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Bright light therapy in focus: lamp emission spectra and ocular safety

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Abstract. In recent years, bright light treatment of seasonal affective disorder (SAD), recurrent depressions in fall and winter, has been discovered. Newer applications include circadian sleep phase disorder, shift work and jet lag. Apart from creating the visual signal, light can modify retinal structure and physiology. UV and visible light lead to distinct lesions of ocular tissues under certain experimental und naturalistic conditions. In light therapy, a large variety of fixtures is used but the spectral emission of lamps is mostly unknown to the user and clinician leading to the potential hazard of ocular lesions. Therefore, we have analyzed a wide selection of light sources commonly used for treatment. We measured the spectral emission and calculated irradiant doses for several light therapy regimens. Based on these measurements, potential hazards are analyzed, physiological mechanisms of light action are discussed and safety measures for bright light therapy are proposed. They include recommendations for lamps devoid of damaging spectral emissions and standardized therapy fixtures, ophthalmological monitoring of patients with eye diseases and control by optometrists for patients with healthy eyes who are likely to undergo light treatment for extended periods.

1. Introduction

Recent years have seen the discovery and widespread application of bright light therapy for seasonal affective disorder (SAD), a syndrome marked by recurrent depression in fall and winter [1]. Newer applications of bright light include treatment of circadian sleep phase disorder [2] and countermeasures for shift work maladaptation [3] and jet lag [4]. Most research has concentrated on SAD, in which exposure to artificial bright light may be scheduled daily for up to half the year for many years. Patients with sleep phase disorders might use the method continuously, without an annual break. By

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contrast, shift workers and air travellers would receive only intermittent treatment, albeit for up to weeks at a time.

Light exposure primarily creates the signal for vision and circadian synchronization in the eye. It also exerts, however, non-visual effects by modifying retinal structure and physiology. Retinal stimulation directly inputs the rhythm-generating master clock of the central nervous system (CNS), in the hypothalamic suprachiasmatic nuclei. Light may thus alter the input stage of the circadian system [5]. Light exposure can also lead to distinct lesions of ocular structures under specific experimental and naturalistic conditions.

A large variety of lighting fixtures is used in light therapy. The spectral emission of light sources and the transmission characteristics of diffusing screens are mostly unknown to the user and clinician and can vary considerably. Apart from the effects on circadian timing, the underlying mechanisms of light treatment are also largely unknown.

In the present study we have analyzed a wide selection of light sources and diffusing screens commonly used for light treatment. We measured spectral emission and screen transmission and calculated irradiant doses for several light therapy regimens. Based on these measurements the potential hazards of extensive exposure to bright light are outlined. Furthermore, physiological mechanisms of light action in the context of chronobiology are discussed.

2. Background: effects of ultraviolet and visible light on the eye

Only an absorbed photon can trigger a photochemical reaction in the absorbing molecule with subsequent responses in cells and tissues. The eye contains a variety of chromophores that could potentially initiate photochemical reactions with ensuing tissue damage. Examples include the visual pigments in rods (absorption peak: 500 nm) and cones (absorption peaks: 426 nm, 535 nm, 575 nm), the mitochondrial cytochromes in photoreceptors and retinal pigment epithelium, the age pigment lipofuscin in the retinal pigment epithelium, the accumulating pigments of ageing lenses and derivatives of the aminoacid tryptophan in younger lenses.

There is widespread evidence for acute and chronic damage to ocular tissues by both UV and visible light [6–8]. Although there are few unequivocal action spectra available for the human eye, photochemical, photophysical and animal studies have revealed action spectra that suggest basic damage mechanisms. Exposure regimens, intensity and spectral range determine the extent of lesions.

Given the likelihood that patients will use bright light therapy for decades, two types of potential lesions must be considered: acute lesions and chronic cumulative damage. The cornea and conjunctiva are the primary loci of UV-induced acute lesions such as the keratoconjunctivitis in snow blindness or after exposure to welding arcs. A number of studies report acute and subacute light damage to the human retina [7].

