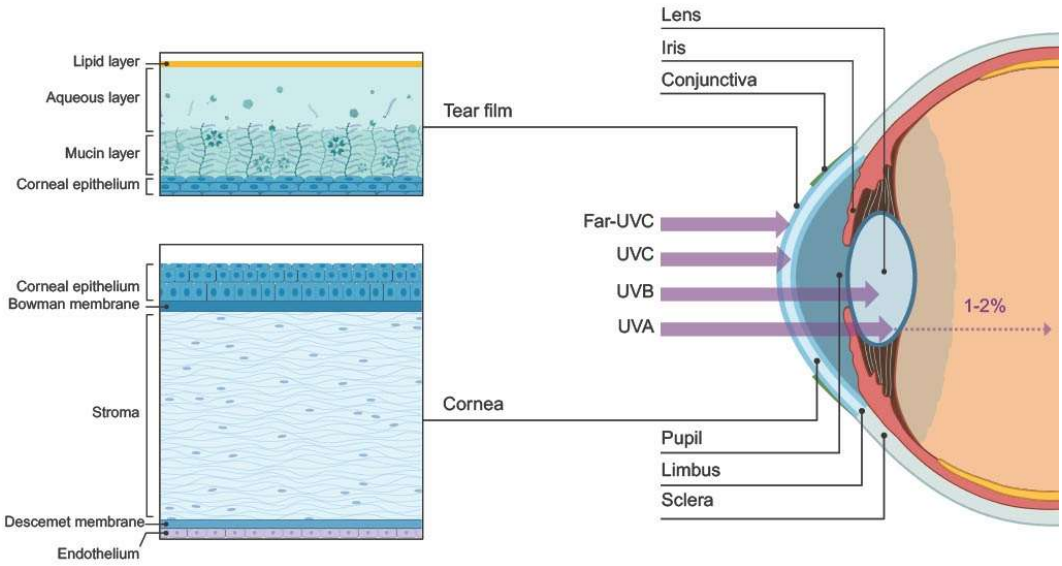


**Figure 2.** Diagram of the penetration depths of optical radiation in the human eye at different UV wavelengths (created with BioRender).

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The effects of exposing the eye to radiation at wavelengths below 254 nm have been examined in only few studies to date (Sliney et al., 1991, Kaidzu et al., 2019, Pitts, 1974, Pitts and Tredici, 1971, Sugihara et al., 2023, Sugihara et al., 2025b, Zamudio Diaz et al., 2025b). The threshold for photokeratitis in the rabbit cornea was determined by Sliney et al. (1991) at a wavelength of 193 nm using an excimer laser. The authors conclude that the penetration depth of the applied shortwave radiation in the epithelial cell layer is limited and an intact tear film can help protect the cornea against the weak scattered laser radiation. Pitts and Tredici (1971) found that the cornea is most sensitive to UV radiation of 270 nm and reacts with symptoms of photokeratitis. With the aid of slit-lamp biomicroscopy, staining and surface mapping, Kaidzu et al. determined thresholds for photokeratitis on a rat eye model 24 hours after exposure to four UVC wavelengths (207 nm, 222 nm, 235 nm, and 254 nm) and one wavelength in the UVB spectral range (311 nm) (Kaidzu et al., 2019, 2021). An effect on the cornea was detected here for 207 nm and 222 nm above the threshold values of 15000 mJ $cm^{-2}$ and 5000 mJ $cm^{-2}$ respectively. Such exposures far exceed the ELs (International Commission on Non-Ionizing Radiation, 2004) that are currently applicable (about 300 and 220 times higher, respectively). Kaidzu et al. also performed a histological assessment of the cornea using CPDs as a marker for DNA damage (Kaidzu et al., 2019, 2021). The depth of the observed CPDs varied with the wavelength; at 207 nm and 222 nm, the CPD marker was only found in the upper superficial cells, which are shed within a few days during the normal life cycle of the corneal epithelium. In the eye, the cells of the cornea that are susceptible to cancer are found in the limbus, i.e., the transition zone between the cornea and the sclera. UV radiation reaches the corneal endothelium at a wavelength of 254 nm and 311 nm, the middle section of the corneal stroma at 235 nm, and the outer portion of the corneal epithelium at 207 nm and 222 nm (Kaidzu et al., 2019, 2021). In the cornea of pigs the $UVC_{222}$ radiation reached only the superficial layer of the limbal epithelium (Kaidzu et al., 2023). According to the authors, since cornea of pigs is similar in size and structure to that of the human eye, no harmful effects on stem cells in the basal portion of the corneal limbus are to be expected. It should, however, be mentioned that the pig cornea is thicker than the human cornea. While Doughty and Zaman (2000) report a central human corneal thickness of 534 µm, the mean porcine corneal thickness published by Sanchez et al. (2011) and Heichel et al. (2016) amounts (833 ± 40) µm and (878 ± 14) µm, respectively. In the study of Jay et al. (2008) porcine corneal thickness almost twice that of the human corneal thickness was published, i.e., 1013 (± 10) µm. The thresholds for corneal injuries from far-UVC radiation reported by Kaidzu et al. (Kaidzu et al., 2019, 2021) are significantly higher than those reported by Pitts (1974), who compared exposure data from 39 human eyes against data from rabbits and primates. Arden et al. (2025) examined the ocular effects of chronic far-UVC exposure in 48 hairless, immune-competent SKH-1 mice over 66 weeks (five days/week, eight hours/day). Mice were exposed to 400 mJ $cm^{-2}$, 130 mJ $cm^{-2}$, 55 mJ $cm^{-2}$ or no $UVC_{222}$ radiation. No significant differences in visual acuity, contrast sensitivity, intraocular pressure, or corneal neovascularization were observed between unirradiated animals and exposure groups.

Zamudio Diaz et al. (2025b) described the wavelength dependent penetration of far-UVC in human and porcine cornea samples by quantification of induced DNA damage (CPDs and 6-4PPs). Using the optical properties of porcine corneal samples with an epithelial thickness of 110 µm they simulated the penetration depth of far-UVC in human cornea with 50 µm epithelial thickness. These simulations suggest that in intact human corneas, damage-relevant intensity of $UVC_{222}$ reaches the middle of the epithelium, while for $UVC_{233}$ the basal layer was reached. A similar effect was observed in immunohistologically stained samples of reconstructed human cornea epithelium (RHCE, 79 µm thickness) after irradiation with $UVC_{222}$ and $UVC_{233}$. In the RHCE samples multiple irradiations with $UVC_{222}$ and $UVC_{233}$ led to accumulation of CPDs (not of 6-4PPs) resulting in a doubling of damaged cells after a fourfold dose. Repeated exposure to far-UVC in public indoor spaces may be of concern with regard to this accumulation. A DNA-repair effect, although not statistically significant, was observed in human cornea samples (with a very thin epithelium of 26 µm) 24 hours after irradiation. A reduction of CPD-damaged cells by 11% and 27% after $UVC_{222}$- and $UVC_{233}$-irradiation, respectively, was observed. After irradiation with $UVC_{233}$, a reduction by 40% of 6-4PP-positive cells

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in the epithelium was shown. In these very thin cornea samples, irradiation with $UVC_{233}$ reaches the upper stroma, inducing 14% damaged cells, and only $UVC_{254}$ reaches the endothelium inducing 69% CPD-positive cells. In porcine cornea samples, after irradiation with $UVC_{254}$ and $UVC_{233}$, 82% and 20% CPD-damaged and 12% and 22% 6-4PP-damaged cells were induced in the epithelium, respectively. While $UVC_{233}$ showed a mean maximum penetration of the epithelium of 28 µm, $UVC_{254}$ showed a mean penetration into the upper stroma by 71 µm inducing 20% CPD-damaged cells.

