Scientific Committee on Emerging and Newly Identified Health Risks

SCENIHR

Health Effects of Artificial Light

The SCENIHR adopted this opinion at its 17th plenary meeting on 19 March 2012

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SCENIHR

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All Declarations of working group members and supporting experts are available at the following webpage:
http://ec.europa.eu/health/scientific_committees/emerging/members_wg/index_en.htm

ABSTRACT

A: *Potential health impacts on the general public caused by artificial light*

In general, the probability is low that artificial lighting for visibility purposes induces acute pathologic conditions, since expected exposure levels are much lower than those at which effects normally occur, and are also much lower than typical daylight exposures.

Certain lamp types (including also incandescent light bulbs) may emit low level UV radiation. According to a worst case scenario the highest measured UV emissions from lamps used in offices and schools, but not the very low emissions lamps used for household lighting, could add to the number of squamous cell carcinomas in the EU population.

There is no evidence that blue light from artificial lighting belonging to Risk Group 0 ("exempt from risk") would have any impact on the retina graver than that of sunlight. Blue light from improperly used lamps belonging to Risk Groups 1, 2, or 3 could, in theory, induce photochemical retinal. There is no evidence that this constitutes a risk in practice. Other damages to the eye from chronic artificial light exposure during normal lighting conditions are unlikely. Exposure to light at night (independent of lighting technology) while awake (e.g. shift work) may be associated with an increased risk of breast cancer and also cause sleep, gastrointestinal, mood and cardiovascular disorders.

B: *Aggravation of the symptoms of pathological conditions*

UV, and in some patients, visible light can induce skin lesions of true photodermatoses. Although sunlight is reported by most patients as the main trigger of disease activity, artificial lighting is reported to play a role in some cases.

The blue or UV components of light tend to be more effective than red components in aggravating skin disease symptoms related to pre-existing conditions such as lupus erythematosus, chronic actinic dermatitis and solar urticaria. UV and/or blue light could also possibly aggravate the systemic form of lupus erythematosus.

It is recommended that all patients with retinal dystrophy should be protected from light by wearing special protective eyeware that filters the shorter and intermediate wavelengths.

The previous SCENIHR opinion on Light Sensitivity stated that modern CFLs are basically flicker-free due to their electronic high frequency ballasts. However, some studies indicated that perceivable flicker can occur during certain conditions with both CFLs and incandescent lamps. This statement is still valid.

There is no scientific evidence available to evaluate if conditions such as Irlen-Meares syndrome, myalgic encephalomyelitis, fibromyalgia, dyspraxia, autism, and HIV are influenced by the lighting technologies considered in this opinion.

C: *Risk estimates and mitigations*

Short-term UV effects from artificial lighting on healthy people are thought to be negligible. A proper assessment of long-term risks due to daily low level UV exposure is not possible since relevant exposure data are lacking. A worst case scenario, with assumptions of validity in extrapolation from animal to studies to human conditions, involved workplace/school exposure to double- or single-capped fluorescent lamps with the highest identified UV radiation. Such exposure may add to the annual UV dose (e.g. comparable to the increased dose obtained during an annual one week vacation in a sunny location), and increase the risk of squamous cell carcinomas correspondingly.

Improper use of lamps belonging to Risk Groups 1-3 could cause retinal damage, which would be avoidable with appropriate measures.

The current standardization of lighting lamps and luminaires in four risk categories appears sufficient to limit the personal short-term risk. However, Risk Group 0 should not be taken to imply adequate protection of the general population as a whole from long-term UV-exposure effects.

There are a number of patients (around 250,000 EU citizens; SCENIHR 2008) that are exceptionally sensitive to UV/blue light exposure. The risk for this group of patients includes all light sources with significant UV/blue light emissions. It may be advisable to make sufficient information on the emitted spectrum for individual lamp models available to the healthcare professionals and the patients to allow them to choose their lighting solutions optimally.

D: *Potential research needs*

Several areas where relevant data are lacking regarding the effects of specific lighting technologies on medical conditions have been identified. The most important areas where knowledge gaps have to be filled in order to be able to draw firm conclusions are outlined in the opinion.

Keywords: artificial light, incandescent lamps, fluorescent lamps, compact fluorescent lamps, halogen lamps, LED, public health, human health, SCENIHR, Scientific Committee on Emerging and Newly Identified Health Risks

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EXECUTIVE SUMMARY

The SCENIHR delivered an opinion on Light Sensitivity on 23 September 2008 (SCENIHR 2008), which identified blue light and ultraviolet radiation "as a potential risk factor for the aggravation of the light-sensitive symptoms in some patients with such diseases as chronic actinic dermatitis and solar urticaria". The committee also noted that "some single-envelope compact fluorescent lamps (CFLs) emit UVB and traces of UVC radiation. Under extreme conditions (i.e. prolonged exposures at distances less than 20 cm) these CFLs may lead to UV exposures approaching the current workplace limit set to protect workers from skin and retinal damage." However, "the use of a second lamp envelope or similar technology could largely or entirely mitigate both the risk of approaching workplace limits on UV emissions in extreme conditions and the risk of aggravating the symptoms of light-sensitive individuals."

The purpose of the present opinion is to update the conclusions of the SCENIHR opinion on Light Sensitivity as appropriate and to carry out an analysis of a wider range of lighting technologies and of associated potential health risks. In addition, if available data do not allow such analysis, the SCENIHR is asked to identify relevant research needs.

The opinion is based on a scientific rationale which has taken into account the relevant scientific literature and other accessible and reliable information on physical and technical characteristics of lighting technologies, principles of optical radiation, as well as biological and health effects of optical radiation. Health effects due to optical radiation have been considered both for the general population and for persons with photosensitive or other pathological conditions. Since the assignment also includes evaluation of possible health effects of various types of lighting technologies, additional data regarding lamp emissions were requested and some were obtained from stakeholders. In addition, for assessment purposes, data regarding exposure patterns were sought, but found to be virtually lacking. This lack of information has seriously hampered efforts to perform specific risk assessments.

The SCENIHR's answers to the questions given in the Terms of Reference are given below:

> ***A:*** *To explore and report scientific evidence on potential health impacts on the general public caused by artificial light of which the main purpose is to radiate in the visible range (as opposed to artificial light where the invisible part of the radiation is the main purpose, e.g. suntanning lamps or infrared lamps). The impacts of the light from all available electrical lighting technologies should be studied, both in the visible and invisible range (with specific analyses of the ultraviolet radiation subtypes UVA, UVB and UVC).*

A combined assessment of natural and artificial light shows that adverse health effects due to optical radiation can either occur acutely at certain levels of exposure, or after long-term repeated exposures at lower levels. Depending on the effect (endpoint) of concern (e.g. skin burn, skin cancer, retinal damage, cataract) either intensity or duration of exposure is of most relevance. In general, the probability that artificial lighting for visibility purposes induces any acute pathologic conditions is low, since the levels of maximum exposure are normally much lower than those where such effects are known to occur in healthy people and certainly much lower than in typical summer daylight.The available lamp emission data show that for all investigated hazard outcomes, the absolute majority of lamps are classified as Risk Group 0 (RG0; "exempt from risk"). Most of the rare exceptions are classified as Risk Group 1 (RG1; "low risk"). The very few lamps assigned to higher Risk Groups were either measured without the required UV-shielding glass cover, or at a very short distance (20 cm) which is not the intended use distance for this lamp type.

A common exposure situation, such as most household lighting, would involve an illumination level which is so low that exposure to potentially hazardous radiation is considered negligible. However, according to a worst case scenario, the highest measured emissions of UV from fluorescent lamps used typically indoors in professional environments, although well below the limits for RG0, could be contributing to the number of squamous cell carcinomas in the EU population. This is in comparison to a hypothetical situation where the same population is not exposed to UV radiation from artificial light indoors. The annual erythemal UV dose expected from the worst case scenario approximately corresponds to the dose one would get from a half week sunshine holiday. The fluorescent lamps measured in the reviewed studies emitted from less than 1% to at most 33% of the UV radiation assumed in the worst case scenario.

UV emissions may occur from certain lamp types (quartz halogen lamps, single- and double-capped fluorescent lamps as well as incandescent light bulbs), although at low levels. These emissions may, in some cases, in particular for certain halogen lamps with poor UV filtering, also include UVC in addition to UVA and UVB. Most action spectra on skin and eye effects include UVC. Hence, biologically effective doses take UVC into account and are thus considered in the categorisation of the Risk Group. However, detectable levels of UVC do signal a considerable overall output of biologically harmful short wavelength UV radiation.

Regarding a possible need for separate UVA, UVB or UVC radiation limits for tungsten halogen lamps and other light sources that emit UV radiation, the Scientific Committee considers that there is no scientific basis for making such specific recommendations beyond the established dose limits.

Evidence from in vitro experiments suggest that blue light at 10 W/m^2 induces photochemical retinal damages (Class II) upon acute (hours) exposure, and animal experiments and *in vitro* studies suggest that cumulative blue light exposure below the levels causing acute effects also can induce photochemical retinal damage. There is no consistent evidence from epidemiological studies regarding the effect of long-term exposure to sunlight (specifically the blue component of sunlight) and photochemical damage to the retina (particularly to the retinal pigment epithelium), which may contribute to age-related macular degeneration (AMD) later in life. Whether exposure from artificial light could have effects related to AMD is uncertain.

There is no evidence that artificial light from lamps belonging to RG0 or RG1 would cause any acute damage to the human eye. Studies dedicated to investigating whether retinal lesions can be induced by artificial light during normal lighting conditions are not available. Lamp types belonging to RG2 and higher are usually meant to be used by professionals in locations where they do not pose a risk.

Chronic exposure to blue light from improperly used lamps could, in theory, induce photochemical retinal damage in certain circumstances. There is however no evidence that this constitutes a risk in practice. It is unlikely that chronic exposures to artificial light during normal lighting conditions could induce damage to the cornea, conjunctiva or lens.

Besides the beneficial effect of light, e.g. through synchronising the day-night rhythm, there is mounting evidence suggesting that exposure to light at night while awake (especially during shiftwork), may be associated with an increased risk of breast cancer and also cause sleep, gastrointestinal, mood and cardiovascular disorders and possibly through circadian rhythm disruption. Importantly, these effects are associated with light, without any specific correlation to a given lighting technology.

> ***B:*** *To update the SCENIHR report on Light Sensitivity (from 23 September 2008) in light of further evidence, and to examine the aggravation of the symptoms of pathological conditions in the presence of lamp technologies other than compact*

fluorescent lamps (including conventional incandescent and halogen lamps, halogen lamps with improved efficiency and light emitting diode lamps).

The SCENIHR opinion on Light Sensitivity (SCENIHR 2008) identified that some pre-existing conditions (epilepsy, migraine, retinal diseases, chronic actinic dermatitis, and solar urticaria) could be exacerbated by flicker and/or UV/blue light. However, at that time there was no reliable evidence that compact fluorescent lamps (CFLs) could be a significant contributor. More recent studies indicate a negative role for certain CFLs and other artificial light sources (sometimes including incandescent bulbs) in photosensitive disease activity. There are no published data on the effect of exposure of a photosensitive patient to light from halogen lamps.

There is strong evidence that UV, and in some patients, visible light can induce skin lesions of true photodermatoses. Although sunlight is reported by most patients as the main trigger of disease activity, occasionally severely affected patients over the range of endogenous (and exogenous) diseases report a provocative role for artificial lighting.

There is evidence that the shorter wavelength light components (blue or UV) tend to be more effective than the longer wavelength components (red) in aggravating skin disease symptoms related to pre-existing conditions such as lupus erythematosus, chronic actinic dermatitis and solar urticaria. In the case of lupus erythematosus, UV or and/or blue light possibly also aggravate the systemic disease.

With the considerable variability of UV/blue light components for lighting technologies of the same or a similar kind, no general advice can be given and individual optimisation of the lighting technology is advised for these patients. Notably, LED type of lighting is on technical grounds providing a sharper cut-off at shorter wavelengths than any of the incandescent (halogen and non-halogen) and fluorescent (compact and conventional) light sources. Generally, double envelope CFLs emit much less UV radiation than single envelope CFLs.

The effect of light is variable depending on the genetic alterations that cause inherited retinal degeneration. For specific conditions such as Stargart disease, the retinal pigment epithelial (RPE) cells are particularly sensitive to Class II photochemical damage, which is induced by peaks at shorter wavelengths. In other retinal dystrophies, light does not exert any aggravating effect. However, since the causative mutation is seldom known, and because there is no clear correlation between genotype and phenotype, it is recommended that all patients with retinal dystrophy should be protected from light by wearing special protective eyeware that filters the shorter and intermediate wavelengths.

The previous SCENIHR opinion on Light Sensitivity stated that modern CFLs are basically flicker-free due to their electronic high frequency ballasts. However, it was also noted that studies indicated that residual flicker can occur during certain conditions. This statement is still valid.

There is no scientific evidence available to evaluate if conditions such as Irlen-Meares syndrome, myalgic encephalomyelitis, fibromyalgia, dyspraxia, autism, and HIV are influenced by the lighting technologies considered in this opinion.

***C:** If health risks are identified under points A or B, to estimate the number of EU citizens who might be at risk and identify the level of emission/exposure safeguarding the health of citizens and/or means to mitigate or entirely prevent the impact of the problematic parameter of the light technology in question.*

Short-term UV effects from artificial lighting on healthy people are thought to be negligible. A proper assessment of long-term risks due to daily low level UV exposure is not possible, because data on actual personal indoor UV exposure are lacking. A worst case scenario examined in this opinion involved workplace/school exposure to double- or single-capped fluorescent lamps in ceiling-mounted open luminaires. This scenario assumes the validity of extrapolating from studies on animals with short lifespans to life-

time human exposures.. Furthermore, it assumes the appropriateness of dose-level extrapolation from experimental studies to real human exposures and that all individuals in a population experience the same risk independent of susceptibility factors. Exposure from lamps with the highest measured UV output (still well within Risk Group 0) adds the equivalent of 3 to 5 days vacation in a sunny location to the average annual UV dose. Although this would lead to an increase in the personal risk of squamous cell carcinoma, such an increase would remain small (a few % over a lifetime in Denmark). Population-wide exposure to such lamps could, however, add approximately 100 cases of squamous cell carcinomas a year to a base line of 900 cases/year in Denmark. It should be stressed that the UV output of most of the fluorescent lamps tested fall well below this level, and are not expected to affect squamous cell carcinoma incidences to any appreciable extent. Improper use of lamps belonging to Risk Groups 1-3 (due to missing or disregarded user information, non-professional installation) could cause retinal damage. While the number of such cases remains currently unknown, appropriate measures could be considered to ensure that these lamps are not misused.

The current standardization of lighting lamps and luminaires in four risk categories appears sufficient to limit the personal short-term risk. However, RG0, as it is based on acute effects, should not be taken to imply adequate protection of the general population as a whole from effects after long-term exposure to UV radiation. Nevertheless, it would be useful to communicate information on risk categories to the consumer.

It was stated in the previous SCENIHR opinion (SCENIHR 2008) that there are a number of patients that are exceptionally sensitive to UV/blue light exposure. The number of EU citizens with light-associated skin disorders that would be affected by exposures from CFLs was estimated in the report to be around 250,000. Clearly, the risk for this group of patients is not limited to CFLs, but includes all light sources with significant UV/blue light emissions. The lack of proper data precludes any improvement of the estimate of the size of the affected group.

Also photosensitive patients undergoing photodynamic therapy might be expected to react to CFL and LED sources to a greater extent than to incandescent lighting. This is due to a combination of greater sensitivity of porphyrins to blue light coupled with an enhanced blue light emission of these sources. Such patients need careful management.

For patients with light-associated skin disorders, the previous SCENIHR opinion recommended that if using CFLs, a double envelope type is preferable since this reduces the UV component of the particular lamp type. Available data, however, show a high variability of UV and blue light emission due to different internal design parameters even in presence of a second envelope. For these patients, retrofit LED lighting, which does not emit UVR, would provide an even better option than CFLs. Double envelope CFLs generally emit much less UV radiation than single envelope CFLs and are much less likely to induce a reaction in patients with light-associated skin disorders. The UV/blue light irradiation from halogen lamps is also highly dependent on the lamp type. With lamps other than incandescent retrofit halogen bulbs, attention needs to be given to the proper installation as they are at times sold by the manufacturer to be installed at larger distances or in conjunction with special luminaires or filters against e.g. UV or IR irradiation. While it is unlikely that there would be a significant UV risk from halogen lamps for the general public, provided that protective measures are complied with, the UV content can rise to levels which are of concern for patients with light-associated skin disorders at close operating distances and long exposure times.

In view of the large number of patients affected by photosensitive diseases it may be advisable to make sufficient information on the emitted spectrum for individual lamp models available to the healthcare professionals and their patients to allow them to choose their lighting solutions optimally.

D: *To identify potential research needs related to the areas where the lack or scarcity of scientific evidence prevents SCENIHR from coming to firm conclusions.*

The Scientific Rationale has identified a number of areas where relevant data are lacking regarding the effects of specific lighting technologies on medical conditions. The most important areas where knowledge gaps have to be filled in order to be able to draw firm conclusions related to this opinion include:

- Emission data (ranging from UVC up to 800 nm) characterizing the different lighting technologies – a challenge due to the variation of manufacturing parameters, and a database of these characteristics of specific lamps on the European market.
- Exposure database on indoor visible light radiance to the eye and personal UV exposures from various lamp types compared to ambient outdoor exposure. Such a database should be established in view of the potential conditioning of the eye due to the largely different voluntary exposure to sunlight from one individual to another, and also for the very different use patterns for UV/light protective eyewear between individuals and populations.
- Attention should be paid to develop a risk group categorisation that takes into account potential chronic effects like SCC.

- Eye conditions:

  a. Epidemiologic studies of artificial light exposure and ocular pathologies (including AMD); and

  b. Retinal effects of chronic exposure to artificial light for visibility purposes (animal studies).
- The role of various types of artificial lighting sources in photosensitive skin diseases (provocation studies).
- Mechanisms and consequences of exposure to artificial light in the late evening, at night and in the early morning, including circadian disruptions in both shift-workers and in the general population.
- Flicker induced health effects from the residual high frequency (100-120 Hz) intensity modulations.
- The particular role of UVC components in artificial lighting for skin diseases taking into account especially sensitive populations and the role of prior exposure to sunlight.
- The effects of non-incandescent light sources, in particular those with very inhomogeneous or biased spectral distribution on colour rendition, fatigue, and other components of the human visual perception.

1. BACKGROUND

Within the context of the promotion of the wide-spread use of energy saving lamps such as compact fluorescent lamps, and the upcoming phase-out of incandescent lamps, the Commission mandated SCENIHR in April 2008 to look into the claims of light sensitive citizens' associations such as Right to Light, Spectrum Alliance and Lupus UK that the symptoms of some diseases are, or could be, aggravated in the presence of energy saving lamps (mainly compact fluorescent lamps).

In reply to this mandate, SCENIHR delivered an opinion on Light Sensitivity on 23 September 2008 (SCENIHR 2008), which identified blue light and ultraviolet radiation "as a potential risk factor for the aggravation of the light-sensitive symptoms in some patients with such diseases as chronic actinic dermatitis and solar urticaria". The committee also noted that "some single-envelope compact fluorescent lamps (CFLs) emit UVB and traces of UVC radiation. Under extreme conditions (i.e. prolonged exposures at distances less than 20 cm) these CFLs may lead to UV exposures approaching the current workplace limit set to protect workers from skin and retinal damage." However, "the use of a second lamp envelope or similar technology could largely or entirely mitigate both the risk of approaching workplace limits on UV emissions in extreme conditions and the risk of aggravating the symptoms of light-sensitive individuals."

Since the publication of the opinion and the adoption of Commission Regulation 244/2009 setting ecodesign requirements for non-directional lamps (in practice phasing out incandescent lamps by 2012), further claims and facts relating to light sensitivity and to the potential health effects of artificial light have been brought to the attention of the Commission:

1. Light sensitive citizens' associations have contested some of the conclusions of the SCENIHR opinion on Light Sensitivity, arguing that a wider range of disease states are affected by the light of compact fluorescent lamps than those identified by SCENIHR. They also question the effectiveness of the technology supposed to prevent the aggravation of their symptoms in the presence of compact fluorescent lamps (second lamp envelope or other similar technology).
2. Light sensitive citizens' associations claim that some of their members experience an aggravation of their symptoms when exposed to the light of screw-base halogen bulbs with improved efficiency. The potential effect of light emitting diode (LED) lamps on light sensitive patients is also largely unknown, although there are already reports of LED lamps aggravating symptoms.
3. Measurements provided by the European Lamp Companies Federation show that the proportion of UVC radiation emitted by tungsten halogen lamps may be quite high compared to the total UV radiation (up to 67% of total UV radiation for a 70 W halogen capsule with G9 cap, i.e. 0.074 mW/klm UVC out of 0.11 mW/klm total UV). For higher wattage lamps without UV block, UVC radiation is also high in absolute terms (0.973 mW/klm for 300 W lamps). The harmonised standards applicable to the UV radiation of tungsten halogen lamps (EN 60432-2 and EN 60432-3)[1] allow up to 2 mW/klm total UV radiation; however they do not have separate requirements for UVC radiation. To the Commission's knowledge, it has not yet been explored whether it would be justifiable to set out separate UVA, UVB and UVC radiation limits for tungsten halogen lamps and other light sources and if so, what those values should be.

[1] EN 60432-2:2000 Incandescent lamps — Safety specifications — Part 2: Tungsten halogen lamps for domestic and similar general lighting purposes and Amendment A1:2005 to EN 60432-2:2000.

EN 60432-3:2003 Incandescent lamps — Safety specifications — Part 3: Tungsten-halogen lamps (non-vehicle) and Amendment A1:2005 to EN 60432-3:2003.

4. Some press articles claim that according to recent research, artificial light with a strong blue component could affect human circadian cycles and the hormonal system, and could result in diseases ranging from sleep disorders, immune system disorders, macular degeneration, cardiovascular diseases, diabetes and osteoporosis to breast cancer. Some comparisons of the light of different artificial light sources claim further health disadvantages related to fluorescent lamps as compared to incandescent lamps.

Taking into account the above, it is considered necessary to ask SCENIHR to update the conclusions of its opinion on Light Sensitivity as appropriate and to carry out an analysis of a wider range of lighting technologies and of associated potential health risks. Considering the scarcity of scientific evidence in relation to many of the questions raised, the assessment of the plausibility of the alleged health effects followed, if required, by the identification of potential research needs, is likely to be an important part of SCENIHR's work.

2. TERMS OF REFERENCE

Against the above background, SCENIHR is requested:

A. To explore and report scientific evidence on potential health impacts on the general public caused by artificial light of which the main purpose is to radiate in the visible range (as opposed to artificial light where the invisible part of the radiation is the main purpose, e.g. suntanning lamps or infrared lamps). The impacts of the light from all available electrical lighting technologies should be studied, both in the visible and invisible range (with specific analyses of the ultraviolet radiation subtypes UVA, UVB and UVC).

B. To update the SCENIHR report on Light Sensitivity (from 23 September 2008) in the light of further evidence, and to examine the aggravation of the symptoms of pathological conditions in the presence of lamp technologies other than compact fluorescent lamps (including conventional incandescent and halogen lamps, halogen lamps with improved efficiency and light emitting diode lamps).

C. If health risks are identified under points A or B, to estimate the number of EU citizens who might be at risk and identify the level of emission/exposure safeguarding the health of citizens and/or means to mitigate or entirely prevent the impact of the problematic parameter of the lighting technology in question.

D. To identify potential research needs related to the areas where the lack or scarcity of scientific evidence prevents SCENIHR from drawing firm conclusions.

The scope of the analysis under points A and B should cover all electrical lamp technologies, including conventional incandescent and halogen lamps, halogen lamps with improved efficiency, single-capped (compact) and double-capped fluorescent lamps, high-intensity discharge lamps and light emitting diode lamps. The full range of possible lamp luminous fluxes and lamp voltages (mains voltage, extra low voltage and other low voltages) should be covered by the analysis of each technology, and if appropriate, separate conclusions should be drawn for the different voltage/luminous flux categories.

3. SCIENTIFIC RATIONALE

3.1. Introduction and scope

Ever since man started to consciously use fire and thus light for improving vision during dark parts of the day, humans have strived to improve on the quality of light sources. With the advent of electricity it was possible to develop the technology that has been used for incandescent light bulbs. The present day society displays a plethora of further developments in light technology (see Annex I – Technical Characteristics of Lighting Technologies) where energy aspects as well as ergonomic and other considerations have their place. It is well established that humans and other biological entities are sensitive to light to various degrees, and that normal physiological processes can be, and are, influenced by light from natural or artificial sources. Typical for the modern society is that the all-encompassing use of artificial light sources disturbs the normal conditions of light at day and darkness at night. This has the potential to disturb circadian rhythms, but to what extent, if any, the common light sources also have negative effects on human health is unclear.

The impact of one type of modern light technology, compact fluorescent lamps (CFLs), on human health issues was covered by an earlier SCENIHR opinion (SCENIHR 2008). The main conclusions from that opinion were that there were no direct scientific data on the relationship between this specific form of energy saving light bulb and a number of symptoms in patients with various conditions. For some of these conditions (epilepsy, migraine, and retinal diseases), it was identified that either flicker and/or UV/blue light could exacerbate the effects. The evidence in regard to the skin conditions chronic actinic dermatitis and solar urticaria was found to be related to UV/blue light emissions only. However, at the time of writing the report, there was very little reliable evidence that emission from fluorescent tubes was a significant contributor. Furthermore, it was noted that certain CFLs can under specific conditions emit UVB and traces of UVC radiation. In addition, CFLs emit a higher proportion of blue light than incandescent lamps. Both these types of emissions can be risk factors for the aggravation of symptoms in some patients suffering from chronic actinic dermatitis and solar urticaria.

The purpose of the scientific rationale is to take into account relevant scientific data from the fields of physics, engineering, biology, and medicine, and assess whether optical irradiation from all types of common light sources can cause disease conditions or aggravate already existing conditions.

The use of the light sources discussed in this opinion may also expose the general public to risks not originating from the optical radiation. These potential risks (e.g. fire hazards, cuts, heat, electric shocks, electromagnetic fields, mercury, etc.) are either well known and/or discussed in other opinions from the Scientific Committees (SCENIHR 2008, SCHER 2010). Furthermore, recent media reports on emissions of certain chemicals from energy saving lamps are not discussed in this opinion, but will be dealt with by other public bodies. Also exposure to light from specialized technologies such as operating lamps is not included in this opinion. Thus, the focus of this opinion is on possible effects from optical radiation emanating from artificial light sources.

3.2. Methodology

The health risks of artificial light have been investigated through different approaches such as epidemiologic studies, experimental studies in humans, experimental studies in animals, and cell culture studies. A health risk assessment evaluates the evidence within each of these areas and then weighs together the evidence across the areas to generate a combined assessment. This combined assessment addresses the question of whether or not a hazard exists, i.e. if there is a causal relationship between exposure and some adverse health effect. The answer to this question is not necessarily a definitive yes or no, but may express the weight of evidence for the existence of a hazard. If such a

hazard is judged to be present, the risk assessment should also address the magnitude of the effect and the shape of the dose-response function used for characterizing the magnitude of the risk for various exposure levels and exposure patterns. Detailed criteria that are used to evaluate the documents which the opinion is based on and criteria for the weighting process will be described in a forthcoming SCENIHR memorandum/position statement (to be published in 2011).

Information has primarily been obtained from reports published in international peer-reviewed scientific journals in the English language. Additional sources of information have also been considered, including web-based information retrieval, and documents from Governmental bodies and authorities, non-Governmental organizations (NGOs), and the lighting industry. Furthermore, during the work process it was deemed necessary to issue a call for information regarding the physical characteristics of light emissions from the various types of lamp that have been considered. The call was initially published for a four week period on 11 May 2010 and was later extended until 2 July 2010. The request allowed stakeholders to provide specific information on the spectral power distribution of current technology light sources given both as spectral irradiance and as spectral radiance. Responses were received from several individuals, organizations, and companies. The information received has been evaluated carefully and was useful for writing the opinion.

The weight of evidence for a particular outcome is based on data from human, animal and mechanistic studies (the primary evidence) along with exposure. For each line of evidence, the overall quality of the studies is taken into account, as well as the relevance of the studies for the issue in question. The weighting also considers if causality is shown or not in the relevant studies. In the present opinion, the following categories are used to assign the relevant weight of evidence for the specific outcomes.

Strong overall weight of evidence: coherent evidence from human and one or more other lines of evidence (animal or mechanistic studies) in the absence of conflicting evidence from one of the other lines of evidence (no important data gaps).

Moderate overall weight of evidence: good evidence from a primary line of evidence but evidence from several other lines is missing (important data gaps).

Weak overall weight of evidence: weak or conflicting evidence from the primary lines of evidence (severe data gaps).

In addition to these three categories, based on the available data, it is noted that there is a lack of data to scientifically weigh the evidence in certain cases. This might be because there was either a general lack of studies, or most studies that were available were classified as being inadequate for the risk assessment.

Throughout the opinion, consistency and adherence to SI (International System of Units, Système International d'unités) regarding the use of terms and units has been attempted. However, since photobiology and photochemistry represent the fusion of several scientific disciplines it has occasionally been necessary to accept the use of a technical terminology which is not standardized, but the convention in a certain discipline.

3.3. Physical characteristics of artificial light sources

3.3.1. Physical principles

Light is electromagnetic (EM) radiation in the range from 400 to 780 nm (1 nm is 10^{-9} m) that is visible to the intact adult human eye (see also CEI/IEC 62471/2006). Light, like EM radiation in general, is emitted by the transition of quantum states if excess energy is to be released in this specific wavelength range. Light sources experienced in nature include different physical phenomena involving atomic/electronic de-excitation

processes induced e.g. by heat, inelastic collisions and nuclear reactions. Examples include: (1) the glowing appearance of fires, flames and other sources e.g. volcanic hot material, where thermal radiation is released; (2) the photochemical light generation of animals such as the glow-worm; (3) the Nordic light (aurora borealis) when showers of elementary particles are trapped by the earth's magnetic field and hit the outer atmosphere; (4) the bright sensation of the electric discharge through the air in lightning, and last but not least (5) the light emitted by the sun, which emerges from the hot plasma induced by hydrogen to helium fusion.

The light which is incident to a certain user or observer is not only dependent on the initial light emission characteristics in the light source, but also on the typical and often frequency dependent light absorption properties of the medium between the light emission and the observer. This is normally provided by the medium in which light is generated as well as its envelope or surrounding (air). A further aspect, which is important to consider, is the geometric arrangement of the source and the user/observer, possibly wearing eyewear, as well as the geometry and reflective properties of the room and/or the luminaire.

Lighting by flames (e.g. candles and oil lamps) was historically the predominant source of light, until electrical heating of filaments (carbon and then tungsten) came to dominate the field. Common to all these lighting applications is that matter is heated to a suitable temperature in order to emit thermal broadband radiation. Numerous thermal excitation and de-excitation processes occur, and are involved in, the generation of light, which leads to a characteristic "bell shaped" spectrum governed by Planck's law of radiation. This law predicts that with increasing temperature of the irradiating material, the peak intensity of the irradiated electromagnetic spectrum is observed at higher characteristic frequencies. This implies that at about 5,000 K the emitted spectrum is similar to that of the sun's radiation through clear skies at midday. Therefore each lamp (and each spectrum) can be associated with a "colour temperature", which describes the sensation of this light on the human eye and on a photographic film, and also affects the colour perception. Also characteristic to the emission from these sources is an increasing fraction of blue light and ultraviolet radiation with a higher operating temperature.

3.3.2. Artificial light technologies

For centuries, mankind has essentially used burning or heated materials as light sources (incandescence). However, it was well known that light could also be generated without heating (luminescence). Thus, bio-luminescence (fire-flies, glow-worms, glowing mushrooms, etc.), phosphorescent minerals, as well as lightning were observed by prehistoric human beings. Today, **flame-operated lamps** (mainly kerosene, carbide and gas lamps) and candles are still in use. Such lamps use a chemical reaction to heat material (soot particles in oil lamps, carbide lamps, and barium oxide particles on a glow-body for gas lamps). The emitted spectrum is continuous and further characterized by a correlated colour temperature, which is often low due to the limited temperature of the irradiating component and by a poor luminous efficacy.

Beyond flame-operated lamps, which are still used in everyday life by approximately 1.6 billion people who do not have access to an electrical grid, the major part of the world population uses **electrical-powered lamps** for producing artificial light. In 2005, 3,418 TWh of electricity, which represents roughly 19% of world electricity production, was used for producing 133 Plmh (peta-lumen-hours) of artificial light (Brown 2009, Waide and Tanishima 2006). According to the same authors, on average 43% of this electricity is used for illuminating tertiary-use buildings, 31% for residential lighting, 18% for industrial buildings, and finally 8% for outdoor stationary illumination and signalling. As shown in Figure 1, two technologies are mainly in use today: Incandescent and Luminescent Lamps. The latter category can be further divided into Discharge/Fluorescence Lamps and Solid State Lighting Devices, respectively.

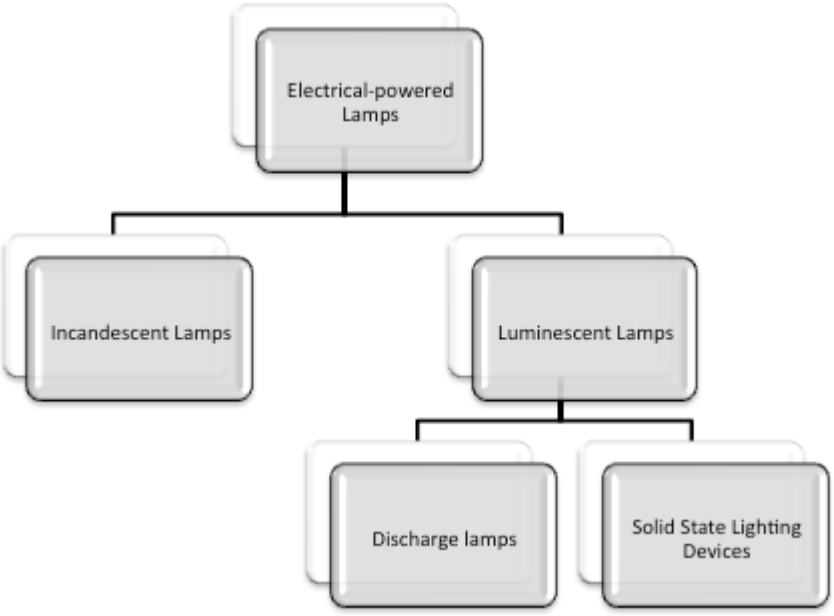


Figure 1 Electrical lighting sources technologies

A thorough overview of different lamp types is presented in Annex I, which includes descriptions of the fundamental technologies and their areas of use as well as some examples of emission spectra when such are available. It should however be noted that there is such a diversity of products among each lighting technology available on the market, that it is, in many cases, very difficult to present emission spectra which are "typical" for a given lamp type.

In the case of some lamp technologies, a second bulb or glass envelope is sometimes present. Most glass types absorb a large fraction of the UV radiation, and the UV transmittance depends on the thickness of the glass. UVB and UVC, as well as the shortest UVA wavelengths, do not penetrate ordinary glass. Whereas pure glass SiO_2 does not absorb UV light, soda-lime glass does not allow light at a wavelength lower than 400 nm (UV) to pass. Even Pyrex and other more ordinary forms of heat-resistant glass can be used as shields to block UVB and UVC. Using additional UV-blocking dopants can enhance this blocking behaviour. High efficacy incandescent lamps and some ceramic metal halide lamps often use a second bulb made of soda-lime glass. Compact fluorescent lamps are also made of borosilicate glass and a specific type called "look-alike" has a second shaped envelope in order to mimic the appearance of a classic incandescent lamp. This external envelope is usually made of polycarbonate. Most plastic materials (acrylic, polycarbonate, plexiglass) used for lamp bulbs give more protection than soft glass. Polycarbonate is almost completely transparent throughout the entire visible region until 400 nm, and if intact, successfully blocks UV radiation (UVB, UVC and more than 90% of UVA). Linear fluorescent lamps are also made of soda-lime glass, but it is rather unusual for these lamps to have a second protective envelope. However, this type of lamp can be placed in luminaires that have polycarbonate protection. In addition, it is possible to use a specific filter (GAM 1510 UV shield) that exists in the form of a gel or Rosco film (03114); this can be used to envelop the lamp and eliminates more than 95% of UVA radiation. Last but not least, when a lamp is placed in a luminaire or fixture, UV blocking elements (such as soda-lime glass or polycarbonate layers) may be introduced into the system and drastically reduce UV output. However, as this type of situation is optional rather than a rule, it is suggested to evaluate UV risks for bare lamps.

