

BLUE LIGHT RISK AND LIGHT THERAPY

David H. Slaney

US Army Center for Health Promotion and Preventive Medicine

Aberdeen Proving Ground, MD, USA

ABSTRACT

Light therapy for the treatment of sleep disorders and jet-lag, as well as for seasonal affective disorder (SAD) has been shown to be effective for many subjects. However, concerns are sometimes expressed about the safety of viewing relatively bright light boxes used in this type of light phototherapy treatment. Typically, a patient is illuminated—normally not on-axis—by an illuminance of approximately 2,500 lux to 10,000 lux as measured at the corneal plane of the eye for periods ranging up to two hours, or even longer each day. Normally there is little ultraviolet radiation from either fluorescent or LED-array light sources. The greatest concerns have centered on the potential retinal hazard from the “blue-light hazard,” as described in photobiological lamp safety standards. Currently evaluated, diffused sources do not pose a hazard.

Keywords: phototherapy, light therapy, seasonal affective disorder, ocular hazards, eye safety

INTRODUCTION

Light therapy for seasonal affective disorder (SAD) and for treatment of jet-lag and sleep disorders has been shown to be effective for many subjects; however, some users occasionally ask the question: “But is it safe?”[1,2] Typical treatments incorporate the patient being illuminated to a level of 2,500 lux to 10,000 lux measured at the eye for periods ranging up to two, or even four, hours a day. The illumination may come from a light box or a head-mounted light source.[2] Normally there is little ultraviolet radiation from either fluorescent or LED-array light sources. The greatest concerns have centered on what has been referred to as the “blue-light hazard,” as described in lamp safety standards. This has become all the more important with the elucidation of the action spectrum of melatonin suppression which is in the blue part of the visible spectrum [3].

Human exposure limits exist for the entire optical radiation spectrum [4-8]. These employ CIE standardized radiometric quantities, terms and units [9] with the addition of spectral weighting factors.[10-11] Since retinal irradiance is directly proportional to the radiance of any light source being viewed, The radiometric quantity radiance is used for retinal safety standards whenever it is necessary to answer the question of how safe it is to stare at a bright light. Lamp safety standards exist, such as the CIE Standard S009/E-2002, “Photobiological Safety of Lamps and Lamp Systems,” [10] and these are based upon exposure limits for viewing intense optical sources from the ACGIH (American Conference of Governmental Industrial Hygienists) and from the ICNIRP (International Commission on Non-Ionizing Radiation Protection), which in term are based upon general health criteria documents such as those by the World Health Organisation.^{6,7,10-11} There are seven different optical hazards from the ultraviolet to infrared and each is limited to certain CIE photobiological spectral bands.⁹ Each is separately assessed in the CIE lamp safety standard, and for the most part ultraviolet and blue-light hazards have been the most often cited as potentially limiting the unrestricted use of lamps by the lamp safety standard.¹¹⁻¹⁵

The guidelines for human exposure to optical radiation are based upon laboratory studies of thresholds for acute effects upon the eye and skin, but also take into account the known hazards from chronic exposure. With regard to chronic light exposure of the retina, the ICNIRP and ACGIH exposure guidelines were based not just upon laboratory studies of acute exposure of periods of seconds, minutes or hours—or even days, but also were based upon epidemiological studies and dosimetric studies of retinal light exposure in different environments. Artificial lighting can hardly

compete with exposure from outdoor sunlight! For each hazard to be assessed, the photobiological action spectrum has to be known, and most of these have become standardized. The chromophore, the target molecule that undergoes a chemical change when a photon is absorbed, in many human tissue responses is important to know, but unfortunately, several are either unknown or under debate. DNA is a key target chromophore for delayed effects from ultraviolet radiation. For photochemical damage of ocular tissue, photometric quantities [i.e., the spectral weighting of visible light to obtain quantities such as luminous power (unit: lumen) or illuminance (unit: lux)] are not very helpful for optical hazard assessment, as the quantities do not predict the relative hazard or effectiveness.¹⁴ Photobiological hazards depend not on just wavelength, but also exposure duration, source radiance and exposure geometry.¹⁴⁻¹⁵ Thermal and photochemical mechanisms require different hazard assessments. Only pulsed lamps are of a concern for thermal effects and thermal effects are not of concern during light therapy.