Chronic degenerative diseases linked to UV-exposure include climatic droplet keratopathy in the cornea, the pterygium and pinguecula of the conjunctiva, and cataracts of the lens [9]. Light exposure might accelerate ageing processes in the retina or potentiate age-related macular degeneration [9]. A crucial factor that influences ocular irradiant dose is the individual exposure geometry. Disparate results of epidemiological studies may be attributed to inadequate specification of individual's exposure conditions [10].

Therefore, in order to clarify exposure conditions, future study standards promulgated by the World Health Organization [11] require ascertainment of lifetime light exposure history and dosimetry for every subject. As those measurements are not yet available, the current epidemiological evidence for

radiation induced ocular changes is controversial [12]. However, the potential for UV – and light induced lesions in the human eye cannot be ignored. The epidemiological evidence is only one albeit important factor in the range of theoretical, experimental and clinical considerations which support the inherent danger of photochemical lesions in the human eye [7].

Several factors increase light induced lesions. In particular, certain drugs and other compounds cause photosensitization of ocular tissues. They show absorption in the near UV and visible ranges, pass the blood-ocular interfaces and may bind to ocular tissues. The chemical structures include tricyclic-heterocyclic, amphiphilic-lipophilic and porphyrin ring systems [13,14]. Exogenous melatonin has been administered at specific times of day to assist circadian phase adjustment [2,15] and it will likely be used in conjunction with light treatment. This hormone is widely investigated in chronobiology [16] and is thought to provide a physiological "dark signal". It may increase retinal sensitivity by suppressing light adaptive retinal dopamine [17]. Indeed, melatonin administration in animals increases susceptibility to light induced retinal lesions [18,19]. Under supervised clinical use the timing of melatonin and bright light would be strictly separated, because their phase-response curves bear an approximate inverse relation. However, if the drug were taken at high dose or concurrently with bright light exposure there would be a potential retinal hazard. Non-prescribed use of the hormone with inadequate instructions is currently widespread in the United States.

3. Materials and methods

Fluorescent tubes frequently used in light treatment were selected for the measurement of their spectral emission: Osram Lumilux deluxe L36/12, Osram Biolux L26/72, Philips TLD36/95, Philips TLD 36/96, Duro-Test Truelite 40 TH 12, Duro-Test Truelite Plus 40 TH 10, Duro-Test Color Gard 50 (40 W), Duro-Test Color Gard 50 (65 W).

The spectral irradiances were established using two monochromators (concave grating with a 1 m focal length, 1 nm resolution), one for the UV and one for the visible range. The output signals were measured with either a UV-photomultiplier or a CCD array. The tubes were located at 70 cm distance from the entrance slit and were allowed to warm up for 30 minutes. Three measurements per tube were performed and the mean of those was taken. Stray light was minimized by means of external baffles. The irradiances were compared to those obtained with a calibrated 800 W halogen incandescent bulb driven at 230 V. Absolute irradiances were derived from a photometric spectral integration compared to the value obtained with a calibrated luxmeter (LMT, Germany).

The plastic diffusing screen materials mainly applied for light therapy fixtures were selected for the measurement of their diffuse transmission using a dual beam spectrophotometer fitted with an integrating sphere (Perkin-Elmer, Germany): Standart white diffusor, white wafer cloth of 0.11 mm, Virgin Acrylic (smooth white flat sheet), Lexan of 0.66 mm, Virgin Acrylic (prismatic conical prisms), plexiglas 233 (transparent colorless, 3.0 mm), plexiglas 060 (translucent, 0.33 mm), plexiglas 010 (medium white, 0.3 mm), Acrylite OP-3 (transparent), See-More (transparent yellow).

4. Results and discussion

4.1. Emission spectra

In general, a quarter to a third of the lamp spectral power is provided by UV and blue light, which has greater photon energy than the longer wavelengths. In sunlight, the corresponding proportion is

1.5% UVB and 6.3% UVA, whereas 91.7% of spectral power derives from visible light and IR. The proportion of UV and blue in sunlight impinging on the human eye, however, varies greatly with time of day and environmental conditions including grass or trees and reflecting surfaces such as snow or cement. Furthermore, protective behavioral factors such as aversion and squinting strongly modify the exposure of the eyes to natural sunlight in an outdoor environment [10,20]. Thus, there is no simple correspondence to the conditions of artificial bright light therapy despite the fact that light levels used for therapy are below or within the illuminance range encountered on a bright sunny day outdoors.