3.3.3. Lamp emissions

A critical aspect of any risk assessment of the potential health effects of lighting technologies is the availability of exposure data for the general population as well as occupational exposure. Unfortunately, data regarding actual exposure are sparse, which stresses the need for reliable data regarding emissions from the various lamp types. During the writing of this opinion, a call for information was launched regarding inter alia emission data (see section 3.2 for further details regarding the call for information). Relevant information was obtained, based on measurements performed by, or requested from, different stakeholders. Two contributions provided substantial information which could be used in this opinion (spectral UV emissions from the Belgian Federal Public Service of Health with a focus on CFLs and more global and graphical information from the European Lamp Companies Federation). In addition a recent study from Schulmeister et al. (2011) provided valuable relevant detailed spectral information. Measurement data from all three sources are according to the measurement methodologies recommended by Standard EN 62471.

Furthermore, detailed emission spectra (with nm resolution) were only provided in the study from Schulmeister and co-workers (2011). There is thus only scattered knowledge regarding the full emission spectrum from all available lamp types. It is not possible at present to perform a much needed comparative assessment of the different lamp types.

Based on emissions from the lamp, the Standard EN 62471 (and also IEC 62471 and CIE S009, since they are all identical in this sense) categorizes the lamps according to the photo-biological hazard that they might pose.

The different hazards are:

1. Actinic UV-hazard for eye and skin (see section 3.4.3.2);

2. UVA-hazard for the eye (section 3.4.3.2);

3. Blue-light hazard for the retina (section 3.5.2.3);

4. Thermal retina hazard (section 3.4.3.1) and

5. IR-hazard for the eye (sections 3.4.3.1 and 3.4.3.2).

According to the standards, measurements should be performed according to two approaches; viz. at a distance where a light intensity of 500 lx is obtained and also at a distance of 20 cm (see also section 3.4.2.2 for additional discussion regarding measurement). Based on these measurements, lamps are then classified according to the "Risk Group" (RG) to which they belong. RG0 (exempt from risk) and RG1 (minor risk) lamps do not pose any hazards during normal circumstances. RG2 (medium risk) lamps also do not pose hazards because of our aversion responses to very bright light sources, or due to the fact that we would experience thermal discomfort. RG3 (high risk) include only lamps where a short-term exposure poses a hazard. This classification is based on acute exposure responses (a single day, up to 8 hours) and applies only to individuals of normal sensitivity.

The material received from the Belgian Federal Public Service of Health included measurements on 70 CFLs, and also Ecodesign UV functional requirements (Ecodesign regulation 299/2009). Four lamps were classified as RG1 for actinic UV-hazard at 20 cm, whereas lamps were otherwise classified as RG0. However, UVC emissions could not be determined according to Ecodesign, since background levels were higher than the Ecodesign requirements.

The contribution from the European Lamp Companies Federation (ELC) included six lamp types from eight manufacturers, considered by ELC to be "representative lamp types". Risk group classification was carried out in accordance with EN 62471. Results were presented for the following lamp types:

- Tubular fluorescent (4,000 K and 6,000 K);

- CFL (2,700 K 11W with and without envelope);
- LED (3,000 K, retro-fit, and 6,000 K);
- Halogen (two high voltage, one without UV filter, and three low voltage);
- High pressure discharge (two metal halide and one sodium); and
- Incandescent (60 W clear).

A summary of important parameters for each lamp is shown in Table 1.

Table 1 Lamp parameters supplied by the European Lamp Companies Federation

	Φ [lm]	2. envelope	UV filter	Xenon filling	IR coating	voltage	Tc	CRI
Tubular Fluorescent, 4000K, 80W, 16mm	7000	no	*)	n/a	no	230V 1)	4000K	80
Tubular Fluorescent, 8000K, 80W, 16mm	6400	no	*)	n/a	no	230V 1)	8000K	80
CFLi, 2700K, 11W, without envelope	650	no	*)	n/a	no	230V 2)	2700K	80
CFLi, 2700K, 11W, with envelope	630	yes	*)	n/a	no	230V 2)	2700K	80
LED reflector lamp MR16 retrofit, 3000K	n/a	front glass	n/a	n/a	no	12V 2)	3000K	80
LED high power single 1W, 1mmx1mm new	100	no	n/a	n/a	no	n/a	6000K	80
LED incandescent retrofit, diffuse bulb	810	yes	n/a	n/a	no	230V 2)	2700K	80
Halogen HV 230V, 42W, ECO with outer bulb	630	yes	*)	yes	no	230V	2800K	100
Halogen HV 230V, R7s, 230W, without UV filter	5000	no	no	yes	no	230V	2950K	100
Halogen LV 12V, 50W, UV reduced, without refl.	900	no	yes	no	no	12V	3000K	100
Halogen LV 12V, 50W, 12V, MR16 dichroic refl.	n/a	front glass	yes	no	no	12V	3000K	100
Halogen LV 12V, 35W, ECO with IR coating	860	no	yes	yes	yes	12V	2950K	100
Metal halide discharge, 70W, 830 new	7300	no	yes	n/a	no	230V 1)	3000K	80
Metal halide discharge, 70W, 942 new	6800	no	yes	n/a	no	230V 1)	4200K	90
High pressure sodium, 70W new	6600	no	yes	n/a	no	230V 1)	n/a	n/a
Incandescent, 60W, 230V, clear	710	no	*)	n/a	no	230V	2700K	100

*) standard glass (bulb or tube) filters UV, 1) separate ballast, 2) integrated ballast

According to ELC, under normal conditions of exposure, all lamps are classified as RG0 (exempt from risk) or RG1 (low risk) from UV and IR emissions, with the exception of one lamp. This halogen lamp is intended to be used with additional glass shielding, which was tested without the glass shield, and was characterized as RG2-RG3 at 20 cm. The metal halide lamps are RG1 or RG2 at 20 cm, but these are not intended for use at such close distance according to ELC.

Concerning blue light emission, ELC considered all lamps as belonging to RG0 or RG1. This includes the 6,000 K LED ("high power" LED) which is RG0 when analysed as a "small source". The use of the "small source" approximation is valid because the eye moves rapidly without our knowing it ??).. This means that the image of the source is smeared over a larger area of the retina than the area of the image itself and the light emitted is averaged over an angle of 11 mrad, which is the effective angular subtense taking account of eye movement. This means that treating the LED as a "small source" and averaging over an effective angular subtense of 11 mrad is acceptable in accordance with EN 62471. It follows that the 6,000 K LED in the ELC dataset is correctly classified as RG0.

The metal halide lamps are also RG2 when measured at 20 cm, but these lamps are not intended to be used at such close distance according to ELC.

ELC reported that the provided data were measured in an accredited laboratory according to ISO/IEC 17025 and so the measurement procedure should be reliable and the results reproducible. Furthermore, it is stated that the lamps were selected such that they are typical, mid-range samples from the quality control process.

The results presented in the ELC report suggest to SCENIHR that there is little or no risk to individuals of normal sensitivity from the UV, IR or blue light optical radiation emission from lamps which are considered to be "representative" of the type of lamps selected to replace incandescent lamps. SCENIHR however considers that "non-representative" lamps may emit levels that are much higher than those included in the report; however quality control limits applied by lamp manufacturers were not reported. Further consideration should also be given to the "intended" vs. "reasonable foreseeable" use of lamps. For risk assessment purposes, most light sources should be assessed at the distance corresponding to 500 lux illumination. It is inappropriate to classify high output lamps at a distance of 20 cm when they are designed to illuminate a large area, e.g. a factory. Only those lamps that are intended to be used in close proximity to the skin should be assessed at 20 cm.

Further consideration also needs to be given to the risk classification of high power LEDs. Also, halogen lamps that are intended to be used with an external glass filter must not be used without the filter because of the risk of exposure to UV radiation.

Schulmeister et al. (2011) measured UV emission characteristics, as far as we can judge according to EN 62471 with a nm resolution, in 96 different types of light sources (including CFLs, LEDs, halogen lamps, fluorescent tubes, high-pressure discharge lamps, and incandescent lamps). One high pressure mercury lamp intended for industrial lighting was classified as RG1 (actinic UV) at 500 lx, whereas some of the high-pressure discharge lamps were assigned to higher RGs at 20 cm. These lamps are however not intended for use at such close distances.

Again, SCENIHR considers that "intended" vs. "reasonably foreseeable" use should be considered for the lamps classified as higher RGs at 20 cm.

It is important to know whether the risk categories designed to protect the general public provide adequate protection to photosensitive patients. In a preliminary study, it has been shown that single envelope CFLs may cause an erythematous reaction in patients with a photosensitive disorder (Eadie et al. 2009). The published report does not contain data on the risk classification of the lamp. Subsequent analysis of the lamp used in that investigation (Moseley, personal communication) shows that it is RG1 at 20 cm. In the study reported by Eadie et al. (2009), the lamp was used at a distance of 5 cm because it was argued that in practice this was quite reasonable for task lighting, particularly since there is very little heat emitted. At this distance the lamp would be RG2. Since lamps which are intended to be used in close proximity to the skin are classified at a distance of 20 cm, it is clear that a single envelope CFL classified as RG1 may be hazardous to a photosensitive patient if used closer than 20 cm to the skin. All of the CFL lamps included in the ELC report were RG0 or RG1. It is difficult to predict how an individual patient will respond to light from a particular lamp because of the range of response that individual patients exhibit when exposed to different wavelengths. However, RG1 lamps cannot be considered safe for use by photosensitive patients. Further work into optical radiation emission from 167 CFLs (103 single envelope and 65 double envelope) demonstrated double envelope lamps generally emitted much less than single envelope lamps (Moseley, personal communication). Taking the highest emitting lamp of each model tested, the mean UVB irradiance was 4 mW/m^2 (double envelope) and 101 mW/m^2 (single envelope).

Schulmeister et al. (2011) also reported on UV emission levels from halogen lamps. These lamps have a smoothly decreasing spectrum at UV wavelengths. Although there are no published data on the effect of exposure of a photosensitive patient to light from a halogen lamp, it is unlikely that there would be a significant risk provided the protective filter was in place. However, it should be noted that some halogen lamps may be used without the filter attached which would increase the chance of an adverse reaction.

Conclusions

Optical radiation emission data from three different laboratories (representing public authority, industry, and a commercial research enterprise) have been obtained and considered for the conclusions in this opinion. Data from more than 180 different lamps were provided and represent all major lamp types that are used for general lighting purposes. Regarding specific lamp types, CFLs are well represented in the samples assessed, whereas LEDs are measured in only a few cases. All other lamp types are represented mostly in small numbers.

The photobiological hazard from each lamp has been determined according to Standard EN 62471. For all investigated hazard outcomes, the absolute majority of lamps are classified as RG0 (exempt from risk). Most other lamps are classified as RG1 (low risk). The lamps assigned to higher risk groups were either measured without a UV-shielding glass cover, or at a short distance (20 cm) which is not the intended use distance for this lamp type.

SCENIHR considers that further consideration needs to be given to the representativeness of the measured lamps and to the question of whether the intended use can be ensured for those lamps classified as RG2 or RG3 at a distance of 20 cm.

LEDs were under-represented in the present analysis of lamps. Further assessment of LED retinal hazards should be evaluated at 20 cm taking into account that LED luminaires can be used at this distance for domestic lighting.

3.4. First principles and biology

3.4.1. Optical radiation

Wavelengths of visible EM radiation range from 400 to 780 nm (1 nm = 10^{-9} m), spanning the visible range from violet to red light (see CEI/IEC 2006/62471, Directive 2006/25/EC[2]). In article 2a of Directive 2006/25/EC the visible range is positioned more broadly between 380 and 780 nm.

Light can be manipulated by a variety of optical devices or elements; most characteristically a beam of light can be focused or diverged by optical lenses made of crystal (quartz) or glass, as in binoculars, telescopes and cameras. ***Optical radiation*** encompasses light but also includes EM radiation of wavelengths well beyond the visible range: ultraviolet (UV) radiation is below 400 nm down to 100 nm and infrared (IR) radiation is above 780 nm up to 1 mm. UV and IR radiation can also be manipulated by optical devices and elements such as optical lenses (sometimes optical radiation is referred to as "light", and one then speaks of "UV light" and "IR light", next to "visible light"; here the latter is considered a tautology and the former two are consequently oxymora).

The UV band is sub-divided in three wavelength regions (CIE 2006/62471):

- UVA from 400–315 nm
- UVB from 315–280 nm
- UVC from 280–100 nm

The IR band is similarly sub-divided in three wavelength regions (CIE 2006/62471):

- IRA from 0.78 to 1.4 μm (μm = 10^{-6} m)
- IRB from 1.4 to 3.0 μm

[2] http://eur-lex.europa.eu/LexUriServ/site/en/oj/2006/l_114/l_11420060427en00380059.pdf.

- IRC from 3.0 µm to 1 mm

Formally, this leaves the stricter range of 400-780 nm as the wavelength range of visible radiation, light.

Although the sun emits optical radiation over the full wavelength range, the earth's atmosphere blocks UVC and part of the UVB irradiation below 290-295 nm (mainly by oxygen and stratospheric ozone) and IRC of wavelengths over 30 µm (by water vapour). Interestingly, the sun's spectrum peaks over the visible range. Although UV is classified as non-ionizing radiation, it can cause chemical reactions, and causes many substances to fluoresce. Most people are aware of the effects of UV irradiation through the painful condition of sunburn, but UV irradiation has many other effects, both beneficial and damaging, to human health.

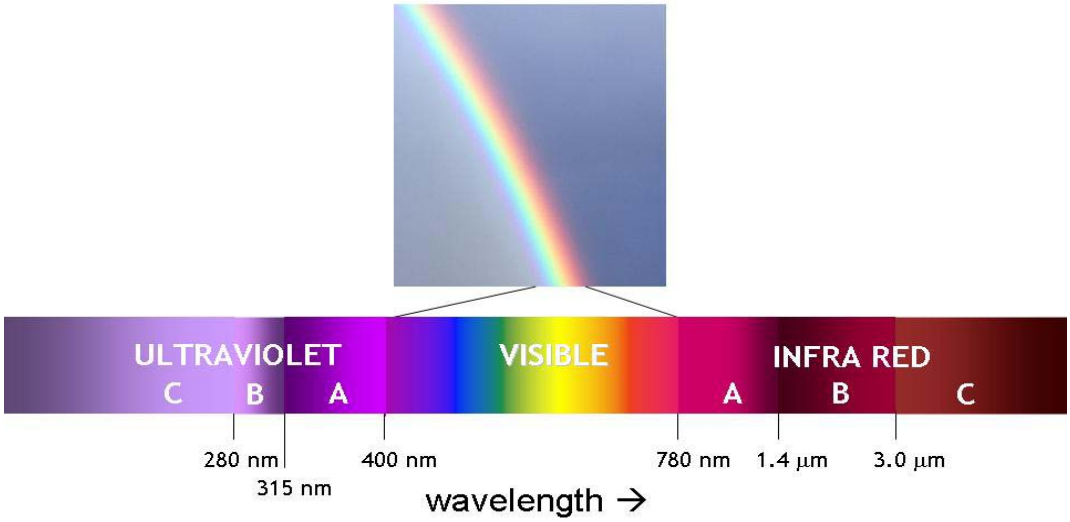


Figure 2 Wavelength regions in optical radiation

3.4.2. Radiant energy absorption

3.4.2.1. Mechanisms

For optical radiation to have an effect on matter the radiation needs to be absorbed, i.e. the radiant energy needs to be transferred to the material in which the effect is to occur. Two main mechanisms can be distinguished through which the absorbed radiant energy can take effect:

a) ***Heat***: radiant energy is converted into molecular motion (kinetic energy) such as vibration, rotation and translation. Thus the temperature is increased (photothermal effect). Here, the radiant energy (measured in Joules, J) absorbed per unit time (s) in a certain volume determines the rise in temperature, i.e. the absorbed radiant power (J/s = Watt, W) per unit volume (m^3) or the (specific) absorption rate (W/m^3) is the determining factor (next to how fast the absorbing volume is cooled by heat exchange with its environment).

b) ***Photochemistry***: radiant energy can cause excitation of atoms or molecules by moving the outermost (valence) electrons to higher orbital energy levels. This energy can subsequently be utilized in (photo-)chemical reactions, yielding "photoproducts". The

radiation needs to be within a certain wavelength range (the "absorption band") for the excitation to take place as the radiant energy is absorbed in discrete quanta, "photons", which must match the energy required for the excitation. The (part of the) molecule that absorbs the radiation is dubbed the chromophore. Not every excited molecule will cause a chemical reaction: the energy may be lost through fluorescence (emission of radiation of longer wavelengths) or dissipated as heat. This implies that only a certain fraction of the absorbed radiant energy is channelled into the (photo-)chemical reaction: this is represented by the quantum efficiency (the number of photoproducts formed per photon absorbed; a ratio usually <1). The absorbing molecule is not necessarily the molecule that is chemically altered; the energy can be transferred to another molecule, which may then become chemically reactive (e.g. radicals and reactive oxygen species may thus be formed). In general, the total radiant energy (radiant power times exposure time in *W x s=J*) absorbed by the proper chromophores determines to what extent the photochemical reaction has evolved, i.e. the amount of photoproduct formed.

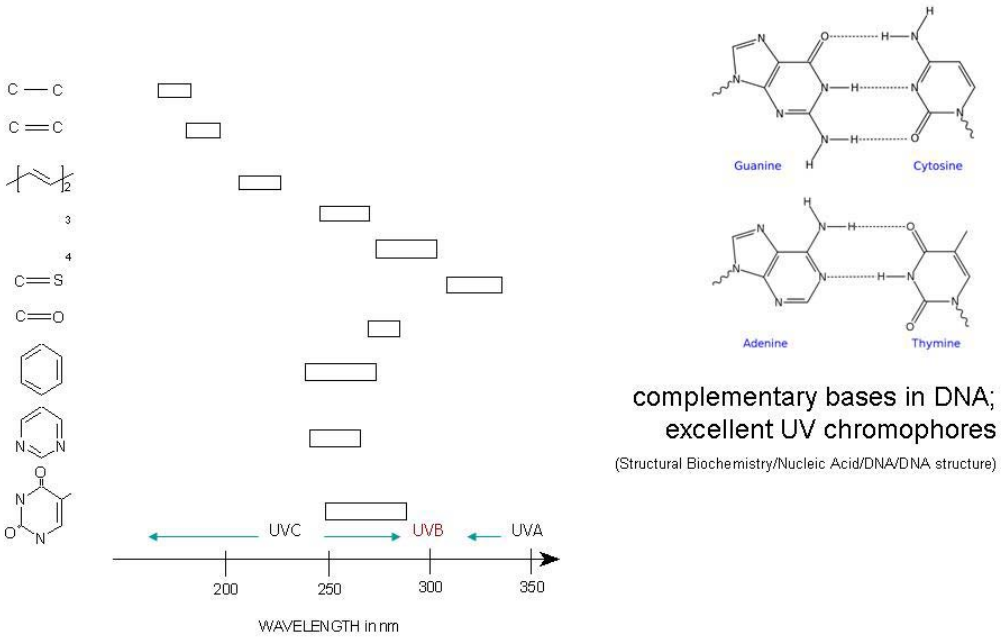


Figure 3 Chromophores and their absorption bands (adapted from Jagger 1967)

Of the three types of optical radiation, UV radiation is photochemically most active (the photons carry the highest energy), and it is absorbed by certain common chromophores in organic molecules (e.g. C=O, C=S and aromatic rings; the latter are abundantly present in DNA (Figure 3)). Clearly, light is also photochemically active in the eye: visual perception starts with the photo-isomerisation of opsin proteins (in G-protein coupled receptors which trigger the neural signalling). In the skin there are also other chromophores that absorb light. For example, heme-ring structures are present in enzymes, such as cytochrome-c oxidase in the mitochondria. This enzyme is even sensitive to IRA radiation of wavelengths around 820 nm (Karu et al. 2004) by excitation of a copper atom. However, by and large, IR radiation is not capable of moving valence electrons to higher energy levels (the energy transferred per photon is too low for excitation of valence electrons) and thus initiate photochemical reactions. Most IR effects are heat-mediated.

The light interacts with eye tissues and molecules through different mechanisms. Some of the eye tissues or pigments can absorb light and thus reduce retinal exposure. In other parts of the eye or pigment structures, the light can induce oxidative stress damage defined as photochemical and photodynamic effects.

3.4.2.2. Photobiology and dosimetry

In photobiology, optical radiation usually penetrates a body through the outer surface (skin or eye), and the ***exposure*** (radiant energy per surface area in J/m^2) and exposure rate or ***irradiance*** (radiant energy per surface area per unit time in J/m^2s, W/m^2) are the commonly used proper photobiologic metrics by which to quantify the transfer of radiant energy to the body. However, by convention, in some disciplines such as ophthalmology and dermatology the exposure is most often given as mJ/cm^2. This convention is also followed in this document. The eye has the special feature of focusing the light onto the retina whereby the irradiance from the surface of the eye to the retina is increased by several orders of magnitude (up to 200,000-fold; see http://www.safetyoffice.uwaterloo.ca/hse/laser/documents/hazard_eye.html). The irradiance at the retina over the image of a light source (either a lamp or an object reflecting light) is determined by the diameter of the pupil and the radiance of the light source. The radiance is the power transmitted into a solid angle onto the pupil per surface area of the source (in $W/sr.m^2$). Interestingly, the distance from the light source drops out of the equation for a source with a homogeneous radiance over its surface (see Box I) if the light is not attenuated by absorption or scattering in the air between the eye and the light source. At greater distances the pupil catches less of the light from the source, but as the image of the source becomes smaller with larger distances, more of the radiant surface is projected onto a small area on the retina. These loss and gain with distance cancel each other out, leaving the irradiance in the image area on the retina unchanged. It should be noted that a very bright source will cause immediate aversion and thus will not be focused on for any substantial length of time.

The skin remits by back scatter much of the incoming visible and IRA radiation but absorbs most of the UV and IRB and IRC radiation.

The penetration of the optical radiation into the tissue (skin or eye) determines to what depth effects or damage can occur, but also over which volume of tissue the absorbed radiant energy is spread; Figures 4 (a-d) and 5 illustrate the penetration of UV, visible and IR radiation (only depicted for skin) into the eye and the skin, respectively. From these figures it is clear that visible and IRA radiation penetrate deepest into the skin (10-fold reduction at 0.1-0.4 mm depth) and eye (onto the retina), whereas UVA and UVB radiation reach the lens in the eye. Short wavelength UVC and long wavelength IRB and IRC penetrate the skin only very shallowly and do not reach the lens in the eye. The superficial absorption of broad-band IRB and IRC radiation implies that most of the radiant energy is absorbed in a very thin layer which can consequently be heated efficiently.

In the IRB and IRC region of the spectrum, the ocular media is opaque as a result of the strong absorption by their constituent water. Beyond a wavelength of 1.9 μm the cornea becomes the only absorber. Direct exposure to high levels of IRC ($>1W/cm^2$) may induce corneal lesions, particularly of the epithelium.

The human cornea transmits radiant energy only at 295 nm and above (and thus not in the UVC range). Indeed, all UVC (100-280 nm) radiations are absorbed by the human cornea which absorbs radiation. It absorbs light very efficiently, over 90%, between 300-320 nm (UVB range), about 30-40% between 320-360 nm (UVA range) and almost 100% above 800 nm (i.e. IRA, IRB and IRC ranges) (Sliney 2002). Almost no absorption occurs in the spectrum of visible radiation.

However, the part of UVA that is transmitted from the cornea is absorbed in the aqueous humour, the lens and even in the vitreous. Indeed, about 45-50% of the UVA is absorbed

by the lens. Part of the UVA transmitted by the lens is then absorbed by the vitreous, so that only 1-2% of the UVA reaches the retina. In young children (at about or just below 9 years of age, where the limit is approximate since no study has clearly defined it), a window exists that allows transmission of about 2-5% of UV at 320 nm to the retina (Gaillard et al. 2000). At older ages no UV at this wavelength reaches the retina (Dillon et al. 2004). The other main difference in young children compared to adults and older children is the transmission of blue light by the lens. Around 15% of 400 nm and about 65% of 460-480 nm wavelengths reach the retina in children less than 9 years of age, compared to 60% at 460-480 nm at 10 years. In the age group of 60-70 years, only ca. 1% at 400 nm and 40% at 460-480 nm reaches the retina. This difference is explained by the fact that the colour of the lens becomes more and more yellow with increasing age (Gaillard et al. 2000). It is important to note that even without any clinically detectable cataract, changes in transmission are occurring in the lens (Ham et al. 1978). The age at which transmission of blue light decreases may be variable due to genetic, nutritional and exposure factors. Therefore the percentages given in these schemes are approximate and intended to give a range of ocular media transmission as a function of age and spectrum.

Figures 4 a-d below show the penetration/absorption of radiation by the eye for different age groups (all figures adapted from Sliney 2002).

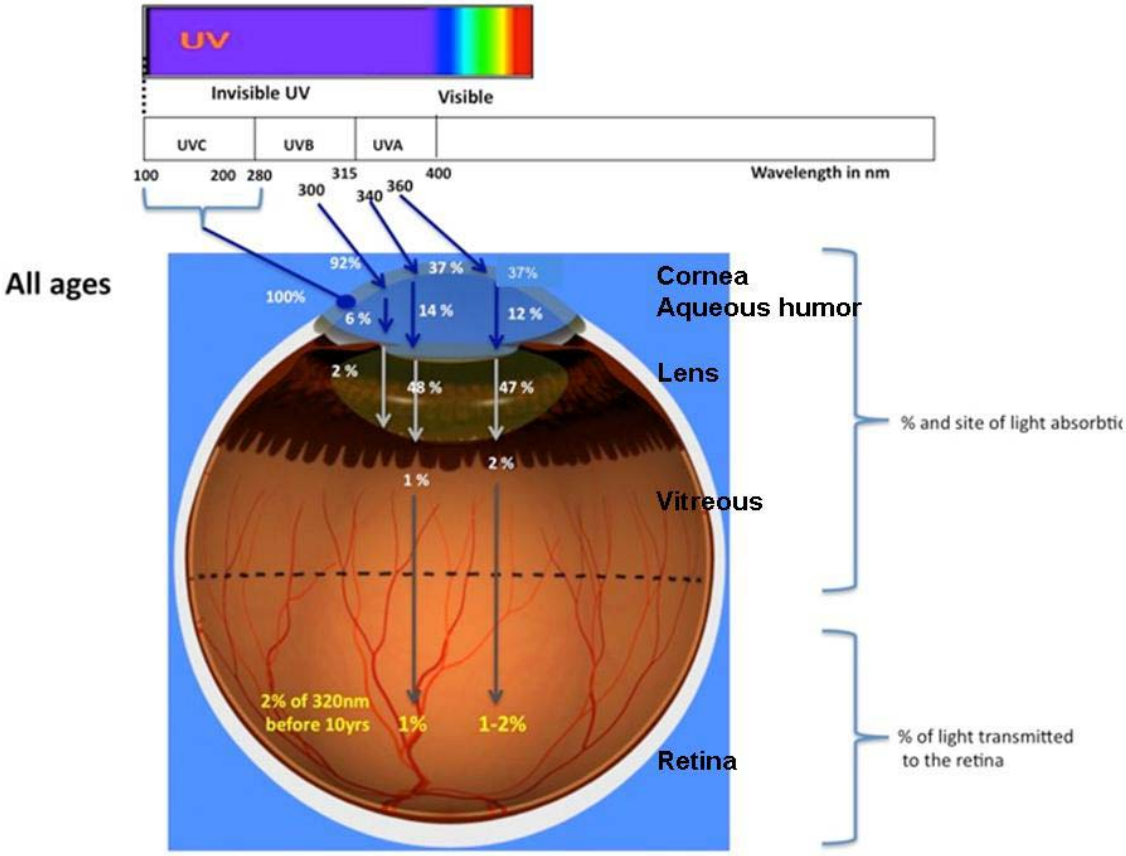


Figure 4a Interaction of UV radiation with the human eye at all ages (adapted from Sliney 2002)

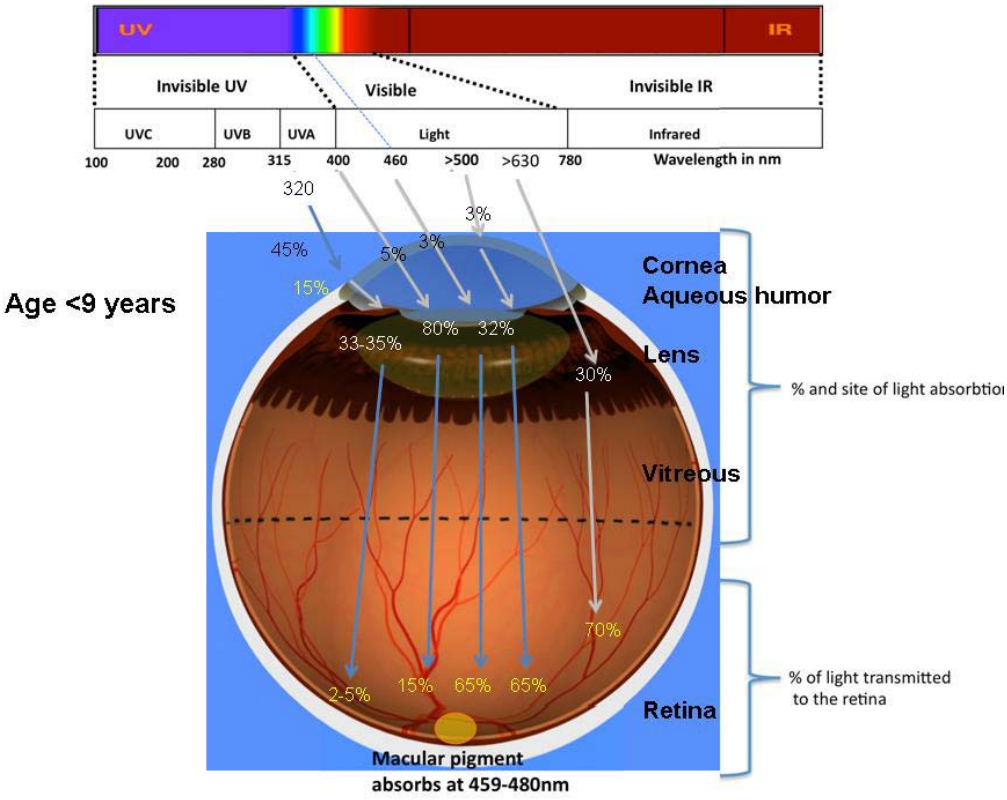


Figure 4b Specificity of optical radiation interaction with the eye of children below 9 years of age (adapted from Sliney 2002)

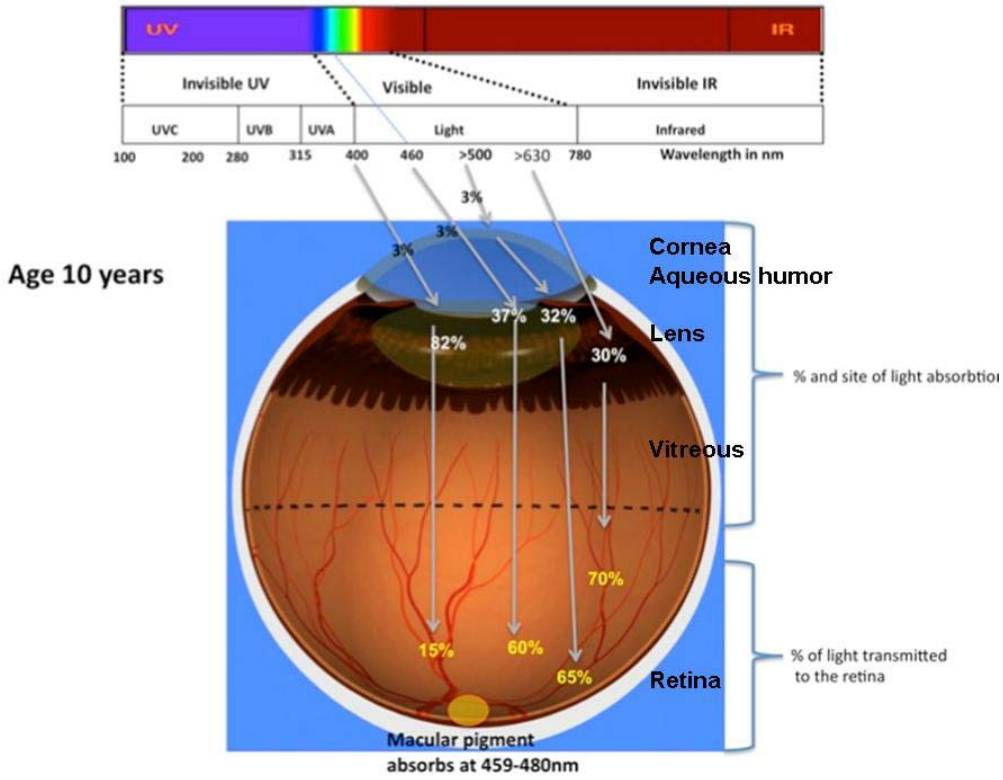


Figure 4c Optical radiation interaction with the young human eye (10 years old up to young adulthood) (adapted from Sliney 2002)

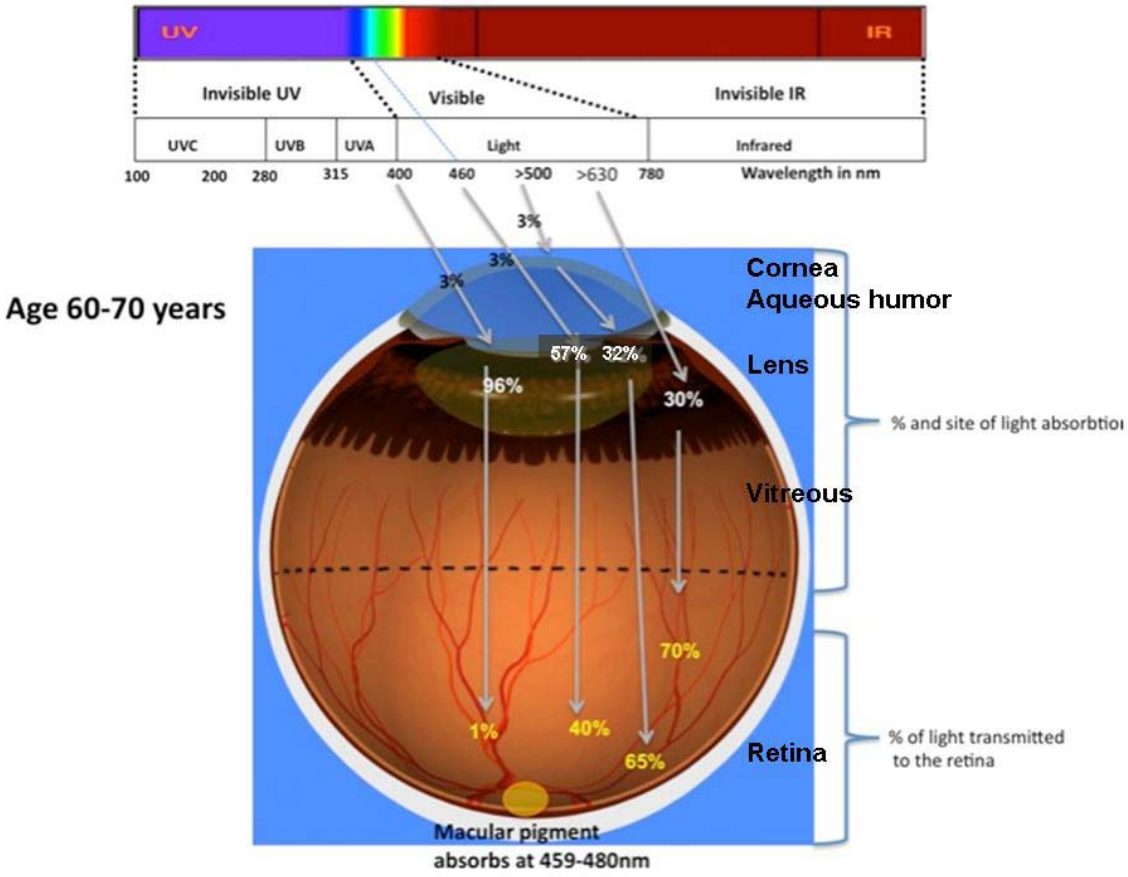


Figure 4d Optical radiation interaction with the eye of an aging human (adapted from Sliney 2002)

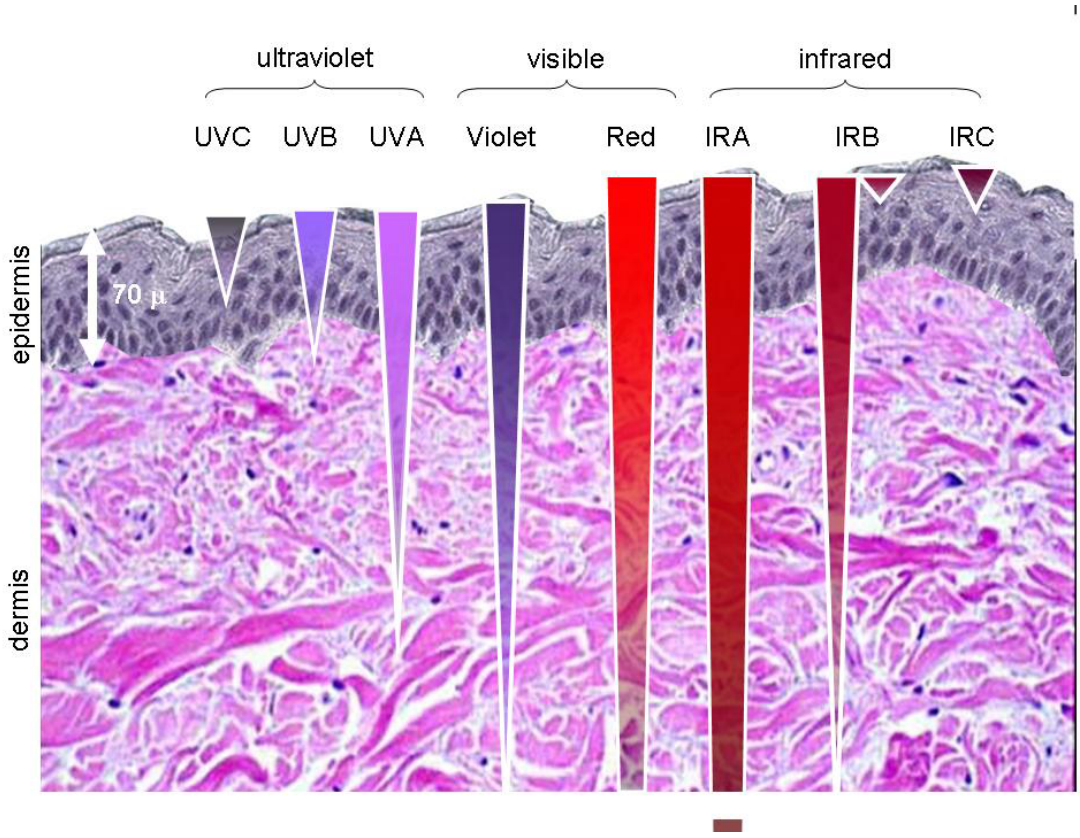


Figure 5 **Light penetration in the skin (attenuation down to 1% occurs for light wavelengths of 250-280 nm at around 40 µm depth; for 300 nm at 100 µm; for 360 nm at 190 µm; for 400 nm at 250 µm; for 700 nm at 400 µm; for 1.2 µm at 800 µm; for 2 µm at 400 µm; for 2.5 µm at 1µ; and for 400 µm at 30 µm)**

Although some photochemically mediated biological effects may depend on the total amount of photoproducts irrespective of the spatial distribution, others may depend on the density of photoproducts, i.e. the amount per surface area or volume. If the photoproducts are removed from the tissue (dead cells in days) or repaired (DNA damage in hours to days), the effect in the tissue will evidently depend on how quickly the photoproducts are generated. After absorbing light, visual pigments (opsins) take minutes to get regenerated (Sandberg et al. 1999). Following exposure of the eye to very intense illumination, a greatly elevated visual threshold is experienced, which requires tens of minutes to return completely back to normal. The slowness of this phenomenon of "dark adaptation" has been studied for many decades, yet is still not fully understood. Upon photon excitation, rhodopsin undergoes photoactivation and bleaches to opsin and all-trans-retinal. To regenerate rhodopsin and maintain normal visual sensitivity, the all-trans isomer must be metabolized and reisomerized to produce the chromophore 11-cis-retinal. This constitutes the visual cycle, which involves the retinal pigment epithelium, where all-trans retinoid is isomerized to 11-cis-retinol. The time-course of human dark adaptation and pigment regeneration is determined by the local concentration of 11-cis retinal. After intense light exposure, the recovery is limited by the rate at which 11-cis retinal is delivered to opsin in the bleached rod outer segments.