In a study with six subjects exposed to $UVC_{222}$ radiation for about 6.7 hours per week for 12 months, Sugihara et al. (2023) observed no acute or chronic eye damage. The applied radiant exposure of about 2.8 mJ $cm^{-2}$ per day did not exceed the EL of 22.7 mJ $cm^{-2}$ at 222 nm for protecting the eyes against actinic UV hazards (International Commission on Non-Ionizing Radiation, 2004). Sugihara et al. (2025a) extended the initial observation period from 12 to 36 months for four subjects exposed to $UVC_{222}$ radiation for (5.5 ± 4.1) hours per week. The radiant exposure remained within the above mentioned EL of 22.7 mJ $cm^{-2}$ at 222 nm, and no evidence of photokeratitis or other UV-related eye conditions was observed.

Since effects on the cornea in conjunction with exposure to far-UVC radiation also depend on tear film thickness, it is important to note that no significant interindividual difference was found in the thickness of the muco-aqueous tear film or the lipid tear film between healthy subjects and patients with dry eye in a study of tear film thickness involving 49 subjects (Segev et al., 2020). According to Masoudi (2022), further investigation of the "normal" tear profile and the functionality of the tear film that depends on this profile would require the participation of a broad spectrum of sample populations, whereby age, ethnicity, sex, geographical and environmental parameters would need to be considered.

Sugihara et al. (2025b) performed a prospective, interventional study involving five subjects aged 29 to 47 years, who were exposed to a $UVC_{222}$ radiant exposure up to 75 mJ $cm^{-2}$. The subjects were monitored using custom-made glasses with a UV-cut filter on one eye to serve as a control. Ocular examinations (visual acuity, refractive error, corneal endothelial density, corneal erosion scores, and conjunctival hyperemia scores) were performed prior and 24 hours post-exposure as well as after 1, 3, and 6 months. The study found no clinically significant photokeratitis or long-term eye damage across the five subjects, even at the highest radiant exposure of 75 mJ $cm^{-2}$. However, epiphora sensation, dry eye sensation, epiphora, eye discomfort, conjunctival hyperemia and mild pain were reported. These symptoms started 2 to 2.5 hours after the start of irradiation and subsided within 4.5 to 11 hours after irradiation.

Kousha et al. (2024) investigated immediate and delayed eye discomfort with 38 subjects in a simulated office environment equipped with ceiling-mounted $UVC_{222}$ sources (filtered KrCl excimer lamps) directed downwards into the occupied space. Discomfort was assessed immediately post-exposure and several days after exposure. The results showed no significant eye discomfort or adverse effects. However, the study does not provide data on actual eye exposure, as only the irradiance on the subjects' heads was measured. The highest radiant exposure to which 25% of the subjects were exposed was between20 mJ $cm^{-2}$ and 50 mJ $cm^{-2}$. The radiant exposure of the eyes can only be a small percentage of that at the top of head, as shown by Duncan et al. (2023). In a mannequin study, they showed that the eye received on average 5.8% of the radiant exposure measured at the top of the head.

A summary of the studies into the effect of UVC radiation on the eye is provided in Table 3.

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**Table 3.** Studies into the effect of UVC radiation on the eye.

| Publication | Study object | UV source / wavelength (filtering, if applicable) | Radiant exposure | Effect | |
|---|---|---|---|---|---|
| | | | | Eye damage | CPDs and 6-4PPs |
| Kaidzu et al. (2019)† | Albino rats | KrCl excimer lamp / 222 nm, filtered | 30 mJ $cm^{-2}$, 150 mJ $cm^{-2}$ 600 mJ $cm^{-2}$ (irradiance 5.7 mW $cm^{-2}$) | No corneal damage | |
| | | Mercury vapour lamp / 254 nm | 30 mJ $cm^{-2}$ (irradiance 1.1 mW $cm^{-2}$) | No corneal damage | |
| | | | 150 mJ $cm^{-2}$ (irradiance 1.1 mW $cm^{-2}$) | Keratitis | |
| | | | 600 mJ $cm^{-2}$ (irradiance 1.1 mW $cm^{-2}$) | Corneal erosion | |
| Kaidzu et al. (2021)† | Albino rats | KrBr eximer lamp / 207 nm, filtered | 30 mJ $cm^{-2}$ to 15000 mJ $cm^{-2}$ (irradiance 0.83 mW $cm^{-2}$) | Corneal damage above 15000 mJ $cm^{-2}$ | CPDs only in the uppermost layer of the cornea |
| | | KrCl excimer lamp / 222 nm, filtered | 30 mJ $cm^{-2}$ to 5000 mJ $cm^{-2}$ (irradiance 4.2 mW $cm^{-2}$) | Corneal damage above 5000 mJ $cm^{-2}$ | CPDs only in the uppermost layer of the cornea |
| | | Xenon lamp / 235 nm, filtered | 10 mJ $cm^{-2}$ to 300 mJ $cm^{-2}$ (irradiance 0.077 mW $cm^{-2}$) | Corneal damage above 300 mJ $cm^{-2}$ | CPDs in the cells of the middle layers of the corneal epithelium |
| | | Mercury vapour lamp / 254 nm | 10 mJ $cm^{-2}$ to 300 mJ $cm^{-2}$ (irradiance 1.3 mW $cm^{-2}$) | Corneal damage above 20 mJ $cm^{-2}$ | CPDs in all cells of the cornea |
| | | Narrowband UVB lamp / 311 nm | 30 mJ $cm^{-2}$ to 600 mJ $cm^{-2}$ (irradiance 2.3 mW $cm^{-2}$) | Corneal damage above 600 mJ $cm^{-2}$ | CPDs in all cells of the cornea |
| Kaidzu et al. (2023)† | Albino rats (CPDs in upper limbus 24 | KrBr excimer lamp / 207 nm, filtered | 1500 mJ $cm^{-2}$ (irradiance 0.18 mW $cm^{-2}$–0.25 mW $cm^{-2}$) | | No CPD-positive cells in the basal cells of the limbus |
| | | | 2500 mJ $cm^{-2}$ | | Weakly CPD-positive cells in the limbus |

