

CURRENT STATE OF KNOWLEDGE REGARDING HEALTH EFFECTS OF ARTIFICIAL LIGHTING

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Abstract

Purpose of this paper is to outline the risk and effects of artificial lighting in simple terms as current state of knowledge regarding the short and long term health effects of artificial lighting.

It will cover UV, IR and visible light effects on the body in both physical and physiological, in the form of melatonin suppression. A fast growing topic of concern related to the increasingly counts of breast cancer. Issues pertaining to pre existing medical conditions are mentioned but not covered under this topic.



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Introduction

In recent years there has been an increase in research on the possible negative health effects of modern lighting. This has been spurred on by an increasing understanding of the possible long-term effects of artificial light exposure (Lewy, Wehr, Goodwin, Newsome, & Markey, 1980; Parrish, Anderson, Urbach, & Pitts, 1978). However, despite this increasing body of knowledge, it is surprising that definitive, substantial theories about the health effects of light have yet to be developed. This theoretical gap is largely a result of the complexities inherent in extrapolating data gathered from a range of different lamp types that are developed from different technologies and used for varying applications. Each of these varying factors can result in different biological outcomes.

Due to this complexity, a comprehensive review of all health effects is beyond the scope of this paper. Instead, this paper will focus on exploring the major underlying components behind the primary health effects of modern lighting.

In order to fully appreciate the influence that modern lighting can have on human biology, it is important to remember that human beings have evolved to their current state over millions of years. Our eyes, skin and biological functions have evolved to be carefully tuned to our natural environment. For much of our evolution we, as mammalian creatures, have adapted to the cycle of the rising and setting of the sun (Waide P, 2006). The discovery of fire eventually extended productive hours and soon after (on an evolutionary scale), we created gas lamps. After this point, the discovery and exploitation of electricity led to rapid advancements in human technology, which has resulted in our current society now being dependant on copious amounts of artificial lighting to fuel our 24 hour dominance over our surroundings.

Indeed, in 2005, our modern society consumed 3,418 TWh of electricity for the production of 133 Plmh (peta-lumen-hours) of artificial light (Waide P, 2006). Additionally, flame operated lamps are used by approximately 1.6 billion people who do not have access to an electricity grid (Waide P, 2006). The use of artificial lighting is now ubiquitous, however, our exposure to it has only been for the blink of an eye when compared to the millions of years of evolution it has taken for human beings to evolve to their current form. It is, therefore, not surprising to discover that with only such a short exposure (on an evolutionary scale) to artificial lighting, human beings may be having unexpected, negative reactions to modern lighting.

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However, before we delve into an exploration of these issues, we must first understand what light is and how it functions so that we can gain a fuller appreciation of how something as ubiquitous as light influences human biology

Light By Definition

To begin to understand the effect of light we must first identify what light is. For the purpose of this paper, we will use the term light to describe electromagnetic radiation that is of the visible spectrum (that is wavelengths of 380nm to 760nm). Outside this range there are two spectrums that are also often referred to as light, although they are not visible. Throughout this paper, such light will be referred to as ultraviolet radiation and infrared radiation. Ultraviolet radiation (UVR) refers to the wavelengths 100nm-400nm. These are shorter than those of visible light and can be further broken down into UVA – 400nm-315nm, UVB-315nm- 280nm and UVC 280nm- 100nm, (CIE, 2009). Infrared radiation (IRR) refers to wavelengths longer than that visible to the human eye. For infrared radiation, the range between 780 nm and 106 nm is commonly subdivided into IR- A 780 nm- 1400 nm, IR-B 1400 nm - 3000 nm, and IR-C 3000 nm - 106 nm

(CIE, 2009). A graphical depiction of the radiation spectrum is shown in Figure 1 below.