Radiations of different wavelengths will generally differ in the efficiency by which they trigger a chemical reaction or evoke a biological response; i.e. the wavelength at which a smaller exposure is required for a certain (level of) response is more efficient (such differences largely depend on the absorption spectrum of the relevant chromophore and the transmission of the radiation through the medium or tissue to the chromophore). The wavelength dependence of this efficiency is dubbed an "**action spectrum**" (a

wavelength by wavelength plot of the inverse of the exposure needed for a certain response). Such an action spectrum can be used for spectral weighting of the exposure to a source to ascertain the biologically **effective exposure** or photobiologic **dose** (for details in formulae see Box I).

The European Standard EN 62471 recommends evaluating the Photobiological Risk Group for General Lighting Systems (GLS) at a distance where the horizontal illuminance is 500 lx. However, the same standard underlines that for all other lamp types this evaluation has to be carried out at 200 mm. The two recommendations are consistent with two distinct risks: the first (500 lx) corresponds to the situation for a worker in a well-illuminated environment without direct view of the light source; the second (200 mm) is more appropriate for evaluating the risk of a person looking directly in the direction of the light source. Following this reasoning, it is recommended to evaluate the risk class based on the potential use of the light source by the end-user. For example, light sources within ceiling fixtures or indirect lighting can be characterized at 500 lx level, whereas task lights, downlights, etc. that can be in the line of sight should be evaluated at 200mm.

BOX I: Metrics of optical radiation and (bio-)effectiveness

Here follows a concise representation of the physical and bio-effective metrics of optical radiation as discussed in 3.4.2.1 and 3.4.2.2 in mathematical formulae with physical dimensions given in square brackets,"[ ]".

Given the spectral irradiance $E(\lambda)$ in [W/m^2 /nm] at the wavelength λ in [nm], we find the total irradiance, E, in [W/m^2] by integration over the wavelengths:

$$E = \int E(\lambda)\, d\lambda,$$

If the spectral irradiance varies with time, we find the spectral exposure, $H(\lambda)$ in [J/ m^2 /nm] at wavelength λ by integration over time, t in [s]:

$$H(\lambda) = \int E(\lambda,t)\, dt$$

and the exposure, H, by integration over λ:

$$H = \int\int E(\lambda,t)\, d\lambda dt = \int H(\lambda)\, d\lambda.$$

which simplifies to:

$$H = \varepsilon \int E(\lambda)\, d\lambda = \varepsilon.E$$

if $E(\lambda)$ is constant over the exposure time ε in [s].

To ascertain the bio-effectiveness of the radiation we define a dimensionless action spectrum $S(\lambda)$ to weight the spectral exposure. $S(\lambda)$ is inversely proportional to the exposure $H_r(\lambda)$ required at a certain wavelength λ for a certain level of biological response (level), and normalized to equal "1" at λ_{max}, the most effective wavelength to induce this response with smallest $H_r(\lambda)$, i.e.

$$S(\lambda) = H_r(\lambda_{max})/H_r(\lambda).$$

The effectiveness spectrum is then defined as:

$$E_e(\lambda) = S(\lambda).E(\lambda),$$

The (bio-) effective irradiance as:

$$E_e = \int S(\lambda)\, E(\lambda)\, d\lambda,$$

and the (bio-) effective exposure or photobiologic "dose" as:

$$H_e = \int S(\lambda)\, H(\lambda)\, d\lambda,$$

where $E_e(\lambda)$, E_e and H_e are then given in equivalents of [W/m^2 nm], [W/m^2] and [J/m^2], respectively, at wavelength λ_{max}.

Note that this procedure of spectral weighting requires "additivity" to hold, i.e. that separate effective doses from different wavelengths can be added up to a total effective dose.

The irradiance at the retina of the eye from a radiant source is strongly dependent on the imaging, i.e. focusing, of the source onto the retina which can cause up to a 200,000-fold increase in irradiance from the surface of the eye to the retina[3]. When we consider a source (a lamp or a reflecting surface), its spectral radiant power in [W/nm] emitted per radiant surface area in [m^2] into a solid angle in steradian [sr], i.e. the spectral radiance, $L(\lambda)$, in [W/sr m^2 nm], towards the pupil of the eye is crucial to the retinal irradiance E_{ret} in [W/m^2]:

$$E_{ret} = (\pi/4)\ (d_p/f)^2 \int L(\lambda)\ \tau(\lambda)\ d\lambda,$$

where $\tau(\lambda)$ is the transmittance from the source to the retina at wavelength λ, d_p denotes the pupil diameter in [m] and f is the focal length of the eye in [m]. Note that the distance of the source from the eye drops from the equation (as the source is moved away from the eye the image spot on the retina becomes smaller but the irradiance remains constant if the radiance is constant over the surface of the source). L is also referred to as the source's "intensity". The eye tremor (around 80 Hz with amplitudes of 0.2 to 2.5 μm) spreads a point source over a larger image area on the retina (about 25 μm in diameter in 1 sec and 190 μm in 100 s, subtending about 11 mrad). It should also be noted that in a realistic assessment, for example of a human who is reading, the source is a page at a certain distance from a lamp, i.e. not the lamp itself. The eyes rarely focus on a primary light source, a lamp, and strongly evade looking into bright ones (eyes closing and causing the face to turn away from the source).

To ascertain the bio-effectiveness on the retina $E_{ret}(\lambda)$ and $L(\lambda)$ can be weighted by an action spectrum $S(\lambda)$. For example, taking $S(\lambda)$ as the photopic* luminous efficiency function, $V(\lambda)$, (normalized to 1 at λ_{max} = 555 nm) one converts a physical radiant power spectrum, or spectral flux $\Phi(\lambda)$ in [W/nm], into a luminous flux, Φ_v, in lumens [lm] representing the visual effectiveness:

$$\Phi_v = 683.002\ [lm/W] \int V(\lambda)\ \Phi(\lambda)\ d\lambda.$$

Illuminance (the photometric equivalent of irradiance) equals the luminous flux per surface area in lux, [lx] = [lm/m^2].

The photopically weighted radiance, luminance $L_{ph,}$ of a source is given in candela per surface area [cd/m^2] = [lm/sr m^2]. Luminance is also loosely referred to as the "brightness" of a radiant source, i.e. the visual intensity.

* *Photopic refers to daylight vision, i.e. with a light-adapted eye; scotopic, on the other hand, refers to night vision, i.e. with a dark-adapted eye.*

3.4.3. Biological effects

Overexposure can cause dysfunction or outright destruction of tissue, either through heating or photochemical reactions. As implied by the term "overexposure", a certain threshold of tolerable levels of exposure or irradiance is surpassed: the irradiance can become too high and cause thermal damage or the accumulated exposure carries a photochemical reaction to a toxic level. It should be stressed that this does not imply that there is no biological effect below the threshold level, but the damage is minor and tolerable (non-destructive) and/or the absorbed radiant energy causes a functional biological response (receptive absorption).

[3] http://www.safetyoffice.uwaterloo.ca/hse/laser/documents/hazard_eye.html.

Below we present biological effects from "receptive absorption" or by "destructive or toxic overexposure".

3.4.3.1. Photothermal effects

A. Reception

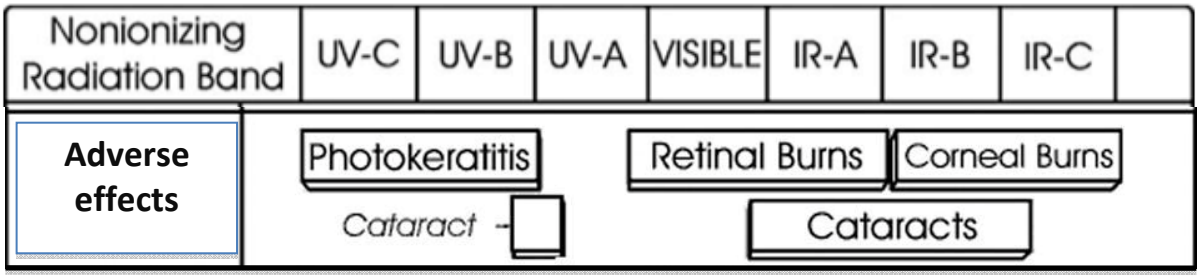
Absorption of optical radiation by the skin will cause heating which can raise the temperature. The skin can sense temperature differences smaller than 0.1°C on the face, especially on the lips (Jones 2009, Stevens and Choo 1998). The skin is innervated by axons (nerve endings from neurons residing in the spine) which carry transient receptor potential (TRP) ion channels that are sensitive to temperature changes in their cell membranes. Some axons carry TRP channels that are activated below certain temperatures, sensing cold, whereas others are activated above certain temperatures thus giving a hot sensation (some of these TRPs are also present on the tongue and respond to menthol, a "cool" sensation, and capsaicin in pepper, a "hot" sensation (Denda et al. 2010). Very recently, transient receptor potential vallinoid (TRPV) channels have also been found in human cornea cells (Mergler et al. 2010). They may be involved not only in thermo-sensation, but also in the regulation of cell proliferation. In the retina, TRPV channels have been identified that are more sensitive to pressure than temperature (Sappington et al. 2009).

B. Damage

Proteins can become denatured (loss of tertiary structure) at high temperatures and, cells and tissue irreversibly damaged in 15 to 60 minutes at 45°C (Kampinga et al. 1995) and in a matter of seconds at 60-70°C (Biris et al. 2009, Priebe et al. 1975). Pain and retraction reflexes evidently serve to limit the damage. Blisters may develop first due to loss of adherence between skin layers. Limited superficial thermal wounds, as from cosmetic or therapeutic skin ablation by laser treatment, can be restored from deeper and neighbouring layers of skin, but extensive third degree deep burns need special medical care and skin transplants. The immune system will respond to thermal damage by an inflammatory reaction in the skin.

Regarding thermal damage to the eye, only pulsed lamps are of concern. If the rate of energy deposition is faster than the rate of thermal diffusion (thermal confinement), then the temperature of the exposed tissue rises. If a critical temperature is reached (typically about 10°C above basal temperature), then thermal damage occurs. Thermal injury is caused mainly by absorption of light wavelengths >450 nm by the retinal pigment epithelium; the effects are usually immediate. Thermal burn is rare unless the light source is pulsed or in near contact with the eye. Thermal damage usually does not occur with domestic lights but can be induced by pulsed lamps and lasers. In such cases, retinal damage is primarily induced via thermal mechanisms for exposures shorter than 5 seconds. During longer exposure times both thermal and photochemical damage takes place.

Figure 6 shows the typical adverse effects of light on eye tissues as a function of wavelength.



Threshold levels for photokeratitis: 3-4 mJ/cm^2 (270 nm), 10 mJ/cm^2 (300 nm).

Threshold levels for cataracts: 600 mJ/ cm^2 (300 nm), 2 J/cm2/ cm^2 (>315 nm), 4 W/cm^2 for IR.

Retinal thermal damages (burns): 1-1,000 W/cm^2 depending on spot size.

Figure 6 Adverse effects of light on eye tissues as a function of wavelength (adapted from Sliney 2001, Sliney et al. 2005a)

3.4.3.2. Photochemical effects

A. Physiological responses

A1. In the eye

The iris

The iris responds to light by constriction, the pupillary reflex, thus reducing light entry into the eye. This mechanism is extremely important and efficient for protecting the retina against light damage. Pupillary constriction is highly dependent on the wavelength. Lucas et al. (2001) showed that the pupillary light reflex in mice was driven by a non-rod, non-cone photoreceptive system using a photopigment with peak sensitivity around 479 nm (melanopsin). The work of Hattar et al. (2003) recognized the melanopsin-associated photoreceptive system as being responsible for conveying photic information for accessory visual functions such as pupillary light reflex and circadian photo-entrainment. In humans, light pupillary constriction is achieved at a peak sensitivity of 482 nm and the sustained, post-stimulus pupil constriction is mediated predominantly by the melanopsin-driven, intrinsic photoresponse and not by sustained rod activity resulting from bleached rhodopsin as had previously been suggested. Light pupillary constriction is observed at 5 cd/m^2 at 482 nm in humans and primates (Gamlin et al. 2007).

The retina

The peak of absorption of the retina is between 400 and 600 nm and its transmission is between 400 and 1,200 nm. Rods are present across the retina except for the very central region (the foveola), and provide scotopic (night) vision. Their sensitivity is 10^{-6}-1 cd/m^2, with comparatively low resolution and high sensitivity, but lacking colour information. Their absorption peak is at 498 nm (blue), but in vivo, if the lens absorption and macular pigments are taken into account, the effective maximum sensitivity of the rod integrated in the eye is shifted to 507 nm. Cones are responsible for daylight (photopic) vision. Their sensitivity varies in a wide luminance range, from 10^{-3}-10^{8} cd/m^2. Maximal absorbance for blue cones is around 450 nm, 530 nm for green cones and 580 nm for red cones.

The visual pigment in the rod is rhodopsin, which consists of opsin and the vitamin A aldehyde 11-cis-retinal. Phototransduction is triggered by the photic conversion of 11-cis-retinal to all-trans-retinal in the rhodopsin molecule. The activation of rhodopsin starts a cascade of events that leads to the closure of sodium channels, hyperpolarization of the photoreceptor membrane, and a decrease in the concentration of intracellular calcium (Pepe 1999). The phototransduction system can be modulated by several proteins (such as S-modulin [recoverin], S-antigen [arrestin], guanylate cyclase-activating protein, phosducin, and calmodulin) in a calcium-dependent manner, inducing light and dark adaptation. Rhodopsin is regenerated in the retinal pigment epithelial (RPE) cells through the visual cycle of retinoid metabolism (Bok 1990, Saari et al. 1994).

A2. In the skin

In the skin (solar) UV radiation drives the formation of pre-vitamin D3 from pro-vitamin D3 (7-dehydrocholesterol, a precursor of cholesterol). At skin temperature the pre-vitamin D3 isomerizes to vitamin D. Prolonged UV exposure however does not continue to raise vitamin D3 levels. Instead, surplus vitamin D3 is converted into inert substances and further UV exposure increases the risk of undesirable effects such as burning (Webb et al. 1989), see below section 3.5.2.1 (Figure 8). Regular and moderate sun exposure in summer appears optimal for adequate vitamin D3 production.

B. Damage

B1. In the eye

The cornea

Exposure of the cornea to UVA and UVB usually induces reversible lesions of the corneal epithelium. UVC can induce lesions of the corneal stroma and the Bowman membrane leading to corneal opacity and potentially to corneal neovascularization. IR usually only causes irritation but may, at high energy levels (>3 mJ/cm^2), also cause deep stromal lesions and even perforations. Protection from IR and UV components of the sunlight is therefore recommended in certain instances (Sliney 2001).

Upon prolonged exposure to UV (sunlight), climatic droplet keratopathy and cortical cataracts (opacification of the cortex of the lens and not the nucleus) can occur. On the conjunctiva, pterygium and conjunctival neoplasms can be observed. Ocular melanoma (mostly uveal melanoma) might also be induced by UV overexposure. Evidence for an association between ocular melanoma and sun exposure comes from Australia. A national case-control study of ocular melanoma cases diagnosed between 1996 and mid-1998 demonstrated an increase in risk of the cancer with increasing quartile of sun exposure prior to age 40 (relative risk (RR) in the highest quartile 1.8; 95% CI 1.1–2.8), after control for phenotypic susceptibility factors (Vajdic et al. 2002).

The subclinical photokeratitis level normalized to the UV-hazard action spectrum peak at 270 nm wavelength is approximately 4 mJ/cm^2 (as defined by ACGIH and ICNIRP, and stated in EU Directive 2006/25/EC). The radiant exposure at 300 nm that would be equivalent to the corneal exposure of 4 mJ/cm^2at 270 nm is 10 mJ/cm^2. Between 315-400 nm, the exposure guideline limit is 1 J/cm^2 for t <1000 s.

The lens

The lens absorbs near UV and far infrared light (<400 and >800nm) (Boettner and Wolter 1962). It is known that UV light induces cataracts (Hockwin et al. 1999, Sasaki et al. 1999) with a damage threshold of 600 mJ/cm^2 at 350 nm.

A corresponding value for 310 nm is 750 mJ/cm^2. Blue light may induce photodynamic damage in lenses which have accumulated photosensitive debris or drugs. Other compounds that accumulate in the aging lens may act as antioxidants (Balasubramanian 2000). Infrared may also cause cataracts (Roh and Weiter 1994). Cortical cataracts have been associated with UV exposure. Furthermore, it seems that exposure to UV at younger ages also predisposes individuals to nuclear cataracts later in life (Neale et al. 2003).

Whether cumulative UV exposure from artificial lighting, added to the natural light exposure, might increase the incidence of cataract at younger ages was never investigated. The absorption spectrum of the lens changes with age. In young children, more than 80% of blue light is transmitted to the retina. At around 25 years of age, only 20% of the light between 300 and 400 nm and 50% of wavelengths between 400 and 500 nm is transmitted. With increasing age, the yellow filters of the lens increase and absorb most of the blue light. The peak of absorption of the lens is around 365 nm in young adults and around 400 nm at 60 years. This natural retinal protection of the lens, increasing with age, tends to be replaced in the case of cataract surgery by yellow intraocular lenses (Margrain et al. 2004).

The retina

Light (particularly short wavelengths) can interact with photoreceptor associated opsins and retinoids and cause damage via the overproduction of reactive oxygen species (ROS) (Boulton et al. 2001), but such damage can also arise outside the photoreceptors.

In the retina, photochemical damage through oxidative stress takes place when the incident radiation has a wavelength in the high energy portion of the visible spectrum. The retina, which contains a large concentration of cell membranes, is particularly sensitive to oxidative stress because lipid peroxidation breaks down membranous structures. The photochemical damage spreads from the absorbing molecule to other molecules in an uncontrolled molecular chain reaction. There are two classes of photo-damage (see also overview in Table 2 below):

- Class I damage has an action spectrum that is identical to the absorption spectrum of the visual pigment, and it appears after exposure (of several hours to weeks) to irradiances below 10 W/m^2 of white light estimated at the retina. For comparison, an approximation calculated for this document suggests that the retinal illuminance caused by the sun shining on snow or white sand on a clear day is in the order of 30-60 W/m^2. The initial damage is mainly located in the photoreceptors, where reactive oxygen species (ROS production) can be measured upon blue light exposure in vitro (Figure 7). However, depending on the species, it seems that both RPE and photoreceptor cells can be the primary target sites.
- Class II damage has an action spectrum that peaks at shorter wavelengths, and this type of damage occurs following exposure to high irradiances of white light, at or above 100 W/m^2. The initial damage is generally confined to the retinal pigment epithelium (lipofuscin-mediated) but may then extend to the photoreceptors. In RPE cells, lipofuscin granules are converted to melanolipofuscin in aging eyes, and the lipofuscin becomes much more phototoxic and particularly sensitive to blue light with increasing age, meaning that more free radicals may be produced in the eyes of elderly people. The damage that occurs in RPE cells and subsequently in photoreceptor cells is irreversible. The damage occurrence depends on the anti-oxidant status of the retina and also on the local oxygen tension in the outer retina. The photooxidative damage taking place in the outer retina is cumulative.

Table 2: Overview of the classes of photodamage to the retina

	Class I damage ("Noell damage")	Class II damage ("Ham damage")
Exposure duration	> 1.5 hours	< 4 hours
Retinal irradiance	< 1 mW/cm2	> 1 mW/cm2
Source spectrum	Incandescent white	White and laser lines
Primary investigated animal species	Rats	Primates
Anesthesia	No	Typically yes
Size of exposure source	Large	Small
Site of major impact	Photoreceptors	RPE

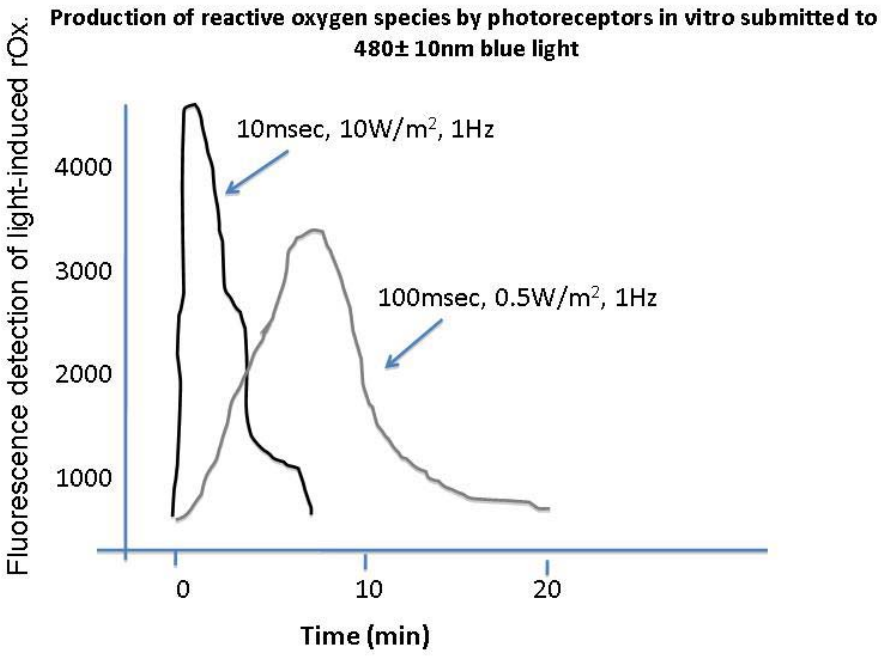


Figure 7 Production of reactive oxygen species (ROS) by rod photoreceptors exposed to blue light in vitro (adapted from Yang et al. 2003)

Other pigments exist in mitochondria in all tissues but are particularly susceptible to photochemical damage in ganglion cells that receive light directly on the retinal surface. Their peak absorption is also in the blue spectrum (e.g. peak at 450 nm for flavine).

Macular pigments

In the macula of the retina, yellow pigments located in the inner retinal layers are particularly concentrated in the fovea. The lutein and zeaxanthin pigments efficiently

absorb blue light between 400 and 500 nm (Whitehead et al. 2006). Lutein protects against oxidative damage and is a scavenger for singlet oxygen (Davies and Morland 2004, Krinsky et al. 2003, Li et al. 2010, Wooten and Hammond 2002). However, humans cannot synthesize macular pigments. They are highly concentrated in the macula of children and additional amounts of macular pigment can only be achieved through nutrient intake. Nutrient supplements have been shown to increase macular pigment density in older patients and are therefore considered to reduce the risk for progression of age-related macular degeneration (AMD) (Carpentier et al. 2009, Loane et al. 2008).

Lipofuscin

RPE cells are polarized epithelial cells with long microvilli on their apical surfaces, interfacing with the outer segments of photoreceptor cells. The tight junctions between RPE cells constitute the outer blood-retinal-barrier, selectively controlling the passage of water and ions between the subretinal space and the choroids. RPE cells play a crucial role in the phagocytosis of photoreceptor outer segments and regeneration of visual pigments (Bok 1990). At their apical side, RPE cells contain intracellular melanin granules (eumelanin and pheomelanin) as well as many microperoxisomes and antioxidative enzymes, which act as protective and anti-oxidative mechanisms. Particularly, melanin absorbs the excess of photons from 300 to 700 nm.

Lipofuscin is a mixture of chromophores that accumulates in the retinal pigment epithelium with age and in the case of several retinal disorders. It is a potent photosensitizer capable of inducing photodynamic effects and subsequent photochemical processes (Boulton et al. 1990, Wang et al. 2006), possibly causing permanent damage to RPE and photoreceptors (Wassel et al. 1999). The major fluorescent component of lipofuscin, A2E, has been identified (Sakai et al. 1996). A2E is formed in rod outer segments by a sequence of reactions that is initiated by the condensation of two molecules of all-trans-retinaldehyde with phosphatidylethanolamine. It has a visible absorption maximum between 430 and 440 nm, depending on the solvent, and generates light induced ROS (Parish et al. 1998, Reszka et al. 1995). Interestingly, age-induced changes in the lipofuscin composition and structure increase its photodynamic effect upon illumination, resulting in higher oxidative damage (Wu et al. 2010).

Several other native retinal chromophores including melanin (Margrain et al. 2004), protoporphyrin (Gottsch et al. 1990), all trans-retinal (Delmelle 1978, Wielgus et al. 2010) and other lipofuscin components (Gaillard et al. 1995, Reszka et al. 1995, Wassel et al. 1999) have been suggested to act as photosensitizers of damage. The maximal potential phototoxic retinal damage is expected to occur with blue light wavelengths between 430 and 460 nm.

It has been recognized that retinal mitochondria also contain photosensitizers capable of generating ROS under blue light stimulation in photoreceptors (Chen et al. 1992a, Chen et al. 1992b), RPE cells (King et al. 2004, Youn et al. 2009) and in retinal ganglion cells (Osborne et al. 2006). A recent hypothesis suggests that retinal ganglion cells, which are particularly rich in mitochondria, may suffer from light-induced damage, particularly in pathologic conditions such as glaucoma (Osborne 2010). ROS generation by retinal mitochondria is mostly stimulated by the shorter blue light wavelengths (404-420 nm).

The interaction between light of different wavelengths and eye structures, and the possible consequences is presented in Table 3 below.

Table 3 Interaction of light with eye tissues and chromophores

Tissue/ molecule	Wavelength (nm)	Mechanism	Consequence
Cornea	<300 and >800	Heat dissipation	Keratitis, droplet keratopathy
Iris	Melanin: 380-700	Heat dissipation	
Lens	Peak at 365 at 8 years Peak at 450 at 65 years	Heat dissipation	Cataract (nuclear and/or cortical)
Retina	400-700 Rhodopsin: 507 SWS: 450 MWS: 530 LWS: 580	Photochemical damage type I: max at 507 nm type II: max at shorter wavelengths Toxicity potential	Solar retinitis Maculopathy Aggravation of retinopathy
RPE	Melanin: 380-700	Heat dissipation	Potentiation of lipofuscin toxicity (melanolipofuscin)
Lipofuscin	355-450 A2E: peak at 430-440	Photodynamic effect Retinal toxicity	Solar retinitis Age-related maculopathy (probable) Aggravation of retinopathy
Xantophylle pigments	Lutein: 446 Xanthine 455 Zeaxanthine 480	Heat dissipation	Reduced blue light toxicity Protects against AMD

Notes: RPE = retinal pigment epithelium; SWS = short wavelength sensitive cones (blue); MWS = medium wavelength sensitive cones (green); LWS = long wavelength sensitive cones (red); and AMD = age-related macular degeneration.

B2. In the skin

As pointed out earlier, UV radiation is very (photo-) chemically active on a large variety of organic molecules, most prominently on DNA. Next to direct damage to molecules like DNA, UV radiation can generate reactive oxygen species and various kinds of radicals which can then damage cell components. At low UV exposure levels the skin is perfectly capable of coping with this UV challenge through antioxidants, radical scavengers and repair mechanisms, and the exposure will have no direct noticeable effect (see de Gruijl 1997). If the exposure, and the damage, increases to levels where the functions of the cell become seriously disturbed, the cell may become apoptotic (undergoing programmed cell death). The UV radiation at higher levels has a clear toxic impact which evokes an inflammatory reaction. In the long term, sub-acute damage may cause accumulation of gene mutations in the (stem) cells of the epidermis (causing cancer) or cause loss of collagen in the dermis with a subsequent gradual loss of elasticity ("photo-aging"). Specific UV signature mutations (at sites of neighbouring pyrimidine bases in the DNA) were found in p53 tumour suppressor genes in a majority of human skin carcinomas, providing direct evidence that UV radiation had contributed to the development of these tumors (de Gruijl et al. 2001).

3.5. Adverse health effects in the general population

Besides the short-term local effects presented in section 3.4.3, optical radiation (or lack of it) can cause systemic or long-term adverse health effects.

3.5.1. Photothermal effects

Acute thermal damage (burns) to the skin is usually prevented or minimized by aversion responses. Such burns may be evoked by extremely intense sources of optical radiation,

such as lasers or high-power flash lamps. Under extreme conditions (environmentally and/or physically) high levels of (solar) visible light and/or IR (as in IR saunas) may heat the skin and thus contribute to a breakdown of the body's thermoregulation through the skin resulting in a "heatstroke". If not treated properly such a systemic hyperthermia may be fatal. However, such extreme heat assaults are not to be expected from artificial optical sources intended for lighting purposes.

Regular localized heating of the skin (stoves under the feet or a hot water bottle on the stomach), not necessarily causing burns, can cause a skin condition dubbed "erythema ab igne", reddish to brown colouration (Edwards et al. 1999) and has anecdotally and in clinical case reports been associated with the development of skin cancer, "turf fire cancer" or "cangri cancer" (ICNIRP 2006a). In mouse experiments IR and higher room temperatures were found to enhance skin tumour formation from chronic UV radiation (van der Leun and de Gruijl 2002); epidemiologic data of skin cancer incidence in different geographic locations indicate that this may also be true in humans (van der Leun et al. 2008).

3.5.2. Photochemical effects

3.5.2.1. Vitamin D status

UV **deprivation** can lead to adverse health effects. As described in 3.4.3.2 A2, vitamin D3 is produced naturally in the skin from exposure to UVB radiation in sunlight. As the nutrient vitamin D is contained in inadequate quantities in our modern Western diet, the vitamin D status shows considerable seasonal variation in temperate climates because of ineffective solar UV exposure in wintertime. Vitamin D needs to be metabolised in order to become active as a hormone: it is hydroxylated to 25 hydroxyvitamin D3 and 1,25 hydroxyvitamin D3 in the liver and kidney, respectively. The latter metabolite binds to the "vitamin D receptor" in gut epithelial and bone cells. This is known to regulate calcium absorption and mobilisation in the gut and bones. Over the last few decades it has, however, become clear that 1,25 dihydroxyvitamin D can be formed outside the kidneys (extrarenally) in various tissues and immune cells, and the vitamin D receptor is present in a plethora of different cell types triggering various responses. Thus, vitamin D may potentially have a broad impact on health, but the evidence for this is generally inconclusive. Although many observational epidemiological studies have reported decreases in the risk of various diseases and conditions (e.g. schizophrenia, autism, multiple sclerosis, diabetes, respiratory tract infections, influenza and certain types of cancer) associated with elevated levels of vitamin D or increased sun exposure, the available data in humans are mostly either too sparse or inconsistent, and generally inadequate to assert any causal relation (Norval et al. 2011, Zhang and Naughton 2010). The evidence for colorectal cancer due to vitamin D deficiency is mounting and found to be "persuasive" but "limited" by the International Agency for Research on Cancer (IARC 2008), although some experts have recently qualified it as "sufficient" (Dutch Cancer Society 2010).

Furthermore, UV deprivation in wintertime causes a loss of skin photo adaptation which in some individuals suffering from photosensitivity disorders such as polymorphic light eruptions may predispose to springtime provocation of their condition upon renewed UV exposure in springtime or higher levels of exposure during summer holidays (see section 3.6.1.1).

