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The risk of ultraviolet radiation exposure from indoor lamps in lupus erythematosus

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Abstract

It is well known that ultraviolet radiation can exacerbate skin disease in patients with lupus erythematosus. While many patients are advised to avoid sunlight and artificial tanning, it is not clear how best to counsel patients regarding the use of indoor lamps. Indeed, many of the light bulbs commonly used in the home and workplace emit low-dose ultraviolet radiation. The irradiance is considerably lower than that of the sun, however the exposure time can last for hours and is typically repeated on a daily basis. Therefore, it is possible that this chronic exposure could ultimately result in a significant accumulation of damage.

Keywords

Lupus erythematosus; ultraviolet radiation; halogen; incandescent; fluorescent

Take-Home messages

- UVA2 and UVB can exacerbate skin disease in patients with lupus, while UVA1 may be protective.
- The lupus subsets most associated with photosensitivity are tumid lupus erythematosus and subacute cutaneous lupus erythematosus.
- Halogen lamps emit significant levels of ultraviolet radiation and should be doped or covered with glass prior to use.
- Incandescent bulbs emit low-dose ultraviolet radiation.
- Fluorescent bulbs emit varying levels of ultraviolet radiation, and patients should strive to use bulbs with the lowest irradiance.
- Chronic, low-dose UV exposure can cause cumulative skin damage. Additional studies must be done to determine the lowest dose capable of inducing damage in photosensitive patients.

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1. Introduction

It has long been known that ultraviolet radiation (UVR) can induce or exacerbate skin lesions in patients with lupus erythematosus (LE). The mechanism has been reviewed extensively [1,2] and seems to involve the Ro60 autoantigen, which promotes cell survival after exposure to UVR [3,4]. The most obvious source of these damaging rays is the sun, and for years patients have been warned to avoid direct sun exposure [3,5–7]. Little is known, however, about the potential danger of chronic exposure to indoor lighting sources. In 1990, Diffey elaborated the most common sources of UVR, listing sunlight and cosmetic tanning units first and indoor lamps last [8]. It is becoming increasingly clear, however, that the effects of indoor lamps are more substantial than was once assumed; though the level of UVR emitted is considerably lower than that of the sun, the total exposure time is much longer, which could result in a significant amount of cumulative damage.

2. The Lupus Action Spectrum

UVR is typically classified into three major groups based on wavelength: UVA (320–400 nm), UVB (290–320 nm), and UVC (200–290) [9]. Whether or not a particular bulb is considered safe depends on which type of UVR it emits. It is therefore important to understand which wavelengths are considered photobiologically active.

In the sixties and seventies, it was determined that UVB was capable of inducing skin lesions in patients with systemic lupus erythematosus (SLE) [10,11]. Thus, for many years it was believed that UVB was a danger to SLE patients, while UVA was presumed to be innocuous. This changed in the nineties, when it became apparent that broad spectrum UVA was also capable of exacerbating skin disease [12,13]. This was a particularly important finding because most sunscreens and glass screens did not protect against UVA irradiation. Soon thereafter, McGrath demonstrated that not all UVA causes damage— in fact, longer wavelength UVA1 actually decreased disease activity [14]. In a series of clinical trials, he demonstrated that UVA1 mitigated systemic symptoms, photosensitivity, and even facilitated the healing of preexisting skin lesions [15].

In summary, it is now understood that UVA2 and UVB pose a risk to lupus patients, whereas UVA1 may be beneficial (Table 1).

3. The Standard Erythema Dose

Because the ability of a light source to induce erythema depends strongly on wavelength, simply listing irradiance without specifying the relative contribution of UVA, UVB, and UVC does not provide sufficient information. The standard erythema dose (SED) was developed as a means of addressing this issue; it is equal to an erythemal effective radiant exposure of 100 J/m^2, which takes both irradiance and wavelength into account. As a point of reference for future discussion, it would take 4 SED to elicit erythema in previously unexposed fair skin [16]. Depending on the exact solar altitude, it takes between 5.4 and 33 minutes of sun exposure to receive 1 SED [Klein et al, submitted for publication].

4. Indoor lamps

With the knowledge that UVB and UVA2 can exacerbate skin disease in patients with lupus, it is critical to understand how patients can avoid exposure to these rays. Though physicians already warn patients to avoid direct sun exposure and artificial tanning booths, many do not counsel their patients about the potential risk of indoor lamps [8,17,18].

4.1 Halogen lamps

Unshielded tungsten halogen lamps emit significant levels of UVA, UVB, and even UVC. At a 1 cm distance from the bulb, the UVA and UVB output mirrors that of the sun, while the UVC output far exceeds that of the sun [19]. Several studies have demonstrated that this has serious biological consequences, both from a molecular and clinical perspective.