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| | hours post irradiation) | | (irradiance 0.25 mW cm$^{-2}$) | | |
| | | | 10000 mJ cm$^{-2}$ (irradiance 0.86 mW cm$^{-2}$ - 0.91 mW cm$^{-2}$) | | Strong CPD-positive cells throughout the limbus, including the basal cells |
| | | KrCl excimer lamp / 222 nm, filtered | 1500 mJ cm$^{-2}$ (irradiance 4.2 mW cm$^{-2}$ - 5.0 mW cm$^{-2}$) | | Slightly stronger CPD-positive cells in the upper layer |
| | | | 2500 mJ cm$^{-2}$ (irradiance 4.2 mW cm$^{-2}$) | | Moderately CPD-positive cells throughout the limbus |
| | | | 5000 mJ cm$^{-2}$ (irradiance 4.2 mW cm$^{-2}$) | | Strong CPD-positive cells throughout the limbus, including the basal cells |
| | | Xenon lamp / 235 nm, filtered | 30 mJ cm$^{-2}$ (irradiance 0.079 mW cm$^{-2}$–0.16 mW cm$^{-2}$) | | Weak CPD-positive cells as far as the middle layer |
| | | | 300 mJ cm$^{-2}$ (irradiance 0.11 mW cm$^{-2}$ - 0.17 mW cm$^{-2}$) | | Cells in the upper layer with even stronger CPD positivity |
| | | | 600 mJ cm$^{-2}$ (irradiance 0.082 mW cm$^{-2}$) | | Cells in the middle layer with strong CPD positivity and cells in the basal layer with weak CPD positivity |
| | | Mercury vapour lamp / 254 nm | 20 mJ cm$^{-2}$ (irradiance 1.1 mW cm$^{-2}$) | | Cells in the middle layer with strong CPD positivity, basal cells with weak CPD positivity |
| | | | 100 mJ cm$^{-2}$ (irradiance 1.1 mW cm$^{-2}$) | | All cells in the limbus CPD-positive, also the basal cells |

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| | | | 300 mJ $cm^{-2}$ (irradiance 1.1 mW $cm^{-2}$) | | Cells with even stronger CPD positivity |
| | | Narrowband UVB lamp / 311 nm | 30 mJ $cm^{-2}$ (irradiance 2.0 mW $cm^{-2}$–2.3 mW $cm^{-2}$) | | No CPD-positive cells |
| | | | 150 mJ $cm^{-2}$ (irradiance 2.0 mW $cm^{-2}$ - 2.3 mW $cm^{-2}$) | | CPD-positive cells in the middle layer and in the basal cell layer |
| | | | 600 mJ $cm^{-2}$ (irradiance 2.0 mW $cm^{-2}$ - 2.3 mW $cm^{-2}$) | | CPD-positive cells throughout the limbus |
| | Pigs' eyes (CPDs in limbus immediately after irradiation) | KrCl excimer lamp / 222 nm, filtered | 600 mJ $cm^{-2}$ (irradiation duration 120 s) | | CPD-positive cells in the uppermost layer |
| | | Mercury vapour lamp / 254 nm | 600 mJ $cm^{-2}$ (irradiation duration 546 s) | | CPD-positive cells in the upper layer and 50 µm - 100 µm deep, but not in the basal layer |
| Arden et al. (2025) | 48 hairless, immune-competent SKH-1 mice | KrCl excimer lamp / 222 nm, filtered | 55 mJ $cm^{-2}$130 mJ $cm^{-2}$400 mJ $cm^{-2}$66 weeks (five days/week, eight hours/day) | | No significant differences in visual acuity, contrast sensitivity, intraocular pressure, or corneal neovascularization observed between unirradiated and exposed animals. |
| Zamudio Diaz et al. (2025b) | Ex vivo human cornea in the presence of human tears from a single | KrCl excimer lamp / 222 nm, filtered above 230 nm | 40 mJ $cm^{-2}$ (irradiation duration 2 min 47 s) | | (60 ± 12)% CPD-positive cells in the epithelium (5 ± 1)% CPD-positive cells in the anterior stroma |
| | | LED array / 233 nm, FWHM 12 | 40 mJ $cm^{-2}$ (irradiation duration 5 min 33 s) | | Approx. 100% CPD-positive cells in the epithelium. |

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| | healthy donor, mean epithelial thickness 26 µm, very thin due to corneabank storage | nm, filtered above 240 nm | | | (14 ± 5)% CPD-positive cells in the anterior stroma |
| | | Mercury vapour lamp / 254 nm | 40 mJ $cm^{-2}$ (irradiation duration 1 min 15 s) | | Approx. 100% CPD-positive cells in the epithelium (21 ± 8)% CPD-positive cells in the posterior stroma (57 ± 19)% CPD-positive cells in the endothelium |
| | | Broadband UVB lamp, 280 nm–400 nm, peak emission at 311 nm | 3 mJ $cm^{-2}$ (irradiation duration 1 min 26 s) | | Approx. 100% CPD-positive cells in the epithelium |
| | Reconstructed human cornea epithelium (RHCE), mean epithelial thickness 79 µm | KrCl excimer lamp / 222 nm, filtered above 230 nm | 150 mJ $cm^{-2}$ (irradiation duration 10 min 25 s) | | Superficial CPD-positive cells (approx. 25%) immediately after irradiation, no significant repair 24 hours after irradiation 6-4PP-positive cells immediately after irradiation, a slight repair 24 hours after irradiation |
| | | | 150 mJ $cm^{-2}$ (irradiation duration 10 min 25 s) Four repeated irradiations with 2 hours breaks in between | | 24 hours after multiple irradiations, CPD levels approximately doubled, but CPD formation did not increase fourfold compared to a single dose No clear trend observed for 6-4PPs |

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| | | LED array / 233 nm, FWHM 12 nm, filtered above 240 nm | 60 mJ $cm^{-2}$ (irradiation duration 8 min 20 s) | | Superficial CPD-positive cells, (approx. 25%) immediately after irradiation, no significant repair 24 hours after irradiation<br>Statistically significant increase in 6-4PP-positive cells immediately after irradiation, a slight repair 24 hours after irradiation |
| | | | 60 mJ $cm^{-2}$ (irradiation duration 8 min 20 s) Four repeated irradiations with 2 hours breaks in between | | 24 hours after multiple irradiations, high CPD levels, but CPD formation did not increase fourfold compared to a single dose |
| | | Broadband UVB lamp, 280 nm–400 nm, peak emission at 311 nm | 3 mJ $cm^{-2}$ (irradiation duration 1 min 26 s) | | Immediately after irradiation, threefold increase (> 75%) of CPD-positive cells in comparison with $UVC_{222}$ and $UVC_{233}$ radiation, no significant repair 24 hours after irradiation<br>6-4PP-positive cells immediately after irradiation, a slight repair 24 hours after irradiation |
| | | | 3 mJ $cm^{-2}$ (irradiation duration 1 min 26 s) Four repeated irradiations with 2 hours breaks in between | | Saturation 24 hours after multiple irradiations |
| | | LED array / 233 nm, | 40 mJ $cm^{-2}$ | | (20 ± 3)% CPD-positive cells in the epithelium, |