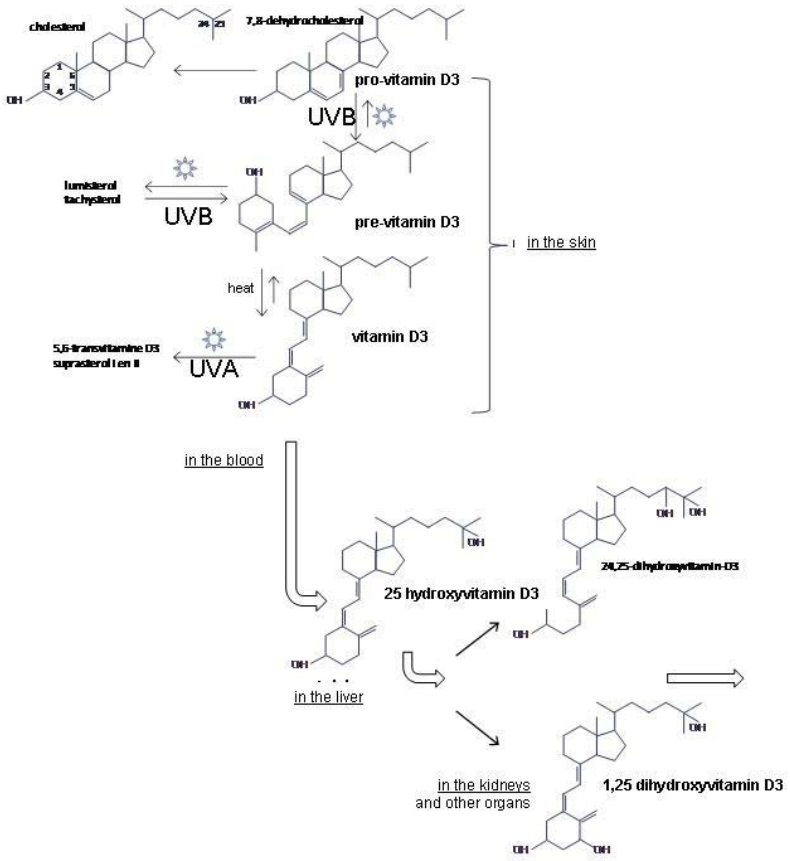


Figure 8 Photosynthesis of vitamin D3 and further metabolism (adapted from Dutch Cancer Society 2010)

3.5.2.2. Assessment of effects on healthy skin

Pain sensation from thermal effects occurs at lower skin temperatures than those at which burns occur. The thresholds for "effective" irradiancies of "thermal radiation" are given in DIN 33403 (DIN 2001), and amount to 1 kW/m^2 for exposure times over 5 minutes (where "effective" refers to the difference between the incoming and emitted radiant flux density at the skin surface; the latter is about 460 W/m^2 at 30°C skin surface temperature and emissivity of 0.97, while about 410 W/m^2 would be incoming in a room with 18°C wall temperature and emissivity of 1). With shorter exposure times (t), this threshold goes up (approximately in direct proportion to $t^{-1/2}$); the International Commission on Non-Ionizing Radiation Protection (ICNIRP) has formulated limits for exposure times up to 10 s (irradiance <20,000 $t^{-3/4}$ W/m^2; t in s), which could be extended to longer exposure times to give very conservative limits (ICNIRP 2006b). No limits were given by ICNIRP at these longer exposure times because effects strongly depend on thermal environmental conditions. Natural avoidance behaviour will restrict exposure times and prevent thermal injury. In practice, thermal pain sensation from an illuminator is quite exceptional and would only occur in very close proximity to very high intensity sources.

Other thermal damage, such as erythema ab igni, would require protracted substantial heating of the skin which is not likely to occur from lighting sources. There are indications that elevated ambient temperatures and/or IR radiation may increase skin cancer formation from sun (UV) exposure (van der Leun et al. 2002, van der Leun et al.

2008) and that IRA may enhance skin aging, but evidence in humans is very weak or lacking, and the effects cannot be quantified in any reliable way.

Overexposure to UVB and UVA radiations cause well known sunburn reactions. The first feature is skin reddening ("erythema") after a couple of hours and at higher doses severe discomfort and, after a couple of days, skin peeling. The UV radiation at these dose levels has a clear toxic impact which evokes an inflammatory reaction causing dilation of superficial capillary blood vessels (increased redness, and skin temperature), leakage of serum through vessel walls (causing oedema) and trafficking of immune cells from the blood vessels into the skin and from the skin into draining lymph vessels. The effective UV dose for sunburn is generally assessed by an action spectrum standardized by the Commission International de l'Éclairage (the erythemal action spectrum; CIE 103/3 Reference Action Spectra for Ultraviolet Induced Erythema and Pigmentation of Different Human Skin Types). The skin can adapt to gradually increasing levels of UV exposure (occurring from spring to summer). This decrease in sensitivity is accompanied by a tanning reaction, but people that do not tan are nevertheless capable of acclimation to increasing UV levels.

The threshold UV dose for a minimal reddening of the skin occurring some 4 to 8 hours after exposure (the minimal erythemal dose, MED) for a fair-skinned Caucasian (skin phototype II) is typically 200-250 J/m^2 when the exposure is spectrally weighted according to the CIE-erythemal action spectrum, which equals 2-2.5 SEDs (SED stands for standard erythemal dose = 100 J/m^2 of CIE-erythemally weighted UV). The solar midday exposure rate is given as the UV index in weather reports, where 1 hour of exposure at a UV index of 7, as in summer in Northern Europe, amounts to 6.3 SEDs and at a UV index of 10, as in the Mediterranean region, to 9 SEDs, i.e. about 2.5 to 4.5 times the typical MED of a previously unexposed, unadapted fair-skinned Caucasian. The actual MED of unexposed fair skin varies, with 95% within the range of 2.5 times below and over the median of about 2.2 SED (based on a median of 34 J/cm^2 and 95% in 14–84 J/cm^2 at 300 nm; Diffey 1991). MEDs also vary over the body. In acclimation of the skin to UV radiation the MED is raised several-fold, preventing sunburns from occurring as the UV index increases from spring to summer. Skin peeling occurs after severe overexposure, >4 MEDs.

As 1 MED of whole body irradiation has been estimated to produce roughly up to 20,000 IU of vitamin D (Holick 2004), and because most fair skinned people maintain adequate levels of vitamin D over summer (>50 nmol/l 25-hydroxyvitamin D) (Frost et al. 2010, Hyppönen and Power 2007, Webb et al. 2010), it has been asserted that regular brief exposures (15–30 minutes) in summer clothing to midday summer sun is adequate for vitamin D, which is supported by recent experimental evidence (Rhodes et al. 2010b). With low UV indices it is not possible to produce adequate levels of vitamin D in winter time, and the majority of people in Northern and Northwestern Europe do not maintain an adequate vitamin D status (Hyppönen and Power 2007, Webb et al. 2010). It was shown that 3.9 SEDs/week from a simulated summer sun to a group of volunteers in summer clothing was sufficient to attain sufficient vitamin D statuses in wintertime (Rhodes et al. 2010b). UV exposure from indoor lighting will commonly fall well below this level; even fluorescent lamps at the top end of CEI/IEC Risk Group 0 for UV emission will not come close in an office setting. Hence, lamps for indoor lighting would most likely need to overstep the current UV emission norm to be effective in vitamin D production. However, a shortage of vitamin D from sunlight in winter can easily be compensated for by oral vitamin D supplements, or by a diet rich in fatty fish.

Overexposure to UV radiation, but also to a lesser degree sub-acute doses (<1 MED), can suppress adaptive cellular immunity (i.e. acquired immunity against a pathogenic agent or substance and effected by direct cell-to-cell contact) which in animal experiments was proven to contribute to skin cancer formation and aggravate bacterial and viral infections (Norval 2006b). Solar overexposure is thus known to cause cold sores in humans, a flare-up of an infection with Herpes Simplex viruses (Norval 2006a, Sayre et al. 2007). On the other hand, UV irradiation is known to boost innate immunity (inborn

defences against infectious agents; UV exposure increases levels of anti-bacterial proteins in the skin) (Gläser et al. 2009). The immunosuppression apparently serves to prevent adverse immune (allergic) reactions to the UV-exposed skin (putatively against photochemically altered molecules) while the boosted innate immunity increases acute defences against exogenous infectious agents.

Episodes of severe sunburns have been found to be associated with increased risk of skin cancer, specifically of malignant melanomas (Gandini et al. 2005), but also of basal cell carcinomas (Kütting and Drexler 2010). UV exposure in childhood is linked to increased risk of melanoma later in life (Armstrong and Kricker 2001).

The action spectrum for the UV induction of squamous carcinomas has been determined in (hairless) mice and resembles that of sunburn (de Gruijl and Van der Leun 1994). Hence, UV indices, based on sunburn effectiveness, given in weather reports also reflect the carcinogenic effectiveness of sun exposure. The action spectrum for photo-ageing is not well defined, and could range from UV to IRA.

In contrast to sunburn, there is no threshold dose known for photo-aging and it does not exist for the induction of skin cancers.

Cancer is the result of a probabilistic process for which increasing UV dose or more excessive UV dosages increase the chance. This complicates defining an "acceptable UV dose" because it requires a choice regarding what is an "acceptable risk". Moreover, the data on skin cancer and related sun (UV) exposures are generally not detailed enough for adequate risk assessments of personal UV exposure. As the experimentally determined action spectrum for the induction of skin carcinomas roughly resembles the sunburn action spectrum (de Gruijl and Van der Leun 1994), relating annual ambient erythemal exposures and typical personal exposures to the actual skin cancer incidence in a population is informative. Skin cancer incidences are quite substantial in Northwestern Europe even though ambient UV levels are considered low. For Denmark the following was recently reported: "Between 1978 and 2007, the age-adjusted incidence of basal cell carcinoma (BCC) increased from 27.1 to 96.6 cases per 100,000 person-years for women and from 34.2 to 91.2 cases for men. The incidence of squamous cell carcinoma (SCC) increased from 4.6 to 12.0 cases per 100,000 person-years for women and from 9.7 to 19.1 cases for men" (Birch-Johansen et al. 2010). These increases are most likely attributable to increases in sun exposure decades earlier. The median annual exposure among 164 Danish volunteers (age 4-67 years) was found to equal 166 SEDs (95% within 37-551 SEDs; Thieden et al. 2004). A quarter of lifetime exposure was received before the age of 20 years. A recent Danish study also reported that sunburning was quite common, with 35% of 3,499 people (age 15–59) reported to have been sunburned in the preceding 12 months (Køster et al. 2010). The incidence of melanoma in Denmark in 2008 was 20.2 and 26.6 per 100,000/y for men and women, respectively. This was the highest among women in Europe (mortality 4.3 and 2.5 per 100,000/year, respectively[4]). Based on Norwegian data and on US skin cancer surveys among non-Hispanic white Caucasians, SCC and BCC incidences increased by 2.3 ± 0.5 and 1.7 ± 0.3%, respectively, per 1% increase in ambient annual erythemal dose (NRPB 2002). Incidences of melanoma are reported to increase by about 0.6 ± 0.4% (Eide and Weinstock 2005, Slaper et al. 1996) per 1% increase in ambient annual erythemal dose. However, although episodes of severe sunburn are linked to an increase in melanoma risk and sunscreen use by adults has been proven to protect against melanoma (Green et al. 2011), whether the erythemal action spectrum is appropriate for melanoma is still debated.

With a large spread, the annual erythemal dose of outdoor workers is about twice that of indoor workers in Northwestern Europe (medians of 3.1 vs 6.7% of ambient dose, excluding holidays, i.e. 138 vs 300 SEDs/y, 95% interval about 3-fold below and over medians; Schothorst et al. 1985). Outdoor professions are commonly associated with

[4] See European Cancer Observatory: http://eu-cancer.iarc.fr/cancer-11-melanoma-of-skin.html,en#block-9-20.

increased risk of SCC and BCC. Thus, it was found that in German males, outdoor occupations brought about a relative risk (RR) = 2.9 (95% CI, 2.2-3.9), for BCC and 2.5 (95% CI, 1.4-4.7) for SCC (Radespiel-Tröger et al. 2009). A recent metastudy on occupational UV exposure found that 16 out of 18 studies reported an increased risk of SCC in outdoor workers (in 12 of the studies there was a significant increase) and an overall odds ratio of 1.8 (95% CI, 1.4-2.2) (Schmitt et al. 2011). This pattern is not entirely consistent (Green et al. 1996, Håkansson et al. 2001), and a recent Danish study even found a lower risk of SCC in outdoor workers, an odds ratio of 0.83 (95% CI, 0.77 – 0.88) (Kenborg et al. 2010). The steady increase in SCC in the general population over decades, most likely owing to increased sun exposure in leisure time, probably gradually evens out the differences with outdoor workers.

No increase in the risk of malignant melanoma (Radespiel-Tröger et al. 2009), except for those on face, hands (Beral and Robinson 1981) and eye (Håkansson et al. 2001), has been observed among outdoor workers. On the contrary, some studies show a significant reduction in risk (Rivers 2004), whereas office workers showed an increased risk on trunk and limbs (Beral and Robinson 1981); plausibly attributable to fewer sunburns in the acclimated skin of outdoor workers, in contrast to more frequent sunburns among indoor workers because of intermittent overexposure of their un-acclimated skin.

An estimate of a 3.9% (1.6-12%) increase in lifelong SCC risk for office workers by exposure to fluorescent daylight lamps was previously calculated (Lytle et al. 1992-1993). The effect is small but not entirely negligible. The study combined measurements of UV spectra of commercially available fluorescent daylight lamps (four different types) on the US market with the above mentioned percentage increase in SCC per percent increase in ambient UV, and estimates of personal UV exposures from sunlight and unfiltered fluorescent lighting in schools and offices. Filtering through acrylic prismatic diffusers, instead of louvers, in the fixtures was found to reduce the effective UV exposure by more than 100-fold, i.e. virtually eliminating any contribution to the SCC risk. More recently, a survey of indoor lighting sources in the USA revealed UV levels of wavelengths around 300 nm in the order of 10^{-5}-10^{-4} W/m^2/nm at a distance of 20 cm, comparable with outdoor summer levels of solar radiation, and even higher levels than in solar radiation at shorter wavelengths (Sayre et al. 2004). In the UVA range the levels were generally orders of magnitude lower than those in summer sun. No erythemally effective dosages were given.

Based on a small sample of CFL spectra (kindly provided by Ms. M. Lukovnikova of the Belgian Federal Public Service of Health) we found that the emission of erythemally effective UV from compact fluorescent lamps varied enormously from one type to the next: from undetectable levels (<0.05 mW/m^2) at 500 lx to substantial levels (a few mW/m^2, i.e. around 100 SED/year with persistent exposure at this level during office hours) (office lighting up to 1,000 lx, which should be compared to the levels in a living room which would typically be around 50 lx, whereas full sunlight would exceed 100,000 lx). These substantial levels of UV emission are easily prevented by producing lamps with UV-absorbing glass envelopes. In actual practice the indoor exposures for most indoor workers are likely to fall well below those corresponding to a continuous level of 500 lx over 8 h/day (Lytle et al. 1992-1993). Unfortunately, comprehensive reliable data on indoor personal UV exposures in Europe appear not to be available, suggesting that UV exposures in offices are in actual practice not commonly monitored and controlled (limits given in Directive 2006/25/EC for indoor workers). Studies performed by the Dermatology Department of the Bispebjerg Hospital in Copenhagen showed very low personal UV exposures in December and January (medians of 0.002–0.003 SED/day; Thieden et al. 2006), but these readings may not have been entirely reliable at such low levels (detection threshold stated at 0.1 SED/h) and may not have adequately included indoor exposures (more directed at solar exposures).

For indoor workers the EU (Directive 2006/25/EC; following the American Conference of Governmental Industrial Hygienists, ACGIH, and ICNIRP) has chosen a daily limit of 30 J/m^2 of "actinic UV" to avoid short-term damage ("sunburn") to skin and eyes. This

actinic UV dose is spectrally weighted according to an action spectrum largely based on photokeratitis of the eye: this dose limit corresponds to 0.75 SED at 298 nm and overall to about one third of a typical MED with broadband UV lamps (Sliney and Bitran 1998). This limit is generally exceeded by outdoor workers, and by people who expose themselves excessively during sunny summer holidays (e.g. with some 10-20 SEDs/day). Thus, this UV limit for indoor workers would ensure that the risk stays well below that of outdoor workers, and only in exceptional cases (e.g. welders) might this limit be reached, i.e. on average the personal risk will be small. However, one should be careful with applying this exposure limit to the indoor UV exposure of the general population: a small increase in personal UV exposure for an entire population may then result in a substantial additional number of skin cancer cases per year. Thus, a life-long increase of 7% in erythemal UV in Northwestern Europe (caused by an ozone depletion) was estimated to increase BCC and SCC incidences by 14 and 25%, respectively, (Madronich and de Gruijl 1993) which would have amounted to about 2,000 and 750 additional cases of BCC and SCC in the Netherlands in the year 2000 in a population of about 16 million people (de Vries et al. 2005). Following CEI/IEC (CEI/IEC 62471, 2002), the EU (EN 62471, 2008) has adopted UV emission standards for lighting lamps where the lowest risk category (Risk Group 0), which is assumed "safe", is based on the exposure limit for indoor workers: i.e. 30 J/m^2 of actinic UV over 8 h at 500 lx, which corresponds to 2 mW/klm. However, even sources complying with this UV emission limit, may contribute considerably to the annually accumulated UV dose at 500 lx. As an illustration, we present worst case scenario studies of population-wide extensive exposures to fluorescent lamps currently on the EU market to assess the potential impact on incidence of SCCs; see section 3.7.

Conclusions on effects on healthy skin

Thermal effects from visible or IR radiation emitted by lighting sources are unlikely to cause any serious health effects in healthy skin; problems may arise only with excessively intense sources with exposures in close proximity of the source (adherence to DIN 33403 for pain thresholds or more conservative expansion of the ICNIRP limit to exposure times over 10 s).

Considering the data on UV effects, the sunburn reaction would appear to be the practical key for proper control of UV exposure levels on the skin, both for short- and long-term health effects. Minimizing sunburn reactions is advisable to prevent acute discomfort from inflammatory skin reactions, and to minimize possible adverse effects from immune modulation. In the long term it is likely to lower the risk of the most fatal skin cancer, melanoma. Limiting the daily erythemal dose will further limit the life-long accumulated dose which is likely to lower the risk of the skin carcinomas, SCCs and BCCs.

Although a risk assessment of SCC risk from indoor UV exposure appears feasible, SCENIHR had to resort to simplified worst case scenarios (see section 3.7) as adequate data on personal exposures were not available. The worst case scenarios presented in section 3.7 suggest that lamps that comply with current limits on UV emission (preventing acute/short-term adverse effects) may have a substantial impact on SCC incidences when a population is subjected to extensive and large scale exposure to these lamps.

A comprehensive database of emission spectra (including UV) of lamps on the European market, with routine checks and updates, together with data on actual personal exposures would be very useful for public health monitoring. Future measurements of actual personal indoor UV exposures may indicate whether more vigilance is warranted and if current regulation on indoor UV exposures of the general public is appropriate.

The current categorization of UV emissions from lighting lamps in risk groups is primarily based on a UV exposure limit for indoor workers. This exposure limit has been translated into an emission limit of 2 mW actinic UV per klm. Lamps below this emission limit are in

the first and lowest risk group which is considered "safe" and exempt of any liability. Although personal risks may be low under these exposure and emission limits, adopting these limits for the general population can, nevertheless, conceivably result in a substantial number of additional cases of skin carcinomas each year (section 3.7). Any acceptable limit on population-wide risk should be translated into UV exposure limits for the general population and corresponding limits on UV emissions from lamps for lighting purposes. This retracing of a risk limit to an emission limit would require reliable data on personal (UV) exposures from lamps and luminaires in actual practice (with known spectral output in UV and VIS, and known UV radiant power over luminous flux ratios [W/lm] and illuminances [lx]). As such detailed data are currently lacking, a UV emission limit can now only be based on worst case scenarios like those presented in section 3.7.

With any of these potential health effects from artificial lighting sources, it is always advisable to take sun exposure (however variable it may be) as a reference. Designing light sources to include UV and stimulate vitamin D (such as "Full Spectrum Fluorescent Lighting", Hughes and Neer 1981, as referred to by McColl and Veitch 2001) and related health effects may introduce unwarranted long-term risks to eyes (cataracts) and skin (carcinomas). In contrast to persons deliberately exposing themselves to sunbeds for cosmetic or presumed health effects, persons staying indoors do not expect to be exposed to UV radiation from the lighting, and lighting lamps should therefore evidently be adequately low in UV output.

3.5.2.3. Assessment of effects on the healthy eye

There are no studies available on the possible effects of domestic artificial light on the human eye.

However, studies have been performed on hazards of specific artificial lights, mostly ophthalmologic instruments, on human eyes.

A considerable number of studies are also available on the effects of artificial light on the retina of laboratory animals (e.g. mice, rats, monkeys, dogs…). These studies have been the basis of the understanding of light induced toxicity to the retina and have defined the wavelengths responsible for photochemical damage to the retina.

A. Light and cornea and lens pathology

UV and IR radiation can cause corneal and lens lesions. However, medical and/ or surgical treatments are available for these pathogenic events and permanent vision decline would only in exceptional cases result from UV or IR induced permanent damages. Lesions differ whether they result from acute or chronic exposure.

In contrast, putative light-induced retinal pathology is in the majority of the cases non-reversible and not treatable.

i) Corneal and conjunctival lesions induced by UV exposure

The ocular UV environment is a function of both the direct as well as the diffuse UVR. The diffuse component of UVR has a strong effect on the eye since it is incident from all directions. Compared to visible light, UVR is strongly scattered, as the amount of scatter increases greatly with decreasing wavelength. On average 40% of the total global UVB dose is diffuse radiation. This fact and the natural aversion of the eye from direct bright radiation mean that the majority of UVR arriving at the cornea is from diffuse scatter and not from direct sunlight (Parisi et al. 2001, Sliney 1999). For acute UV exposure (normally limited to wavelengths below 315 nm in the UVB-UVC bands) the only effect on the normal eye is photokeratitis (Bergmanson and Sheldon 1997) (see also Figure 6). In

severe cases of this condition, an anterior stromal edema can be observed. Rarely, endothelial lesions can occur as a result of UV keratitis (Dolin and Johnson 1994). Lamps used for normal lighting purposes that belong to RG0 or RG1 would not be expected to cause photokeratitis because toxic thresholds will not be reached (0.03-0.06 J/cm^2).

Chronic exposure to UV and other environmental factors such as sand, dust, wind, and dry conditions induce climatic droplet keratopathy. Its occurrence is more frequent in people with previous solar keratitis and the process progresses as the individuals continue their light exposure.

Climatic droplet keratopathy is a degenerative process characterized by golden-brown translucent material found in the anterior corneal stroma, Bowman's layer, and subepithelium. Initially, the deposits are found near the limbus of the cornea within the interpalpebral zone. They may progress as large nodules up to the central cornea resulting in decreased vision. Deposits may also infiltrate the epithelium and the conjunctiva and become painful (Cullen 2002).

UV exposure can also induce conjunctival lesions such as pingueculae and pterygium. The former are elevated masses of conjunctival tissue, almost always occurring within the interpalpebral zone at the 3 and/or 9 o'clock limbal area. The mass consists of basophilic subepithelial tissue. The lesions are usually bilateral but tend to be more frequent nasally due to increased UV radiation exposure from reflection off the nose. Occasionally, the pingueculae may become inflamed and cause ocular discomfort. A pterygium is typically triangular in shape with the apex extending onto the cornea. The tissue is fibrous and tends to be highly vascularized. Bowman's layer below the tissue is destroyed as it crosses the cornea. There is strong evidence that pterygia are caused by UV. Pterygia are found with more frequency nasally. Pterygium can cause decreased vision as it progresses on the visual axis. It also causes inflammation and discomfort and astigmatism. Outdoor work is a recognized factor for pterygium development (Luthra et al. 2001, Shiroma et al. 2009). These studies showed that pterygium was almost twice as frequent among persons who worked outdoors, but was only one fifth as likely among those who always used sunglasses outdoors. The Blue Mountains Eye Study examined 3,654 residents aged 49+ years during 1992 to 1994 and then re-examined 2,335 (75.1% of survivors) after 5 years to assess the relationship between baseline pterygium and pingueculae and the 5-year incidence of age-related maculopathy (ARM) (Pham et al. 2005). The study found that pterygium was associated with a 2- to 3-fold increased risk of incident late and early ARM.

There are, however, no studies available that have specifically investigated the effect of indoor lighting on these conditions. Furthermore, the available scientific literature has focused on acute effects of UV and not evaluated effects of chronic exposures.

ii) Cataracts

A cataract is defined as a decreased transparency of the ocular crystalline lens or its capsule. Cataracts are divided into three subtypes; subcapsular cataract occurs just behind the anterior capsule of the lens or in front of the posterior capsule, cortical cataract affects the cortex of the lens, and a nuclear cataract is an opacification of the lens nucleus that in addition causes refractive myopic changes. These subtypes can occur concurrently in any combination.

Cortical cataracts have been associated with chronic, but not acute, UV exposure (Taylor et al. 1988).

Indeed, in a case-control study within the Nambour (Australia) Trial of Skin Cancer Prevention conducted between 1992 and 1996, 195 cases with a nuclear opacity of grade 2.0 or greater were compared with 159 controls (Neale et al. 2003). Structured questionnaires were used to ascertain lifetime sun exposure history, eyeglasses and sunglasses use, and potentially confounding variables such as education and smoking.

There was a strong positive association of occupational sun exposure between the ages of 20 and 29 years with nuclear cataract (odds ratio = 5.9; 95% confidence interval = 2.1-17.1). Exposure later in life resulted in weaker associations. Wearing sunglasses, particularly during these early years, afforded some protective effect.

When the pupil is dilated (i.e. when wearing sunglasses), the incident sunlight reaches the germinal epithelial lens cells. Most cortical opacities are located in the lower nasal quadrant, most likely owing to the convergence of sideways incident solar rays (the "Coroneo effect", Coroneo et al. 1991).

iii) Conclusions on light and cornea and lens pathology

The only effect of acute exposure to UV, particularly UVB and UVC below 300 nm, is photokeratitis of the cornea and conjunctiva, a condition which would not be expected to be caused by lamps used for normal lighting purposes and belonging to RG0 or RG1.

Chronic UV exposure from sunlight may cause corneal lesions (climatic droplet keratopathy) as well as cortical and nuclear cataracts of the lens.

B. Light and retinal pathology

i) Sunlight and retinal pathology

Acute exposure: Solar retinitis

The visual disturbances caused by a few minutes of directly looking into the sun or to a solar eclipse have been known for many years (Young 1988). Eyes from patients who volunteered to stare at the sun prior to enucleation had various degrees of injury in the RPE cells 38-48 hours later, and only minor changes of the outer segments and inner segments of the photoreceptors. This explains the good vision shortly after exposure. The damage to the RPE was very similar to photochemical damage observed in the RPE of the monkey 48 hours after exposure to blue light (Ham et al. 1978).

Whilst RPE and the blood retinal barrier restores rapidly, permanent degeneration of the photoreceptors was observed some time after the exposure (Tso and La Piana 1975) inducing various degrees of visual disturbances and central scotoma. It is now well recognized that sunlight-induced retinal lesions result from chemical damage similar to that observed after blue light exposure, and not from thermal injury.

At noon in the summer the sunlight may reach 100,000 cd/m^2 but the overhead protection of the cornea by the upper lid and the natural avoidance reflex protects from direct exposure. However, in some specific situations, increased retinal exposure may occur from ground reflections even during decreased luminance conditions, which may enhance the lid opening. Indeed, ground surface reflection is the most important environmental exposure factor (Sliney 2005b). For example, prolonged exposure to a hazy sky on fresh snow may induce overexposure and lesions. Unprotected soldiers exposed to sand reflexion for several months in the desert have shown macular lesions similar to solar eclipse retinitis. Even if nobody stares directly at the sun, cumulative chronic low intensity exposure to the sunlight (particularly with ground reflexion) may induce similar lesions (Gladstone and Tasman 1978).

Glare

The eye continuously adapts to light, which allows humans to see about 10 orders of magnitude of illuminance, from almost total darkness to highly luminous environments. Nevertheless, at a given time, vision is possible and comfortable only within a two or three order of magnitude range. Glare occurs with too much light. It is empirically

divided into two types (see Marshall and Sliney (1997) for a comprehensive review). *Discomfort glare* does not impair visibility but causes an uncomfortable sensation that causes the observer to look away from the glaring source. It increases when the light source is facing the observer. *Disability glare* is due to the light scattering within the ocular media which creates a veil that lowers any contrast and renders viewing impossible. High luminance light sources generate a veiling glare with a luminance which decreases as the inverse of the angle between the direction of the point source and the direction of the gaze.

The luminance of the sky is rather stable at about 5,000 cd/m^2. This value can be exceeded on bright surfaces on clear days when luminance can reach several tens of thousands cd/m^2. The sun is never viewed directly except when it is at sunrise or at sunset when its luminance is about the same as the sky and its colour temperature low or moderate. It is when both the luminance and the colour temperature of the light are high that the blue light hazard increases.

The spectral sensitivity to glare at night has its maximum around 507 nm which is the most efficient radiation for the rods. However, the mechanisms for glare are not fully understood and the role of the recently discovered intrinsically photosensitive retinal ganglion cells (ipRGCs) which are active during daytime is not clear. Nevertheless, light with a relatively high content of blue is liable to generate glare both during daytime and night-time.

Whatever the type of glare and its source, it is not in itself a health effect, but an inconvenience that can substantially affect the vision.

Blue light hazard

As detailed above in section 3.4.3.2 B1, the interaction of blue light with molecules constituting the retina or accumulating in the retina with age or in pathological conditions can induce damage to RPE cells, photoreceptor cells, and to ganglion cells. This "blue light hazard" was identified more than 40 years ago (Noell et al. 1965, Noell et al. 1966). Subsequent studies have shown that the shortest wavelengths in the visible spectrum are the most dangerous ones for the retina (e.g. Gorgels and Van Norren 1998, Ham et al. 1976, Ham et al. 1979) and the mechanisms of light-induced damage have been reviewed previously by others (Organisciak and Vaughan 2010, Wu et al. 2006).

A recent review (van Norren and Gorgels 2011) has furthermore analyzed the relevant studies on action spectra of photochemical damage to the retina. Data from four different species were included, and the outcome of the analysis is that most studies agree that retinal damage is higher at shorter wavelengths and decreases with increasing wavelength. The review furthermore stresses that there are significant knowledge gaps in several areas related to retinal damage.

The potential phototoxic retinal damage is expected to occur with wavelengths in the blue light spectrum between 400 and 460 nm (Algvere et al. 2006, Ham 1983, van Norren and Schellekens 1990). The first evidence for retinal light toxicity of blue light came from the observation of Noell in 1965, who accidentally discovered that the retina of albino rats can be damaged irreversibly by continuous exposure for several hours or days to environmental light within the intensity range of natural light. The intensity of light that damages the retina is several orders of magnitude below the threshold of thermal injury in pigmented animals. The same damage as in albinos of different strains is produced in pigmented rats when the pupils are dilated (Noell et al. 1966). Other investigators have by now characterized non-thermal retinal damage in several species (see Organisciak and Vaughan (2010) for a recent review).

The wavelength is considered to be one of the factors that enhance the susceptibility to light damage in animal studies. Thus, for both Class I and Class II photochemical

damage, the action spectrum peaks in the short wavelength region, providing the basis for the concept of blue light hazard.

Laboratory studies have suggested that photochemical damage includes oxidative events. Grimm et al. (2001) have shown that blue light causes photopigment-mediated severe retinal damage under experimental conditions (Class I damage). Indeed when light hits a photoreceptor, the cell bleaches and becomes useless until it has recovered through a metabolic process called the "visual cycle." Absorption of blue light, however, has been shown to cause a reversal of the process in rodent models. The cell becomes unbleached and responsive again to light before it is ready. This greatly increases the potential for oxidative damage, which leads to a buildup of lipofuscin in the retinal pigment epithelium (RPE) layer.

Further evidence that light damage is mediated by photopigments was provided by studies in which monkeys were exposed to different wavelengths to bleach predominantly the pigment in one particular class of photoreceptors. The damage to the blue cones was permanent whilst damage to green cones was reversible (Sperling and Harwerth 1971, Sperling et al. 1980). More recently, murine retinal explant cultures were irradiated with visible blue light (405 nm) with an output power of 1 mW/cm^2 (Roehlecke et al. 2011). Live retinal explants displayed an increase in reactive oxygen species production after 30 min of blue light exposure. Longer exposures were accompanied by photoreceptor cell death.

Besides a possible wavelength dependency, it has been suggested that photochemical damage depends on the total dose received. This implies that the light intensity and the duration required to cause a certain level of damage are correlated, and that longer light exposure can substitute for the use of a lower intensity (Williams et al. 1985). However, the reciprocity appears to hold for Class II damage but not for Class I damage. Noell and co-workers (1966) demonstrated the cumulative effect of light in retinal damage. They showed that a 5-minute exposure does not produce a significant effect, whereas three and four exposures, each of 5 minute duration and each followed by a 1-hour dark interval, lead to significant damage. However, the cumulative effect does not take place if the retina recovers sufficiently from subliminal damage before the next exposure is applied. The cumulative nature of light damage has been observed in several subsequent investigations. Thus, Organisciak et al. (1989) have confirmed that intermittent light exposure can result in greater photoreceptor damage than continuous exposure, and that it exacerbates Class I photochemical damage in rats.

Susceptibility to light damage increases with age in a process that is distinct from age-related degenerative changes. O'Steen et al. (1974) exposed rats of different age to the same duration of light and compared the histology of the retina. Animals aged 6-7 weeks showed minor structural changes localized in the central retina; those aged 8 weeks had some photoreceptor destruction in 80% of the circumference of the retina; those aged 9-10 weeks showed discontinuities in the outer retinal layer, and these became progressively more severe in animals aged 11-14 weeks. Approximately 95% of the photoreceptors were damaged in adult retinas (16-24 weeks).

Importantly, all animal experimental studies analyzing retinal light toxicity have been performed using artificial light and not sunlight exposure. However, due to the fact that the retina of animal models, mostly rodents, differ from the human retina and to the fact that monkey studies are performed on anesthetized animals directly exposed to light with dilated pupils, extrapolation of doses to human exposure is not possible.

In summary, studies indicating a blue light hazard within the intensity range of natural light to the retina are based on animal experiments. They have shown that Class II damage is strictly mediated by blue light illumination, while Class I damage can be mediated by different photopigment wavelengths, although only blue cone damage was seen to be permanent. Therefore, for both classes of damages, blue light seems to be more dangerous than other components of white light. The relevance of these

experimental data for human pathological conditions is not totally clear, although these studies are suggestive of retinal damage due to blue light also in humans.

Epidemiologic studies have provided conflicting results regarding the relationship between sun exposure and retinal pathologies, mostly due to the fact that dosimetry is difficult to evaluate during long-term exposure and is highly dependent on geometric factors. High quality epidemiologic studies are needed to evaluate the real impact of light on retinal diseases (age related macular changes, age related macular degeneration, and also other retinal and macular pathologies).

ii) Artificial light and retinal damage

In humans, the only direct evidence for acute light toxicity due to artificial light exposure has been observed after acute accidental exposures to ophthalmologic instruments and to sunlight. No epidemiologic studies have evaluated the potential hazards of artificial light exposure for the eye.

Ophthalmologic instruments

Exposure to ophthalmologic instruments has caused accidental overexposures and subsequent retinal lesions, which has led to threshold exposure limits and guidelines.

The risks of retinal damage to patients in the operating room were recognized about 20 years ago. Operating microscopes can induce paramacular lesions, very similar to those induced experimentally by intense blue light exposure in animals. Moreover, filtration of blue light has been seen to significantly reduce the risks, although not entirely eliminating them. Increased duration of illumination of the retina through dilated pupils increases the risk of retinal damage. In 1983, on a series of 133 patients, it was shown that at 6 months post surgery, visual acuity was significantly higher in patients operated on with a fiberoptic light attenuated in the blue range as compared to a high-intensity tungsten filament microscope (Berler and Peyser 1983). Since then, several reports have identified blue light output as the major risk for the retina when compared to red and UV wavelengths (Cowan 1992).

Welder exposure

Arc welding exposes workers to UV and to blue light. Radiation in the UV range is absorbed mostly by the cornea and lens if welders are unprotected and gives rise to "arc-eye" or "welder's flash" (keratoconjunctivitis), well known as an occupational hazard for welders. Even if very painful, this condition is not expected to induce any permanent ocular damage. On the other hand, visible light, particularly in the blue range may expose welders to retinal photochemical damage. Okuno and co-workers (2002) evaluated the blue-light hazard for various light sources and found that arc welding was among the highest effective hazardous sources. Blue-light hazard effective irradiance has a mean value of 18.4 W/m^2 (300-700 nm) at 100 cm with a t_{max} (allowed exposure time) of 5.45 s. Exposure times of 0.6-40 s are typical, which may be very hazardous to the retina (Okuno et al. 2002). Several case reports have been published stressing that welding should be performed in good background lighting and with permanent adequate protection since pupillary constriction in response to striking the arc is too slow to block the initial surge of radiation.

iii) Chronic exposure to sunlight and Age-related Macular Degeneration (AMD)

Oxidative stress and sub-clinical local inflammation have been suggested to be associated with aging processes in the retina (Chen et al. 2010), and benzo(a)pyrene

toxicity through smoking has been shown to contribute to the development of AMD (Fujihara et al. 2008, Sharma et al. 2008, Wang et al. 2009a). The involvement of photochemical damage of the retina in AMD progression is also suggested by the observed protective effects of macular pigments and vitamins (Desmettre et al. 2004). However, due to lack of support from epidemiological studies, there is no consensus regarding sunlight exposure, which also generates oxidative stress and AMD (see Mainster and Turner 2010). One of the exceptions is the Beaver Dam Eye Study, where a correlation between sunlight and 5-year incidence of early AMD was observed. The study showed that leisure time spent outdoors while persons were teenagers (aged 13-19 years) and in their 30s (aged 30-39 years) was significantly associated with the risk of early AMD. People with red or blond hair were slightly more likely to develop early AMD than people with darker hair (Cruickshanks et al. 2001). A population-based cohort study with a 10-year follow-up confirmed the finding that, controlled for age and sex, exposure to the summer sun for more than 5 hours a day during the teens, the 30's, and at the baseline examination led to a higher risk of developing increased retinal pigment damage and early AMD signs as compared to exposure for less than 2 hours during the same period (Tomany et al. 2004).