Early evidence illustrated that halogen lamps are genotoxic to bacteria. The UV induces base-pair substitutions and frameshift errors at a rate that surpasses that of natural sunlight [20]. In addition, they are clastogenic to human cells; uncovered halogen lamps increase the frequency of micronucleated lymphocytes in peripheral blood, a marker of genotoxicity [21]. It causes an array of chromosomal abnormalities, including breaks and interchanges between chromatids [22]. The DNA damage is sufficient to cause neoplastic transformation of human cells in culture and induce the growth of skin tumors in animal models [23–25]. The mechanism by which this occurs includes the formation of pyrimidine dimers, anchorage independent cellular growth, and the loss of function of the p53 tumor suppressor [19,23,24].

The effects of halogen lamps extend beyond the laboratory. In addition to the subtle molecular changes, they are also capable of inducing erythema in humans. At a distance of 10 cm, a 100 Watt quartz halogen bulb can elicit erythema in just fifteen minutes. Over the course of a lifetime, this represents a 3.4-fold increase in the risk of developing a cutaneous malignancy [26]. In the lupus population, where patients are already hypersensitive to the toxic effects of light, it is likely that an even more pronounced reaction would be observed.

Luckily, the genotoxic, clastogenic, and carcinogenic effects of halogen lamps can be prevented entirely if the bulb is shielded with a silica glass cover [20,21,25,27]. This discovery prompted the scientific community to demand compulsory shielding of all manufactured halogen bulbs [25]. Now, most halogen bulbs are covered with glass or “doped” with a special coating that filters out UV. However, these treated bulbs still emit UVA2, UVB, and UVC, though significantly less than the unshielded bulb [28]. Not surprisingly, the doped lamps are still mildly genotoxic to bacteria and can induce some chromosomal abnormalities [22,27]. Thus they are not as protective as a silica glass covering, which seems to absorb all UV, but they are safer than an unshielded bulb.

4.2 Incandescent

The safety of incandescent bulbs has not been studied extensively, and the results reported in the literature are conflicting. In general, the emission spectrum of an incandescent lamp begins at a discrete point and then increases monotonically. The starting point, however, is under debate. Chignell et al recently demonstrated that a 60 Watt incandescent bulb will begin to emit UV at 375 nm, a point comfortably past the dangerous UVC, UVB, and UVA2 [29]. However, another study indicates that the emission spectra of incandescent bulbs begin as low as 280 nm, which would be considered a risk to photosensitive patients [28]. The discrepancy between the two is due in part to the spectroradiometers used to measure the bulb output, with the latter being far more sensitive to UV than the former.

Even using the more sensitive spectroradiometer, the level of irradiance is quite low. Assuming eight hours of exposure per day, it would take close to two weeks to receive 1 SED [Klein et al, submitted for publication].

4.3 Fluorescent

An anecdotal case report in the early eighties suggested that fluorescent light could induce rashes in patients with SLE [30]. This observation was substantiated in 1985, when Cole et al demonstrated that commercially available fluorescent lamps emitted significant levels of UVB

and UVC. Of note, an acrylic diffuser, but not a glass envelope, blocked transmission of all short-wave UVR [31]. These results proved to be clinically relevant in 1992, when Rihner and McGrath H Jr. established that photosensitive SLE patients reported worsening rash, arthritis, and fatigue after exposure to fluorescent light. These same patients, however, denied symptoms when the fluorescent lamps were covered with an acrylic diffuser [32]. Thus is appeared that naked fluorescent bulbs could cause significant flaring of cutaneous and systemic LE, unless UV transmission was blocked with an acrylic diffuser.

In 2004, Sayre et al made quantitative measurements of UV emission from fluorescent light bulbs. He tested the bulbs commonly used at home and in the workplace, including unshielded tube lamps and energy-saving compact fluorescent lamps (CFL). His results confirmed the observations made previously. He found that all emitted appreciable levels of UVA and UVB, and several even emitted UVC [28].

Recently, Sayre's group tested several commonly used, commercially available, enveloped compact fluorescent bulbs. They sought to determine which emitted the least UVR and would therefore be safest for photosensitive patients. They found that nearly all of the bulbs emitted UVB and UVA2, despite being covered with a glass envelope. The exceptions were two Philips "Bug-A-Way" bulbs, which did not emit any detectable short-wave UVR. However, these bulbs emit a yellow light, which is not aesthetically pleasing. The remaining bulbs exhibited a surprising degree of variation in the amount of UV emitted; assuming eight hours of exposure per day, the total UV dose (250–400 nm) ranged from 73 to 634 mJ/cm^2, while the UVB-only dose (290–320) ranged from 0.01 to 15 mJ/cm^2. Under these conditions, it would take between eight days and six months to receive 1 SED, depending on the particular bulb. These results indicate that even within the same class of bulbs, there are sufficient differences between specific models that patients may benefit from using those that have been shown to emit the lowest levels of UVR [Klein et al, submitted for publication].

5. Clinical Relevance

It is apparent that most indoor lamps emit UVR, but the question remains as to whether or not this level of UVR is clinically relevant. While the irradiance is significantly lower than the sun, people spend much more time exposed to light bulbs than they do in direct sunlight. It is therefore important to understand what doses of UV are capable of eliciting damage and to appreciate the cumulative effects of chronic, low-dose UV exposure.