Light as Energy

It is important to understand that the full effects of light often go unnoticed. Light is electromagnetic radiation that carries energy. This energy has the potential to affect the surfaces that it lands on (a common example can be seen in the molecular changes of skin cells that result in sunburn). Electromagnetic radiation transfers energy via quantum particles knows as photons. Photons impact on molecules and transfer energy inciting a reaction. Such reactions can be simple kinetic reactions causing molecular motion such as vibration. This type of reaction is commonly used to create heat (for example, a radiant heater which emits high quantities of IRR that vibrate molecules in the surfaces they land upon). However, unlike UV, IR is not capable of exciting valence electrons to create a photochemical reaction. The molecules that react to photon energy are called chromophores. The resultant of the reaction between the photons and chromophores is the photoproduct. Outermost valence electrons can be excited into higher orbital levels by the certain wavelengths or 'absorption' band' of radiation. Not every excited molecule will cause a chemical reaction; the energy may be lost through longer wavelength radiation or heat. Additionally, the absorbing molecule may not be the molecule that is chemically altered. Instead, energy can be transferred to another molecule, which can then become chemically reactive (Cleary & Department of Physiology & Biophysics). Such photochemical effects can instigate molecular changes that result in cancerous cells in mammalian biology.

It has been long known that UVR has detrimental effects on human skin; however, there are many more health implications of UV and IR Radiation. Radiation can effect mammals through absorption via the eyes and skin (though not all radiation can penetrate to have a lasting effect). Radiation interacts with eye tissues and pigment molecules via different methods. Radiation can induce oxidative stress as photochemical and photodynamic effects in some parts of the eye, while other parts of the eye or pigment structures can absorb light, which reduces retinal exposure (Sliney, 2002).

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The exposure (radiant energy per surface area in J/m2) and exposure rate or irradiance (radiant energy per surface area per unit time in J/m2s, W/m2) are the commonly used photobiologic metrics by which to quantify the transfer of radiant energy to the body. However, in some disciplines (such as ophthalmology and dermatology), the exposure is most often given as mJ/cm2. It is important to quantify exposure and exposure rates as cells can with stand or repair thresholds of radiation. The CIE has outlined these safe thresholds and readers interested in gaining a full understanding of these limits and how they are calculated are referred to this standard (CIE, 2009). While a full exploration of the research into exposure limits is beyond the scope of this paper, it is important to understand that the effects of electromagnetic radiation is proportionate to transferred energy (joules of emitted radiation) and duration of exposure (time). Thus, we can see that damage is caused not in an instant but over time. Both the skin and eyes have varying exposure times attributed to wavelength spectra. These variations are partly due to the penetrative nature of radiation and the absorbing effect of organisms. The following images provided by the Scientific Committee on Emerging and Newly Identified Health Risk (SCENIHR, 2012) depict penetrative examples of varying radiation on the skin and eyes.





Figure 2a Interaction of UV radiation with the human eye at all ages.





Figure 2b

Specificity of optical radiation interaction with the eye of children below 9 years of age.





Figure 2c

Optical radiation interaction with the young human eye (10 years old up to young adulthood)





Figure 2d Optical radiation interaction with the eye of an aging human



Figure 3 Light penetration in the skin

(attenuation down to 1% occurs for light wavelengths of 250-280 nm at around 40 μ m depth; for 300 nm at 100 μ m; for 360 nm at 190 μ m; for 400 nm at 250 μ m; for 700 nm at 400 μ m; for 1.2 μ m at 800 μ m; for 2 μ m at 400 μ m; for 2.5 μ m at 1 μ ; and for 400 μ m at 30 μ m) (SCENIHR, 2012).

Figures 2a, 2b, 2c and 2d show the penetration/absorption of radiation by the eye for different age groups while Figure 3 shows the penetrative effects of optical radiation through the skin. It is important to understand that:

"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 3 (a-d) and 4 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." (SCENIHR, 2012) p. 25.

From this information we can extrapolate that IRA, UVA and UVB have an increased potential for harm due to their ability to penetrate deeper into the structure of the skin (through epidermis into dermus) and the eye (1% of UVA reaching the retina). The photochemical effects of UVR is of the greatest concern, because this can result in unrepairable molecular changes such as squamous cell carcinoma (SCC) which is the world's second most common form of skin cancer. It should be noted that with regular safe doses of UV exposure, the skin increases its own resistance to UV damage and also increases its ability to repair the damage caused by exposure (de Winter, Vink, Roza, & Pavel, 2001).