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| | | | | | |
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| | Ex vivo porcine cornea, mean epithelium thickness 110 µm | FWHM 12 nm, filtered above 240 nm | (irradiation duration 5 min 33 s) | | with a mean penetration of (28 ± 5) µm<br>(12 ± 0)% 6-4PP-positive cells in the epithelium |
| | | Mercury vapour lamp / 254 nm | 40 mJ $cm^{-2}$ (irradiance 0.54 mW $cm^{-2}$, irradiation duration 1 min 15 s) | | (82 ± 8)% CPD-positive cells in the epithelium<br>(22 ± 10)% 6-4PP-positive cells in the epithelium<br>CPD-positive cells in the anterior stroma, with a mean penetration of (71 ± 2) µm |
| | | Broadband UVB lamp, 280 nm–400 nm, peak emission at 311 nm | 3 mJ $cm^{-2}$ (irradiation duration 1 min 26 s) | | (72 ± 27)% CPD-positive cells in the epithelium<br>(16 ± 2)% 6-4PP-positive cells in the epithelium |
| Sugihara et al. (2023)† | Human (6 subjects, 5 of whom wore glasses) | KrCl excimer lamp / 222 nm, filtered | The lamps were alternately on for 200 s and off for 1600 s.<br>Maximum irradiance on the subjects' eyes 0.002 mW $cm^{-2}$<br>12 months, mean length of stay 6.7 h per week (approx. 1 h per day, radiant exposure 2.8 mJ $cm^{-2}$ per day) | No acute or chronic effects | |
| Sugihara et al. (2025b)† | Human (5 subjects) | KrCl excimer lamp / 222 nm, filtered | 22 mJ $cm^{-2}$<br>50 mJ $cm^{-2}$<br>75 mJ $cm^{-2}$ | Epiphora sensation, dry eye sensation, epiphora, eye discomfort, conjunctival hyperemia, and mild pain were reported. The symptoms started 2–2.5 h | |

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| | | | | | |
|---|---|---|---|---|---|
| | | | | after the start of irradiation and disappeared 4.5–11 h after the start of irradiation. No clinically significant photokeratitis after 24 h. No long-term eye damage after 1, 3 and 6 months. | |
| Sugihara et al. (2025b)[†] | Human (4 subjects) | KrCl excimer lamp / 222 nm, filtered | 36 months, subjects exposed (5.5 ± 4.1) hours per week. Radiant exposure within EL of 22.7 mJ $cm^{-2}$. | No evidence of photokeratitis or other UV-related eye conditions. | |

[†] The authors declare a conflict of interest.

*3.4. Conclusion Drawn from the Skin and Eye Studies*

In healthy human skin, far-UVC radiation only reaches as far as the outermost layer, which consists of dead cells (corneal layer, stratum corneum). The prerequisite is that longer-wave radiation is reliably filtered out. With filtered $UVC_{222}$ radiation, premutagenic lesions were only detected in the uppermost cells of the epidermis, which in any case are shed within a few days. Essential for the protective effect is a well-established, intact corneal layer, the thickness of which is still very thin in infants. Studies to date have shown that the suprabasal cells and cells of the basal layer of the human epidermis are not damaged. Concerning the thickness of the corneal layer, the available data does not produce evidence of the existence of significant subgroups of people who are systematically more susceptible to UVC exposure than the average population according to age, sex, health status and genetics. In accordance with mouse studies, it is therefore assumed that the risk of skin cancer from far-UVC radiation is not increased. However, no epidemiological studies into skin carcinogenesis from UVC radiation are available to date.

Concerning the effect of far-UVC radiation on the eye, only few studies are available. Unlike the skin, the outermost layer of the cornea also contains intact epithelial cells. The tear film above the corneal epithelium absorbs only a minimal amount of far-UVC radiation; hence, an intact tear film offers only very limited protection. So far, animal studies have not demonstrated that far-UVC radiation damages the deeper cells. A long-term study (66 month) in mice (Welch et al., 2023) did not show an enhanced skin cancer risk or other non-cancer lesions. Considering the well-documented physiological differences between murine and human skin, long-term studies in human subjects are nevertheless missing.

When assessing the effects of far-UVC radiation on the skin and eyes and thus its use in the presence of humans, the lack of robust data on the effects of regular or prolonged exposure to far-

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UVC radiation in humans, e.g., on injured or damaged skin or in vulnerable populations such as children, must be taken into account. Due to the intense absorption of far-UVC radiation by proteins and possibly by ROS formation, harmful effects cannot be ruled out. These could affect the proteins of the tear fluid in the eye in particular and this can lead to health consequences, e.g., in terms of immunological protection against pathogens (Masoudi, 2022). There has been little study so far into the damage caused to proteins by far-UVC radiation and the resultant, potential inhibiting effect on enzymatic processes. Research is needed to this end. In addition to the potential harmful effects on cells, the influence of far-UVC radiation on the microbiome of the skin and eyes must also be investigated.

## 4. Radiation Protection Aspects Concerning Use of Far-UVC Radiation in the Presence of People

### *4.1. Exposure Limits*

As mentioned in the introduction, in 2004 the ICNIRP published recommendations for exposure limits (ELs) to protect the skin and eyes from the harmful effects of incoherent UV radiation (International Commission on Non-Ionizing Radiation, 2004) from artificial sources and solar radiation. The effect of UV radiation on the skin and eyes depends on the effective radiant exposure $H_{eff}$, a product of the effective irradiance $E_{eff}$ and the duration $t$ of the irradiation. The effective irradiance $E_{eff}$ denotes the spectral irradiance weighted with the actinic UV action spectrum $S(\lambda)$ (Figure 3), taking into account the wavelength dependence of the hazardous effects of UV radiation on skin and eye, and based on skin and corneal injury thresholds. To protect the skin and eyes from the actinic UV hazard in the spectral range between 180 nm and 400 nm, an EL expressed as radiant exposure of $H_{eff} = 30$ J m$^{-2}$ (3 mJ cm$^{-2}$) is recommended. This EL applies to single or repeated exposures within an 8-hour period. The corresponding 8-hour ELs for monochromatic radiation are illustrated in Figure 4. Over the last three decades, the ELs of ICNIRP have been internationally accepted and were e.g., identical to those of the American Conference of Governmental Industrial Hygienists (ACGIH).