When irradiance levels are measured from single fluorescent bulbs in 1 nm steps at 1 m distance contrasting emission spectra are obtained with prominent variations in the short-wavelength ranges considered to be potentially dangerous (Fig. 1). Out of nine lamps analyzed, seven showed distinct patterns of UVA and UVB emission (Fig. 2). Most of the bulbs also showed a strong component of blue and green light (Fig. 1, Table 1), a wavelength range that has caused damage to photoreceptors and the pigment epithelium in laboratory studies [6]. Bulb no. 9 showed minimal levels of UV emission and moderate levels of blue and green light. Furthermore, the spectral pattern was relatively evenly

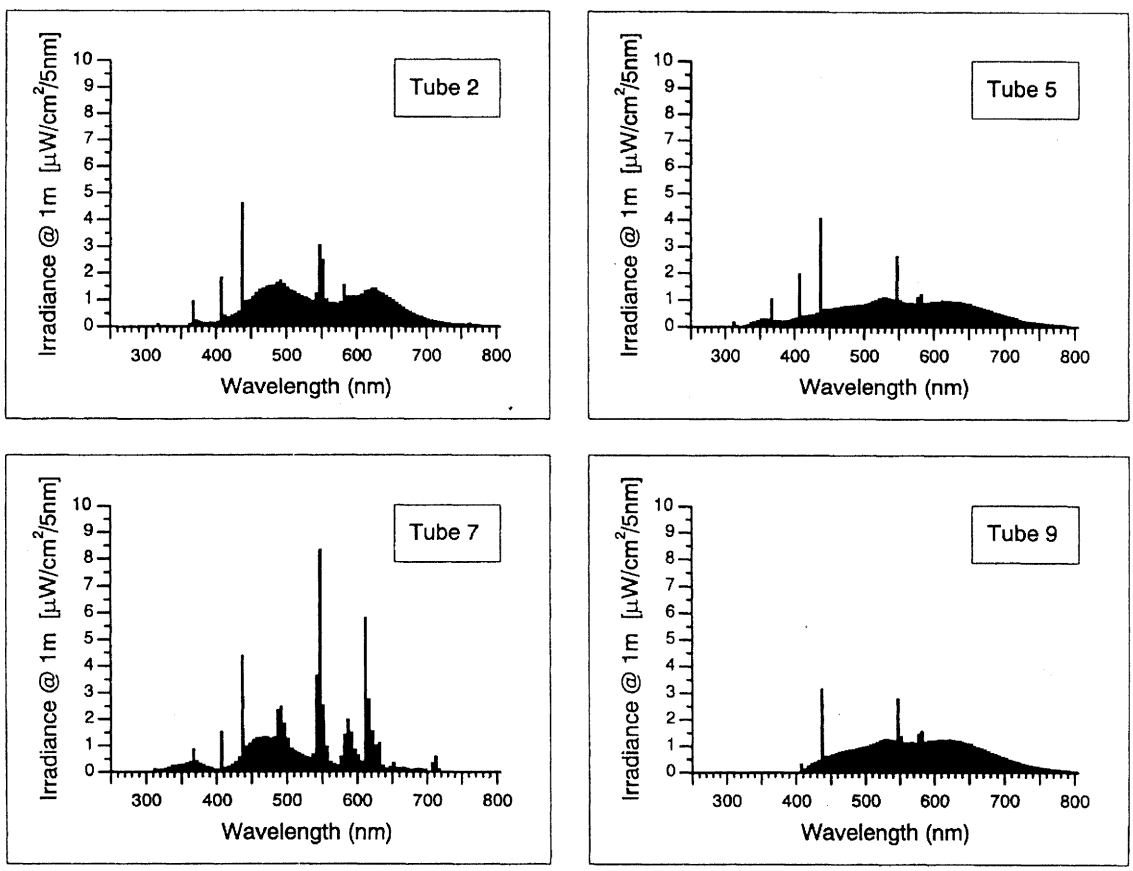

Fig. 1. Emission spectra from fluorescent lamps frequently used in light therapy boxes. The emission was measured with a spectroradiometer in 1 nm steps at 1 m distance from the bulb and is expressed as microwatts per square centimeter per 5 nm. The spectra show different degrees of UV emission and various peaks over the range of the visible spectrum. Bulb no. 9 shows a fairly even distribution of spectral ranges and no UV emission. Tube 2: Osram Biolux L 36/72, Tube 5: Duro-Test Truelite 40 TH 12, Tube 7: Duro-Test Truelite 40 TH 10, Tube 9: Duro-Test Color Gard 50 (65 W).

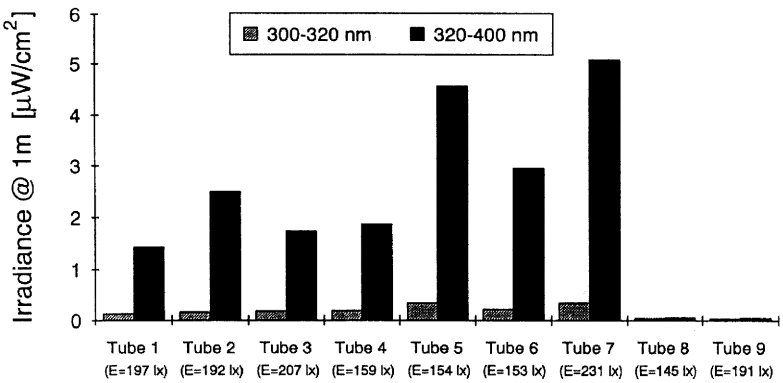

Fig. 2. Emission of UVB and UVA by fluorescent lamps frequently used in light therapy boxes. Measurements were made with a spectroradiometer in 1 nm steps at 1 m distance from the bulbs. The emission of UVB from 300–320 nm and the emission of UVA are shown. Bulb nos. 5 