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The Effects of Artificial Light on Human Biology

IRR

Many lamps have high IR output. A prime example of this is the common 12v MR16 halogen downlight (Osram, 2000). The IR output component of this type of lamp is harmless to humans unless it is misused (for example, pointed) directly in the eye at very short distance or directly aimed at the skin at very short distance). The intended use and design of these lamps should mean that these circumstances do not arise. Indeed, it is even included in most installation requirements that they are not to be mounted where they may shine directly onto a flammable or ignitable surface at a proximity closer than approximately .02m so to avoid heat damage over time. Such protections can be argued to apply to the skin. For example, the effects of IRR would be felt in the form of heat or burning on the skin well before any damage has occurred (at which point the individual would simply remove themselves from harm). For these reasons, the IRR component of lamps shall not be considered as a health concern. Indeed, it can be concluded that IRR from artificial lighting has negligible health effects on the population other than through the deliberate misuse of existing technology.

UVR

The UVR (particularly UVA and UVB) can, however, leave unnoticed lasting damage to humans. The UVR photochemcical effects of the sun have been directly linked to DNA and molecular conditions such as basal cell carcinomas (BCC), squamous cell carcinomas (SCC) and malignant melanomas (Pfeifer & Besaratinia, 2012). Many lamp types do emit wavelengths of radiation in the UV spectrum. While, these levels are deemed safe according to the CIE safe levels (SCENIHR, 2012), long term exposure is likely to represent a greater risk. Lytle and colleagues (1992) found that due to constant or repeated low level exposure of many years, office workers found themselves at an increased risk of SCC. Indeed, Lytle and colleagues stated:

"The lifetime exposure of indoor workers to typical fluorescent lighting (if unfiltered) may add 3.9% (1.6-12%) to the risk from solar UV, resulting in the induction of an additional 1500 (600-4500) SCC per annum in the United States. This calculated

projection must be compared with the 110,000 SCC caused by solar exposure. Thus, this analysis suggests there may be a small increased risk of SCC from exposure to UV-emitting fluorescent lamps." (Lytle et al., 1992)

The effect, though small, is not entirely negligible. With the introduction of acrylic prismatic diffusers, instead of louvers in the fixtures, the effective UV exposure is reduced by more than 100-fold, to virtually nothing. This is due to the filtering effect of diffusers or a second glass envelope on CFL lamps to absorb UVC and UVB Radiation (SCENIHR, 2008). There are also health benefits attributed to UVR exposure, including protection against the development of Hodgkin and non-Hodgkin lymphomas and vitamin D production (Lucas, Repacholi, & McMichael, 2006). This would lead us to think that complete reduction of UVR exposure to individuals who are not getting regular solar exposure may require small amounts of UVR (particularly UVA) responsible for vitamin D production) from artificial sources. However, because vitamin D production is directly effected by skin pigmentation, with fair skin creating higher vitamin D production from smaller solar exposure than dark skin for a similar level of exposure (de Gruijl 1997), the required UVR exposure for humans needs to be individually catered or self monitored. As such, this cannot be considered in a lighting design, except to say that maintaining minimal artificial UVR and a balance of day lighting would be highly preferred.

UV Radiation is photochemically active on a large variety of organic molecules including DNA. UVR can create various kinds of radicals that damage cell components. The skin is capable of coping with this damage via antioxidants, free radical scavengers and repair mechanisms (de Gruijl 1997). However, if the level of exposure and the resultant damage reaches the level where the functions of the cell have become seriously disturbed, the cell may undergo a process of programmed cell death. UV radiation at high levels has a clear toxic impact that evokes an inflammatory reaction. In the long term, damage may cause carcinogenic accumulation of gene mutations in the cells of the epidermis or cause loss of collagen in the dermis with a subsequent gradual loss of elasticity ("photo-aging"). Specific UV mutations (at sites of neighbouring pyramidine bases in the DNA) have been found in a majority of human skin carcinomas, providing direct evidence that UV radiation had contributed to the development of these tumors (de Gruijl, van Kranen, & Mullenders, 2001).

While there is high potential of long-term harm as a result from UVR exposure, the impact of this threat from artificial lighting devices is actually quite mild, and is far outweighed by the impact of the more imminent threat of spending a weekend sunbathing while on holidays. Indeed, cases of vitamin D deficiency in some office workers highlight the fact that there is not a high enough UV component in the lighting of office environments to help maintain the recommended healthy level of exposure to UVR required to promote vitamin D and protect against skeletal disease. Thankfully, modern medical science is aware of the importance of sun exposure and that UVR is required for healthy living and so our modern society is no longer suffering from the epidemics of rickets that was prevalent in the 1930's (Lucas et al.,2006).