In their 2021 publication, Sliney and Stuck argue that, when setting the above ELs by ICNIRP, a very conservative safety factor was used in the UVC spectral range (Sliney and Stuck, 2021). They discuss the need to revise the ELs in the UVC spectral range, as several studies (Sliney et al., 1991, Buonanno et al., 2017, Narita et al., 2018c, Kaidzu et al., 2019, Yamano et al., 2020, Fukui et al., 2020, Kaidzu et al., 2021, Eadie et al., 2021) seem to indicate that the thresholds for the skin and eyes are higher in this wavelength range than the thresholds in the study by Pitts (1974). As an arc-lamp monochromator with a large spectral bandwidth was used in this study, Sliney and Stuck assumed that the derived UV action spectrum was strongly flattened. Based on the new studies listed above, the authors propose modified different curves for the UV action spectra for the skin and eyes at wavelengths below 300 nm, which result in part a significant increase of the ELs for both skin and eyes. The UV action spectrum for the skin would apply if eyes are not exposed.

At the end of 2021, the ACGIH adopted the proposed new UV action spectra and amended its ELs (ACGIH, 2022). They therefore deviate significantly from the UV action spectrum $S(\lambda)$ of the ICNIRP guidelines (International Commission on Non-Ionizing Radiation, 2004) and the corresponding ELs in the UVB and UVC spectral range. Figure 3 shows the new ACGIH UV action spectra for the skin and eyes, as well as the action spectrum $S(\lambda)$ for the actinic UV hazard of ICNIRP. In Figure 4a and Figure 4b the new ELs for monochromatic radiation as per ACGIH for the skin and eyes are compared against the corresponding ELs of ICNIRP. In addition, the measurements from various studies are presented that are designed to substantiate the curve for the new ELs according to the interpretation by Sliney and Stuck (2021). In Table 4, the ELs of International Commission on Non-Ionizing Radiation (2004) are shown together with the new ELs of ACGIH (2022) for monochromatic radiation at 222 nm, showing that the ELs recommended by the ACGIH for protecting the skin and eyes in the far-UVC spectral range have significantly increased.

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The ACGIH states clearly that the curves do not represent a legal norm, nor are they supported by the ACGIH for such use. The ACGIH also points out that all the ELs given are designed as guidelines or recommendations for protecting workers in the workplace and serve no other purpose. Nevertheless, they are frequently already considered to be new, valid ELs by some manufacturers who advocate the use of far-UVC radiation for disinfection in the presence of people in public indoor spaces.

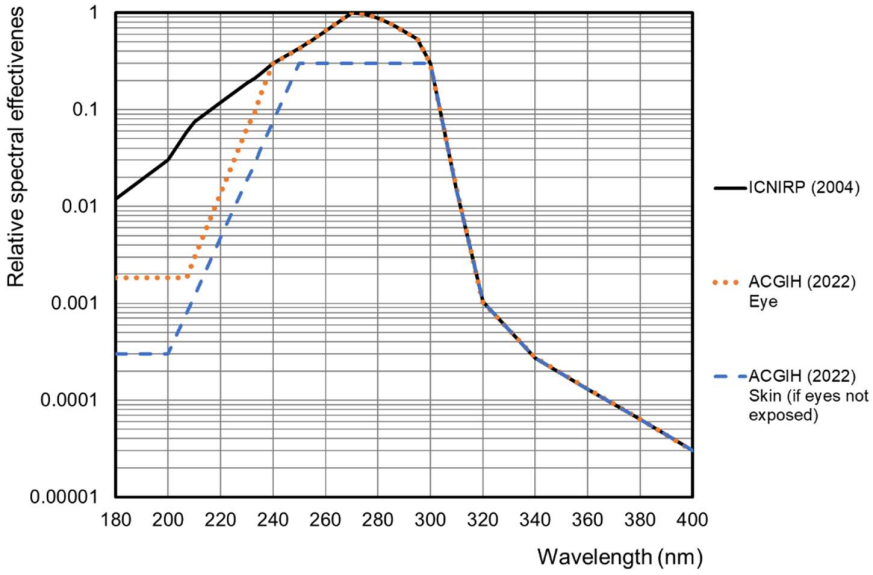


**Figure 3.** Relative spectral effectiveness for the actinic UV action spectrum $S(\lambda)$ of International Commission on Non-Ionizing Radiation (2004) (solid line) compared to the relative spectral effectiveness according to ACGIH (2022) for the skin (if the eyes are not exposed, dashed line) and the eyes (dotted line).

The argument by Sliney and Stuck (2021) that the ELs specified to date were estimated very conservatively due to a lack of available data with high measurement uncertainties appears justified at first. However, it is questionable whether recent studies cited by the authors actually improve the data situation sufficiently. In some studies, self-tests were conducted (Eadie et al., 2021) or only a small number of subjects were examined (Fukui et al., 2020). In the case of various studies (e.g., Narita et al., 2018b, Kaidzu et al., 2019, Kaidzu et al., 2021, Yamano et al., 2020, Fukui et al., 2020), the question arises as to whether appropriate radiometric measurements were carried out. For instance, for the measurements at 222 nm it is not permissible to use a broadband radiometer (e.g., USHIO VUV S172/UIT-250), which is by default calibrated by the manufacturer for radiation in the vacuum ultraviolet at 172 nm. Broadband radiometers have to be calibrated directly for the radiation source spectrum for which they will subsequently be used or, alternatively, the spectral responsivity of the radiometer can be calibrated to the dominant wavelength of the source to be measured (e.g., 222 nm for KrCl excimer lamps). A broadband radiometer calibrated at 172 nm would therefore clearly indicate a false reading in the case that a correction factor for 222 nm is not applied. More recent studies, such as Welch et al. (2025), use radiometric measurement equipment that is specially adapted for irradiation at 222 nm.

In general, it should be noted that radiometric measurements in the spectral range below 250 nm are associated with high levels of measurement uncertainties. For the correct measurement of irradiance originating from narrowband radiation, a correspondingly high level of technical expertise is required and experience in the field of UV radiometry is an essential prerequisite. Most of the studies mentioned do not specify the actual effort involved in measurements, the quality of the radiometric measurements is often not assessable and, consequently, an estimation of the reliability of the given radiometric quantities cannot be made.

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In 2024 Gutierrez-Bayona et al. published an erythema action-spectrum determined in mouse skin in a spectral range from 200 nm to 270 m describing comprehensible radiometric measurements (Gutierrez-Bayona et al., 2025). The authors calculated exposure doses for erythema as an acute skin reaction resulting in high ELs compared to the recommendations of ICNIRP. It should be noted that pre-mutagenic DNA damage already occurs at lower (suberythemal) doses. Unfortunately, this endpoint was not investigated in the present study.