and 7 show the highest values for UVA and UVB, while bulb nos. 8 and 9 emit practically no UV. Tube 1: Osram Lumilux deluxe L 36/12, Tube 2: Osram Biolux L 36/72, Tube 3: Philips TLD 36/95, Tube 4: Philips TLD 36/96, Tube 5: Duro-Test Truelite 40 TH 12, Tube 6: Duro-Test Truelite II 40 TH 10, Tube 7: Duro-Test Truelite Plus 40 TH 10, Tube 8: Duro-Test Color Gard 50 (40 W), Tube 9: Duro-Test Color Gard 50 (65 W).

Table 1
Cumulative doses for two contrasting lamp types for typical regimens in SAD treatment and shift-work phase adjustment

Winter depression (SAD) 10,000 lx, 0.5 h/session		Cumulative doses in J/cm²			
		per day 1 session/day	per month 30 days/month	per 5 months 30 days/month	per 10 years 5 months/year
Tube 5	UVB (300–320 nm)	0.06	1.18	5.88	58.76
	UVA (320–400 nm)	0.80	16.02	80.09	800.86
	Blue (400–470 nm)	2.30	46.07	230.34	2303.41
Tube 9	UVB (300–320 nm)	0.005	0.10	0.48	4.82
	UVA (320–400 nm)	0.006	0.13	0.63	6.35
	Blue (400–470 nm)	1.372	27.45	137.24	1372.45
Shift work phase-adjustment 5000 lx, 3 h/session		Cumulative doses in J/cm²			
		per day 1 session/day	per month 5 days/month	per year 12 months/year	per 10 years 5 days/month
Tube 5	UVB (300–320 nm)	0.12	0.59	7.05	70.51
	UVA (320–400 nm)	1.60	8.01	96.10	961.03
	Blue (400–470 nm)	4.61	23.03	276.41	2764.09
Tube 9	UVB (300–320 nm)	0.010	0.05	0.58	5.78
	UVA (320–400 nm)	0.013	0.06	0.76	7.62
	Blue (400–470 nm)	2.745	13.72	164.69	1646.94

balanced across the broad range of visible wavelengths (Fig. 1), a fact that would increase subjective comfort at high intensity. Therefore, this lamp type may be preferable for therapeutic applications.