Research has explored the potentially negative impact of both IR and UVR on both an organism and cellular level. While it is important to understand that both IR and UVR have the potential to impact human cellular biology, in practice, the effects are negligible due to the radiant output and intended use of modern lighting.

The Influence of Light on Circadian

Rhythms and Melatonin Suppression

The term 'circadian' is derived from the Latin words circa, meaning 'about', and dies, meaning 'day', which form the meaning of 'a daily cycle'. Circadian rhythms pertain to functions that cycle within a 24 hour period revolving around day and night. These functions control various systems within the wake and sleep cycle to assist in daily functions and survival. Such biological clocks can be found in all living creatures, even plants (Paul, Saafir, & Tosini, 2009), and it has been long known that humans have evolved to possess this feature. Circadian rhythms have a vast and, as yet not fully understood, impact on the interaction of hormones, organs, tissue and cells that have multiple functions throughout the human body. While we have known of the existence of circadian rhythms for a long time, it is only recently that we have begun to understand the full extent and importance that they have in our lives and daily functioning.

One of the primary effects that circadian rhythms exert on human biology is in the suppression of melatonin. Melatonin is a hormone released by the pineal gland in the base of the brain that acts as the master signal to numerous other glands and tissues thoughout the body to initiate action (Brzezinski, 1997). In recent years, a link between artificial light and melatonin suppression has been uncovered. Berson and colleagues (Berson, Dunn, & Takao, 2002) furthered our understanding of the biological link between retinal ganglion cells (RGC), neural signaling, and light absorption in the eye. The function of the RGC, located at the rear of the eye behind the rods and cones, is to relay presence of light signals to the supra-charismatic nuclei (SCN) which is located in the hypothalamus at the base of the brain (Pauley, 2004).

Unlike the rods and cones of the eye, the RGC do not give visual detail of light, they merely denote the presence of light and operate completely independently of the visual functions. In functionally blind transgenic mice that lack virtually all known photoreceptors (rods and cones), photic entrainment persists with undiminished sensitivity (Berson et al., 2002). The RGC are highly sensitive; levels of as little as 0.2 lux of white light or 0.1lux of monochromatic blue light at 464nm has been shown to suppress melatonin production (Pauley, 2004). This incredibly low level helps to reinforce the subtlety and frailness of the biological system that has evolved over millions of years.

Humans have evolved from their ape-like counterparts over 6 million years, and more closely in the homo-sapien state for approximately 2 millions years (Pontzer, 2012). It has only been in the past 187 years since the innovation of the incandescent light bulb that we have existed with extended light hours (Waide P, 2006). From that time in 1827, our technology has rapidly improved to the point that we have multitude types of lamps that are in almost constant use. Lamp technology has allowed society to function around the clock, with cities and industries lighting up the night sky for the universe to see 24 hours a day. However, as mammals, we are still function on a 24 hour daily cycle and a circadian rhythm that keeps our bodies in check. Like other mammals our 24 hour cycle is effected by light stimulating our SCN. In mammals the SCN acts as a "master pacemaker" of the hypothalamus and peripheral oscillators located through out the organism. These independent oscillators exist within almost every cell and tissue of the body including the liver and heart. The SCN drives and manages the peripheral clocks through hormonal and neural signals (Paul et al., 2009). These 'clocks' control the release of various other hormones throughout the body including melatonin (which is secreted during the night

whilst we sleep), cortisol, thyroid stimulating hormone (TSH), and prolactin (PRL), to note only a few (Shechter & Boivin, 2010). By extending our light hours or stimulating RGC during the night via the presence of light, the excretion of melatonin is suppressed. In humans, melatonin secretion naturally increases soon after the onset of darkness, peaks in the middle of the night (between 2am and 4 am). This inhibits the body's natural repair cycle when we sleep.

Environmental lighting does not cause the rhythm but entrains it (alters its timing). Light has two effects on melatonin: day-night light cycles modify the rhythm of melatonin secretion and brief pulses of light of sufficient intensity and duration abruptly suppress melatonin production (Brzezinski, 1997). In normal subjects, exposure to light inhibits melatonin secretion in a dose-dependent manner. The threshold is approximately 250-400 lux (equivalent to ordinary fluorescent light), and maximal inhibition occurs after exposure to intense light (600 lux or higher) for one hour (see figure below).