Concerning effects of UVC radiation on the eye, Sliney und Stuck state that there are hardly any recent studies available. A self-experiment is then described which resulted in a sensation of dry eye and ultimately tear production at high radiant exposures. To assess the risk from far-UVC radiation, comprehensive studies are still essential—especially with respect to the effects on the eye.

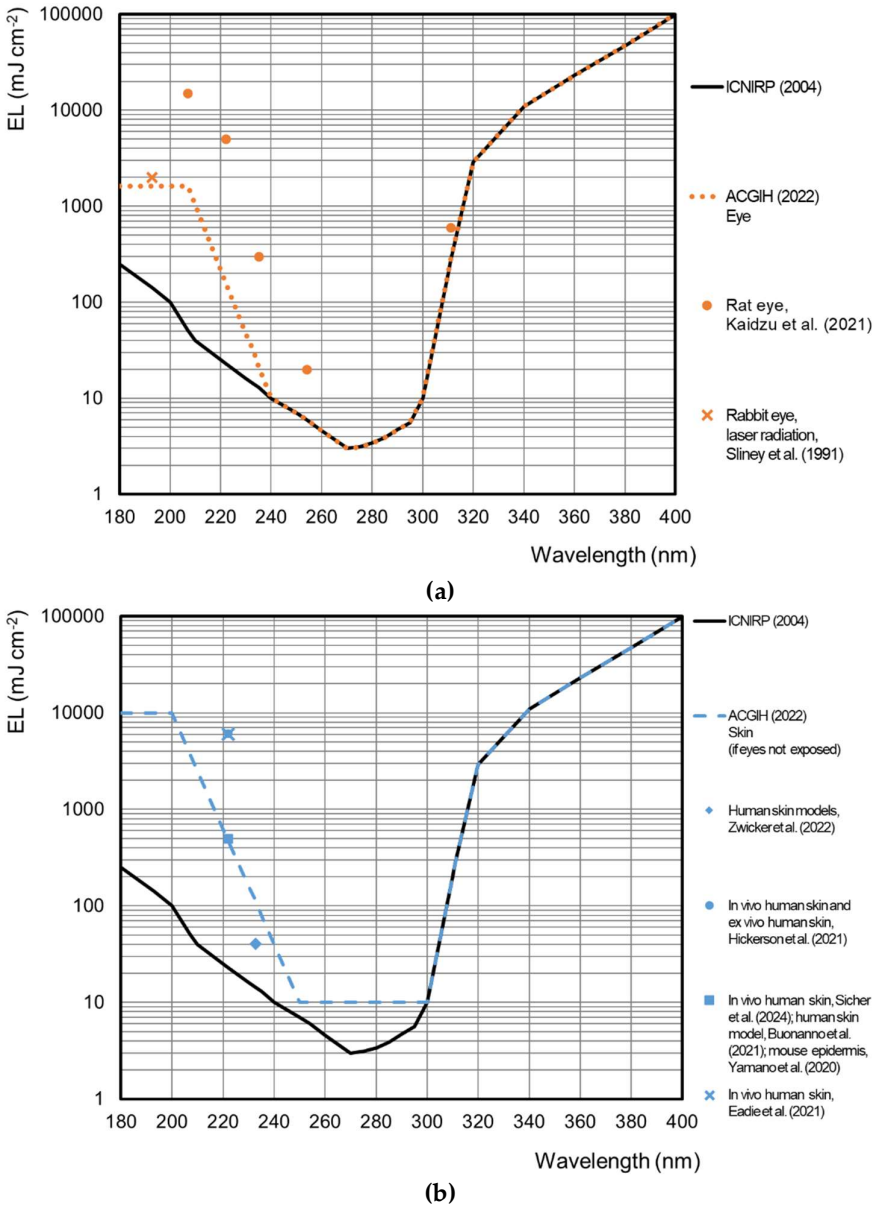


**Figure 4**. ELs for monochromatic radiation according to International Commission on Non-Ionizing Radiation (2004) (solid line) compared with the ELs of ACGIH (2022) to protect the eyes (**a**, dotted line) and the skin, if eyes are not exposed (**b**, dashed line). The maximum radiant exposures for monochromatic radiation are given from some studies where no notable damage from UV radiation was observed.

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**Table 4**. ELs for 222 nm according to International Commission on Non-Ionizing Radiation (2004) compared with the ELs of ACGIH (2022) to protect the eyes and the skin, if eyes are not exposed.

| Wavelength | EL International Commission on Non-Ionizing Radiation (2004) | EL ACGIH (2022) Skin, if eyes are not exposed | EL ACGIH (2022) Eye |
|---|---|---|---|
| 222 nm | 22.7 mJ $cm^{-2}$ | 479 mJ $cm^{-2}$ | 160.7 mJ $cm^{-2}$ |

*4.2. Further Aspects of Radiation Safety*

The ICNIRP emphasizes that the recommended ELs are aimed at protecting workers and that, with due caution, they might be applied to the general population. ICNIRP states that they can certainly not be applied to over-photosensitive individuals or individuals exposed to photosensitising substances. As no studies are available for these groups of people, particular caution is required since adverse health effects can occur at significantly lower radiant exposures.

In the study of Christou et al. (2025), patients with photosensitivity disorders underwent monochromator phototesting at different UV wavelengths with four radiant exposures below ICNIRP EL with subsequent visual assessment of the exposed skin area. The photosensitivity disorders of the patients, characterized by abnormal skin responses, were photodermatosis, polymorphic light eruption (PLE), photo-aggravated atopic dermatitis (PAD), lupus erythematosus, actinic folliculitis, resolved cases of chronic actinic dermatitis (CAD), solar urticaria (SU), drug-photosensitivity, UVA and UVB sensitivity, actinic prurigo and erythropoietic protoporphyria. Of 83 Patients that underwent monochromatic phototesting, photosensitivity was observed in 22 individuals (n = 22/46) at 305 nm, 32 individuals (n = 32/46) at 335 nm, 37 individuals (n = 37/46) at 365 nm, 13 individuals (n = 13/44) at 400 nm and 4 individuals (n = 4/39) at 430 nm. At 222 nm none of the tested individuals showed an effect after radiant exposure up to 23 mJ $cm^{-2}$. The authors conclude that far-UVC exposure, within ICNIRP EL, is unlikely to pose a risk to patients with above-mentioned photosensitivity disorders. The investigation is limited exclusively to skin responses—specifically acute erythema—and does not address either potential long-term effects or the critical impacts on ocular tissues. Due to possible uncontrolled radiation exposures in occupied environments complete verification of far-UVC safety would require substantially higher doses and studies with repeated exposures.