has not been completely developed. The current clinical convention has been to describe illuminance in lux (lx) at the patient's eyes and the exposure duration.¹⁻³ This approach has underlying assumptions that the action spectrum for this effect is that of photopic (daylight, cone) vision and that illumination at the patient's face adequately describes the total retinal exposure. Both assumptions have been shown to be incorrect and a more rigorous analysis of exposure dose requires use of the melatonin-suppression action spectrum or other effectiveness curve. One could quip that the state of dosimetry for SAD treatment is in a sad state. Retinal exposure depends on source brightness,^[12-13] and corneal illuminance in lux does not predict instantaneous retinal illumination, although it can be argued that illuminance can be indicative of an averaged retinal exposure for small sources. Since retinal irradiance is directly proportional to source radiance, it may be more appropriate to calculate a retinal effective dose from the source radiance. On the other hand, if the subject constantly moves his or her fixation during the therapy, and the source is relatively small, an irradiance may still be the most appropriate. For certain, corneal illuminance is completely inappropriate to predict a potential light hazard.¹²⁻¹³

To calculate retinal exposure, as the retinal irradiance is directly proportional to the radiance (brightness) of the source being viewed, the retinal irradiance E_r in $W \cdot cm^{-2}$ is:

$$E_r = 0.27 L \cdot \tau \cdot d_e^2 \quad [1]$$

where L is the radiance in $W \cdot cm^{-2} \cdot sr^{-1}$ and τ is the transmittance of the ocular media. It might be noted that the definition of the quantity of retinal illumination as expressed in Trolands (td) is just the luminance L ($cd \cdot m^{-2}$) multiplied by the square of the pupil diameter (in mm), thus employing an engineering form for Equation 1 for photometric quantities. As an example of typical retinal illumination, the ambient outdoor illumination of the retina is of the order of 0.02-0.1 mW/cm^2 and these levels are just comfortable to view. The retinal illuminance outdoors is $\sim 5 \times 10^5$ td. The retinal irradiance in the sun's image is a million times greater. Our measurements of sky brightness (luminance) and retinal

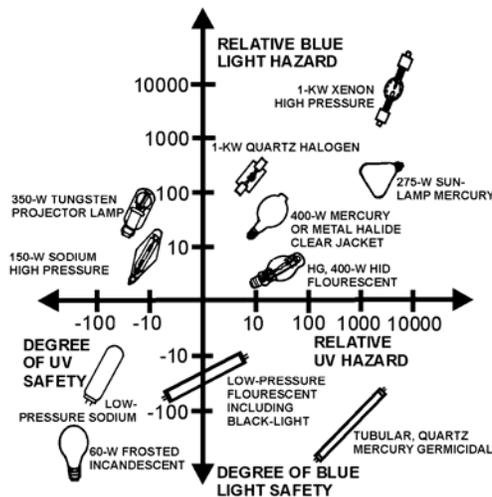


Figure 1. The relative risk of staring at different lamps. In both instances the duration for safe exposure depends inversely on the relative risk.

An added complication in describing potential hazard levels to light-phototherapy patients has been that the dosimetry to quantify the appropriate light exposure dose

illumination show that these vary greatly early in the morning at about 07:30 for different seasons if looking out the window at breakfast at my home at latitude 40 N. The light levels in the summer are ten times brighter in the summer than in winter at that breakfast hour.^{14,16} The sky was simply not bright enough in winter to provide for SAD phototherapy. Measurements of skylight luminance reported at this meeting by Wengraitis were far more similar at midday across the seasons; thus, the period of high luminance exists for a longer duration in spring and summer months.

Measurements of most phototherapy light sources show no potential optical radiation hazard. Indeed, concern over chronic exposure to “blue light” is not justified, since the limited studies of chronic light damage suggest rhodopsin as the primary chromophore for “Type 1” photochemically induced retinal injury, which relates to the repeated, lengthy (> 12 h/day over many days). This means that “white light” or green light is as hazardous or more so than a blue-light source of the same clinical effectiveness. Current thinking is that only the blue-light hazard (referred to as “type 2 light damage”) is relevant to humans—and are therefore treated in the safety standards.[15] The “type 1 light damage” studies of Noel and others with rats viewing bright fluorescent tubes at close range are not realistic conditions for humans, but if there is an underappreciated risk of staring at bright light, it would be more likely to be related to white-light stress (better measured in lux) of the visual system.

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Author:

David H. Sliney, Ph.D.
US Army Center for Health Promotion and
Preventive Medicine
MCHB-TS-OLO; Bldg. E1950
Aberdeen PG, MD 21010
Phone: 410-436-3002
Fax: 410-436-5054
E-mail: david.sliney@att.net