The present spectra and irradiance levels were derived from measurements of single fluorescent bulbs without any interposed filters or reflectors. The number of lamps in a light box and the characteristics of rear-reflectors and plastic diffusing screens all contribute to the irradiance level and the spectral composition of the emitted light. Therefore, we also measured the transmission characteristics of several common plastic diffusing screens, which varied greatly (Fig. 3).

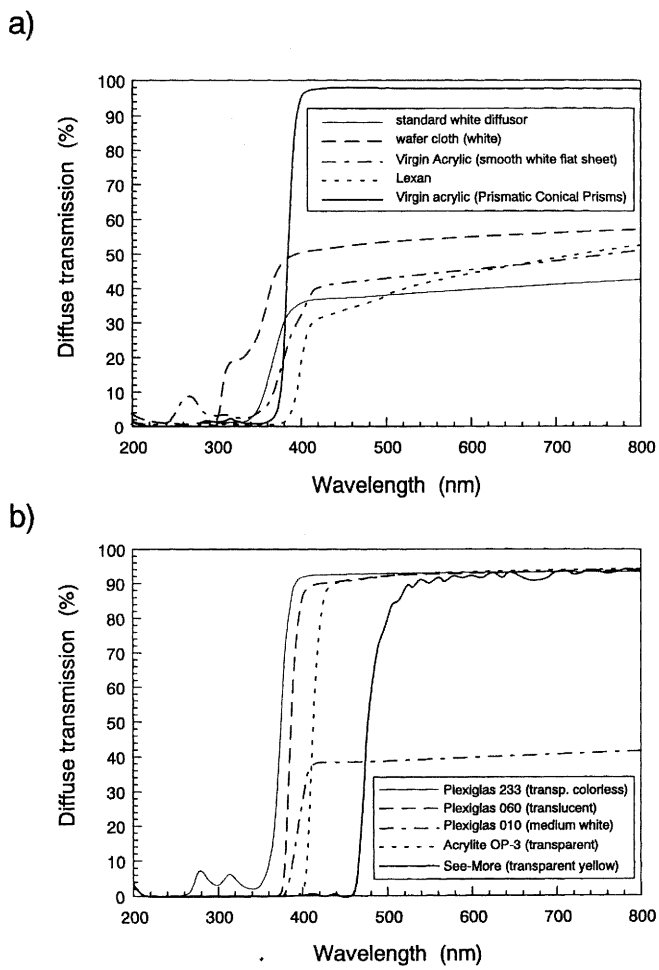

Fig. 3. Spectral transmission curves of selected plastic (a) and plexiglas (b) diffusing screen materials used in light therapy boxes. The transmission was measured in individual samples with a spectrophotometer in 1 nm steps and expressed as percent transmission. Distinct differences in the transmission of UVA and blue light is apparent, which underscores the importance of a standardization not only of fluorescent lamps but also the interposed screens. "See-more" represents an ideal transmission property for ocular safety.

4.2. *Dosing and potential hazards*

The radiometric measurements have to be translated into the therapeutic situation, where a patient is supposed to receive about 2500 lux or 10,000 lux, respectively, at eye level. We have calculated the approximate irradiant doses for UVB, UVA and blue light for different therapeutic regimens over a wide range of treatment durations for two contrasting bulbs, one of the recommended type and another that showed unfavorable emission characteristics (Table 1). Bulb no. 5 shows a high UVA emission with 4.6 $\mu W/cm^2$. With treatment at 10,000 lux, the corresponding irradiance would be approximately 220 $\mu W/cm^2$. A treatment session of 30 minutes using bulb no. 5 would result in a UVA exposure of about 0.8 J/cm^2; with 30 sessions per month the cumulative dose would be 16.0 J/cm^2. A treatment period of five months per year over 10 years would yield a cumulative dose of 800.9 J/cm^2. By comparison, bulb no. 9, with its strict cut-off near 400 nm, would yield a cumulative