In accordance with occupational health and safety regulations, workers have to be informed of the risks associated with UV exposure, and various technical, organisational and individual protective measures have to be taken to prevent excessive exposure at the work place. With the objectives of the prevention and timely detection of any adverse health effects, as well as the prevention of any long-term health risks, appropriate health surveillance of workers shall be ensured. Protective measures shall be adapted to the requirements of workers belonging to particularly UV sensitive risk groups. While for workers a high level of protection against UV radiation in the workplace is foreseen, including even individually adapted protective measures, this cannot be guaranteed for people in public spaces. While workers generally belong to a limited age group and are predominantly healthy individuals, the general population also includes other age groups such as children and the elderly. It is highly likely, at least in the elderly population, that vulnerable groups will exist, such as individuals with pre-existing conditions or injuries to the skin and eyes (also due to surgery) as well as individuals taking photosensitising medication. None of these individuals would be aware of the far-UVC exposure in public indoor spaces without the relevant information and would not be able to take any protective action, as already observed by Abboushi et al. (2025). Therefore, the health and safety regulations should also be applied in the presence of far-UVC radiation in public indoor places in order to inform people about the possible risks. However, individual protective measures would not be enforceable in this case.

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It is important to underline that—regardless of the level of the ELs—when using far-UVC radiation in the presence of people in public spaces, an exceedance of the ELs cannot be ruled out because the exposure of individuals cannot be adequately controlled in this case. As the total radiant exposure depends on the exposure duration, prolonged or repeated periods of exposure can cause the ELs to be exceeded. In particular, for anyone who is already exposed to UVC radiation in the workplace, exposure to far-UVC radiation in public spaces would imply additional UVC radiant exposure (cumulation).

Furthermore, monitoring of the technical reliability of the devices employed, e.g., filtering, is not regulated. Several studies have demonstrated that optical filtering is essential when using KrCl excimer lamps, to minimise the proportion of harmful UVB and UVA radiation. The reliability of the lamps and the efficiency of the filtering in the long-term have not yet been studied, however. In addition, there are hardly any standards or regulations on certification or type approval and on monitoring the technical reliability of far-UVC systems. Consequently, there is a risk of uncontrolled circulation of unfiltered or technically unreliable devices through online retail, especially in the private sector.

## 5. Effects of Far-UVC on the Environment

The potential use of far-UVC radiation through unshielded sources of radiation in public indoor spaces raises further questions about the impact on the environment that also need to be considered.

Whether multi-resistant bacteria can develop resistance to UV radiation after repeated exposure was investigated in the study by Choi et al. (2022). They exposed methicillin-resistant *Staphylococcus aureus* (MRSA), carbapenemase-producing *Klebsiella pneumoniae* and metallo-beta-lactamase-producing *Klebsiella pneumoniae* to the UV radiation of a mercury vapour lamp ($UVC_{254}$ radiation) and a xenon lamp. After 25 cycles of UV irradiation, no significant genetic changes were found in these three bacterial strains upon sequencing analyses of the entire genome. However, in this study very high UV radiant exposures were used: 56160 mJ $cm^{-2}$ (irradiance 93.6 mW $cm^{-2}$ for 10 minutes) in the case of the mercury vapour lamp and 11430 mJ $cm^{-2}$ (irradiance 38.1 mW $cm^{-2}$ for 5 minutes) in case of the xenon lamp. If the applied UV radiation is high enough to eradicate the entire bacterial population, no UV-resistant bacterial variants can develop. In studies by Alcantara-Diaz et al. (2004) and Shibai et al. (2017), where *Escherichia coli* bacteria were exposed to lower radiant exposure of UV radiation from a mercury vapour lamp (up to 15 mW $cm^{-2}$ and 10 mW $cm^{-2}$, respectively), the *Escherichia coli* produced mutant strains that were more resistant to UV radiation than the parental strains. When using far-UVC radiation in public indoor spaces—especially at greater distances from the radiation source—radiant exposure sufficient to completely eradicate bacterial populations cannot be guaranteed. Hence, the possible emergence of UVC-resistant bacterial variants as a result of repeated exposure to such radiation must be investigated and taken into account.

The influence of $UVC_{222}$ and $UVC_{254}$ radiation on Arabidopsis plants was investigated in the study of Otake et al. (2021) by irradiating seedlings that were seven days old. In plants exposed to $UVC_{222}$ radiation, the following was observed four days after irradiation: The leaves curled after a radiant exposure of 100 mJ $cm^{-2}$ and were significantly bleached at 1000 mJ $cm^{-2}$. These effects were not observed in non-irradiated plants or plants exposed to $UVC_{254}$ radiation. Furthermore, functional guard cells (which are important to the plant for gas exchange with the surrounding air) were found in the leaves of non-irradiated plants and plants exposed to $UVC_{254}$ radiation with radiant exposure of 100 mJ $cm^{-2}$ and 500 mJ $cm^{-2}$. Conversely, most guard cells were deformed after the same radiant exposure of $UVC_{222}$ radiation. The studies indicated that mitochondria are inactivated or fragmented and that chloroplasts are damaged. The authors conclude that the $UVC_{222}$ radiation severely damages the guard and epidermal cells and that such damage can inhibit growth.

Experiments on a transparent model organism, nematode *Caenorhabditis elegans* were conducted by Yoshiyama et al. (2023). At radiant exposure of 20 mJ $cm^{-2}$ and 40 mJ $cm^{-2}$ ($UVC_{222}$ and $UVC_{254}$), only $UVC_{222}$ was found to cause serious neurodegenerative effects and neuronal damage in the sensory neurons located just 1 µm–2 µm below the surface. The authors assume that $UVC_{222}$-induced

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ROS are primarily responsible for this damage. Comparable damage to the neurons was not observed with $UVC_{254}$ at such radiant exposures, but damage to the oocytes located 20 μm to 60 μm below the surface was reported.

Drungilas et al. (2023) investigated the long-term effects of $UVC_{222}$ radiation on the color and mechanical degradation of materials used to construct public bus interiors. In the bus tested, the interior walls were made of fiber-reinforced composite (FRC; E-glass fiber and vinyl ester resin matrix), the floors of polyvinyl chloride (PVC), passenger seats cover of velour fabric, and passenger handholds were powder coated. Applied irradiances were between from 0.27 mW $cm^{-2}$ and 0.93 mW $cm^{-2}$. The $UVC_{222}$ radiation caused significant color degradation of the FRC and the PVC after 150 hours (radiant exposure of approximately 290 J $cm^{-2}$). These two materials showed significant changes in mechanical properties as well (elasticity modulus, tensile strength, elongation at ultimate strength and elongation at break). A particularly large decrease in elongation at break (up to 26%) was observed in the FRC material. Hurtado Macias et al. (2024) investigated the aging, i.e., the changes in material's properties of polyamide 66 (high-performance plastics frequently used in the automotive, transportation, consumer goods, electrical and electronics industries) caused by $UVC_{254}$, $UVC_{222}$ and $UVB_{313}$ radiation. Fourier transform infrared spectroscopy showed the formation of O-H and/or N-H bonds, more evident in samples exposed to $UVC_{254}$, followed by samples that were irradiated with $UVC_{222}$ radiation. Samples that were aged with $UVB_{313}$ did not show changes even after 500 hours of irradiation.