The Beaver Dam Eye Study and the Blue Mountains Study, respectively, provided data on a total of 11,393 eyes from 6,019 subjects undergoing cataract surgery (Cruickshanks et al. 2001, Tomany et al. 2003, Tomany et al. 2004). Of these patients, 7% developed AMD in the 5 years following cataract surgery as compared to 0.7% in the phakic population (with the natural crystalline lens present). The cataracted lens is a strong blue light filter. However, more recent studies such as the large prospective AREDS study in 2009 (Chew et al. 2009) did not confirm this finding.

To better control for light environmental conditions, the effect of sun exposure on AMD was evaluated on 838 watermen on the Chesapeake Bay (Taylor et al. 1992). In this specific population, it was possible to estimate the relative exposure to blue light and UV. Compared with age-matched controls, patients with advanced age-related macular degeneration (geographic atrophy or disciform scarring) had significantly higher exposure (estimated to 48% higher) to blue or visible light over the preceding 20 years, but were not different with respect to exposure to UVA or UVB. This suggests that blue light exposure could indeed be related to the development of AMD, particularly in the more advanced ages. However, these associations were not found in other studies such as the French POLA study (Delcourt et al. 2001).

In the light of newly discovered genetic susceptibility factors for AMD, associations between sunlight exposure and genetic markers are relevant to study. Since polymorphisms in genes encoding proteins involved in the control of inflammation in the choroid/retina are strongly associated with the risk of developing AMD, the effect of light on these populations should allow better analysis of the risk of sunlight on AMD.

iv) Blue light and glaucoma or other optic neuropathy

Osborne et al. (2008) showed that mitochondrial enzymes such as cytochromes and flavin oxidases absorb light and generate ROS. Because retinal ganglion cells are unprotected from visible light, they are directly exposed to such photo-oxidative stimuli. In vitro, ganglion cells have been seen to undergo a caspase-independent form of apoptotic death due to light exposure. Studies of the effects of broad-band light exposure (400-700 nm) in rats have shown that only blue light exposure induced signs of ganglion cell suffering (Osborne et al. 2008).

Moreover, because melanopsin-containing ganglion cells participate in the light-induced pupil response, patients with ganglion cell dysfunctions owing to anterior ischemic optic neuropathy, demonstrated global loss of pupil responses to red and blue light in the affected eye, suggesting that in those patients retinal illumination could be enhanced, increasing the blue light hazard (Kardon et al. 2009). However, to-date no epidemiologic

study has evaluated the correlation between sunlight, or blue light, exposure and the progression or occurrence of glaucoma or other optic neuropathy.

v) Conclusions on light and retinal pathology

There is strong evidence from animal and in vitro experiments that blue light induces photochemical retinal damage upon acute exposure, and some evidence that cumulative blue light exposure below the levels causing acute effects can induce photochemical retinal damage.

In humans, there is direct evidence of acute light-induced damages to the retina from accidental high-intensity artificial or sunlight exposure. Regarding long-term exposure at sub-acute levels, there is no consistent evidence for a link between exposure from sunlight (specifically blue light) and photochemical damage to the retina, particularly to the retinal pigment epithelium.

Taking into account that AMD primarily affects the choroids and the retinal pigment epithelial cells, future epidemiologic studies should focus on the impact of light on retinal diseases (age-related macular changes, age-related macular degeneration, and also other retinal and macular pathologies) in particular after long-term light exposure at lower intensities. On the basis of the action spectrum of blue light in animal studies, blue light exposure may be considered a risk factor for long-term effects which should be investigated further in dedicated case-control and cohort studies.

There is no consistent evidence that sunlight exposure early in life may contribute to retinal damage which can lead to AMD later in life. Available epidemiologic studies are also not consistent regarding aggravation of AMD. Whether light exposure from artificial light could induce similar lesions remains to be demonstrated.

There is no clinical or epidemiological evidence that blue light causes neuropathy.

C. Conclusions on effects on the healthy eye

Under specific circumstances, exposure to sunlight or artificial light can cause acute as well as chronic effects and damage to various structures of the eye.

Acute UV exposure, particularly UVB and UVC below 300 nm, may cause photokeratitis of the cornea and the conjunctiva. In experimental studies, it is also shown that acute photochemical damage to the retina can occur due to blue light exposure. Acute damage to the human retina can occur due to accidental high-intensity artificial light or sunlight exposure.

Chronic UV exposure from sunlight may cause damage to the cornea (climatic droplet keratopathy) and the lens (cataracts). There is no consistent evidence that long-term exposure to sunlight (especially blue light) may be involved in retinal lesions that can develop into AMD.

There is no evidence that artificial light from lamps belonging to RG0 or RG1 would cause any acute damage to the human eye. It is unlikely that chronic exposures to artificial light during normal lighting conditions could induce damage to the cornea, conjunctiva or lens. Studies dedicated to investigating whether retinal lesions can be induced by artificial light during normal lighting conditions are not available.

3.5.3. Circadian rhythms, circadian rhythm disruptions, sleep and mood

3.5.3.1. Circadian rhythms

From an evolutionary perspective, exposure to artificial light is very new (Stevens 1987, Stevens and Rea 2001). Thus, life on earth has for billions of years been organized around the 24-hour day with a normal period of approximately 12 hours of light and 12 hours of dark at the equator, which varies with latitude and seasonal changes throughout the year (Stevens et al. 2007). Hence, almost all organisms on earth show 24-hour circadian and biological rhythms in adaptation of their biochemical systems to the rotation of the earth around its axis. This fundamental component of our biology, with the main function of coordinating biological rhythms, is controlled by endogenous biological clocks, and this periodicity has a profound impact on biochemical, physiological, and behavioural processes in almost all living organisms (Reddy and O'Neill 2010, Reppert and Weaver 2002).

In mammals, these rhythms are primarily generated by the master circadian pacemaker located in the suprachiasmatic nucleus (SCN) of the hypothalamus in the brain. The SCN clock can function autonomously, without any external input, with a period close to 24 h in all species studied (Dunlap et al. 2004). The clock is, however, not independent from the environment, as it is synchronized to the 24 h day through daily resetting by environmental cues ("Zeitgebers" = time givers), in particular light in mammals. Thus, the SCN receives input from both internal and external stimuli and its period may be entrained by these time cues. Information on light, by far the most potent synchronizer, reaches the SCN exclusively via the retinohypothalamic tract in the eyes in mammals, including humans. The visual rod and cone photoreceptor systems, necessary for normal vision, seem only to have a minimal role in circadian photosensitivity (Brainard et al. 2001a, Brainard et al. 2001b, Thapan et al. 2001). Circadian photoreception is primarily mediated by intrinsically photosensitive melanopsin (a vitamin-A photopigment) containing retinal ganglion cells (ipRGCs) distributed in a network across the inner retina (Berson et al. 2002, Brainard et al. 2001a, Brainard et al. 2008, Hattar et al. 2002, Hankins et al. 2007, Guler et al. 2008). In the absence of these two systems (classical photoreceptors and ipRGCs), the circadian timing system is free-running, expressing its own endogenous rhythmicity (Hattar et al. 2003).

Melanopsin contained in ipRGCs is a rhabdomeric photopigment, and possesses response properties of the invertebrate opsins. A unique property of rhabdomeric photopigments is the dual function as sensory photopigments and photoisomerases (Koyanagi et al. 2005). The photopigment chromophore is regenerated by light (conversion of *all-trans* back to *11-cis* retinal). Due to this property, melanopsin is resistant to "light-bleaching", and retains its ability to respond to light at high levels of irradiance and for long duration exposures. ipRGCs project mainly to the SCN, but also to other structures involved in non-visual responses, including, but not limited to, the pretectum (the pupillary reflex), the VLPO (sleep), the amygdala and the hippocampus (mood, memory). These photopigments require high irradiances, display a high degree of inertia in their responses, and show a peak of sensitivity between 460 and 484 nm in all vertebrates studied so far, including humans.

Melatonin, N-acetyl-5-methoxytryptamine, is a ubiquitous hormone in all groups of organisms. In vertebrates, including humans, it is primarily synthesized in the pineal gland and immediately secreted into the blood. Its 24-h rhythm is directly driven by the circadian clock through a polysynaptic sympathetic output pathway from the SCN to the pineal gland. Thereby, in normally entrained individuals, pineal melatonin is synthesized during the night (normal peak 1-3 a.m.), whereas during the day, production is virtually null. The primary role of melatonin is considered to provide an internal biological signal ("the third eye") for the length of night (Wehr 1991), and a signal for dawn to dusk (Arendt 2006, Arendt and Rajaratnam 2008, Brzezinski 1997). In addition, melatonin has been shown in studies in vitro to have antioxidant properties, including scavenging of

free radicals, direct antiproliferative effects, enhancing the immune response, and possibly an epigenetic regulator, which may influence certain metabolic diseases (Brzezinski 1997, Korkmaz et al. 2009, Reiter et al. 2010). In a recent study, it was shown that exposure to room light (<200 lx) in the evening before bedtime had a profound effect not only by a suppressed melatonin level, but the exposure also shortened the duration of melatonin production by about 90 minutes, and thus induced a shortened internal biologic night (Gooley 2011).

Melatonin has been suggested to function as a protective agent against "wear and tear" in several tissues. It has been shown that in normal retinas, melatonin exerted protection against free radical damage. Moreover MT1-type melatonin receptors were found in photoreceptor cells and MT1 knock-out mice demonstrated a loss of photoreceptors at 12 and 18 months of age, suggesting that lack of melatonin may be involved in retinal degeneration (Baba et al. 2009). Also in the brain, melatonin is suggested to have protective functions, including protection against oxidative damage (Kwon et al. 2010) and also by inhibiting the intrinsic apoptotic pathway (as reviewed by Wang 2009b).

In addition to acutely inhibiting melatonin synthesis at night via the SCN sympathetic output pathway, light resets the phase of the circadian timing system (advances and delays the 24-h rhythms of temperature, melatonin, cortisol etc.). The response of the circadian system to light, generally quantified by the degree of melatonin phase shift and suppression, is dependent on the timing of light exposure, duration, intensity and spectral composition (Gronfier et al. 2004, Gronfier et al. 2007, Lockley et al. 2003, Rimmer et al. 2000, Thapan et al. 2001). Short wavelength blue light (460-480 nm) has been shown to exert a stronger effect on light-induced melatonin suppression at equal photon density than green light (555 nm; Figueiro and Rea 2010, Lockley et al. 2003). West et al. (2011) has recently shown that narrow-bandwidth blue light (469 nm, 20 $\mu W/cm^2$) is significantly more efficient in night-time melatonin suppression in humans than polychromatic white light (4,000 K) as well.

However, both light intensity and other spectral components seem to influence nocturnal melatonin suppression in studies on human volunteers (see for example, Duffy and Czeisler 2009, Gooley et al. 2010, Revell and Skene 2007) suggesting that although melanopsin is the primary circadian photopigment, it is not alone in regulating melatonin production levels and the circadian phase. The important role of melanopsin is nevertheless suggested in many studies since a greater effect of monochromatic blue (460 nm) light compared to green (560 nm) light has been frequently documented. This includes phase shifting of the melatonin rhythm (Lockley et al. 2003), enhancing alertness, temperature, and heart rate (Cajochen et al. 2005), activating PER2 gene expression (Cajochen et al. 2006), phase shifting PER3 gene expression (Ackermann et al. 2009), enhancing psychomotor performances, and activating waking EEG (Lockley et al. 2006). Blue light also affects sleep structure (Münch et al. 2006), and activates brain structures, including the hippocampus and the amygdala that are involved in cognition, memory and mood (Vandewalle et al. 2007a, Vandewalle et al. 2007b, Vandewalle et al. 2009, Vandewalle et al. 2010).

Recently, ten core circadian clock genes (*CLOCK, CSNK1E, CRY1, CRY2, PER1, PER2, PER3, NPAS2, BMAL1, TIMELESS*) have been discovered (Cermakian and Boivin 2009, Fu and Lee 2003) with direct control of at least 10% of the genome (Bellet and Sassone-Corsi 2010, Storch et al. 2002). Their main function is to be responsible for generating the rhythmic oscillations on a cellular level. They seem also to play critical roles in many disease-related biological pathways including cell cycle, DNA repair and apoptosis (Fu and Lee 2003). The SCN orchestrates temporal alignment of physiology by transmitting daily signals to multiple mainly self-sustained clocks in peripheral tissues (Panda et al. 2002). Ill-timed light exposure (late evening, night or early morning), e.g. during fast transmeridian travelling or night shift work, the central oscillator in the SCN, however, tends to shift more rapidly than the peripheral oscillators, resulting in transient uncoupling of the peripheral oscillators from the central oscillator leading to internal de-

synchronisation among circadian periodic physiologic variables within the body (Haus and Smolensky 2006, Wood et al. 2009).

3.5.3.2. Circadian rhythm disruptions

Appropriate exposure to electrical light during periods of the day with local environmental darkness has, over the last 100 years, become a milestone of modern life. "Ill-timed light exposure" (i.e. late evening, night, early morning), in addition to light exposure during the day, may, however, result in attenuation of melatonin production and disruption of normal circadian rhythms (Czeisler et al. 1990), dependent on duration, wavelength and intensity of light exposure (Stevens et al. 2011). Circadian disruption is mainly characterized by desynchronization between internal (circadian rhythms) and external time (environmental clock time), including desynchrony of the master pacemaker (SCN) with the sleep cycle and with the peripheral oscillators in tissues throughout the body (Dibner et al. 2010). A desynchronization of the SCN with peripheral oscillators will persist for a variable period of time depending on the exposure pattern and the characteristics of the individual, e.g. age and chronotype (i.e. morning or evening preferences) (Davidson et al. 2009). Light exposure induces phase advances, and phase delays at different points in the circadian cycle, i.e. depending on the time during which light exposure occurs (on average in humans, light between about 5 a.m.-5 p.m. advances, and light between about 5 p.m.-5 a.m. delays the clock(Khalsa et al. 2003)). Thus, consecutive ill-timed light exposures may induce inappropriate phase shift of the circadian system, not allowing for its complete synchronization to the actual light conditions, and leading to circadian disruption.

Recent studies indicate that ill-timed exposures to even low levels of light in house-hold settings may be sufficient for circadian disruptions in humans. A comparison between the effects of living room light (less than 200 lx) and dim light (<3 lx) before bedtime showed that exposure to room light suppressed melatonin levels and shortened the duration of melatonin production in healthy volunteers (18-30 years) (Gooley et al. 2011). Cajochen et al. (2011) compared the effects of a white LED-backlit screen with more than twice the level of blue light (462 nm) emission to a non-LED screen on male volunteers. Exposure to the LED-screen significantly lowered evening melatonin levels and suppressed sleepiness. In another study from the same group (Chellappa et al. 2011) 16 healthy male volunteers were exposed to cold white CFLs (40 lx at 6,500 K) and incandescent lamps (40 lx at 3,000 K) for two hours in the evening. The melatonin suppression was significantly greater after exposure to the 6,500 K light, suggesting that our circadian system is especially sensitive to blue light even at low light levels (40 lx). However, no study has investigated whether the impact of warm white CFLs and LEDs (2,700-3,000 K) on melatonin suppression is in any way different from that of incandescent lamps.

Disruptions of fundamental circadian rhythms including communication between different cell types (Cermakian and Boivin 2009) may have the potential to significantly affect human health. Circadian disruptions, including decrease of melatonin levels, have been suggested to play an important role in development of chronic diseases and conditions such as cancer (breast, prostate, endometrial, ovary, colo-rectal, skin and melanomas, non-Hodgkin's lymphomas), cardiovascular diseases, reproduction, endometriosis, gastrointestinal and digestive problems, diabetes, obesity, depression, sleep deprivation, and cognitive impairment (Bass and Takahashi 2010, Boyce and Barriball 2010, Frost et al. 2009, Haus and Smolensky 2006, IARC 2010, Kvaskoff and Weinstein 2010, Mahoney 2010, Poole et al. 2011, Rana and Mahmood 2010, Stevens et al. 2007). It is, however, difficult to study directly the effects of ill-timed light exposures and long term health consequences, especially because virtually all humans are, to a various degree, exposed to artificial ligtht in the period between dusk and down. Therefore, epidemiologic studies can mainly provide indirect support for the theory. Thus, regarding breast cancer, it has in four out of five prospective cohort studies been observed that women with the lowest

concentration of the main melatonin metabolite sulfatoxymelatonin, have the highest risk (Travis et al. 2004, Schernhammer & Hankinson 2005, Schernhammer et al. 2008, Schernhammer et al. 2009, Schernhammer et al. 2010). Further, some relatively consistent epidemiological support has been found from other very different aspects of light exposure and potential circadian disruption: 1) increased risk in night-shift workers, and in 2) flight attendants potentially suffereing from both jet-lag and night shiftwork; 3) decreased risk in blind women, 4) and by long sleep duration; 5) increased risk by ambient light during the night in bedroom, and 6) high community light level, e.g. in cities; and decreased risk 7) for persons living in the arctic with long winters without or with only little light (Stevens 2009). Since light exposures are only measured indirectly in existing epidemiologic studies, other factors than light may, however, be involved at least partly in the observed breast cancer risk (Fritschi et al. 2011; Kantermann & Roenneberg, 2009).

It has also been suggested that melatonin deficits, e.g. caused by exposure to light at night, could be part of the etiology of osteoporosis. However, in vitro and experimental in vivo studies are inconsistent in their outcomes (Sánchez-Barceló et al. 2010). One single prospective study on nurses has investigated the association between hip and wrist fractures and duration of rotating night shift-work. Overall, nurses with at least 20 years of night shift-work, followed up from 1988 to 2000 for hip and wrist fractures, had an adjusted relative risk of 1.10 (0.87-1.42) compared to nurses who had never had shift-work; no dose-response relationship appeared by duration of exposure (Feskanich et al. 2009). In sub-analyses, including 8 years of follow-up and 20 or more years of night shift-work, a significantly increased relative risk (2.36; 1.33-4.20) was observed in nurses who had never used hormone replacement therapy and who had a body mass index <24. Overall, there is inadequate, or no evidence, for an association between exposure to light and risk of osteoporosis.

So far, the most comprehensive evidence of an association between circadian disruption and disease is found for breast cancer in night-shift workers. Night-shift work which may occur for several years, affects about 10-20% of the EU-workforce, is the most extreme source of ill-timed exposure to light and thereby simultaneous reduction of melatonin production, sleep deprivation and circadian disruption (Costa et al. 2010). An expert group convened by IARC in October 2007 concluded that "shift-work that involves circadian disruption is probably carcinogenic to humans, Group 2A", based on sufficient evidence in experimental animals for the carcinogenicity of light during the daily dark period (biological night), and limited evidence in humans for the carcinogenicity of shift work that involves night work and strong bio-mechanistic support (IARC 2010, Straif et al. 2007). In a recent meta-analysis based on eight published studies of shift-work and female breast cancer risk, a significantly increased risk of 40% (95% confidence interval: 1.2-1.7) was found (Viswanathan and Schernhammer 2009). The majority of included shift-work studies have been adjusted for potential confounders, including two large independent prospective cohort studies of high quality (Schernhammer et al. 2001, Schernhammer et al. 2006). After the IARC evaluation, three studies of shift-work and breast cancer have provided further support for the light-at-night hypothesis (Pesch et al. 2010;Lie et al. 2011;Hansen and Stevens 2011), whereas one new study did not provide further support (Pronk et al., 2010).

Furthermore, three independent studies of breast cancer risk after exposure to non-occupational light-at-night in the home have recently been published (Davis et al. 2001, Kloog et al. 2011, O'Leary et al. 2006), and significant associations were found for women who did not sleep during the period of the night where melatonin levels are normally peaking (Davis et al. 2001), or who frequently turned on the light during the night (OR=1.65; 1.02-2.69; O'Leary et al. 2006). An increased breast cancer risk was also correlated with increasing bedroom light levels (Kloog et al. 2010). All results are adjusted for potential confounders, but these three studies are based on self reports of light exposure and therefore prone to recall bias, which may limit interpretations. Due to the frequent exposure to light at inappropriate times (ill-timed exposure) there is an urgent need for further multidisciplinary research on occupational and environmental

exposure to light-at-night and risk of certain diseases (Blask 2009, IARC 2010, Stevens et al. 2007).

Conclusions

There is a moderate overall weight of evidence that ill-timed exposure to light (light-at-night indirectly measured by night shift work), possibly through melatonin suppression and circadian disruption, may increase the risk of breast cancer. There is furthermore moderate overall weight of evidence that exposure to light-at-night, possibly through circadian disruption, is associated with sleep disorders, gastrointestinal and cardiovascular disorders, and with affective disorders. The overall evidence for other diseases is weak due to the lack of epidemiological studies.

3.5.3.3. Sleep

Circadian rhythms, including melatonin rhythms, are involved in different aspects of facilitation of sleep (Cajochen et al. 2005, Dijk et al. 2001). A number of comprehensive reviews deal with the effects of acute light exposure on sleep (see e.g. Antle et al. 2009, Bjorvatn and Pallesen 2009, Czeisler and Gooley 2007). The effect of blue light on sleep is the subject of some recent work. Mottram et al. (2011) compared effects between exposures to 17,000 K (blue-enriched white light) and 5,000 K (white light) for 4-5 weeks on personnel at a research station in Antarctica. The blue-enriched higher colour temperature lamps significantly influenced sleep onset (earlier) and reduced sleep latency. This result, suggesting that blue-enriched white light synchronized the circadian timing system, is in accordance with some other studies, showing that blue-enriched light is more efficient in melatonin suppression than other wavelengths (Figueiro and Rea 2010, Gooley et al. 2011) and induces a circadian phase delay persisting into sleep (Münch et al. 2006). This latter study furthermore shows that monochromatic light exposure before bedtime increases slow wave activity (sleep depth) at the end of the subsequent night of sleep, with a greater effect of blue (460 nm) than green (555 nm), suggesting that light before bedtime can affect sleep.

Conclusions

The effects on sleep are sparsely investigated, making it difficult to draw any conclusions regarding effects of specific wavelengths, although one single study clearly shows that exposure to light artificially enriched in blue before bedtime affects subsequent sleep structure. However, such blue-enriched light does not emanate from common light sources limiting the relevance of the study for the general public.

3.5.3.4. Mood, alertness and cognitive functions

Seasonal affective disorder (SAD), "winter depression", is a mood syndrome or depression, particularly occurring in people living in areas with significant differences in exposure to natural light during summer and winter. Patients occasionally experience depressive symptoms in the winter with remissions in summer (Lurie et al. 2006). Disruption of circadian rhythms by insufficient light exposure seems to be involved (Monteleone et al. 2010). Several studies have shown that light therapy may be an efficient treatment for SAD (international committee recommendation; Monteleone et al. 2010, Westrin and Lam 2007, Wirz-Justice et al. 2005). Recent reports have shown that short wavelength blue light from LED sources (Anderson et al. 2009, Glickman et al.

2006, Howland 2009, Strong et al. 2009) has similar clinical effects to white light sources.

Since humans are day-living organisms, light is linked with a state of wakefulness or alertness. A number of studies have investigated specifically the effect of light on alertness (Dijk et al., 2009). Typically, such studies have been using subjective measures to assess alertness of subjects, but more and more, neurophysiological tools such as EEG, EOG, and also fMRI and PET have also been used.

Cajochen reviewed the evidence for alerting effects of light recently (Cajochen 2007) and pointed out that light exerts an alerting effect both during night and daytime conditions. The night-time effect is normally ascribed to suppression of melatonin levels, whereas the daytime effect is more difficult to explain. The intensity requirement has been investigated (Cajochen et al. 2000, Zeitzer et al. 2000), revealing that white light has an acute alerting effect at 50% of the maximal alertness (achieved with a 10,000 lx light exposure) already around 100 lx. The wavelength dependency of alertness effects has also been studied. Thus, several studies report that shorter wavelengths (460-470 nm) are significantly more efficient in generating alertness responses than longer (555 nm) wavelengths (Cajochen et al. 2005, Lockley et al. 2006, Revell et al. 2006, Vanderwalle et al. 2007a). A recent study by Figueiro et al. (2009) recorded alerting effects by both blue (470 nm) and red (630 nm) light. They investigated 14 volunteers with both neurophysiological and psychomotor tests, self reporting and measurements of salivary melatonin, in a within-subject study with two levels of intensity (10 and 40 lx at the cornea). Also the red light exposure exerted alerting effects at the higher level. However, only the blue light reduced the melatonin levels. The authors concluded that alertness may be mediated by the circadian system, but that this might not be the only light-sensitive pathway that can affect alertness at night. Viola et al. (2008) performed an occupational study where subjects spent the working day (4 weeks) either in a 17,000 K (blue-enriched white light) environment or in a white light environment (4,000 K). A number of subjective measures of alertness, mood, performance, fatigue, etc. improved in the blue-light condition as compared to the white light condition.

There are a few studies suggesting that short wavelength (blue) monochromatic light has an effect on cognitive functions via affecting circadian rhythms or directly through brain structures involved in memory, cognition and alertness (An et al. 2009, Vandewalle et al. 2006, Vandewalle et al. 2007b, Vandewalle et al. 2010). In a study where psychological effects of light were tested, cognitive effects elicited by blue light exposure were found to be different from the effects caused by exposure to longer (red) wavelengths (Mehta and Zhu 2009), but neither the light spectra nor the light intensities used were reported, making it difficult to compare with the aforementioned studies.

Conclusions

There is moderate evidence that monochromatic blue light or light artificially enriched in blue has an effect on cognitive functions, memory, and mood that is stronger than other lights. Whether these studies are relevant for evaluation of effects of common light sources is unclear, since monochromatic or blue-enriched light of this type is not produced by lamps for the general population.

3.5.3.5. Overall conclusions on circadian rhythms, circadian rhythm disruptions, sleep and mood

Light is typically installed for the beneficial purpose of illuminating space to allow for leisure, entertainment or work. Similarly shutters and windows are often used to prevent exposure to daylight and facilitate prolonged sleep, particularly with children. Importantly bright light enables better vision and affects mood which is desired in almost any

illuminated public or private environment. Notably also the colour temperature is typically adapted to the specific environment which is an important feature of light design and architecture. By doing so, an individual is exposed to light which affects the circadian rhythm with immediate and medium term psychological effects.

This behaviour builds on the cyclic behaviour of the spectrum and intensity of solar light, and has been increasingly used with the emergence of artificial light sources. Only recently have these effects on psychologic conditions and wake/sleep cycles been studied systematically. In general, levels of light intensity remain well below the peak intensity of the sun on a clear day, while in some applications (stage-art, film, TV recordings) it may be essential, and necessary, to surpass this "natural" reference value. In this context however it needs to be noted that these effects are not a feature of a lamp technology of concern, but of lighting and light design in general which suggests the need to provide appropriate information to citizens, as well as increasing alertness for the issue of light pollution. Light (at night) and elsewhere may be of beneficial or essential use for some while simultaneously negatively affecting others.

Despite the beneficial effects of light, there is mounting evidence that suggests that ill-timed exposure to light (light-at-night), possibly through circadian rhythm disruption, may be associated with an increased risk of breast cancer and also cause sleep disorders, gastrointestinal, and cardiovascular disorders, and possibly affective states. Importantly, these effects are directly or indirectly due to light itself, without any specific correlation to a given lighting technology.

Specifically under certain conditions blue light may be more effective in influencing human biological systems than other visible wavelengths. Thus, monochromatic blue light or light artificially enriched in blue is particularly effective in melatonin phase shift and suppression. However monochromatic or blue-enriched light of this type is not produced by lamps for the general population, so the relevance for the evaluation of effects of common light sources is unclear.

3.6. Adverse health effects in persons with pathological conditions

3.6.1. The Photosensitive skin diseases

In contrast to light effects on the skin of the normal population, there are two patient groups (Table 4) who react abnormally to sunlight. Those whose disease is induced by ultraviolet/visible/infrared; and others who have a pre-existing skin disease which can be photo-aggravated. Essentially, skin photo-testing is abnormal in the true photosensitivity disease group and normal in the photo-aggravated group. The wavelength dependency, where known, is described in Table 5. The majority of patients are aware of the relationship to sunlight exposure, but skin disease activity following incidental artificial light exposure is much less commonly described.

3.6.1.1. The photodermatoses

Photosensitive diseases induced by light are sub-divided into endogenous and exogenous groups (Table 4). They represent a wide range of diseases which, for space reasons, are succinctly described below.

Table 4 "Light related" skin diseases

Light Induced Diseases	**(The photodermatoses)**
Endogenous	- **idiopathic** (or immune based) Polymorphic light eruption Chronic actinic dermatitis Actinic prurigo Solar urticaria Hydroa vaccineforme Lupus erythematosus – (n.b. may also be photoaggravated) Porphyrias - **genophotodermatoses** Xeroderma pigmentosum Bloom's syndrome Cockayne's syndrome Rothmund Thomson syndrome Smith-Lemli-Opitz syndrome **- porphyrias**
Exogenous	- Drug induced photosensitivity - Phytophotodermatitis - Chemical induced

Photoaggravated Dermatoses

"Classical" photoaggravated dermatoses

- Lupus erythematosus
- Atopic dermatitis
- Psoriasis
- Jessner's lymphocytic infiltrate
- Dermatomyositis
- Lymphocytoma cutis
- Actinic lichen planus
- Erythema multiforme
- Acne vulgaris
- Pemphigus and chronic benign familial pemphigus
- Darier's disease, acantholytic dermatoses
- Disseminated superficial actinic porokeratosis
- Pellagra
- Viral exanthema, including herpes simplex

Other photoaggravated dermatoses

- Allergic contact dermatitis
- Seborrhoeic dermatitis
- Rosacea
- Melasma
- Mycosis fungoides
- Vitiligo
- Bullous pemphigoid
- Linear IgA disease
- Dermatitis herpetiformis
- Chronic ordinary urticaria
- Facial telangiectasia
- Pityriasis rubra
- Reticulate erythematous mucinosis
- Keratotic pilaris
- Actinic granuloma

Table 5 Wavelength dependency in photosensitive diseases

Condition	Wavelengths (nm)			
	Ultra-Violet Radiation		Visible Radiation	
	UVB (280-315)	UVA (315-400)	Visible (blue) (400–500)	Visible (red) (500-780)
Actinic prurigo	■	■		
Chronic actinic dermatitis	■	■	■	
Hydroa vacciniforme		■		
Lupus erythematosus	■			
Polymorphic light eruption	■	■	■	
Porphyria			■	■
Solar urticaria	■	■	■	
Xeroderma pigmentosum	■	■		

Notes: Shaded areas indicate the regions of the spectrum that have been shown to induce the disorder. UVC has not been included as very little data are available on UVC sensitivity. This is because the main purpose of phototesting is to investigate sensitivity to wavelengths that are present in sunlight.

A. The Endogenous photodermatoses

This group is sub-divided into the idiopathic and genophotodermatoses.

i) Idiopathic photodermatoses

Although the exact mechanism is unknown, this group of conditions is believed to be immunologically based. Little prevalence data exists. These diseases have been extensively reviewed (Ferguson and Dover 2006, Honigsmann and Hojyo-Tomoka 2007).

This group of light induced disorders have good clinical evidence for a UV induction role as seen in photoprovocation testing conducted with solar simulator, monochromator and broadband sources.

With the exception of Xeroderma pigmentosum and Lupus erythematosus, animal models do not yet exist for this group of diseases. This undoubtedly has held up understanding of individual diseases.

What is clear from the clinic is that there is a wide range of individual disease severity with differing amounts of UV being required to provoke lesions in patients. It is also evident in this particularly susceptible group that the main concern with the change from the use of incandescent to low energy light sources relates to the UV content of CFLs. Newer LED illumination lamps do not emit in the UV region and are therefore not such an issue for these UV sensitive patients. This explains the CFL emphasis of this section.

Polymorphic light eruption (PLE) or polymorphous light eruption (PMLE)

PLE which is the most common of all the photodermatoses, usually affects females and presents in spring/early summer (or when taking a sunshine holiday) with an itchy red spotty rash on sunlight exposed areas. It usually develops on exposure to between half to a few hours of sunlight, with symptoms often appearing several hours later. The condition settles in one to two weeks without scarring. Although the prevalence is often stated to increase with the distance from the equator (Pao et al. 1994), a recent multicentre European study reported a high overall prevalence of 18% (Rhodes et al. 2010a) without variation between mainly fair skinned populations at different latitudes.

The range of severity and wavelength dependency varies greatly between individuals. Many have a relatively minor disease triggered by UVA whereas a minority have a severe, disabling problem triggered by UVB/A extending into the visible wavebands (Bilsland et al. 1993, Frain-Bell 1985, Lindmaier and Neumann 1991). Although there are no written reports of PLE induced by artificial lighting other than sunbeds, an occasional PLE patient will comment on a possible role.

Low dose ambient sunlight exposure may be associated with improvement via a hardening process of skin thickening and pigmentation. For this reason some patients are free of activity on the face and hands.

Conclusions

Although in the majority of patients it is unlikely that artificial light sources will induce this skin disease (activity during winter months being rarely reported), there may be a small number of patients in whom UV and/or visible light emitting artificial sources could produce the eruption. It is also possible in others that low dose chronic UV radiation could contribute to a hardening process and thereby produce a degree of protection.

In the absence of clinical data, it is reasonable to assume that in a small minority of individuals, provocation of PLE may follow artificial light UV exposure.

Chronic actinic dermatitis (CAD)

This uncommon condition which may be incapacitating, particularly affects males over the age of 50 years (Hawk and Lim 2007). It has a prevalence which has only been studied in Scotland where 16.5:100,000 (Dawe 2009) were affected. The skin is sensitive to multiple contact allergens as well as UVA and UVB and in 50% of patients also visible light (Dawe and Ferguson 2003, Ferguson 1990).

Another type of uncommon chronic actinic dermatitis (CAD) (prevalence unknown) has been identified in atopic dermatitis sufferers (Russell et al. 1998). It appears that these young patients in their teens and early 20s have CAD with a breadth and severity which varies greatly between patients. A role for fluorescent lighting has been commented upon within the literature (Hawk and Lim 2007). The problem is perennial in about 50% (of the elderly male type), which suggests a role for artificial lighting.

An open study has revealed some patients to have the potential of CFL induced skin flares (Eadie et al. 2009). No similar work with unfiltered halogen sources is reported. It seems likely that they too would be capable of the same problem.

Conclusions

Severe and perhaps even moderately affected individuals with this condition may, when exposed to artificial UV or visible light, experience induction of CAD.

Actinic prurigo (AP)

This is an uncommon scarring condition that particularly affects American Indians and less frequently Caucasian and Asian populations (Honigsmann and Hojyo-Tomoka 2007). With an age of onset usually in the first decade it predominantly affects females. Patients complain of a perennial problem with deterioration during spring and summer. Pruritic, oedematous erythema with papules is evident following exposure to sunlight (Ross et al. 2008). Repetitive UVA provocation testing is capable of lesion induction. Management of AP is more difficult than that of PLE. Some cases benefit from a UV desensitisation course early in spring (Gambichler et al. 2005). Its prevalence is estimated at 3.3:100,000 of the Scottish population (Dawe 2009).

No formal provocation study with low energy CFL or other lamps has been conducted.

Conclusions

Severe cases may potentially be at risk from CFL or other UV emitting sources (Eadie et al. 2009).

Solar urticaria

This is an uncommon potentially serious skin disorder that affects males and females (Horio and Holzle 2007). It may arise in any age group but is particularly common in the first four decades of life. The condition is of long duration with about one third of patients failing to respond to anti-histamine and other treatments. It has a wavelength dependency most commonly in the UVA region extending into the visible and occasionally also affecting the UVB region. The life threatening risk is of generalised urticaria with anaphylactic shock. The prevalence in Scotland has been estimated to be 3.1:100,000 (Beattie et al. 2003). Provocation of the lesions is relatively straightforward in the most sensitive group. Light in the visible region (green) in some patients may inhibit eruption induction. Patients with severe UVA visible light sensitivity have reported indoor lighting triggered disease activity (Harber et al. 1985, Horio and Holzle 2007).

Conclusions

Severely affected patients may be at risk from CFL and unfiltered halogen sources producing UV/visible radiation. It should be noted that incandescent light sources also cause problems in some patients.

Hydroa vaccineforme

This condition is rare, arising in 1:300,000 of the Scottish population (Dawe 2009). It is a blistering eruption of sun exposed skin affecting both sexes which heals with characteristic scarring (Gupta et al. 2000, Honigsmann and Hojyo-Tomoka 2007). Occasionally, the eyes can be affected with photophobia and conjunctival inflammation and scarring. Some patients spontaneously resolve in childhood, others continue into adulthood. It appears that UVA wavelengths are particularly effective when used repetitively to induce the characteristic skin lesions (Eramo et al. 1986).