 $100 \cdot$







The samples from Aoki and colleagues' study were taken after light exposure in the morning following sleep under light levels of less than 10lux (Aoki et al., 1998). We can see that as we wake to light, (offset) melatonin levels drop proportionate to light intensity and duration. While this research does show how effectively our body suppresses melotonin in the presence of light, it doesn't reflect the impact of the onset of melatonin while approaching sleep. A further study in 2011 clearly shows that exposure to greater intensity of light prior to sleep sufficiently delays onset of melatonin secretion but does not effect the offset (Joshua J. Gooley, 2011). Intensities of as low as 200lux (room light) before sleep can reduce nightly melatonin production by up to 1.5 hours by delaying its onset. To add to this the presence of light of only approximately 200lux during the night whilst sleeping also produces a 50% reduction in melatonin (Joshua J. Gooley, 2011). This reduction is felt throughout the night as the RGC in the rear of the eyes, even when closed, still react to the presence of light. The disruption of sleep during the night and subsequent exposure to light can reduce melatonin secretion levels which can take up to 40 minutes to return to normal dark room levels (Lewy et al., 1980). However, if one is woken during the night and is not exposed to any light, melatonin secretion is uneffected (Aoki et al., 1998). This fact supports that proposition that while light is not the only stimuli for modifying our circadian rhythms, it is by far the primary trigger.

So Light Effects Melatonin: What Does this Mean?

Melatonin has been proven to be a pivotal hormone involved in a growing number of the mammalian cell culture systems. In recent studies, melatonin has been shown to have implications on immune function and cancer initiation and growth (the newly discovered free radical scavenging and antioxidant activities of melatonin) (Reiter, 2003). Melatonin also plays an important role in the 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 (SCENIHR, 2012). While each of these is an important and growing area of research, a critical example of the effects of melatonin suppression is the recently identified relationship between artificial lighting and breast cancer.

Breast Cancer

The link between exposure to light during traditional sleeping hours and an elevated risk of breast cancer was first identified in shift-working women and has since been extensively tested (Stevens, 2009). Physiological concentrations of melatonin have been shown to inhibit the growth of human breast cancer cells (Gammon & John, 1993). Additionally, low serum melatonin concentration have been found in women oestrogen-receptor-positive breast cancer.(Brzezinski, 1997). Furthermore, studies in experimental animals have shown that uninterrupted visual exposure to light increases the risk of mammary cancer (Mhatre, Shah, & Juneja, 1984).

The mechanisms involved in the apparent protective effects of melatonin against tumors are thought to include an enhanced immune response and the scavenging of free radicals (Brzezinski, 1997). An interesting and informative scenario is found in the case of totally blind women who do not detect light through the eyes and consequently do not have inhibition of melatonin secretion. Such women have been shown to have an approximately 50% lower relative risk of breast cancer than other women (Feychting, Österlund, & Ahlbom, 1998). This provides biologically plausible evidence for the melatonin hypothesis as related to the association between chronobiological disturbance and breast cancer (Brzezinski, 1997). In laymen's terms, the suppression of melatonin via the disruption of the circadian cycle causes a reduction in the female hormones that contribute to fighting off breast cancers in women. Indeed, regular long term exposure (defined as a minimum of 6 years) has been found to increase the risk of breast cancer diagnosis by 50% (Hansen, 2001).

As a result of growing concern raised by such research, the World Health Organization released a statement concluding that "shift work is a highly probable carcinogen" (Bulletin of the World Health Organization 2013;91:626-627. doi: http://dx.doi.org/10.2471/BLT.13.020913) This statement was corroborated in 2007 by the International Agency for Research on Cancer who also concluded that "shiftwork that involves circadian disruption is probably carcinogenic to humans" (Straif et al., 2007). However, it is also clear that not all shiftwork systems and schedules are equally disruptive to circadian rhythmicity and health (Bambra, Whitehead, Sowden, Akers, & Petticrew, 2008). This implies that with further research we can, in the future, create work schedules that have fewer negative impacts on human health.