The impact of far-UVC radiation on indoor air quality has also been investigated.Peng et al. (2023)conducted a model evaluation of the secondary chemistry initiated by $UVC_{254}$ and $UVC_{222}$ radiation in a characteristic indoor environment for different ventilation levels. The evaluation revealed that the $UVC_{254}$ radiation can photolyse ozone molecules ($O_3$) and produce $OH^{\bullet}$ radicals. They oxidise volatile organic compounds (VOCs) and lead to the production of oxygen-containing VOCs (OVOCs) and a secondary organic aerosol (SOA), which can have negative effects on the health (Collins and Farmer, 2021). $UVC_{222}$ radiation with the same virus-inactivation rate has less impact on indoor air quality at moderate to high ventilation levels, mainly due to the lower UV irradiance required and also due to the less efficient $OH^{\bullet}$-generating photolysis than in the case of $UVC_{254}$ radiation. However, if the ventilation level is poor (which can frequently be the case, e.g., in schools), $UVC_{222}$ radiation has a greater impact on room air quality than $UVC_{254}$ radiation due to the low but significant photochemical production of $O_3$ at 222 nm. The authors emphasize that only a very limited number of chemical substances were modelled in the study and that further studies are needed to better assess the toxicity of gas-phase products. Consideration must also be given to interiors with polluted air where the concentration of VOCs can be very high. Further, Liang et al. (2024) studied secondary aerosol formation by UVC radiation on $SO_2$, a precursor of outdoor sulfate aerosol, and found that the formation of sulfate nanoparticles was much more effective at $UVC_{222}$ than at $UVC_{254}$ at the same average irradiance. Goss and Kroll (2025) revealed new particle formation under $UVC_{222}$ radiant exposure of 45 mW $cm^{-2}$, that differs substantially from chemistry expected from reaction with ozone. The authors recommend keeping $UVC_{222}$ radiant exposure to the lowest effective levels when used in indoor spaces. Also, Narouei et al. (2025), who conducted a series of experiments to assess formation of SOA in an office environment induced by $UVC_{222}$, recommend the use of $UVC_{222}$ at minimum irradiations required for disinfection in conjunction with adequate ventilation rates in order to minimize the formation of air pollutants in furnished indoor environments.

## 6. Conclusions

In summary, given the novelty of the use and the potentially harmful photobiological effects of far-UVC radiation, it appears that there is insufficient data to rule out health risks to the population from its use in the presence of people in public indoor spaces.

Currently available studies into potential health effects on humans do not cover important aspects for assessing the risks of far-UVC radiation. The vast majority of studies published to date have dealt solely with short-term exposure to far-UVC radiation, while chronic exposure has only

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been investigated in a few studies. The effect of far-UVC radiation on eye has so far mainly been investigated in animal models. There are very few studies that directly involve the human eye, e.g., Sugihara et al. (2025b), reporting temporary sensations of dryness and epiphora. There are no studies that include potentially vulnerable groups and likewise also no studies into the effect of far-UVC radiation on injured or damaged skin (Busch et al., 2023). No epidemiological data is available concerning the effect of far-UVC radiation on the skin and eyes.

While the studies to date relate primarily to cellular DNA damage, hardly any consideration is given to other potential targets of far-UVC radiation. Since low penetration depth of far-UVC radiation in biological tissue is attributable to the high level of absorption by proteins, some of the inactivating effects, especially with respect to viruses, are attributed to protein damage. In the human tear fluid more than 1500 proteins were identified (Zhou et al., 2012). Therefore, the tear fluid, with its complex composition and diverse tasks, could also be adversely affected by inhibition of enzymes (proteins responsible for catalysis of biochemical reactions), and such inhibition, as well as the underlying photochemical mechanism, are not thoroughly understood.

Also, the harmful effect of far-UVC radiation on the microbiome of the skin and surface of the eye has not been studied. Even when taking into account the frequent use of hand disinfection to reduce the risk of infectious diseases, the effect of unplanned, prolonged exposure of the skin and eyes to far-UVC radiation on the microbiome must be viewed critically. If people are exposed to far-UVC radiation in public indoor spaces, it cannot be ruled out that this will lead to a disruption or even inactivation of the microbiome of the exposed skin and eye surface and thus to “hyper-disinfection”. Such an effect should be investigated more closely, especially with respect to the latest data on the importance of the microbiome in and on humans.

Furthermore, the majority of the studies to date show ambiguities with respect to radiometric measurements, since the validity of the specified radiant exposure has not been adequately demonstrated and information on irradiance levels, irradiation duration and radiometric measurement methods is often absent. In some cases, the information on the employed measuring devices raises the question of whether it was even possible to correctly determine the applied radiant exposure.

Last but not least, since conflicts of interest have been declared in numerous relevant studies, the conclusions may be subject to a relevant bias.

The potential use of open sources of radiation in the presence of people in public indoor spaces requires also fundamentally new radiation safety considerations as to how and with what exposure duration far-UVC radiation can be used. First of all, when using UVC radiation in the presence of people, the ELs recommended by the ICNIRP and legally specified for workers must be complied. However, there is no concept for verifying this compliance in public indoor spaces, as the exposure of individuals there cannot be adequately controlled and therefore exceeding the ELs cannot be ruled out. Secondly, precautionary measures cannot currently be defined to minimise or prevent harmful effects on the health of specific groups of people, such as children, the elderly, individuals with pre-existing skin or eye diseases, or individuals taking photosensitising medication, as the existing ELs do not consider such vulnerable groups.

It should also be noted that the ICNIRP explicitly supports the principle that any decision that changes the exposure situation should bring more benefit than harm (International Commission on Non-Ionizing Radiation, 2020). According to current knowledge, however, it cannot be assumed that the use of far-UVC radiation for disinfection in the presence of people would comply with this basic principle. Technically and economically viable approaches already exist which achieve significant pathogen inactivation while minimizing or eliminating direct human exposure to UVC radiation. For instance, closed-unit systems or shielded UVC sources ensure effective disinfection without exposing occupants to radiation. In their review of methods for managing infectious aerosols to reduce transmission and protect the public, Lerner et al. (2025) recommend upper-room UVC in settings where most occupants regularly spend a significant proportion of their day.

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Finally, the use of far-UVC radiation in public indoor spaces raises questions regarding the effects on the human environment, such as the possible emergence of UVC-resistant bacterial variants, the impact on flora and fauna, the long-term influence on indoor materials/plastics or the possible emergence of harmful chemical compounds indoors.

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