Conclusions

It is possible that some severely affected patients may be provoked by UVA emitting low energy artificial light sources.

Lupus erythematosus (LE)

Lupus erythematosus is an uncommon clinically significant group of closely related auto immune diseases that involve the skin. They affect all age groups in both sexes and are made up of four recognised sub types:

1. Systemic lupus erythematosus (SLE) is the most serious and potentially lethal form which affects both the skin and systemic organs.
2. Sub acute cutaneous lupus erythematosus (SCLE).
3. Chronic discoid lupus erythematosus (CDLE).
4. Lupus erythematosus tumidus (LET).

There is no doubt that UV exposure plays an important induction or aggravation role in all LE sub types. This field has been extensively reviewed (Hasan et al. 1997, Kuhn et al. 2006, Millard et al. 2000). Many LE patients may not be aware of their photosensitivity. This lack of correlation with experimental UV induction of skin lesions is thought to be due to the need for chronic UV exposure and a delayed time interval of up to a week between exposure and development of the skin lesions.

In SLE, there is evidence that UV exposure induced skin flares arise in 72-85% of patients (Dubois and Tuffanelli 1964) and can be accompanied by a flare of potentially serious internal organ disease e.g. kidney, lung and joint involvement (Léone et al. 1997, Wysenbeek et al. 1989). In the less serious sub-types, UV damage seems localised to the skin, producing disfigurement which is particularly important as the face and hands are affected with both acute erythema and a scarring potential. Incidence and sex distribution of LE varies with sub type. SLE, the most studied, has a prevalence in Europe (per 100,000) of 12.5 to 39. It is more common in the Afro-Caribbean population.

The mechanism of action of UV radiation relates to the believed pathogenesis of this autoimmune group of diseases. Current thinking is that LE arises in a group of individuals who have an antibody response directed against nuclear components of their own cell breakdown products. Cell death may be induced by a number of external factors including UV exposure and well described lupus inducing drugs (Wu et al. 2007).

Early skin provocation work suggested UVB wavelengths to be mainly responsible (Baer and Harber 1965, Cripps and Rankin 1973, Freeman et al. 1969). Further murine work

suggested the waveband extended into the UVA region (Bruze et al. 1985, Gilliam and Sontheimer 1982, Golan and Borel 1984, Wollina et al. 1988). Later attempts to reproduce the skin lesions with artificial UV revealed an abnormal response to both UVB and UVA with 93% of a large group of LE sub types having a positive photoprovocation test. Importantly, it was reported that in patients, multiple doses to relatively large areas of skin coupled with extended readings were required to maximise the positivity of this test (Sanders et al. 2003). In other published work (Hasan et al. 1997) a positive provocation test with artificial UV was achieved in 100% of SCLE patients, 70% of those with SLE and 64% with discoid lupus erythematosus (DLE). The skin of non LE controls did not react. Again, the wavelengths involved were UVB and UVA. In these studies it was noted that the dose for induction varied between patients and was considerably slower in evolution with longer persistence after induction than photo induced lesions of the other photodermatoses (Table 3).

In contrast to the shorter UV wavelengths (UVB/UVA) having a definite role in the induction of lupus, there is unexpected evidence that longer UVA wavelengths i.e. UVA1 (340-400 nm) may have a favourable effect on LE activity. This reasonably robust evidence has been reviewed by Pavel (2006).

Some clinical data exists commenting on the role of artificial lighting inducing skin LE. In one study, SLE flares due to fluorescent lighting were reported (Rihner and McGrath 1992). Thirteen of 30 sun sensitive SLE patients described an increase in skin disease activity following exposure to an unshielded fluorescent lighting source, while the same light source with UV filtering had no effect in the same patients.

Consequently it was recommended that patients with SLE and other photosensitive conditions should avoid unfiltered fluorescent lamps.

Conclusions

It seems reasonable to assume that at least some LE patients, and particularly those with SLE, are at risk from chronic UV exposure from some low energy emitting lamps such as CFLs and unfiltered halogen bulbs. In this context it is noted that LE support groups are already advising the use of double rather than single envelope CFLs.

Porphyrias

This group of mixed inherited and environmentally induced photosensitivity skin diseases (Elder 1998, Murphy and Anderson 2007) relate to an accumulation of a photosensitive porphyrin within the skin. A disease example is erythropoietic protoporphyria, the main feature of which is burning or prickling pain in the sunlight exposed skin. A few minutes of intense visible light are usually enough to elicit symptoms causing the individual to try to escape from the light source and seek relief, for example, using cold water compresses. Erythropoietic protoporphyria develops in childhood, or even during infancy. It should be noted that cutaneous porphyrias are particularly sensitive to the blue light region so there would be an argument that fluorescent lighting would be a greater problem when compared with tungsten bulbs (which have less blue light). Porphyrias are rare disorders. For example, the prevalence of congenital erythropoietic porphyria (Günther's disease) in the UK is approximately 2 per 3,000,000 live births. Erythropoietic protoporphyria prevalence is 1 to 2 per 100,000 inhabitants (Burns et al. 2004, Marco et al. 2007). Although an unusual event, theatre and other visible light sources can produce phototoxic burns in those patients with particularly high levels of porphyrins (Meerman et al. 1994).

Conclusions

Artificial, visible light sources which would include incandescent bulbs may produce skin reactions in the most sensitive patients.

ii) Genophotodermatoses

The diverse group of inherited photosensitive skin diseases given in Table 3 include Xeroderma pigmentosum (XP), Cockayne's, Bloom's, trichothiodystrophy, Rothmund-Thomson Syndrome and the Smith-Lemli-Opitz syndrome, which are all rare (Berneburg and Kraemer 2007, Ferguson and Dover 2006).

Xeroderma pigmentosum, as an example of this group, is reported to have a prevalence of 1:250,000 in Europe and the USA (Robbins et al. 1974). XP in its classical excision repair form has marked photosensitivity to UVB/A wavelengths. The development of skin cancer in early childhood makes photoprotection against these mutagenic wavelengths an essential part of management. This disease is associated with a significantly shortened life span. The other diseases which are rarer than Xeroderma pigmentosum vary in their wavelength susceptibility and degree of abnormal photosensitivity.

Conclusions

UV radiation from artificial light sources is associated with an increased skin cancer risk in XP. Patients are currently advised to avoid all sources emitting UVB/A wavelengths. These would include CFLs and unfiltered halogen bulbs.

B. The Exogenous photodermatoses

i) Drug/chemical induced photosensitivity

Many drugs are known to be capable of inducing photosensitivity (Moore 2002, Selvaag 1997). However, many drugs listed as photosensitizers are infrequent causes of the problem and likely to have an idiosyncratic mechanism (Shields 2004). They do so by a variety of mechanisms, most commonly phototoxicity, which indicates that any individual exposed to a sufficient quantity of a drug and appropriate irradiation will be affected. Other mechanisms result in a small number of individuals being affected. Examples of photosensitizing drugs are listed below. Much less commonly seen is the mechanism of drug-induced photoallergy, which involves a sensitised immune system, which follows topical exposure to a photoallergic drug or chemical (usually sunscreens).

Generally, such reactions are UVA dependent with some drugs extending into the UVB and visible range (Ferguson 1998).

Amiodarone

Amiodarone is a cardiac anti-dysrhythmic agent that causes sunlight induced burning, and a prickling sensation with erythema in approximately 50% of individuals on a high dose. The wavelengths responsible are UVA and visible light. Unsightly slate-grey skin pigmentation may also develop on photoexposed sites (Ferguson et al. 1985).

Phenothiazines

Phenothiazine-derivative drugs have an antipsychotic action, thought to act by blocking dopaminergic transmission within the brain. They produce skin discomfort, erythema and blistering elicited by exposure to UVA. Unsightly skin discolouration may also follow.

Fluoroquinolone antibiotics

This is a large group of drugs that exhibit variable degrees of phototoxicity. Symptoms include erythema and blistering; wavelengths responsible are mainly in the UVA region (Ferguson 2003).

ii) Photofrin and other anti-cancer photodynamic therapy (PDT) agents

Photofrin and Foscan are potent intentional visible wavelength dependent photosensitizers used in photodynamic therapy of internal cancers. These drugs can elicit skin phototoxic responses when exposed to visible radiation from artificial light sources (Hettiaratchy et al. 2000, Moriwaki et al. 2001).

Conclusions

The majority of drugs known to have a phototoxic potential would not be expected to have the problem induced by CFL and unfiltered halogen light sources. However, skin flares whilst taking intentional photosensitizers, as during PDT, would be expected following artificial visible light exposure.

With photofrin, photosensitivity might be expected to occur with CFL and LED sources to a greater extent than that currently seen with incandescent lighting. This is due to a combination of greater sensitivity of porphyrins to blue light (soret band), coupled with an enhanced blue light emission of these sources. However, such patients are aware of their extreme photosensitivity which needs careful management.

iii) Photoallergic Contact Dermatitis

Photoallergic contact dermatitis is an uncommon delayed-type hypersensitivity reaction elicited by low doses of UVA radiation in susceptible individuals. The main groups of photocontact allergens current in the environment are organic sunscreen chemicals, and topical non-steroidal anti-inflammatory drugs. When the diagnosis is made, patients can quickly stop the responsible agent and avoid the provoking wavelengths, usually in the UVA region.

Conclusions

Except in the most photoallergic individuals, UVR artificial light sources are unlikely to be a significant factor in this group of patients.

3.6.1.2. Photoaggravated dermatoses

This diverse group of diseases (Werth and Honigsmann 2007) differs from the true photo dermatoses in that they also arise without UV/visible light exposure. Only a small proportion report that their problem is sunlight exacerbated. Phototesting reveals normal responses to UV and visible radiation.

Atopic dermatitis (AD) is an example of this large group. About 10% of people with atopic dermatitis are aware of light triggered exacerbations. The other 90% have a perennial problem with no evidence of sunlight induction and even may have a tendency to flare in the wintertime. The mechanism and wavelength dependency are unknown as these patients have a normal phototest skin response. In fact, many of these patients respond well to UVB phototherapy. In some patients a coincidental true photodermatoses

may be the explanation. In such cases, induction by light will vary with the wavelength dependency and degree of sensitivity of the true dermatoses.

Conclusions

As can be seen from Table 3, the number of diseases within the potential photoaggravated group is extensive. As the precise role of artificial light sources is unknown, it is perhaps unlikely that they play a significant role.

3.6.1.3. Conclusions on photosensitive skin diseases

There is strong evidence based on phototesting that UV and, in some patients, visible light, induces the skin lesions of the true photodermatoses.

Although sunlight is reported by some patients as the main source of disease activity, occasionally severely affected patients over the range of endogenous (and exogenous) diseases do exhibit or suspect a role for artificial lighting.

For this group of patients, artificial light sources with a considerable UV emission would be best avoided. Therefore, the previous SCENIHR opinion recommended that if using CFLs, a double envelope type is preferable. This is supported in the current opinion. Although a second envelope undoubtledly reduces the UV emissions, the currently available data show a high variability of UV and blue light emission due to different internal design parameters even for the same externally visible architecture, i.e. also in presence of a second envelope. While some compact fluorescent lamps are in the same category, retrofit LED lighting, which does not emit UVR on the physical grounds of the light generation therein, would provide potentially an even better option for such patients. The UV/blue light irradiation from halogen lamps is also highly dependent on the lamp type. Here attention needs to be given to the proper installation of those lamps which are sold by the manufacturer to be installed at larger distance or in conjunction with special luminaires or filters against e.g. UV or IR irradiation or to prevent other hazards like fires. While it is unlikely that there would be a significant UV risk from halogen lamps for the general public, provided that protective measures are complied with, the UV content of these lamps can rise to levels which are of concern for patients with light-associated skin disorders at close operating distances and long exposure times. This, however, is not a very common use pattern for this lamp type.

Unfortunately, due to limited study, there is a lack of controlled skin provocation data using the range of artificial lighting sources. Where some work has been conducted in particularly severely affected individuals, as in the photodermatoses, lupus erythematosus, chronic actinic dermatitis and solar urticaria, there is good evidence of induction of skin disease by single envelope fluorescent light sources.

Such work needs to be confirmed and extended using the range of energy saving lamp types over the different diseases with controlled study methods in greater numbers of patients. Until such data exist, it seems reasonable to assume that the UVR component of artificial lighting in an as yet undefined number of patients, may contribute to induction of their skin disease and in the case of lupus erythematosus possibly also their systemic disease.

3.6.2. Photosensitive eye conditions

Inherited retinal degeneration affects about 1.5 million individuals worldwide. The disorders may be inherited in any one of the recognised patterns, and fall within a spectrum ranging from Retinitis pigmentosa (RP) to macular dystrophies. In RP the initial

symptom is loss of night vision and subsequently loss of lateral vision. In late-stage disease, vision is restricted to a narrow central cone but detailed vision remains good. There is also an intermediate group which is characterized by progressive loss of side and central vision equally. In macular diseases, central vision is lost but side vision remains good. Several hundred disorders exist within this family of diseases that vary in their age of onset, speed of progression and final vision capacity. In severe cases of disease, there may be loss of all useful vision in early life, whilst others may be unaware of the presence of disease even in late life.

Light can accelerate degeneration through non-specific toxicity to photoreceptors already stressed by the effects of a mutation, or through a specific interaction with mutant rhodopsin. Experiments with cultured photoreceptors have suggested that activation of mislocalised rhodopsin could kill rods by stimulating inappropriate signaling pathways (Alfinito et al. 2002).

In two specific human forms of regional (macular) RP, i.e. Ogushi disease and Stargardt disease, light has been recognized as aggravating and light protection as protective. Indeed, mutations of proteins involved in rhodopsin deactivation after light exposure (i.e. rhodopsin kinase, arrestin), induce prolonged insensitivity of rod vision following light exposure and different forms of retinal degeneration ranging from stationary blindness to true RP (Ogushi disease) (Chen et al. 1999, Paskowitz et al. 2006).

In Stargardt disease, a hereditary macular dystrophy whose features often include progressive loss of central vision with onset during the first or second decade of life, macular atrophy and fundus flecks, massive accumulation of lipofuscin and extensive A2E accumulation is seen in RPE cells. The disease shows autosomal recessive inheritance and is caused by mutations in ABCA4, a transporter localised to the rims of photoreceptor outer segment discs. ABCA4 mutations have also been identified in fundus flavimaculatus, autosomal recessive RP and cone-rod dystrophy. A possible link with age-related macular degeneration has been proposed but remains poorly documented. Due to the very high content of A2E in RPE cells, blue light is considered to be an aggravating factor for Stargardt disease (Maeda et al. 2009, Mata et al. 2001).

Since in most patients presenting with RP symptoms, the causative mutation is not known, it may be prudent to avoid unnecessary exposure to bright light in patients presenting RP, particularly the regional RP (affecting mostly the macula).

Conclusions on photosensitive eye conditions

The effect of light is variable depending on the genetic alterations that are causing retinal degeneration. In specific conditions like Stargart disease, accumulation of lipofuscin early in life renders the RPE cells particularly sensitive to type II photochemical damage. In other retinal dystrophies, light does not exert any aggravating effect. However, since the causative mutation is seldom known to the patient or their family, and because there is no clear correlation between genotype and phenotype, it is recommended for all patients with retinal dystrophy to be protected from light by wearing special protective eyeware that filter the shorter and intermediate wavelengths.

3.6.3. Flicker, other conditions

Flicker, modulation of light intensity which can be perceived by the human visual system, has been implicated in certain pathologic conditions, most notably epilepsy. The criticial flicker frequency (CFF; where it is not possible to discern single events) is around 50 Hz for luminance, whereas it is considerably lower, ca. 25 Hz, for chromaticity (Shady et al. 2004; Carmel et al. 2006). Flicker of higher frequencies is invisible to humans since the visual system is averaging over 20 ms or more. Through stroboscopic effects, however, modulated light of higher frequency may be observable if the observer and a patterned

object are in relative motion and are illuminated by modulated light. Even if the flicker frequency is well above the CFF, non-perceived may have effects on visual performance (Veitch and McColl 2001; Shady et al. 2004)). The possible influence of flicker from CFLs on various conditions was discussed in the previous SCENIHR opinion (2008). It was also noted that modulation of light from CFLs at 100 Hz was measured and this may be perceived under certain conditions (Khazova and O'Hagan 2008).

Flicker perception generally depends on the time dependence of the light emitted by a light source and on the circumstances of observation (motion, attention, saccadic eyeball motion, field of view, geometry of the incident light etc.). Flicker has been been reduced considerably for fluorescent lighting with the implementation of 'flicker free' high frequency (kHz) ballasts operating at higher frequency (see also previous SCENIHR opinion). A residual modulation of the light intensity, however, with twice the power line frequency, can still occur if AC components of the supply voltage are insufficiently filtered by the power supply circuitry or by time averaging mechanisms inherent to the light generation. The frequency characteristics of the emitted light depends so strongly on specific engineering parameters, that general conclusions can not be drawn on one versus the other technology. There are lighting technologies available and installed (sodium lamps with ferromagnetic ballasts in street lighting) however, which emit considerably modulated light. This suggests that quality standards for lamps may be considered to set minimal requirements for light quality (intensity modulation, colour rendering, and other parameters in addition to energy efficiency).

Regarding other lighting technologies, it was recently reported (IEEE 2010) that LED-generated light, for example, may undergo periodic fluctuations, flicker, with large amplitude (see also ANSES 2010). This light flicker is mainly due to the electronic power supply (driver) of the LED. It is always periodic and its fundamental frequency is similar to the power frequency fluctuation (which is twice the current variation frequency). Usually, LED drivers convert mains to DC current that supplies the LED. Good quality drivers (applicable to any lighting technology) use Power Factor Correctors (PFC, filters) that limit residual current fluctuation after AC/DC conversion to less than 10% of the root mean square current value. This residual power ripple can induce light flickering at a frequency that is two times higher than the mains (50 or 60 Hz depending on the country, inducing flicker of 100 or 120 Hz). Low quality LED drivers with passive power factor correction stages can be subject to higher fluctuation rates especially at low dimming levels and produce perceivable flicker (lamp switch on/off periodically). Furthermore a large proportion of LED drivers on the market use Power Wave Modulation (PWM) architecture in order to dim the light output. PWM uses short pulses at high frequency (several kHz) with variable duty cycle. Under these conditions, light fluctuation is expected at high frequency (twice the pulse frequency). Generally it is not expected that the end-user will observe any flicker, except for low quality products in very deep dimming situations. However, ambient flicker at imperceptibly high frequencies can penetrate the neural site for flicker adaptation, which is presumed to be in the primary visual cortex (Movshon and Lennie 1979). Indeed, earlier physiological studies have demonstrated activity in the human visual cortex in response to imperceptibleflicker frequencies (Regan 1968, Van der Tweel 1964), but these studies suggested no impact on perception as a result of this cortical activity. The IEEE work mentioned above (IEEE 2010) states that high frequency flicker can induce risks including headaches and eye-strain. The sources of high frequency flicker associated with headache include lighting (formerly principally lighting from gas discharge lamps) and computer screens (formerly cathode ray tube displays, now LED back-lights and lamps).

Various non-skin conditions (Irlen-Meares syndrome, myalgic encephalomyelitis, fibromyalgia, dyspraxia, autism, HIV), which are not otherwise considered in this opinion, were discussed in the SCENIHR opinion from 2008 (SCENIHR 2008). No additional data regarding the effects of CFLs on these conditions have been published since then. There is no scientific evidence linking these conditions to other lighting technologies. There is a need for additional experimental and epidemiological studies before final conclusions can be drawn regarding several of these conditions.

Conclusions

The previous SCENIHR opinion on Light Sensitivity stated that modern CFLs are basically flicker-free due to their electronic ballasts. However, it was also noted that studies indicated that hardly noticeable residual flicker can occur during certain conditions in both CFLs and incandescent bulbs. No further information has become available regarding CFLs and incandescent bulbs since then. Also LEDs are normally flicker-free, although it has been mentioned that some low quality products can produce perceivable flicker. Possibly, flicker of higher frequencies can influence the human visual cortex, although data on this are old and difficult to evaluate. More research on possible flicker emissions and subsequent health effects from CFLs, incandescent bulbs and LEDs seems thus to be warranted.

There is no scientific evidence available to evaluate if conditions such as Irlen-Meares syndrome, myalgic encephalomyelitis, fibromyalgia, dyspraxia, autism, and HIV are influenced by the lighting technologies considered in this opinion.

3.7. Exposure and health risk scenarios

3.7.1. Exposure situations in various indoor lighting settings

A proper risk assessment would include knowledge of the hazards involved, as well as knowledge about actual exposure. There are at present very few data available regarding personal exposures, with a few exceptions regarding occupational exposures to UV, which are also included in the discussion of the scenario presented in section 3.7.2, where a detailed "worst case scenario" of SCC incidence as a function of UV exposure from fluorescent lamps is presented. Due to the lack of knowledge regarding exposure, it has not been possible to perform any proper risk assessment of various environments with different types of lighting sources. Furthermore, in many cases, we also do not have data regarding disease incidence on the European or even the national level. Taken together, it was considered unrealistic to present further risk assessment scenarios.

Based on available data, we here present a number of exposure situations, where the strength of some physical parameters is estimated according to their potential to trigger health impacts.

These exposure situations were chosen because at least one of the present parameters could pose a risk. For instance, most exposure situations in a household setting were ignored as they typically involve an illumination level of 50 lux, a level so low that exposure to potentially problematic radiation is considered negligible.

Two types of health situations are included in Table 6 below; the global skin and eye exposure to the ambient light, and the direct eye exposure to blue light coming from a light source in the line of sight. These situations were chosen since they are realistic situations that are included in the health hazards considered in Standard EN 62471.

1 **Table 6 Examples of exposure situations from artificial light for the general population**

Time/duration of exposure	Location	Type of lighting	Distance to light source	Number of light sources (single versus distributed light sources)	Illuminance level	Physical parameters potentially triggering health effects
8 h	Office	Linear fluorescent and CFLs (LEDs for task lights)	Ceiling fixtures: minimum 1.50 m Task light: minimum 20 cm	Distributed large surface light sources (Except for task lights)	500 lx (general lighting) [up to 1,000 lx for architects and designers working posts]	UVR: Unlikely (1) Blue light: Possible (2) Thermal: None
8 h (for workers) 1-2h for customers on average	Supermarkets/ general stores	Linear fluorescent and CFLs for general lighting LEDs and low power metal halide lamps (spots) for accent-uation lighting	Ceiling fixtures: minimum 2 m Accentuation lighting: variable distance	Distributed, large surface light sources for general lighting Spots and projectors for accentuation lighting	750 lx (general lighting) Accentuation lighting can use high brightness spots (>20,000 cd/m^2)	UVR: Unlikely (1) Blue light: Possible (2) Thermal: None
½-3 h for performers, presenters etc.	TV studios	Linear fluorescent and CFLs for general lighting LEDs and halide lamps projectors	Ceiling fixtures: minimum 2-3 m Projectors: 3-4 m but close to the line of sight	Distributed, large surface light sources for general lighting Projectors with white or/and coloured light	TV-studios: about 520 lx at 90 cm from floor High brightness projectors spotting using metal halide lamps the stage (>20,000 cd/m^2)	(retinal damage) UVR: Unlikely (1) Blue light: Possible (2) Thermal: None Glare from bright head lights may indirectly induce risks
½ to 1 ½ h	Night reading	CFLs, LEDs, incandescent	Minimum distance between 20 and 50 cm	Unique lamp with protection, directional lights (spots)	Variable, on average 100 lx on the book is an indicative value	UVR: Unlikely (1) Blue light: Possible (2), (3) Thermal: None
6-8 h	Kindergarden, schools	Linear fluorescent CFLs and LEDs for general lighting Spots (incandescent, LEDs)	Ceiling fixtures: minimum 2.5-3.0 m	Distributed, large surface light sources for general lighting Spots for specific area lighting	200-500 lx	UVR: Unlikely (1) Blue light: Possible (2), (4) Thermal: None

1-5 min	Night drivers	High beams from car in the opposite direction Discharge lamps, LEDs (in the future)	Distance varies from 100 m to less than 5 m (car crossing situation). Truck drivers are more exposed due to high position relative to the road surface	Projectors with very high brightness	N/A	UVR: Unlikely (1) Blue light: Unlikely (2), (4) Thermal: None Glare from bright head lights may induce accident

Possible health effects related to the exposure parameters:

(1): UV radiation that may escape from artificial light sources may affect photosensitive individuals.

(2): Blue radiation directly from bright cold white light sources in proximity of the workers eyes (e.g. task lights) or strong projectors (floodlights, accentuation and scenic lighting, etc.), or reflected may represent a risk for retinal damage.

(3): The blue light component from cold white reading lights may perturbate circadian rhythm of the user.

(4): A child's crystalline lens is more transparent to short wavelengths than that of an adult, making children more sensitive to blue light effects on the retina.

3.7.2. Worst case scenario of UV exposure of general population from indoor lighting in offices and schools

UV radiation from indoor lighting can potentially increase the risk and incidence of skin cancer. As reviewed in section 3.5.2.2, sunburns appear to contribute markedly to the risk of melanoma, and also to that of basal cell carcinoma. Although sunburns from ambient solar exposure are quite common, healthy individuals will not incur sunburns from exposures to indoor lighting, especially not with lamps in the exempt risk category (RG0). In combination with a UV-related risk from exposures limited to childhood years, melanoma risk is not likely to be notably affected by indoor lighting. This excludes any discernable impact on the mortality from skin cancer which is largely attributable to melanoma. Because the UV-related skin carcinomas are well treatable, any impact on skin carcinomas will mainly concern an increase in morbidity and add to the already increasing load on public health care from these skin cancers. Although accumulated doses from low level UV exposure may contribute to the risk of BCC, the data on risk and incidence of BCC need to be corrected for contributions from sunburn, e.g. by exclusion of BCCs occurring in intermittently exposed skin areas. Selection and analyses of data for a proper correction for sunburn is beyond the scope of this report, and we therefore refrain from including BCC, the most common skin cancer, in a risk assessment of the impact of UV radiation from indoor lighting. Here, we limit ourselves to a simplified straightforward risk analysis for the second most common skin cancer, squamous cell carcinomas (SCCs), in order to generate quantitative information on the potential worst case long-term impact of UV exposure from indoor lighting.

Importantly, this scenario is based on the assumption that the risk over the short life-span of an experimental animal (mouse) can be used to extrapolate to 80 years in a human. It furthermore involves extrapolation from the dose levels in experimental studies to real human exposures, assuming a linear dose-response without a threshold e.g. due to the DNA repair pathways. A constant risk coefficient is applied, assumed to represent the entire population, without variability between individuals

The scenario is based on a risk model with inherent assumptions and uncertainties. A non-linear Weibull model for cumulative hazard of skin cancer (representing multiplicative effects of cumulative UV dose and age) is used, derived from mouse and human studies. The parameter values used for humans are obtained from epidemiological studies: age dependence from age-specific incidence rates of skin cancer and dose-dependence from ecological data (variation in skin cancer incidence in relation to ambient UV levels). These are population-level data that do not take into account changes over time (e.g. behavior) or population dynamics (e.g. migration). An important issue is that inferences from aggregate level (populations at different locations) to the level of individuals can be affected by "cross level bias" which could lead to either an under- or overestimated trend in personal risk with dose. Furthermore, the model pertains to the average effect in a population with a wide distribution of exposures and susceptibilities, with an assumption that the amount of exposure is unrelated to

susceptibility. This simplifying assumption enables assessment of the impact of an incremental annual UV dose to the solar UV dose, where the latter varies widely among individuals.

The accumulated solar UV exposure is the main exogenous determinant of the risk of SCCs of the skin. Although UV exposure rates from indoor lighting will be far lower than those from the summer sun, the steady low level daily exposures may add notably to the annually accumulated UV dose, especially exposures in well lit offices, schools, and public places such as malls etc. Modern lamps emit UV radiation at widely different levels. Although these levels commonly fall well below the emission limit of 2 mW actinic UV per klm (defining the Risk Group 0, "exempt from risk"), preventing acute effects, extensive exposures to some of these lamps may contribute significantly to the annual UV dose. Such exposures may result from lamps in open fixtures or luminaires with reflector and louvers. Glass covers will in general lower effective (erythemal) UV exposures to negligible levels.
The risk model for SCC is basically simple, and is based on the average number of tumors that has occurred per individual at risk in a birth cohort of age "a" in absence of death. This number of tumors is referred to as the (tumor) yield, YLD(a), or cumulative hazard function. The yield can be written as a function of the accumulated effective UV exposure, total dose TD(a), and the age:

$$YLD(a) = (TD(a)/TD_0)^{p1}.(a/a_0)^{p2} \quad (1)$$

where TD_0, a_0, p1 and p2 are constants (de Gruijl and van der Leun 1991, de Gruijl and van der Leun 2002, Slaper et al. 1996), and on average p1 = 2.3 and p2 = 3.8 (NRPB 2002). As pointed out in the previous section 3.5.2.2, we can take the UV exposure spectrally weighted according to erythemal (sunburn) effectiveness (using the CIE erythemal action spectrum) as a proxy of the carcinogenic UV dose.

If the first appearing SCCs occur independently of each other (valid in mice; de Gruijl and van der Leun 1991), the chance for a person of age "a" to have contracted an SCC is:

$$P(a) = 1 - e^{-YLD(a)} \quad (2)$$

And if the risk is small (YLD(a)<<1),

$$P(a) = YLD(a) \quad (3)$$

For the age-specific incidence (the number of new cases per year per individual at risk of age "a") we write

$$I(a) = d\,YLD(a)/da \quad (4)$$

The overall incidence in a population (number of cases per year) over all age groups then becomes

$$I = \int I(a)\, n(a)\, da \quad (5)$$

where n(a) is the age distribution in a population, for which we take the European standard population (http://eu-cancer.iarc.fr/5-glossary.html.en; accessed 1 April 2011); $\int n(a)\, da = 1$.

For the following assessment we make the assumption that the contribution from indoor lighting to the risk has been negligible thus far. We then hypothetically add to existing solar UV exposures a regimen of maximum UV exposure from certain types of fluorescent lamps to assess a worst case impact. Considering the low level illumination and sparseness of direct exposure to fluorescent lamps in the homes, we restrict these additional UV exposures from fluorescent lighting to school days (6 h/day, 5 days/week and 40 weeks/year from 5 till 20 years of age) and working days as adults (8 h/day, 5 days/week and 48 weeks/year from 20 till 65 years of age). The fluorescent lamps can be either single-capped (such as single-ended tubes without integrated ballast) or double-capped (e.g. TL tubes). Based on this basic scenario, we can calculate what the

increase in risk and incidence will be as a result of a certain increase in annual UV doses; see Table 7 below. One could simplify the calculations of increases in SCC incidences by assuming that the annual dose for everyone is increased by the same percentage (4th column of Table 6; a good approximation for an increase in UV irradiance caused by stratospheric ozone depletion, Madronich and de Gruijl 1993, Slaper et al. 1996). However, this does not appear to be a valid assumption for added annual doses of UV radiation from indoor lighting on school and working days. A more plausible approximation is that everyone receives about the same additional annual UV dose on top, and irrespective of, whatever annual dose they receive from sunlight; the solar exposure is known to vary widely among individuals in a population. Here we assume that everybody persists in their sun exposure behaviour throughout life with a log normal distribution in annual personal solar UV doses close to the distribution observed in a Danish study (Thieden et al. 2004); with 95% in the range 3.3-fold over and under the median (for the calculations the distribution was made discrete with nine 10% intervals around the median and two 5% intervals at the extremes; each percentile group was assigned the dose halfway the interval; i.e. at 2.5 and 97.5% in the extreme 5% intervals). It is important to note that according to the computations, 90% of the SCC incidence then stems from people with annual UV exposures greater than the median personal annual exposure. The added annual UV dose from working day exposures is given as a percentage of the median annual personal solar dose (column 1 in Table 6, results in column 5).

Table 7 Percent increase in SCC incidence and risk at 80 years of age due to certain added UV doses (from indoor lighting) to the annual UV dose in school and working years (added UV dose given as % of the annual solar UV dose)

% increase in annual UV dose during working years (age 20-65 yr)	37% lower increase in annual UV dose during school years (age 5-20 yr)	% increase in risk of a person of 80 yrs of having had an SCC with % increase given in columns 1 and 2	% increase in overall incidence of SCC with everybody subjected to % increase given in columns 1 and 2 (EU std population)	% increase in overall incidence of SCC with a wide spread* in annual solar UV dose and with an added annual dose given as % of median in columns 1 and 2
1	0.63	1.6	1.6	0.9
2	1.26	3.2	3.2	1.8
5	3.15	8.0	8.0	4.5
10	6.3	16.4	16.4	9.2
20	12.6	34.1	34.2	19.0

Note: the SCC incidence in Denmark in 2007 was 19.1 10^{-5}/yr in males and 12 10^{-5}/yr in females (Birch-Johansen et al. 2010), the corresponding risk of SCC at the age of 80 years is estimated to equal 0.020 in males and 0.013 in females (using equations 1-5).

** 95% in the range 3.3-fold under and over the median, according to a lognormal distribution similar to that in DK.*

Evidently, an increase of a few tenths of a percent in a small personal risk (0.02 for males, 0.013 for females) as presented in Table 6 is not very alarming for an individual. However, if such an exposure regimen occurs population-wide, the increase in incidence can result in a substantial number of additional cases per year. To this end, we consider the incidence (see legend of Table 8) in the Danish population of 5.8 million people, and find that it comes down to 900 new cases of SCC per year. Outdoor workers contribute to this incidence, but do not appear to run an increased risk in Denmark (Kenborg et al. 2010). As a simplification, we make no correction for outdoor workers. Hence, increases in incidence in the range of 0.9 to 19.0 percent from indoor exposure as given in the last column of Table 6 will add 8.7 to 170 additional cases of SCC per year in Denmark (this

would add from 9 up to 190 additional cases annually for every 1,000 new cases that are diagnosed in Northwestern Europe).

As a next step, we want to consider the potential impact of commercially available fluorescent lamps on the number of cases of SCC. We had access to measurements on 17 double-capped and 61 single-capped fluorescent lamps (Schulmeister et al. 2011). In a conservative approach we assume light levels of 500 lux (common in well-lit offices) from double- or single-capped fluorescent lamps in ceiling-mounted open luminaires (i.e. no filters inserted) and the corresponding levels of erythemally effective UV exposures (expressed in mW/m^2 and SED/h) for 6 h/day on school days and 8 h/day on working days as an adult ("full exposure"). However, these exposure levels pertain to a flat surface at desk top level; exposure to the backs of the hands will probably vary between 50 and 100% of this "full exposure" level, and to the face between 30 and 50% (except for designers and people performing fine assembly or repair work for whom levels may be closer to 1,000 lux and the source closer to hand and face). Moreover, the reflectors in luminaires commonly lower the ratio between UV and visible radiations (personal communication Dr. K. Schulmeister, Lytle et al. 1992-1993); we choose a reduction down to 60%. Combining the effects of reflectors and geometry of exposure we introduce a reduction to 30% of the "full exposure" as a more realistic maximum level of exposure ("30% exposure" in Table 8). Thus, we computed the increased risks at 65 years of age, the increases in incidences and additional number of SCC cases per year in Denmark for double- and single-capped fluorescent lamps with high, median and low levels of UV output: see Table 8.

From Table 8 we conclude that a conjectural fluorescent lamp at the upper UV limit of the CIE/IEC exempt risk category (RG0) yields >342 additional SCC cases per year under these worst case scenarios. Fluorescent lamps on the market commonly fall well below this limit, but even these lamps well within the exempt category (RG0) with the highest UV output and TL tubes of median UV output still yield substantial numbers of additional cases (47 - 126126). The double-capped TL tubes lamps in general tend to have higher UV outputs at 500 lux than the single-capped ones. Based on these computations a restriction to a maximum increase in SCC incidence of 1% among indoor workers would amount to a maximum erythemal UV output of about 0.18 mW per klx (i.e. roughly corresponding with 0.05 mW actinic UV per klx; one fortieth of the upper limit of RG0). On the other hand, it should be realized that a nice sunny Mediterranean vacation of a week can add as a median some 50 SEDs to an annual dose, which under the present worst case scenario is equivalent to the estimated annual exposure in the office from a fluorescent lamp with 4.8 mW erythemal UV per klx (roughly 1.4 mW actinic UV per klx, just below the upper limit of RG0). The difference is, of course, that in the "vacation case" people deliberately expose themselves, whereas people are not aware of any UV exposure indoors, and may even assume themselves to be completely free from exposure to UV radiation. Moreover, not every indoor worker will acquire such added UV dosages from sunny holidays, whereas most indoor workers are inescapably subjected to the indoor lighting at work; i.e. the latter regimen is imposed on a large portion of the population, but it can be controlled to a large extent through limits on the UV emissions of the lamps.