6.1 Low-dose UV

A single exposure to UVB irradiation (280–320 nm) at doses as low as 3 mJ/cm^2 can elicit DNA damage in EBV transformed lymphoblasts [33]. Humans have been shown to develop erythema after five daily exposures to 4.7 mJ/cm^2 of UVB (270–320 nm) [34]. If exposed chronically, hairless albino mice will develop a variety of skin tumors in response to a repeated dose of 5.7 mJ/cm^2 of broad spectrum UVR (~280–360 nm). Interestingly, the prevalence of tumor formation in this study approached 100%, regardless of the dose of UVR administered. However, the amount of time needed to reach this prevalence differed, requiring only three months for a dose of 190 mJ/cm^2 and almost two years for a dose of 5.7 mJ/cm^2 [35].

The doses of UVB capable of eliciting erythema and DNA damage are comparable to those emitted from the CFLs, with some bulbs emitting more UVR and others emitting less. The dose of broad spectrum UV capable of inducing tumors, however, is significantly lower than that which is emitted from CFLs. A direct comparison is difficult, however, because the UV emission spectra of the CFLs contained significantly more UVA1 than the lamp used in the tumor study. Because the UVA1 has relatively low photobiological activity, the higher doses emitted from the CFLs might not be representative of increased risk. Moreover, each of these

studies utilized different equipment, with varying sensitivities, which further confounds any direct comparison. It is also important to note that transformed lymphoblasts and albino mice do not necessarily behave like humans, and the results obtained are therefore only suggestive of risks to patients.

6.2 Cumulative Damage

The principle of cumulative damage was established in the early eighties. When individuals with normal skin are exposed to repeated suberythemal doses of UVA or UVB, they will develop erythema within five days [34,36]. This implies that the damage induced by low-dose UV accumulates with time and eventually becomes clinically apparent. These studies also demonstrated that chronic exposure to low-dose UV sensitizes the skin, such that the MED decreases in a time-dependent fashion [34,36]. Significant cellular changes take place in response to chronic suberythemal UV exposure, including epidermal hyperplasia, stratum corneum thickening, depletion of Langerhans cells, increased dermal inflammatory infiltrate, and deposition of lysozyme on elastin fibers [37].

These studies also indicated, however, that damage will only accumulate when the daily UV dose exceeds a specific threshold— if the irradiance is too miniscule, erythema will not develop, even after repeated daily exposures. For UVA (320–410 nm), the threshold dose was 0.15 MED (3.8 J/cm^2), for UVB (270–320) it was 0.25 MED (4.7 mJ/cm^2), and for UVC it was 0.50 MED (6.5 mJ/cm^2) [34].

Damage accumulates when the skin is not given adequate time to recover from the initial insult. After irradiation with 0.75 MED, it takes 30–48 hours to recover from UVA and 24–30 hours to recover from UVB [38]. If repeated exposures are spaced appropriately, the skin will recover and erythema will not develop. Unfortunately, this is not a practical solution for the average patient, who is exposed to light bulbs on a daily, if not hourly, basis (Table 2).

These studies established the foundation for the concern that patients might be at risk from cumulative, low-dose exposure to light bulbs. However, these results might underestimate the risk posed to lupus patients for two reasons. The first is that subjects used in these studies had normal skin. It is likely that photosensitive lupus patients would have a more robust response to lower levels of UVR and may take longer to recover. The second is that these studies lasted a maximum of nine days, while lupus patients are exposed to bulbs for years. It is possible that the threshold doses listed above are capable inducing erythema after a longer period of time. While these studies provide a nice framework, additional work still must be done to understand the true risk to photosensitive patients.

Conclusion

Various studies indicate that commonly used indoor lamps, including halogen, incandescent, and fluorescent emit appreciable levels of UVR. Even though the dose is very low, the exposure time is relatively long, which may result in significant cumulative damage. This is particularly concerning in patients that are exposed on a daily basis, which does not give the skin adequate time to recover. Although threshold doses have been determined for patients with normal skin, they have not been determined in patients with lupus. Until these studies are done, it will remain difficult to know how best to advise photosensitive patients. It is therefore safest for these patients to use bulbs that emit the lowest levels of UVR with a glass envelope or filter.

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Table 1

Landmark papers establishing the LE action spectrum

UV family	Wavelength (nm)	Effect on skin	Year	First author [Reference]
UVB	290–320	Harmful	1969 1973	Freeman RG [10] Cripps DJ [11]
UVA2	320–340	Harmful	1990 1993	Lehmann P [12] Nived [13]
UVA1	340–400	Protective	1994	McGrath H. Jr. [14]

Table 2

Landmark papers establishing the principle of cumulative damage

Principle Established	Year	First author [Reference]
Repeated exposure to suberythemal doses of UV will eventually result in erythema	1981	Parrish JA [36] Kaidbey KH [34]
Chronic, low dose UV exposure lowers the MED	1981	Parrish JA [36] Kaidbey KH [34]
Threshold doses established	1981	Kaidbey KH [34]
Minimum recovery time determined	1983	Arbabi L [38]
Chronic, low dose UV exposure induces cellular changes	1995	Lavker RM [37]