dose of 6.35 J/cm^2. These values assume that the patient's head position is 1 m from the lamps. Closer distance would increase the irradiant dose: at 30 cm, the distance commonly employed for 10,000 lux treatment, the dose would be approximately ten times higher. These calculations demonstrate that over extended treatment periods the potential for UV-induced ocular lesions cannot be disregarded. For example, calculations were made for SAD-treatment of 2500 lux for 2–6 hrs daily during the winter months resulting in the equivalent of a daily erythemally effective radiant exposure of 40 J/m^2 [21].

Animal data support the conclusion that there is a potential for UV- and light induced ocular lesions: exposure to intermittent light is far more harmful than the same illuminance received in a single dose [22]. Other studies have shown funduscopically visible threshold lesions in the retina of pigmented rats with dilated pupils, using a spectral range of 320–440 nm (UVA and blue) with an irradiant dose of 0.3–100 J/cm^2 for 10–160 min exposures [23]. In terms of cumulative doses, several therapeutic regimens fall into this range (Table 1). Even though the damage threshold may well be lower in laboratory animals, the data underscore the importance of reducing the UV- and blue light emission in therapeutic lamps, especially for young patients with UV- and blue transmitting lenses. For diffusing screens placed in front of the fluorescent lamps, several types provide good protection against UV (Fig. 3). Therefore, light boxes and other light therapy fixtures should not be used without appropriate diffusing screens and standards for therapy lamps and diffusing screens should be mandatory.

A satisfactory therapy regimen would thus include lamps with negligible UV emission, diffusing screens which block UV and reduce blue light, and a careful control of patients potentially susceptible to light induced lesions. Worst case situations would include UV-emitting lamps with insufficient absorption of UV and blue light by the diffusing screen or even the lack of a diffusing screen with therapy applied over extended time periods. Patients taking photosensitizing medication and/or suffering from preexisting ocular disease would be in considerable danger if exposed to UV-emitting lamps with insufficient or absent diffusing screens. In general, because scattering is higher in the UV range than in the visible range of the spectrum, the use of a diffusing screen reduces the risk of radiation hazard. On the other hand, there is a drastic increase of the UV dose with decreasing distance from the screen and light source.

4.3. Current safety regulations

Threshold limit values for light exposure have been established for several exposure conditions [24]. It is doubtful, however, that they apply to bright light therapy. Safety standards have been derived mainly from exposure to laser light. There are hardly any safety regulations for conventional light sources, including broad-band lamps [25]. Light therapy involves diffuse white light and may be used for many years. Cumulative subthreshold changes might eventually lead to manifest lesions that cannot be correlated directly with data for acute exposures or extrapolated threshold limit values.

Clinical guidelines recently issued by the United States Public Health Service Agency for Health Care Policy and Research recommended the use of light therapy in primary care practice for treatment of SAD, but noted that individual consultation would be advisable to determine specific risks (p. 102). A consensus statement of the Society for Light Treatment and Biological Rhythms also outlined certain safety principles [26].

The need for ophthalmological screening of patients prior to beginning bright light therapy, or monitoring during treatment, has been debated even in cases of preexisting disease [27–29]. Discussion of these issues, which has thus far occurred primarily within the mental health community, often has not been fully informative. Furthermore, it has not yet been evaluated how many patients suffering from SAD and preexisting eye disease were treated with bright light.

4.4. Lamp configuration

Bright light treatment most often uses fluorescent bulbs mounted in boxes that vary in size with illuminance sometimes boosted by rear-mounted reflectors. Lamps used for bright light therapy have also included bulbs with specialized phosphors which alter spectral emission (e.g., full spectrum, triphosphor, warm white, narrow-band green). At least one manufacturer has added a UV-emitter to the apparatus on the basis of a presumed clinical efficacy. Because the cornea and lens absorb most of the UV impinging on the eye, the retina would not receive such radiation and the anterior eye would be in potential danger of chronic lesions. Whole-room illumination systems with banks of fluorescent bulbs mounted on the ceiling or wall have also been used for inpatient treatment of SAD and to facilitate shift work rotations. Other apparatus has used incandescent filament lamps, both standard and halogen, including one design which mounts small krypton halogen lamps in a head holder beaming light at the eye at very short distance [30].