In actual practice, exposure to fluorescent lamps will be lower than in the worst case scenarios presented here. To improve on these scenario studies we need actual data on indoor personal exposures. Nevertheless, these worst case scenario studies serve to indicate the potential impacts of these lamps on SCC numbers if these lamps dominated indoor lighting outside the home.

Table 8 Estimates of SCC risk, incidence and cases per year in Denmark under the worst case scenario attributable to exposures to double- and single-capped fluorescent lamps with high, median and low UV emissions in offices and schools added to a basic personal annual solar UV dose, with a median of 166 SEDs/yr and 95% in the range 50 – 551 SEDs/yr. For comparison, the two bottom rows give the effects of adding a Mediterranean holiday to everybodys annual UV dose or having the Danish people living in Australia with the corresponding SCC risk and incidence.

Source (all RG0) at 500 lux	Actinic UV in mW/m^2	E_{ery}/E_{act}, ratio erythemal over actinic UV	Erythemal UV in mW/m^2	SED/h at 500 lx exposure	30% exposure SED/y from working days (+% of median annual solar dose)	% increase in risk at 65y[#] with median solar exposure	% increase in incidence	in DK added # cases/y*
Just compliant with RG0 limit	max 1	> 3	> 3	> 0.108	> 62.2 (37.5)	>87	>38	>342
high UV								
Double-capped tube	0.328	3.66	1.20	0.043	24.9 (15)	31	14.0	126
Single-capped tube	0.191	3.30	0.630	0.0227	13.1 (7.9)	16	7.2	65
median UV								
Double-capped tube	0.141	3.24	0.4565	0.0164	9.45 (5.7)	11	5.2	47
Single-capped tube	0.00291	3.85	0.0112	0.000403	0.23 (0.14)	0.27	0.13	1.2
low UV								
Double-capped tube	0.00834	5.59	0.0466	0.00168	0.97 (0.58)	1.1	0.52	4.7
Single-capped tube	$3.64\ 10^{-6}$	3.93	$1.43\ 10^{-5}$	$5.15\ 10^{-7}$	0.0003 (0.00018)	0.0003	0.0001	<<1
Reference exposures								
Everybody 1 week Mediterranean vacation (50 SEDs) each year throughout life						83	45	405
living in Australia						4,440*** *	3,600	33,500

Note: Scenario with UV exposure from the fluorescent lamps during school years, 6 h/d, 5d/wk, 40 wks/y from 5 till 20 years of age, and during working days as an adult, 8 h/d, 5d/wk, 48 wk/y from 20 till 65 years of age; numbers in columns 2–5 pertain to full exposure to the lamps at 500 lux; annual dose in SEDs stated under "30% exposure" is a more realistic maximum exposure from working days than at full exposure; the first row under "sources" represents a hypothetical fluorescent lamp at the upper UV limit of the exempt risk category, RG0, according to CIE/IEC standardization.

[#] *Overall risk at 65 yrs of age scales to 0.0057 for males and to 0.0036 for females in Denmark, and equals 0.26 for males and 0.17 for females in Australia in 2002 (Staples et al. 2006).*

***Not calculated for median solar exposure, but risk estimated from actual cumulative (age-specific) incidence in the white Caucasian population of Australia (Staples et al. 2006).*

4. OPINION

This opinion is based on a scientific rationale which has taken into account the relevant scientific literature and other accessible and reliable information on physical and technical characteristics of lighting technologies, principles of optical radiation, as well as biological and health effects of optical radiation. Health effects due to optical radiation have been considered both for the general population and for persons with photosensitive or other pathological conditions. Since the assignment also includes evaluation of possible health effects of various types of lighting technologies, additional data regarding lamp emissions was requested and some were obtained from stakeholders. In addition, for assessment purposes, data regarding exposure patterns was sought, but found to be virually lacking. This lack of information has seriously hampered efforts to perform specific risk assessments.

We have received some information regarding emission data, which has been used for our evaluation, for more than 180 different lamps. These lamps represent all major lamp types that are used for general lighting purposes (tubular fluorescent lamps; compact fluorescent lamps (CFLs) with and without a second envelope; halogen lamps that are either high or low voltage; high pressure discharge lamps (metal halide and sodium); light emitting diodes (LEDs); and incandescent lamps, although the degree of representativeness is uncertain. Regarding specific lamp types, CFLs are well represented in this collection, whereas LEDs for example have been measured in only a few cases. Based on the lamp emissions, the Standard EN 62471 (and also IEC 62471 and CIE S009, since they are all identical in this sense) categorizes the lamps according to the photo-biological hazard that they might pose. The different hazards are:

1. Actinic UV-hazard for eye and skin.
2. UVA-hazard for the eye.
3. Blue-light hazard for the retina.
4. Thermal retina hazard.
5. IR-hazard for the eye.

Following the standards, emission measurements should be performed according to two approaches; namely at a distance where a light intensity of 500 lx is obtained and also at a distance of 20 cm. Based on these measurements, lamps are then classified according to the "Risk Group" (RG) to which they belong. RG0 (exempt from risk) and RG1 (minor risk) do not pose any hazards during normal circumstances. RG2 (medium risk) lamps also do not normally pose any hazards, due to our aversion responses to very bright light sources or due to the fact that we would experience thermal discomfort. RG3 (high risk) include only lamps where a short-term exposure poses a hazard. Importantly, this classification is based on acute exposure responses (a single day, up to 8 hours) and applies only to individuals of normal sensitivity. It should be noted, with respect to RG3 that the risk classification does not consider either long-term exposures or particularly sensitive persons in the population.

SCENIHR's answers to the questions given in the Terms of Reference are given directly in connection with the questions below:

> ***A:*** *To explore and report scientific evidence on potential health impacts on the general public caused by artificial light of which the main purpose is to radiate in the visible range (as opposed to artificial light where the invisible part of the radiation is the main purpose, e.g. suntanning lamps or infrared lamps). The impacts of the light from all available electrical lighting technologies should be studied, both in the visible and invisible range (with specific analyses of the ultraviolet radiation subtypes UVA, UVB and UVC).*

A combined assessment of natural and artificial light shows that adverse health effects due to optical radiation can either occur acutely at certain levels of exposure, or after long-term repeated exposures at lower levels. Depending on the effect (endpoint) of concern (e.g. skin burn, skin cancer, retinal damage, cataract) either the intensity or duration of exposure is of most relevance. In general, the probability that artificial lighting for visibility purposes induces any acute pathologic conditions is low, since expected exposures are much lower than the levels where effects are known to occur in healthy people and are also much lower than in typical summer daylight. The available lamp emission data show that for all investigated hazard outcomes, the absolute majority of lamps are classified as Risk Group 0 (RG0; "exempt from risk"). Most of the rare exceptions are classified as Risk Group 1 (RG1; "low risk"). The very few lamps assigned to higher Risk Groups were either measured without the required UV-shielding glass cover, or at a very short distance (20 cm) which is not the intended use distance for this lamp type.

Standard EN 62471 gives limits that are protective against acute effects, while long-term effects are only marginally considered. Thus the emissions in e.g. the UV range may comply with these limits, but may still have an effect on skin carcinoma incidences when a population is subjected to extensive and large scale exposure to these lamps.

A common exposure situation, such as most household lighting, would involve an illumination level which is so low that exposure to potentially problematic radiation is considered negligible (with the possible exception of prolonged task lighting with a lamp close to the body which may lead to UV exposures approaching the current workplace limit set to protect workers from skin and retinal damage). However, according to a worst case scenario developed in the scientific rationale, the highest measured emissions of UV from fluorescent lamps used typically indoors in professional environments, although well below the limits for RG0, could be contributing to the number of squamous cell carcinomas in the EU population. This is in comparison to a hypothetical situation where the same population is not exposed to UV radiation from artificial light indoors. The annual erythemal UV dose expected from the worst case scenario approximately corresponds to the dose one would get from a half week Mediterranean holiday. Fluorescent lamps typically emit less than half of the UV radiation assumed in the worst case scenario. The vast majority of the CFLs tested emit erythemal UV at very low levels, amounting at the most to an extra day of sunbathing a year.

Low levels of UV emissions may occur from certain lamp types (quartz halogen lamps, single- and double-capped fluorescent lamps as well as incandescent light bulbs). These emissions may, in some cases, in particular for certain halogen lamps with poor UV filtering, include UVC in addition to UVA and UVB. UVC is not naturally present on Earth due to the blocking action of the earth’s atmosphere, so any emissions from lamps would provide a novel type of exposure. However, most action spectra on skin and eye effects include UVC. Hence, biologically effective doses take UVC into account and are thus considered in the categorization of the Risk Group, as discussed above. However, detectable levels of UVC do signal a considerable overall output of biologically harmful short wavelength UV radiation. Regarding a possible need for separate UVA, UVB or UVC radiation limits for tungsten halogen lamps and other light sources that emit UV radiation, the Scientific Committee considers that there is no scientific basis for making such specific recommendations beyond the established dose limits.

Evidence from *in vitro* experiments suggests that blue light at 10 W/m^2 induces photochemical retinal damage (Class II) upon acute (hours) exposure, and animal experiments and *in vitro* studies suggest that cumulative blue light exposure below the levels causing acute effects also can induce photochemical retinal damage. There is no consistent evidence from epidemiological studies regarding the effect of long-term exposure to sunlight (specifically the blue component of sunlight) on photochemical damage to the retina (particularly to the retinal pigment epithelium), which may contribute to age-related macular degeneration (AMD) later in life. Whether exposure from artificial light could have effects related to AMD is uncertain.

There is no evidence that artificial light from lamps belonging to RG0 or RG1 would cause any acute damage to the human eye. Studies dedicated to investigating whether retinal lesions can be induced by artificial light during normal lighting conditions are not available. Lamp types belonging to RG2 and higher are usually meant to be installed by professionals in locations where they do not pose a risk.

Chronic exposure to blue light from improperly used lamps could, in theory, induce photochemical retinal damage. There is however no evidence that this constitutes a risk in practice. It is unlikely that chronic exposures to artificial light during normal lighting conditions could induce damage to the cornea, conjunctiva or lens.

Besides the beneficial effect of light, e.g. through synchronising the day-night rhythm, there is mounting evidence suggesting that exposure to light at night while awake (especially during shiftwork), may be associated with an increased risk of breast cancer and also cause sleep, gastrointestinal, mood and cardiovascular disorders possibly through circadian rhythm disruption. Importantly, these effects are associated with light, without any specific correlation to a given lighting technology.

> ***B:*** *To update the SCENIHR report on Light Sensitivity (from 23 September 2008) in light of further evidence, and to examine the aggravation of the symptoms of pathological conditions in the presence of lamp technologies other than compact fluorescent lamps (including conventional incandescent and halogen lamps, halogen lamps with improved efficiency and light emitting diode lamps).*

The previous SCENIHR opinion on Light Sensitivity (SCENIHR 2008) identified that some pre-existing conditions (epilepsy, migraine, retinal diseases, chronic actinic dermatitis, and solar urticaria) could be exacerbated by flicker and/or UV/blue light. However, at that time there was no reliable evidence that compact fluorescent lamps (CFLs) could be a significant contributor. This conclusion needs updating as more recent studies indicate a negative role for certain CFLs and other artificial light sources (including sometimes incandescent bulbs) in photosensitive disease activity. There are no published data on the effect of exposure of a photosensitive patient to light from halogen lamps.

There is strong evidence that UV and, in some patients visible light, can induce skin lesions of true photodermatoses. Although sunlight is reported by most patients as the main trigger of disease activity, occasionally severely affected patients over the range of endogenous (and exogenous) diseases report a provocative role for artificial lighting.

There is a lack of controlled skin provocation studies relating effects to the magnitude and the wavelength components of the light source, although there is evidence that the shorter wavelength light components (blue or UV) tend to be more effective than the longer wavelength components (red) in aggravating pre-existing conditions. Some research work has been conducted in particularly severely affected individuals suffering from photodermatoses such as lupus erythematosus, chronic actinic dermatitis and solar urticaria. This provides good evidence for the aggravation of symptoms related to these pre-existing skin diseases. Such work needs to be confirmed, and also extended using a range of lamp types over a wider range of diseases in greater numbers of patients. Particular attention seems justified for the individual variability of the conditions for aggravation of such diseases. Until such data exist, it seems reasonable to assume that the UV, and in some cases the blue radiation component of artificial lighting in an as yet undefined number of patients, may contribute to the aggravation of symptoms related to their skin disease, and in the case of lupus erythematosus possibly also to the aggravation of their systemic disease.

Generally, double envelope CFLs emit much less UV radiation than single envelope CFLs. Most LEDs in general use emit little or no UV radiation. However, with the considerable variability of UV/blue light components for lighting technologies, even of the same or similar kind, no general advice can be given and individual optimisation of the lighting technology is advised for these patients.

The effect of light is variable depending on the genetic alterations that are causing inherited retinal degeneration. In specific conditions like Stargart disease, the retinal pigment epithelial (RPE) cells are particularly sensitive to Class II photochemical damage, which is induced by peaks at shorter wavelengths. In other retinal dystrophies, light does not exert any aggravating effect. However, since the causative mutation is seldom known to the patient or their family, and because there is no clear correlation between genotype and phenotype, it is recommended for all patients with retinal dystrophy to be protected from light by wearing special protective eyeware that filter the shorter and intermediate wavelengths.

The previous SCENIHR opinion on Light Sensitivity stated that modern CFLs are basically flicker-free due to their electronic high frequency ballasts. However, it was also noted that studies indicated that residual flicker can occur during certain conditions, at times also related to other circuitry like dimmers operated with the light source, in both CFLs and incandescent bulbs. In principle, there can be a residual sinusoidal modulation of the light of any light source at twice the line frequency of e.g. 50-60 cycles. Any light source operated on DC, after transformation from the AC line, is flicker-free. This has been the predominant case for LED operation, but is also applicable to other lighting technologies, e.g. halogen and incandescent lamps. Flicker cannot typically be observed in static settings above about 60-80 cycles, while in conjunction with dynamic scenes, the effect is still visible at higher frequencies.

There is no scientific evidence available to evaluate if conditions such as Irlen-Meares syndrome, myalgic encephalomyelitis, fibromyalgia, dyspraxia, autism, and HIV infection are influenced by the lighting technologies considered in this opinion.

> ***C:*** *If health risks are identified under points A or B, to estimate the number of EU citizens who might be at risk and identify the level of emission/exposure safeguarding the health of citizens and/or means to mitigate or entirely prevent the impact of the problematic parameter of the light technology in question.*

All healthy individuals may be at some risk from UV radiation and blue light from indoor lighting, albeit to different degrees due to differences in genetic background and in the type of light source used. Short-term UV effects on healthy people are thought to be negligible. A proper assessment of long-term risks due to daily low level UV exposure is not possible because data on actual personal indoor UV exposure are lacking. Due to this knowledge deficit, it would appear advisable to be cautious and to develop worst case scenarios. The worst case scenario examined in this opinion involved workplace/school exposure to double- or single-capped fluorescent lamps in ceiling-mounted open luminaires. This scenario is based on a biologically plausible risk model as it appears to be similar in form for mouse and man (hazards being proportional to age and power of cumulative dose). However, the model parameters for humans are derived from rather crude population-level incidence data, and the worst case scenario had to be based on some simplifying assumptions for lack of data. This scenario assumes the validity of extrapolating from studies on animals with short lifespans to life-time human exposures. Furthermore, it assumes the appropriateness of dose-level extrapolation from experimental studies to real human exposures and that all individuals in a population experience the same risk independent of susceptibility factors. If we take lamps with the highest measured UV output (still well within Risk Group 0), such exposure adds the equivalent of 3 to 5 days vacation in a sunny location to the average annual UV dose. Although this would lead to an increase in the personal risk of squamous cell carcinoma, such an increase would remain small (a few % over a lifetime in Denmark). Population-wide exposure to such lamps could, however, add approximately 100 cases of squamous cell carcinomas a year to a base line of 900 cases/year in Denmark. It should be stressed that the UV output of most of the fluorescent lamps tested fall well below this level, and are not expected to affect squamous cell carcinoma incidences. Improper use of lamps belonging to Risk Groups 1-3 (due to missing or disregarded user information, non-

professional installation) could cause retinal damage. While no such cases are known, appropriate measures could be considered to ensure that these lamps are not misused. The current standardization of lighting lamps and luminaires in four risk categories appears sufficient to limit the personal short-term risk. However, RG0, as it is based on acute effects, should not be taken to imply adequate protection of the general population as a whole from effects after long-term exposure to UV radiation. Nevertheless, it would be useful to communicate information on risk categories to the consumer.

The previous SCENIHR opinion (SCENIHR 2008) stated that a number of patients are exceptionally sensitive to UV/blue light exposure. The number of EU citizens with light-associated skin disorders that would be affected by exposures from CFLs was estimated in the report to be around 250,000. Clearly, the risk for this group of patients is not limited to CFL, but includes all light sources with significant UV/blue light emissions. The lack of proper data precludes any improvement of the estimate of the size of the affected group.

Also photosensitivite patients undergoing photodynamic therapy might be expected to react to CFL and LED sources to a greater extent than to incandescent lighting. This is due to a combination of greater sensitivity of porphyrins to blue light (soret band), coupled with an enhanced blue light emission of these sources. However, such patients are aware of their extreme photosensitivity which needs careful management.

For patients with light-associated skin disorders, the previous SCENIHR opinion recommended that, when using CFLs, a double envelope type is preferable. The current opinion supports that position. Double envelope CFLs generally emit much less UV radiation than single envelope CFLs and are much less likely to induce a reaction in patients with light-associated skin disorders. While a second envelope undoubtledly reduces the UV component of any particular lamp, the currently available data, however, documents the high variability of UV and blue light emissions due to different internal design parameters, even for the same externally visible architecture (i.e. also when a second envelope is present). While some compact fluorescent lamps are in the same category, retrofit LED lighting, which does not emit UVR on the physical grounds of the light generation therein, would potentially provide an even better option for such patients. However, for patients whose sensitivity extends into the visible part of the spectrum, it may be necessary to exclude LEDs which have a significant blue component. The UV/blue light irradiation from halogen lamps is also highly dependent on the lamp type. With lamps other than incandescent retrofit halogen bulbs, attention needs to be given to the proper installation, as they are at times sold by the manufacturer to be installed at larger distance or in conjunction with special luminaires or filters against e.g. UV or IR irradiation or to prevent other hazards like fires. While it is unlikely that there would be a significant UV risk from halogen lamps for the general public, provided that protective measures are complied with, the UV content can rise to levels which are of concern for patients with light-associated skin disorders at close operating distances and long exposure times, which is not a very common use pattern for this lamp type.

For individuals with photosensitive skin diseases a list of lamp models (not only types) that are specifically suitable for their situation is needed. The non-representative sample spanning across a wide range of lighting technologies which is provided by Schulmeister et al. (2011) provides a first try. However, important issues like the modification of the emitted spectrum with time after switching on, with progressive aging, and from one to the other manufactured batch are not currently assessed. In view of the large number of patients affected by photosensitive diseases it may be advisable to make sufficient information on the emitted spectrum for individual lamp models available to the healthcare professionals and their patients to allow them to choose their lighting solutions optimally.

D: *To identify potential research needs related to the areas where the lack or scarcity of scientific evidence prevents SCENIHR from coming to firm conclusions.*

The scientific rationale has identified a number of areas where relevant data are lacking regarding the effects of specific lighting technologies on medical conditions. The most important areas where knowledge gaps have to be filled in order to be able to draw firm conclusions related to this opinion include:

- Emission data (ranging from UVC up to 800 nm) characterizing the different lighting technologies – a challenge due to the variation of manufacturing parameters, and a database of these characteristics of specific lamps on the European market.
- Exposure database on indoor visible light radiance to the eye and personal UV exposures from various lamp types compared to ambient outdoor exposure. The database should be established in view of the potential conditioning of the eye due to the largely different voluntary exposure to sunlight from one individual to another, and for the also very different use patterns for UV/ light protective eyewear between individuals and populations.
- Attention should be paid to develop a risk group categorisation that takes into account potential chronic effects like SCC.
- Eye conditions:

  a. epidemiologic studies of artificial light exposure and ocular pathologies (including AMD); and

  b. retinal effects of chronic exposure to artificial light for visibility purposes (animal studies).
- The role of various types of artificial lighting sources in photosensitive skin diseases (provocation studies).
- Mechanisms and consequences of exposure to artificial light in the late evening, at night and in the early morning, including circadian disruptions in both shift-workers and in the general population.
- Flicker induced health effects from the residual high frequency (100-120 Hz) intensity modulations.
- The particular role of UVC components in artificial lighting for skin diseases taking into account especially sensitive populations and the role of prior exposure to sunlight.
- The effects of non-incandescent light sources, in particular those with very inhomogenous or biased spectral distribution on colour rendition, fatigue, and other components of the human visual perception.

5. COMMENTS RECEIVED DURING THE PUBLIC CONSULTATION ON THE HEALTH EFFECTS OF ARTIFICIAL LIGHT

A public consultation on this opinion was opened on the website of the EU non-food scientific committees from 19 July to 30 September 2011. Information about the public consultation was broadly communicated to national authorities, international organisations and other stakeholders.

In total, 16 contributions were received of which four were from public authorities, five from industry, one from academia, two from NGOs and six from individuals and three others.

Most of the material submitted was relevant, contained specific comments and referred to peer-reviewed scientific literature. As a result, each submission was carefully considered by the Working Group. Only three submissions from industry disagreed with the preliminary opinion and the submission from academia showed some disagreement.

The document has been revised to take account of the relevant comments and the literature has been updated with relevant publications. The scientific rationale was clarified and strengthened in certain respects. The opinion, however, remained essentially unchanged.

6. MINORITY OPINION

None

7. LIST OF ABBREVIATIONS

A2E	Major fluorescent component of lipofuscin
ACGIH	American Conference of Governmental Industrial Hygienists
AC	Alternating current
AD	Atopic dermatitis
AMD	Age-related macular degeneration
AP	Actinic prurigo
AREDS	Age-Related Eye Disease Study
ARM	Age-related maculopathy
ATP	Adenosine triphosphate
AUVA	Allgemeine Unfallversicherungsanstalt (General Accident Insurance Institution)
BCC	Basal cell carcinoma
CAD	Chronic actinic dermatitis
CCFL	Cold-cathode fluorescent lamp
CDLE	Chronic discoid lupus erythematosus
CFF	Critical flicker frequency
CFL	Compact fluorescent lamp
CI	Confidence interval
CIE	Commision International de l'Eclairage
CMHL	Ceramic metal halide lamp
CRI	Colour rendering index
DBD	Dielectric barrier discharge
DC	Direct current
DIN	Deutsches Institut für Normung
DLE	Discoid lupus erythematosus
DNA	Deoxyribonucleic acid
ECDC	European Centre for Disease prevention and control
ECHA	European Chemicals Agency
EEG	Electroencephalography
EFSA	European Food Safety Authority
ELC	European Lamp Companies Federation
EM	Electromagnetic (radiation)
EMA	European Medicines Agency
EOG	Electrooculography
EU	European Union
FED	Field emission device
FL	Fluorescent lamps
fMRI	Functional magnetic resonance imaging
GLS	General Lighting System
GPCRs	G-Protein-coupled receptors
HID	High-intensity discharge lamp

HIV	Human immunodeficiency virus
HWL	Mercury mixed-light
IARC	International Agency for Research on Cancer
ICNIRP	International Commission on Non-Ionizing Radiation Protection
IEA	International Energy Agency
IEC	International Electrotechnical Commission
IEEE	Institue of Electrical and Electronics Engineers
INSERM	Institut National de la Santé et de la Recherche Médicale (National Institute of Health and Medical Research)
ipRGCs	Intrinsically photosensitive retinal ganglion cells
IR	Infrared (radiation)
LE	Lupus erythematosus
LED	Light emitting diode
LET	Lupus erythematosus tumidus
LOAEL	Lowest observed adverse effect level
LPS	Sodium low-pressure lamp
LWS	Long wavelength cone opsin, long wavelength sensitive cones (red)
MED	Minimal erythemal dose
MHL	Metal halide lamp
MWS	Medium wavelength cone opsin, medium wavelength sensitive cones (green)
OR	Odds Ratio
NOAEL	No observed adverse effect level
NRPB	National Radiological Protection Board
NGO	Non-Governmetal organization
OECD	Organisation for Economic Co-operation and Development
OLED	Organic light emitting diodes
PCA	Polycrystalline alumina
PDT	Photodynamic therapy
PET	Positron emission tomography
PLE	Polymorphic light eruption
PMLE	Polymorphous light eruption
POLA	Pathologies Oculaires Liées à l'Age (study)
PWM	Power wave modulation
RG	Risk Group
RMS	Root mean square
ROS	Reactive oxygen species
RP	Retinitis pigmentosa
RPE	Retinal pigment epithelial cells
RR	Relative risk
SAD	Seasonal affective disorder
SCC	Squamous cell carcinoma
SCCS	Scientific Committee on Consumer Safety
SCENIHR	Scientific Committee on Emerging and Newly Identified Health Risks

SCHER	Scientific Committee on Health and Environmental Risks
SCLE	Sub acute cutaneous lupus erythematosus
SCN	Suprachiasmatic nucleus
SED	Standard erythemal dose
SHP	Sodium high-pressure discharge lamp
SI	Système International d'unités (International System of Units)
SLE	Systemic lupus erythematosus
SSL	Solid state lighting
SWS	Short wavelength cone opsin, short wave length sensitive cones (blue)
TL	Tube luminescent (French for luminescent tube)
TRP	Transient receptor potential
TRPV	Transient receptor potential vallinoid
UHP	Ultra high performance
UK	United Kingdom
US(A)	United States (of America)
UV	Ultraviolet (radiation)
VLPO	Ventrolateral preoptic nucleus
VUV	Vacuum ultraviolet radiation
XP	Xeroderma pigmentosum

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9. GLOSSARY

Term	Explanation
Absorption	The process by which the energy of a photon, which is the quantum of the electromagnetic field, is taken up by matter, typically the electrons of an atom.
Action spectrum	The rate of a physiological activity plotted against wavelength of light.
A2E	*Lipofuscin* accumulates as a by-product of phagocytosis of photoreceptor outer segments. It accumulates with age and its major hydrophobic fluorophore resulting from the reaction of two molecules of all trans-retinal with ethanolamine is A2E. Upon excitation by blue light A2E generates photodynamic reactions.
Cataract	A clouding that develops in the crystalline lens of the eye or in its envelope, varying in degree from slight to complete opacity and obstructing the passage of light.
Choanoflagellates	A group of free-living unicellular and colonial flagellate eukayotes considered to be the closest living relatives of animals.
Chromophore	The part of a molecule, which is responsible for it colour.
Circadian rhythm	An endogenously driven roughly 24-hour cycle in biochemical, physiological, or behavioural processes.
Conjunctiva	A clear mucous membrane consisting of cells and an underlying basement membrane that covers the sclera (white part of the eye) and lines the inside of the eyelids.
Cornea	The transparent front part of the eye that covers the iris, pupil, and anterior chamber. It transmits above 295 nm.
Correlated colour temperature	Colour temperature is a simplified way of characterizing the spectral properties of a light source. While in reality the colour of light is determined by how much each point on the spectral curve contributes to its output, the result can still be summarized on a linear scale. Low colour temperature implies warmer (more yellow/red) light while high colour temperature implies a colder (more blue) light. *Daylight* has a rather low colour temperature near dawn, and a higher one during the day. Therefore it can be useful to install an electrical lighting system that can supply cooler light to supplement daylight when needed, and fill in with warmer light at night. This also correlates with human feelings towards the warm colours of light coming from candles or an open fireplace at night. Standard units: Kelvin.
Dopant	Also called a doping agent, is a trace impurity element that is inserted into a substance in very low concentrations in order to alter the electrical or optical properties of the substance.
Dose response relationship	The dose-response relationship, or exposure-response relationship, describes the change in response at different levels of exposure.
Dose response curve	Records the percentage of a population showing a given quantum (all or nothing) response such as death when each individual member of the population is subjected to the same

	dose of toxicant (reflecting a given exposure).
Effective exposure or Photobiologic dose	The part of the dose, which actually produces the photochemical effect, considered. Symbol: Heff; unit: spectrally weighted J/m^2 or J/m^2 [effective].
Enzyme	A protein that catalyses chemical reactions.
Eukaryotes	An organism whose cells contain complex structures enclosed within membranes.
Erythemal dose	The amount of radiation which, applied to the skin, makes it turn temporarily red (erythematous).
Excitation	An elevation in energy level above an arbitrary baseline energy state.
Exposure	Radiant energy per surface area in J/m^2 is a photobiologic metric, which quantifies the transfer of radiant energy to the body (CAUTION: the unit used in ophthalmology is mJ/cm^2, which is ten times smaller than J/m^2).
Flagellates	Organisms with one or more whip-like organelles called flagella. A flagellum is a tail-like projection that protrudes from the cell body of certain prokaryotic and eukaryotic cells and is used for locomotion.
Fluorescence	The emission of light by a substance that has absorbed light or other electromagnetic radiation of a different wavelength.
Flux	Flux is defined as the amount that flows through a unit area per unit time.
G-Protein-coupled receptors (GPCRs)	GPCRs comprise a large protein family of transmembrane receptors that sense molecules outside the cell and activate cellular responses. G-Protein-coupled receptors are found in eukaryotes, including yeast, and choanoflagellates.
Incandescence	The emission of light from a hot body due to its temperature.
Iris	A thin, circular structure in the eye, responsible for controlling the diameter and size of the pupils and thus the amount of light reaching the retina.
Irradiance	Exposure rate or radiant energy per surface area per unit time in J/m^2s, W/m^2); a photobiologic metric, which quantifies the transfer of radiant energy to the body.
Isomerisation	The process by which one molecule is transformed into another molecule which has exactly the same atoms, but the atoms are rearranged.
Light	Visible electromagnetic radiation.
Lipofuscin	The name given to finely granular yellow-brown pigment granules composed of lipid-containing residues of lysosomal digestion. It is considered one of the aging or "wear and tear" pigments, found in various cells.
Luminaire	An electrical device used to create artificial light and/or illumination, by use of an electric lamp.
Luminance	A photometric measure of the luminous intensity of light emitted from a source per unit area, and falls within a given solid angle. The SI unit for luminance is candela per square metre (cd/m^2).
Luminescence	Light that usually occurs at low temperatures, and is thus a form of cold body radiation. Chemical reactions, electrical

	energy, subatomic motions, or stress on a crystal can cause it. This distinguishes luminescence from incandescence, which is light generated by high temperatures.
Macula	An oval-shaped highly pigmented yellow spot near the center of the retina of the human eye.
Melatonin	A naturally occurring hormone found in humans, animals, and plants. Known as the "hormone of darkness", it is secreted in darkness.
Mitochondria	Often called the "powerhouse of the cell" because they produce adenosine triphosphate (ATP) from sugar and other organic molecules.
Opsins	Visual pigments, which are a group of light-sensitive 35-55 kDa membrane-bound G-protein-coupled receptors of the retynilidene protein found in photoreceptor cells of the retina, which trigger the neural signalling.
Oxidative stress damage	Photochemical and/or photodynamic damage.
Photokeratides	A condition in which the cornea (the front part of the eye), becomes inflamed by light.
Pupil	A hole located in the center of the iris of the eye that allows light to enter the retina.
Radiance and spectral radiance	Radiometric measures that describe the amount of light that passes through or is emitted from a particular area, and falls within a given solid angle in a specified direction. They are used to characterize both emission from diffuse sources and reflection from diffuse surfaces. The SI unit of radiance is watts per steradian per square metre ($W \cdot sr^{-1} \cdot m^{-2}$).
Retina	Light-sensitive tissue lining the inner surface of the eye.
SI	Système international d'unités (The International System of Units).
Solid angle	The two-dimensional angle in three-dimensional space that an object subtends at a point. It is a measure of how large that object appears to an observer looking from that point. The SI unit for a solid angle is steradian.
Threshold dose	The dose below which no harm occurs in an exposed population; may be approximated by a NOAEL (no observed adverse effect level) or a LOAEL (lowest observed adverse effect level).
Xanthophyll pigments	Lutein and zeaxanthine, known as xanthophylls or macular pigments are yellow pigments that accumulate in the inner layers of the macula and efficiently absorb blue light. Macular pigments reduce with age and can be increased by specific food intake.

ANNEX I – TECHNICAL CHARACTERISTICS OF LIGHTING TECHNOLOGIES

A. Incandescent Lamps

Incandescent lamps consist of a bulb containing a wire filament that is heated as a result of an electrical current flow (Joule effect) and emits light. The emitted spectrum is continuous, but up to 95% of the energy emitted by incandescent lamps is in the invisible infrared region. This type of spectrum is characterized by low correlated colour temperature (2,400-3,100 K) and low luminous efficacy (5-14 lm/W). Today, tungsten is commonly used as the filament because it has a relatively high melting point and a relatively low rate of evaporation at high temperatures. The filament is surrounded by a gas (argon or krypton in standard incandescent lamps) to reduce the tungsten evaporation rate. Incandescent bulbs may have different types of bulb finishes (e.g. clear, frost) to modify the brightness of the filament and shapes. Figure A1 shows the incandescent lamp family.

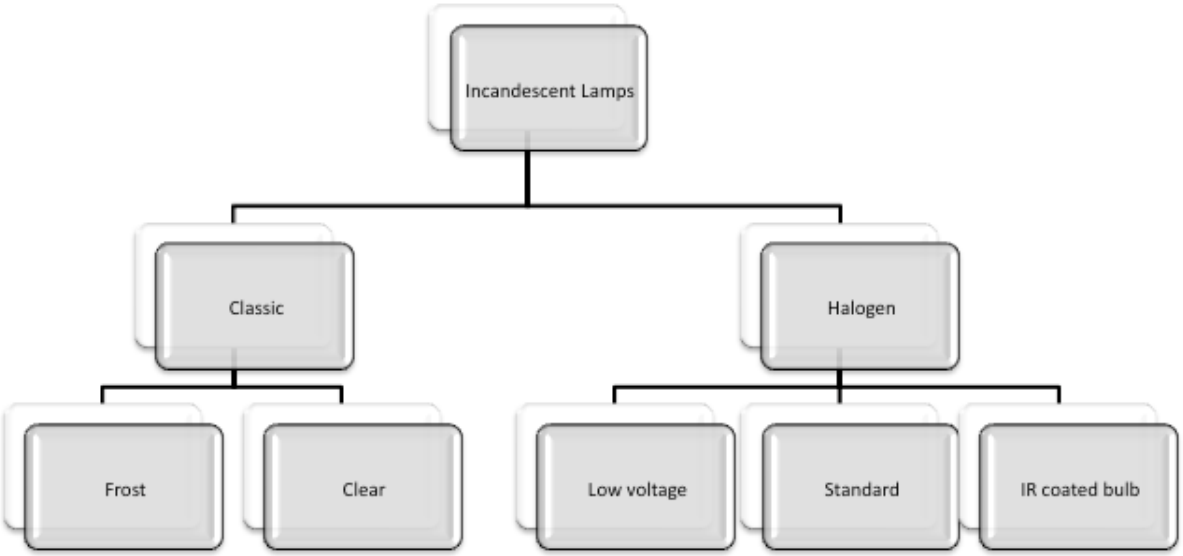

Figure A1 Incandescent lamp categories

A **halogen lamp** is a type of incandescent lamp with a tungsten filament contained within an inert gas and a small amount of a halogen (mainly bromide introduced to the built in methyl-bromide form). The combination of the halogen gas and the hot filament enables a chain of chemical reactions involving metal atoms and halogen, the "halogen cycle", which increases the lifetime of the filament and prevents darkening of the bulb by re-depositing tungsten from the inside of the bulb back onto the filament. This mechanism counteracts filament degradation by sublimation and thus allows operation at higher temperatures with reduced bulb blackening. Higher temperature is responsible for brighter light at higher colour temperatures and higher luminous efficacy (up to 25 lm/W). There are several subsets of halogen lamps that deliver different quantities of light with varying directional spreads which are aimed at a variety of applications. Standard halogen lamps are operated in direct connection to the mains, while low-voltage lamps need a transformer of the electronic power supply. Usually low-voltage lamps are integrated in reflectors; the most common forms employ a conical reflector designed to distribute the light in either very narrow spotlighting applications or slightly broader floodlighting applications. Finally, more efficient lamps use bulbs coated with several thin layers of reflecting materials acting as an interference filter. This filter reflects part of the infrared radiation back into the filament, thus increasing its temperature and enhancing luminous efficacy. The last generation of "energy B-class"

ultra-efficient incandescence uses this technology. Incandescent lamps have quasi-perfect colour rendering, but for fundamental physical reasons, incandescence is much less efficient for converting electricity to light than luminescence and/or fluoresence.