Implications For Designers

As lighting designers, care must now be taken to ensure appropriate lighting design is applied to all tasks and environments with light-at-night concerns in mind. As previously discussed, research has shown that exposure to bright light or light high in blue spectrums prior to sleep has a direct influence on our quality of sleep and melatonin secretion. Therefore, lighting designers have a responsibility to ensure that they consider the health implications of their lighting installations. For example, when designing bedrooms and lounge rooms care should be taken to select lamps with an appropriate low blue representation in spectral output. Additionally, dimming options should be included where available. In the hours approaching sleep, warmly lit environments of dim light are desirable to promote early onset of sleep and melatonin production. Similarly, in hospitals (where human repair mechanisms need to be at their best), care should be taken to remove any unnecessary light sources from a room at night. Monitoring equipment could use a no-light setting for sleep periods in which all displays are blackened until required and external room lighting for corridors should be sufficiently inhibited by doors or curtains to prevent disruption during sleep.

Given our growing knowledge of the health effects of artificial lighting, an

important area of future research will be to further investigate the ideal types of lighting for shift workers. Research must investigate whether lighting for workers should include blue light (e.g., 460-480nm) to help maintain worker alertness, or if lighting during night periods should be altered in order induce some release of melatonin. Such research may help us begin to limit the negative health effects of shift work.

Additionally, as a result of all of this research, there has been an increasing call to better define circadian light(Rea, 2011). In order to allow for lighting designs that help mediate the health effects of modern lighting, it is imperative that a greater quality of spectral output of lamps need to be made standard. Prevention of unwanted spectral bands can only be catered for when we have a clear understanding of what is required and thusly provided for by manufactures.

Lifestyle Carcinogens

While fixed lighting design is the principle concern of many lighting designers, it is important to also consider the various lifestyle factors which concurrently impact upon the individual. Specifically, based upon the knowledge that lighting effects our quality of sleep, it is important to investigate the impact of other sources on our sleep cycle and melatonin production. A large percentage of the population view televisions, laptops, tablets and mobile phones until well into the night. These devices all emit sufficient light to directly impede the onset of melatonin. Therefore, even if lighting designers implement changes to the fixed lighting environment, these changes will be ineffective without making concurrent changes to other lifestyle technologies or making changes to how they are used. Greater education is required to inform people of the possible effects of using such entertainment devices in the late evenings in order to help reduce the long term carcinogenic effects that they may be having on the population at large. Furthermore, education could help mediate the health impacts of artificial lighting by informing individuals about strategies such as dimming their lit environment (or removing the blue wavelength of optical sources) in order to help prepare their bodies for sleep in the evenings.

Conclusion

This paper has provided an overview of some of the health effects of modern lighting on the human body. Direct harm from artificial lighting sources (although evident), are of very little concern. As discussed earlier, IR radiation has little effect on human skin and leaves a damaging effect on the eye in only very particular circumstances. UV radiation, though of greater concern than IR, is also negligible in its effects. Long-term exposure to UV from artificial lights in an office environment could cause an increase in carcinogens; however, these effects are negligible in comparison to normal social behaviours towards sun exposure for the general population.

The most concerning health effect of artificial lighting is the presence of light at night. In modern times, our societal demand for 24 hour services have exposed humans to the previously unforseen risks of extensive night lighting. The availability of light has provided humans with the opportunity to extend our

waking hours well beyond those that we have evolved to adapt to. This exposure to night lighting impacts the body's circadian rhythms and melatonin production which, in turn, can produce carcinogenic effects. However, it is important to note that the light itself is not directly causing harm; the harm arises from our use of it during periods when our body has evolved to expect darkness. While this raises questions about the current state of society and our attitudes towards work and relaxation, it does not mean that there is a vital danger incurred by the mere presence of artificial light.However, although light itself is not a threat to our existence, there are still many ways that we can better design and implement artificial lighting so as to improve our interaction with the lit environment and our relationship with darkness.

Areas not covered in this review,

Effects of artificial light on individuals with pre-existing medical conditions Spectral output of lamps and risk rating Artificial light used for tanning boths -Artificial light for medical purposes Over exposure to medical lighting Effects of light on Light sensitive disorders Effects of miss use - luminares used in the wrong Lighting applications or

medical applications

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