Exposure parameters include the distance of the eyes from the light box, usually between 0.3–1.0 m. For SAD and sleep phase treatment, illuminance levels are usually 10,000 lux for 30–45 min or 2500 lux for 1–4 hrs, in single or dual daily sessions. Some patients adjust illuminance level and exposure duration according to self-perceived needs, often with great incremental dosage [31]. In shift work applications, exposures have been as high as 12,000 lux for 8 hr in a series of 5 daily treatments [3].

Retinal irradiance levels can be calculated if the corneal irradiance, the average pupil diameter during treatment sessions and the axial length of the eye are known [8,32]. Several other ocular factors influence retinal irradiance levels, most importantly the transmission of the ocular media, the mobility of the eye and the retinal magnification factor. Transmission is highest in young eyes, with a gradual decline – especially for shorter wavelengths – in older individuals. Additionally, the patient's distance from the light source and the exposure geometry significantly determine the illuminance level at the eyes [10].

A particular situation is given with the use of the light visor [30]. The variability of illuminance levels at the eyes may be lesser than with other light therapy devices because head tilt, head aversion or eye- and body movements will not reduce the light levels. The average UV-emission of those light sources is low, amounting to 10–100 $\mu W/m^2$ with an increase in the blue-green range and a plateau in the near UV.

4.5. Light-CNS interactions

Apart from local effects on the eye, light exposure may elicit systemic responses mediated by an eye–CNS interaction. Immune responses can show circadian rhythmicity and photoperiod dependency. It is suggested that light may also modulate the immune system via hypothalamic pathways other than CNS-mediated [33]. Visible light treatment for SAD increased the number of peripheral T-4 and T-8 cells, a similar response was observed in the summer season [34,35].

On the basis of one small sample study, which compared the clinical response of patients with SAD to light delivered solely to the eyes or to the skin, it has been assumed that the route of photic input is ocular [36]. UV light was not essential to the antidepressant effect in a study that filtered out UVA with eyeglasses [37]. However, potential contributions of skin-mediated photic responses cannot yet be ruled out. A woman totally blind since she was one year old responded to bright light but not to dim light with alleviation of her winter depression [38]. Apart from UV exposure bright visible light may elicit a skin-mediated release of cytokines which could conceivably directly or indirectly promote

an antidepressant response. Epidermal cells can synthesize and release a variety of mediators such as interleukins or arachidonic acid and its metabolites upon UV exposure [39]. Interleukin 1 can stimulate hypothalamic cells to release sleep inducing hormones [40]. We cannot exclude the possibility that intense visible light exposure has similar effects and interacts with hypothalamic neuroendocrine functions. The secretion of nocturnal melatonin is thought to be suppressed by exposure to light mediated through the eyes [41]. However, a recent study revealed a similar effect in a small group of blind persons [42].

It is currently unclear which receptors transmit eye-mediated non-visual responses. Earlier studies in retinally degenerate rodents demonstrated circadian light responses [43] tentatively suggesting a circadian photoreceptive system apart from that mediating vision. Recent studies, however, show that even in advanced retinal degenerations there are remaining cone cells that could subserve the function of light transduction to the circadian system [44].

5. Conclusion

We propose to develop lamp and treatment standards not only to facilitate controlled studies but also to prevent overexposure to damaging radiation by uncontrolled spectral characteristics of lamps. This can be accomplished by modification of the spectral output of the lamps and use of a plastic diffusing screen, or untinted eyeglasses with UV blockers, blue light attenuation and side shields. Furthermore we propose the identification of patients at risk as determined by ophthalmological consultation, because eyes affected by a preexisting disease may be more susceptible than healthy ones to light induced changes. Finally, an optometric consultation should be sought for patients likely to use light treatment for extended periods in their lives.

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