B. Electrical Discharge Lamps

Luminescent light sources are based on electrical discharge processes in gases. In **electrical gas discharge lamps** free electrical charges created by gas ionization are accelerated by an applied electric field which leads to cascades of atomic and/or ionic excitation/de-excitation processes. Predominantly, it is the pressure of the gas and the atomic or molecular species which dominates the emitted spectrum, and thereby categorizes gas discharge lamps. While at lower pressures single transitions corresponding to sharp emission lines occur, broader bands are generated at higher pressures due to the broadening of these lines by the wide range of the velocity distribution of the emitting species.

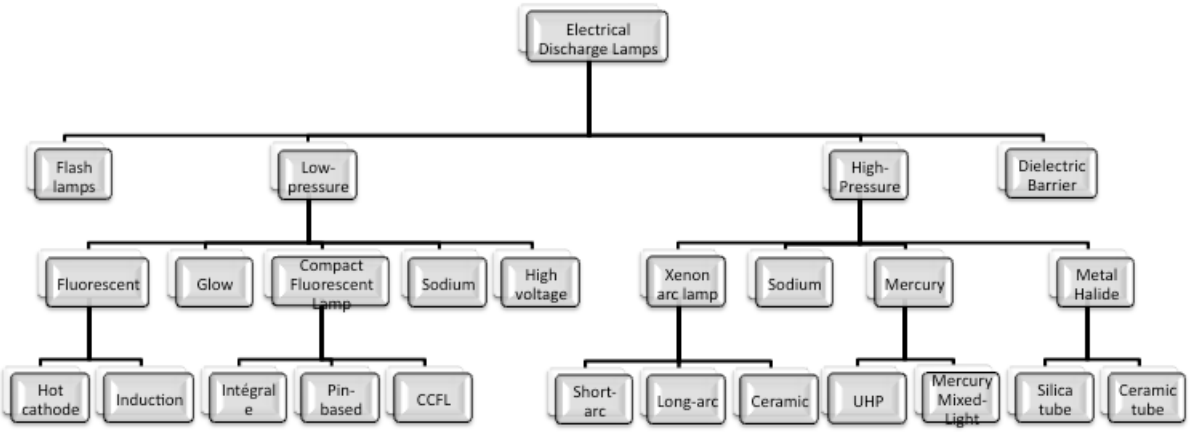

Figure A2 Product classes of electrical discharge lamps

In **low-pressure discharge lamps** the pressure ranges from less than 1 mbar to a few tens of mbar. The physics depends on the balance of collisions and radiative decay of collision induced excited states inside the plasma. The average kinetic energy of the electrons or ions is considerably higher than that of the gas particles. The system is far from local thermodynamic equilibrium conditions. Few sharp emission lines dominate the spectrum that can be tuned in order to better match application needs. Due to the high specificity of the excitation and emission, the luminous efficacy is high, but the colour rendering is very poor.

In **high-pressure gas discharge lamps** the pressure ranges from 1 bar up to 200 bar. In many technical documents this type of lamp is often also named **"high intensity discharge lamp (HID)"**. However, the first term is more accurate and should be preferred. Under these conditions the average kinetic energies of electrons, ions and other components of the gas are closer to each other. Therefore such lamps are operated in a local thermal equilibrium and temperature becomes the key parameter governing all plasma characteristics. This type of lamp is characterized by the considerable broadening

of spectral lines and also by the presence of non-negligible amounts of continuous radiation due to electron bremsstrahlung effect[5].

The luminous efficacy is high, while the fraction of energy irradiated in the IR part of the electromagnetic spectrum is typically increased. The colour rendering ranges from acceptable to excellent. The high pressure cuvettes for these lamps in their current technical implementation mostly require installation and bulb replacement by specially trained staff.

In both cases, the excitation of specific emission lines or bands in the visible range of the electromagnetic spectrum by collisions allows for a considerably higher efficiency of light generation than possible for thermal radiators (incandescence). Overall, lamp engineers can generate a broad range of characteristic spectra suitable for many different applications. Due to the physics involved in the light generation in these sources, the spectral distribution is different from incandescent lighting. While such lamps can be engineered for a continuous emission, some emission lines and bands, dependent on components in the gas, dominate the spectrum. It should be noted that in contrast to incandescent lamps, some constituents are toxic and recycling is strongly advised and required in most countries for most of the discharge lamps.

Glow discharge lamps operate at very low pressures and are characterized by few sharp emission lines due to the excitation/de-excitation of few very specific transitions. Glow discharge lamps can, for example, be operated at mains voltages directly but are of very limited luminosity in confined regions in the vicinity of the electrodes. For their outstanding technical reliability, these lamps are typically used as pilot lamps in a variety of appliances and control electronics but are not typically used in lighting applications.

High voltage low-pressure lamps provide a simple solution to obtain bright and uniform colours at long distances. Long glass tubes are used as discharge vessels and cold tubular cathodes are used as electrodes. High voltage is needed to ignite and maintain the discharge; usually, an electronic ballast ensures the lamp ignition and operation. The tube is filled with various gases (neon, argon, krypton and xenon) and may contain mercury. In some cases phosphor coatings are used in order to change the lamp colour. This type of lamp is often used for signage and commercial advertising, and is therefore of limited applicability for general lighting.

Sodium low-pressure lamps (LPS) have consistently maintained, since their commercial introduction in 1932, their position as the most efficient light source available (200 lm/W under photopic conditions). They are also quite large (about 122 cm long for the 180 W lamp), which makes light distribution from fixtures harder to control. They require a brief warm-up period for the lamp to reach full brightness. These lamps produce a virtually monochromatic light averaging a wavelength of 589.3 nm (actually two dominant spectral lines very close together at 589.0 and 589.6 nm). As a result, the colours of illuminated objects are not easily distinguished because they are seen almost entirely by their reflection of this narrow bandwidth yellow light. Low-pressure sodium lamps have a borosilicate glass gas discharge tube containing solid sodium and a small amount of neon and argon gas acting as Penning mixture to start the gas discharge. The discharge tube may be linear or U-shaped. LPS lamps have an outer glass vacuum envelope around the inner discharge tube for thermal insulation, which improves their efficiency. The key weakness of low-pressure sodium lamps is their very poor colour rendering, which is accounted for by the extremely narrow emission spectrum of light from sodium vapour. They are used where colour is unimportant making them appropriate only for certain types of street lighting and security lighting. Beyond the fact

[5] Electromagnetic radiation produced by the acceleration of a charged particle, such as an electron, when deflected by another charged particle, such as an atomic nucleus. The term is also used to refer to the process of producing the radiation. Bremsstrahlung has a continuous spectrum, which becomes more intense and shifts towards higher frequencies when the energy of the accelerated particles is increased.

that the lamp itself is relatively inexpensive and can be operated on low cost electrical control gear, it contains no mercury.

Fluorescent lamps (FL), also called fluorescent tubes or TL lamps, are low-pressure discharge lamps that use electricity to excite mercury vapour. The excited mercury atoms produce short-wave ultraviolet light (mainly at 253.7 nm) that then causes a phosphor to fluoresce, producing visible light. A fluorescent lamp consists of a soda lime glass tube (straight, U-shaped or circular) internally coated with phosphorous and tungsten wire electrodes coated in a thermionic emitter sealed into each end of the tube. The tube is filled with one or more inert gases (usually argon, krypton or neon) and trace amounts of mercury (less than 5 mg in most cases) in the liquid phase or as a solid amalgam. The operating pressure is less than 10 mbar, while the saturated mercury vapour pressure at operating temperatures remains in the order of few microbars. The inner surface of the bulb is coated with a fluorescent coating made of varying blends of metallic and rare-earth phosphor salts. The bulbs electrodes are usually referred to as cathodes because of their prime function of emitting electrons. In the case of **hot-cathode technology**, electrodes are typically made of coiled tungsten and are coated with a mixture of barium, strontium and calcium oxides chosen to have a low thermionic emission temperature. This type of lamp is dimmable and supports frequent on/off cycles. It should be noted that there exist a variety of fluorescent lamps which are electrode-less and known as **induction lamps**. In these lamps the energy is supplied at high frequency to the plasma via an antenna which is located outside of the bulb.

The overall spectrum emitted in the visible range is relatively poor for any type of fluorescent lamp and crucially depends on the properties of the fluorescent coating. Fluorescent tubes have much higher efficacy levels (60–104 lm/W) and much longer operating lives (7,500–30,000 h) than incandescent lamps, which continue to be characterized as the highest fidelity light source for precise colour rendering (arts, exhibitions etc.). Fluorescent lamps can be designed to provide a large range of correlated colour temperatures ranging from 2,700 K to 7,500 K (daylight) and even more than 8,200 K (skywhite). Fluorescent tubes are the most used electrical light source after incandescent lamps. Fluorescent lamps can operate with both ferromagnetic and electronic ballasts; however, the EU Directive 2000/55/EG bans ferromagnetic ballasts from European market.

Compact fluorescent lamps (CFLs) operate on the same principles as fluorescent lamps. The development of rare-earth phosphors in the late 1970s also enabled the production of CFLs. There are two types of CFLs: with the ballast either integrated (integral) into the lamp or not (pin-based). This terminology corresponds to the "single" and "double-capped" lamps mentioned by the EU directive. CFLs usually consist of two, four or six small fluorescent tubes that are mounted in a base attached to the ballast for ballast-integrated models, or are plug-in tubes for the non-integrated varieties. The lumen-packages of integrated CFLs are designed to match those of equivalent incandescent lamps, but as their efficacies are from four to five times higher, the wattage ratings are proportionately lower. CFL power rating ranges from 4 to 120 W and their efficacies from 35 to 80 lm/W. They have a rated lifespan from 5,000 to 25,000 h. Spectrum, colour temperature and colour rendering are similar to fluorescent lamps.

The **Cold-cathode fluorescent lamps (CCFL)** are like very thin low-pressure fluorescent lamps (a few mm in diameter), but a cold cathode lamp is distinguished from a standard fluorescent lamp since the cathodes are not heated to induce thermionic emission of electrons. Cold cathodes remain popular for LCD backlighting and they have very few applications in general lighting (retail showcases, light boxes, security lighting etc.). They are powered by a direct current supply, usually 12 V, and have efficacy levels around 80 lm/W.

A **mercury high-pressure discharge lamp** is a type of electrical lamp which produces light by means of an electric arc between tungsten electrodes housed inside a transparent fused quartz arc tube. This tube is filled with both gas (usually argon) and mercury in the liquid phase. The gas facilitates the arcs initial strike. Once the arc is

started, it heats and evaporates the mercury forming metallic vapour plasma, which greatly increases the intensity of light produced by the arc and reduces its power consumption. This lamp operates with a ferromagnetic ballast. Originally they produced a bluish-green light, but more recent versions can produce light with a less pronounced colour tint as a result of the addition of phosphors. The power range for these products is from 50 W to 400 W; the luminous efficacy is moderate (23-60 lm/W). The colour temperature is between 3,000 K and 5,500 K, and the colour rendering is also moderate. The lifespan is high and ranges from 6,000 h to 28,000 h. The main use of this type of lamp is outdoor street lighting, but in some cases it can be used for interior lighting for large industrial buildings. However, mercury vapour lamps are falling out of favour and being replaced by sodium vapour and metal halide lamps. The **mercury mixed-light (HWL)** variety contains mercury tungsten blended lamps and has a yttrium vanadate phosphor. These lamps are the perfect alternative to ordinary incandescent lamps because they last longer and do not require either control gear or igniters. Fittings with high-wattage incandescent lamps can be easily upgraded. HWL lamps can therefore provide low-cost lighting installations for factories and other large buildings. The **ultra high performance (UHP**) lamp consists of a small ovoid bulb with interelectrode distance lower than 1 mm. The operating pressure is 200 bar in pure mercury vapour. This lamp doesn't have any lighting applications but it is used for projection applications.

Sodium high-pressure discharge lamps (SHP) contain sodium amalgamated with mercury and some additional rare-gas. The rare-gas (usually xenon) at a low pressure is used as a "starter gas" in the SHP lamp. As sodium is very aggressive for silica, the arc vessel of this lamp is made by polycrystalline alumina (PCA). During the lamp operation some sodium is evaporated, excited and ionised. The sodium D-line is the main source of light from the HPS lamp, and it is extremely pressure broadened by the high sodium pressures in the lamp; because of this broadening and the emissions from mercury, colours of objects under these lamps can be distinguished. The colour of this lamp is pinkish-yellow. Standard high-pressure sodium lamps have the highest efficacy of all high-pressure lamps, with ratings of 80–140 lm/W, but with low- to mid-range colour rendering. Correlated colour temperature ranges from 1,900 to 2,500 K and a lifespan of 5,000 to 28,000 h; typical power ratings range from 40 to 400 W (lamps with power from 600–1,200 W are used for greenhouse lighting and crop growth). High-pressure sodium lamps are now much more commonly used than low-pressure sodium lamps for street and outdoor-lighting applications. A variation of the high-pressure sodium, the white SON, introduced in 1986, has a higher pressure than the typical HPS/SON lamp, producing a colour temperature of around 2,700 K, with good colour rendering. These are often used indoors in cafes and restaurants to create a particular atmosphere. However, these white SON lamps suffer from a shorter lifespan and lower light efficiency (50 lm/W). HPS lamps of any type operate with both ferromagnetic and electronic ballasts and they also need an external starter.

Metal halide lamps (MHL) produce high light output for their size, making them a compact, powerful, and efficient light source. These lamps are closely related to mercury vapour lamps but include other metal elements which are dosed as a metal halide, such as sodium, scandium, thallium, calcium, strontium, indium, praseodymium iodides, in combination with the mercury, which is used as a buffer gas in order to reduce the nominal lamp voltage into typically 90–100 V range. Like most HID lamps, metal halide lamps operate at high pressure and temperature, and require special fixtures to operate safely. There are two technologies: quartz and ceramic tube lamps. The outer bulb also blocks some of the UV light generated by the mercury vapour discharge. The **quartz tube** lamp, as its name indicates operates inside a fused quartz arc tube with two tungsten electrodes (one at each extremity) often doped with thorium. Most types are fitted with an outer glass bulb to protect the inner components and prevent heat loss. The power ranges from 50 W to 2,000 W, the correlated colour temperature varies from 3,700 K to 6,100 K (following the iodide composition); the colour rendering is good, even excellent. The luminous efficacy ranges from 54 to 120 lm/W, but the lifespan is limited to between 4,000 and 8,000 h. Because of their wide spectrum, they are used for indoor

growing applications, in athletic facilities and are quite popular with reef aquarists who need a high-intensity light source for their corals. The problem with quartz tube lamps is the reduced lifespan and the colour stability across the lifespan. This is due to the fact that metal halides in the liquid phase at high temperature (operating conditions) are very aggressive for the quartz. The problem has been solved by replacing the fused quartz by a polycrystalline alumina (PCA) tube. The **ceramic metal halide lamp (CMHL)** is a relatively new source of light that is a variation of the old high-pressure mercury-vapour lamp. The discharge is contained in a ceramic tube. During operation, the temperature of this ceramic tube can exceed 1,200 K. The ceramic tube is filled with mercury, argon and metal halide salts. The metallic atoms are the main source of light in these lamps, creating a bluish light that is close to daylight with a CRI (colour rendering index) of up to 96. The exact correlated colour temperature and CRI depend on the specific mixture of metal halide salts. There are also warm-white CMHLs, with somewhat lower CRI (78-82) which still give a more clear and natural-looking light than the old mercury-vapour and sodium-vapour lamps when used as street lights, besides being more economical to use (Figure A3). The luminous efficacy ranges from 70 lm/W up to 120 lm/W, and the power range of the commercially available products is between 20 W and 400 W. The lifespan ranges from 8,000 h to 16,000 h; the colour stability is excellent. Applications for these lamps include street and architectural lighting, television and film making as well as shop lighting (low power versions), digital photography, etc.

Xenon arc lamps use xenon plasma to produce a bright white light that closely mimics natural daylight. They consist of a glass or fused quartz arc tube with tungsten metal electrodes at each end. The glass tube is filled with xenon gas. Xenon arc lamps can be roughly divided into two categories: short-arc and long-arc lamps. **Xenon short-arc** lamps are low-voltage, high-current, DC devices. Due to the DC operation, the electrodes of these lamps are disymmetric. The white, continuous light generated with this arc is of daylight quality but plagued by a rather low efficiency in terms of lumens per watt. The lamp has a lifetime of around 2,000 h. Many lamps have a low-UV blocking coating on the envelope and are sold as "Ozone Free" lamps. Some lamps have envelopes made out of ultra-pure synthetic fused silica (trade name "Suprasil"), which roughly doubles the cost, but which allows them to emit useful light into the so-called vacuum UV region. These lamps are normally operated in a pure nitrogen atmosphere. Today, almost all movie projectors in theatres employ these lamps with a rating ranging from 900 watts up to 12 kW. When used in Omnimax (IMAX Dome) projection systems, the power can be as high as 15 kW in a single lamp. Xenon short-arc lamps also are manufactured with a **ceramic body** and an integral reflector. Many different wattages are available with either UV transmitting or blocking windows. The reflector options are parabolic (for collimated light) or elliptical (for focused light). They are used in a wide variety of applications such as video projectors, fibre optic illuminators, and search lights. **Xenon long-arc** lamps are structurally similar to short-arc lamps except that the arc-containing portion of the glass tube is greatly elongated. When mounted within an elliptical reflector, these lamps are frequently used to simulate sunlight. Typical uses include solar cell testing, solar simulation for age testing of materials, rapid thermal processing, and material inspection.

Flash lamps are electric glow discharge lamps designed to produce extremely intense, incoherent, full-spectrum white light for very short durations. The lamp comprises a hermetically sealed glass tube (often made of fused quartz, borosilicate or Pyrex), which is filled with a noble gas, usually xenon, and electrodes to carry electrical current to the gas. When triggered, the rare-gas ionizes and conducts a high voltage pulse to produce the light. Additionally, a high voltage power source, usually a charged capacitor, is necessary to energize the gas so as to allow very speedy delivery of very high electrical current when the lamp is triggered. Flashtubes are used mostly for photographic purposes but are also employed in scientific, medical and industrial applications.

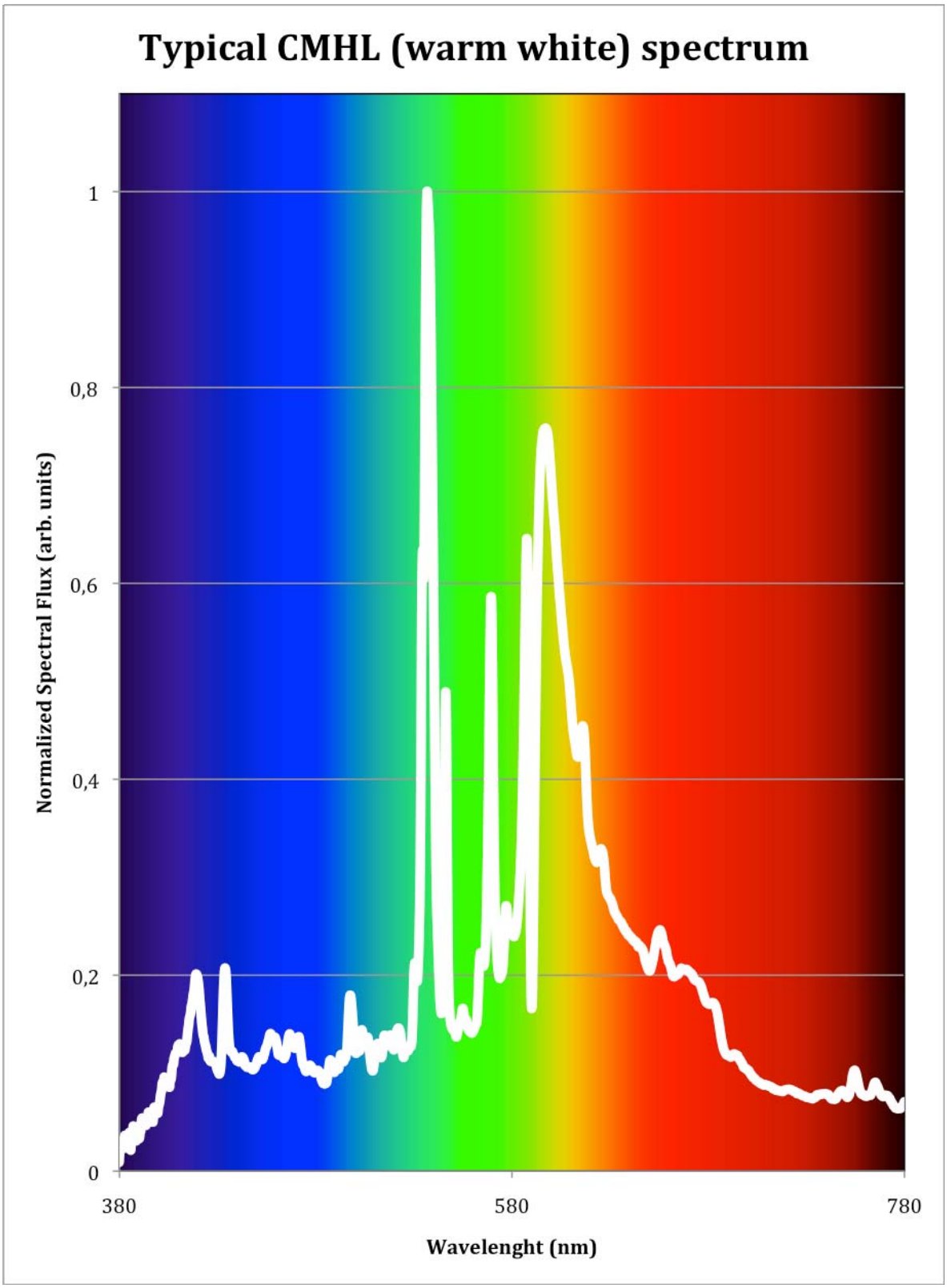

Figure A3 The spectral flux of a ceramic metal halide lamp measured in LAPLACE (EU NumeLiTe project; with permission from G. Zissis)

Dielectric-barrier discharge (DBD) is the electrical discharge between two electrodes separated by an insulating dielectric barrier. DBD devices can be made in many configurations, typically planar, using parallel plates separated by a dielectric, or

cylindrical tube, using coaxial plates with a dielectric tube between them. In a common coaxial configuration, the dielectric tube is shaped in the same form as common fluorescent tubing, filled at atmospheric pressure with either a rare gas or rare gas-halide mix, with the glass walls acting as the dielectric barrier. This type of discharge has some additional advantages compared to a low-pressure burner with electrodes: (a) the burner is free from any metallic part and this allows the use of other molecules that are incompatible with metals; (b) the discharge is established in high pressure but still in non-equilibrium conditions, and this allows the formation of excimer and exciplex molecules that cannot exist in classic conditions; and (c) any geometry is possible. In fact, dielectric barrier discharge (DBD) excimer or exciplex lamps are known as efficient UV and VUV sources with a narrow spectrum. This type of lamp is mainly limited today to industrial applications, but some years ago Osram presented a mercury-free flat lamp, known as Planon™, using Xe_2* excimer radiation at 147 nm. The concept is very interesting and proves that DBDs could be used for lighting applications. However, the Planon system design (including the electronic driver) is very complex and the luminous efficacy remains limited (27 lm/W). Similar configurations have also been demonstrated later with higher luminous efficacies but presenting severe light uniformity problems.

C. Solid state lighting

Currently the development and commercial introduction of a new kind of lighting technology is taking place: solid state lighting (SSL). In the long-term SSL, inorganic and organic light emitting diodes, could become the dominant type of light sources. Figure A4 illustrates synoptically the Solid State Lighiting technologies.

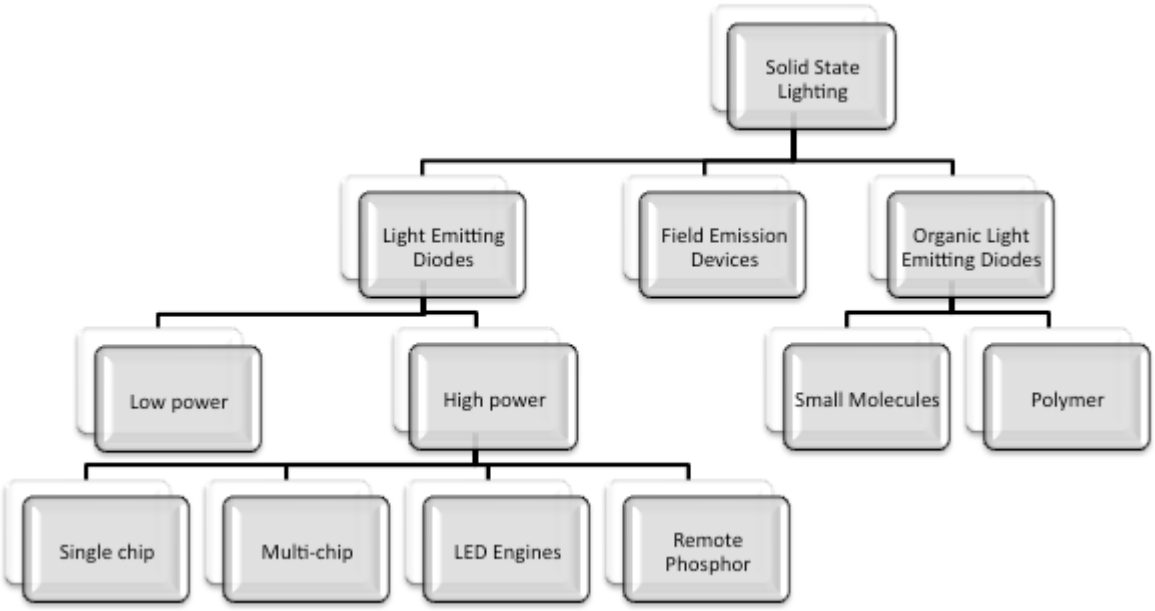

Figure A4 Solid State Lighting Devices product classes

Light emitting diodes (LEDs) generate light by recombination of charge carriers in a semiconductor diode. In light emitting diodes (LEDs), light is produced by a solid-state process called electroluminescence. One way to construct an LED is to deposit three semiconductor layers on a substrate. Between p-type and n-type semiconductor layers, an active region emits light when an electron and a hole recombine. Considering the p-n combination to be a diode, then when the diode is forward biased, holes from the p-type material and electrons from the n-type material are both driven into the active region. When holes meet electrons they can recombine, the energy can be converted into photons. This implies that the electron-hole pair drops into a more stable bound state,

releasing energy in the order of electron-volts by emission of a photon. The wavelength of the emitted photon is dependent on the energy band-gap of the semiconductor material. Thus, a LED is emitting a unique almost monochromatic radiation. For example, AlGaAs emits red light, AlGaInP – orange, AlGaP – green, GaN – blue, InGaN – UV. However, the dominant wavelength and the spectral line width depend on the junction operating temperature. The light conversion efficacy depends on several parameters and theoretically can attain 100% efficiency. However, efficacy is greatly reduced by increasing the junction temperature or the forward current. As no IR radiation is produced during the conversion, serious thermal management is necessary for LED devices in order to evacuate heat by the conduction-convection process. Light emission from any LED junction is directional, with the maximum emitted power in the direction perpendicular to the emitting surface. The typical radiation pattern shows that most of the energy is emitted within 20° of the direction of maximum light. Today LED packages used for lighting for LEDs include plastic lenses or more sophisticated primary optics directly implemented on the semiconductor (micro-lens, plasmons, micro-prisms, etc.) to spread the light over a greater angle of visibility. As mentioned earlier, LEDs produce coloured light; there are three primary ways of producing or obtaining white light needed for general lighting applications: one is to use individual LEDs that emit three primary colours (red, green, and blue) and then mix them to obtain white light. Drawbacks of this technique are low efficacy green LEDs, efficient colour mixing optics, poor colour rendering and coloured shadows. It is possible to mix several colours in order to obtain a better colour rendering. The second way is to mix two complementary colours such as blue and yellow. For this aim a yellow emitting phosphor material is used to convert monochromatic blue light from the LED junction and the two colours are mixed at the end. This is the most often used technique for high brightness LEDs, but negative aspects include important conversion losses, low colour rendering and unacceptably high colour temperature, as well as colour homogeneity and halo problems. It is possible to bypass some of the light quality problems by using additional phosphor layers that may add some red component in the spectrum (dual-phosphor technology). In recent LEDs an additional red emitting junction can be found which modifies the average colour temperature by shifting it towards warm white. The last way to obtain white light is to down-convert UV emission from the LED junction to visible light by using phosphors. This technique can produce good quality white light with excellent colour rendering and a large range of colour temperatures; however few semiconductors are able to emit radiation in the UV region with good efficiency. The few examples include zinc oxide (ZnO) that emits in UV region and gallium nitride (GaN) deposited by homoepitaxy on a GaN subtrate that emits mainly in the violet-near UV region. It is possible that in the near future emission efficiency from these materials will be significantly enhanced. This being the case, emissions in the UV region can no longer be neglected.

Today, for both coloured and white LEDs, two major product classes are commercially available: the **low power LEDs** that have the form of a dome and are sometimes called "5 mm LEDs". The luminous flux produced is very often limited to less than 5 lumens and the absorbed power is less than 1 W. They are mainly used for signage, decorative and festive lighting applications, and indicators. Some low quality "substitution lamps" use a large number of these LEDs clustered on a unique lamp for lighting applications. The **high power LEDs** consume between 1 W to 5 W power (for one chip), have different geometry and are able to produce a high amount of light from a unique package that may contain **single** or **multiple-chips** (1200 lm is the record for white light). **Light engines** cluster several chips under the same phosphor and they can achieve higher luminous fluxes. High Power LEDs are used mainly for lighting applications (emergency, scenic, general lighting, etc.). For commercial white LEDs (high power, "cold white") the luminous efficacy can attain 116 lm/W, and the correlated colour temperature ranges from 2,800 K to more than 12,000 K. The colour rendering ranges from poor to excellent. The nominal lifespan is in the order of 50,000 h for the LED itself, but for a LED lamp or LED system this lifespan is very often limited to 15,000 h – 25,000 h. It should be noted here that nominal values are given for 25°C junction temperature and nominal forward current, as the colour rendering index definitions are not really applicable to LEDs. LEDs

operate under a DC current power supply and require only a few volts (usually less than 10 V). LED devices are fully dimmable and two techniques are used: current aptitude modulation and power wave modulation (PWM). The first is more expensive but more robust; the second is easier to manage but may lead to some undesirable effects such as light flicker.

Figure A5 shows the spectral flux obtained experimentally from three LED light sources[6]. In order to make the values comparable, we have normalized them by dividing all of them by their respective maximal values. The LED lamps have been selected directly for the market. The "cold white" corresponds to a correlated colour temperature of 12,100 K, the "neutral white" to a correlated colour temperature of 5,700 K and the "warm white" to 2,900 K.[7] It should be noted here that cold and neutral white use LEDs with a single phosphor whereas warm white uses dual-phosphor technology for improved colour rendering (this explains the difference in the spectral shape).

Organic light emitting diodes (OLED) pioneered and patented by Kodak/Sanyo, enable full colour, full-motion flat panel displays with a level of brightness and sharpness not possible with other technologies. An OLED consists of an emissive organic material, supplied with an electrical current. In fact, an OLED is a light-emitting diode (LED) in which the emissive electroluminescent layer is a film of organic compounds, which emit light in response to an electric current. This layer of organic semiconductor material is situated between two electrodes. Generally, at least one of these electrodes is transparent. The colour rendering is high and the colour is fully tuneable. This type of light source is flat, thin and light, and it has a large etendue. The last implies a low brightness, which means low glare induced by this type of lamp. Today two technologies exist; the small molecules and the plastic (polymer) OLEDs. The technology is in its early stages with many remaining problems to solve. However, since 2009 OLED products for indoor and residential lighting applications have also been marketed.

Field emission devices (FED) or field emission lamps are based on the same principle as the luminescent phosphor materials used in TV sets. Light is emitted when electrons are driven into the material. Traditional TV sets use a thermal electron gun to fire electrons into a phosphor screen. The new field emission devices use a powerful electric field to extract electrons from the cathode and drive them into the phosphor, which are located close together. The process is dramatically more efficient than the filaments used in electron guns. Field emission lamps could match exactly the spectrum of natural daylight. They are up to five times more energy efficient than existing lamps and do not contain environmentally hazardous materials, such as the mercury vapour used in fluorescent tubes. Lamps based on the material should have a lifetime of up to 30,000 hours. Today this technology is still at an experimental stage and very few devices are commercially available. A use for projectors and indoor lighting applications is expected.

[6] Data obtained by CTSB laboratory in the frame of ADEME-CITADEL project and reproduced here with the approval of CITADEL consortium.

[7] Experimentally determined colour temperatures.

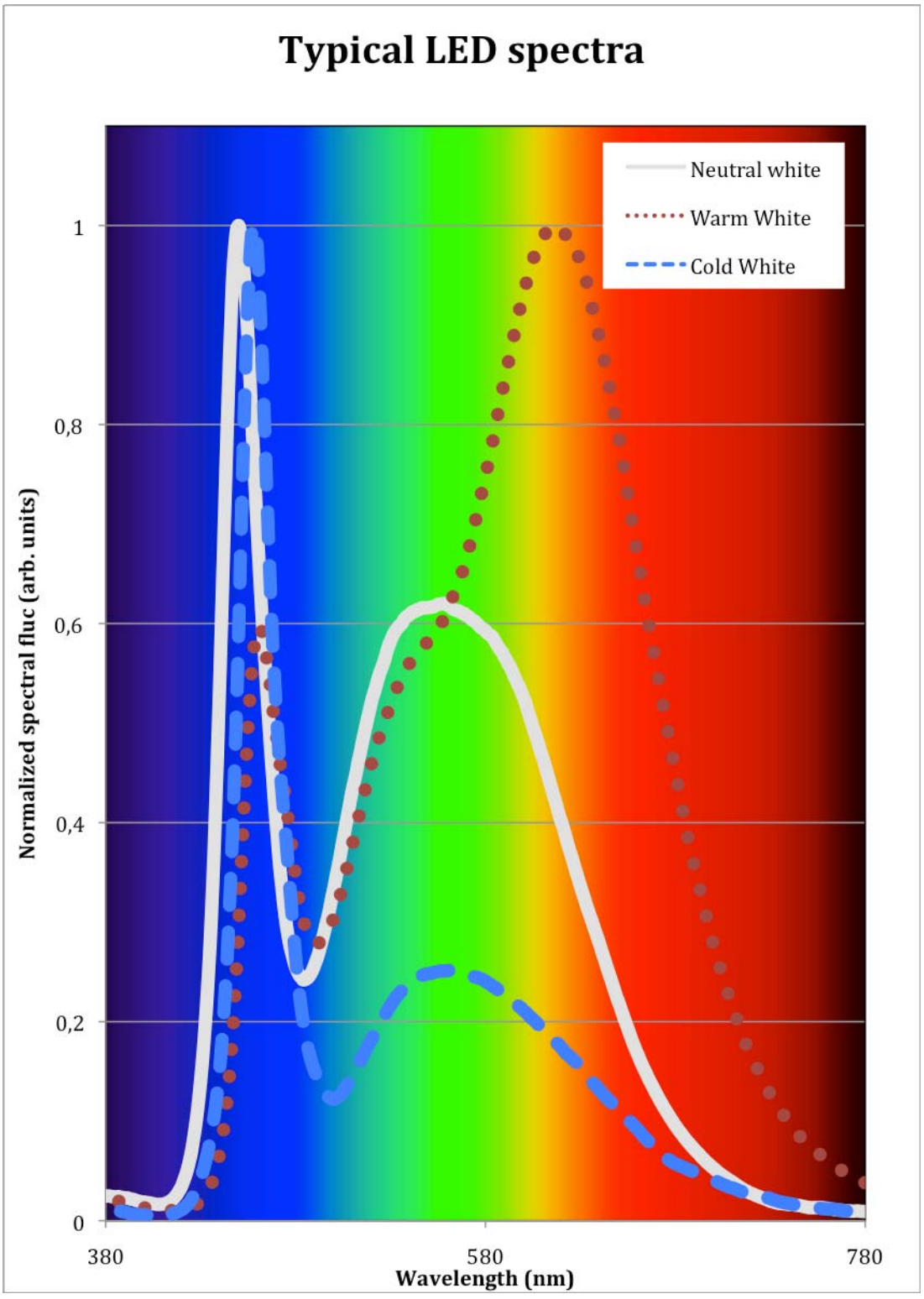

Figure A5 The spectral flux of three different types of LEDs (Measurement data provided by G. Zissis)