Adverse Health Effects of Nighttime Lighting

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Abstract The effects of poor lighting and glare on public safety are well-known, as are the harmful environmental effects on various species and the environment in general. What is less well-known is the potential harmful medical effects of excessive poor nighttime lighting. A significant body of research has been developed over the last few years regarding this problem. One of the most significant effects is the startling increased risk for breast cancer by excessive exposure to nighttime lighting. The mechanism is felt to be by disruption of the circadian rhythm and suppression of melatonin production from the pineal gland. Melatonin has an anticancer effect that is lost when its production is disrupted. I am in the process of developing a monograph that will summarize this important body of research, to be presented and endorsed by the American Medical Association, and its Council of Science and Public health. This paper is a brief overall summary of this little known potential harmful effect of poor and excessive nighttime lighting.

1. Introduction

The following is a brief summary of a longer monograph that will be presented by me to the American Medical Association (AMA) in June of 2012, written with contributions by Dr. Richard Stevens, Dr. David Blask, Dr. Steven Lockley, and Dr. George Brainard.

2. Human health issues

Since the introduction of electricity a little over a century ago, lighting the night has become a priority of modern societies due to many perceived advantages including for commerce and social activity. However, in the past two decades there has emerged a realization that with these benefits have come detriments, some of which may be substantial. The dialogue on electric light in the environment has focused on four topics: 1) esthetics, or loss of the starry night sky, 2) the energy cost of unnecessary electric light, especially at night, 3) the impact of the evolutionarily novel light at night on animal and plant life, and 4) impact of electric lighting on human health, primarily through disruption of circadian biological rhythms.

The Milky Way is no longer visible to the majority of people in the modern world. As societies have increasingly used electricity to light the night, it has

become difficult to see more than a few stars from Earth's surface. Though the major impact of electric light at night is in major metropolitan areas, even the once pristine nights of the U.S. National Parks are beginning to be degraded, more rapidly in the East but also in parks in the West as well.

Electric lighting accounts for about 19% of electricity consumption worldwide and costs about $360 billion annually (OECD/IEA 2006). Much of the light that is produced is wasted, for example by radiating up into space away from the task or environment intended to be illuminated. Estimates of how much is wasted vary; one estimate from the International Dark-Sky Association is 30% in the United States. Such a percentage worldwide would account for an annual cost of about $100 billion.

The 24-hour solar cycle of light and dark is ancient, and all life on the planet has evolved to accommodate it. Human imposition of light at night is a dramatic change in the environment but has only recently begun to attract the attention it deserves. Study of the effects of light at night on animal and plant life is in an early stage but already suggests major impacts on the entire spectrum of life including animal, plant, insect, and aquatic. Electric light at night disrupts the solar light cycle, and can be expected to have impact on any life form that is exposed to it.

About 30% of all vertebrate species and 60% of invertebrate species on Earth are nocturnal, and depend on dark for foraging and mating. Documented wildlife destruction by light at night has been evident on bird species and migrating amphibians. The most studied case is of sea turtle hatchlings on the coast of Florida which historically have scurried from their nest directly to the ocean; with increased development along the coast, and attendant increased electric lighting at night, these hatchlings become confused and often migrate away from shore to the lights. Hundreds of thousands of hatchlings are believed to have been lost as a result of this stray electric lighting at night in Florida.

The circadian biology of plants is as robust as animals, and the impact of light at night on plant life may also be considerable due to the role of light in photosynthesis and the fact that many plants are pollinated at night. In addition, light at night as a vector attractant for diseases such as malaria is beginning to be evaluated.

2. Disability glare and discomfort glare

Disability glare is unwanted and poorly directed light that blinds, causes poor vision by decreasing contrast, and creates an unsafe driving condition, especially at night. Disability glare is a particular problem on the older aging eye. Many older drivers have a difficult time driving at night, unaware of the etiology of their poor night vision, which is, in part, the result of badly engineered lights. A proper understanding of the human eye physiology would allow for engineering safer and better designed street lighting. There are natural causes of disability glare, such as solar glare at sunset on a dirty windshield. We have all

experienced such glare, and attempt to minimize its effects with sunglasses or a cleaning of the windshield. Unfortunately, glare at night while driving is not so easily remedied. Its cause is generally overly bright and unshielded or poorly directed light that enters the eye and then scatters off of eye structures resulting in diminished contrast and impeded vision. Such effects become dramatically worse as the human eye ages due to aging eye structures. As the human eye cannot be so easily fixed as cleaning a dirty windshield it thus behooves us to thus engineer our street lighting to minimize the effects of disability glare.

Disability and Discomfort in the nighttime driving environment have long been a topic of research. Disability glare has been fairly well defined based on the physiology of the human eye and behavior of light as it enters the ocular media. However, discomfort glare has been less defined. Discomfort glare is not based on a physical response but rather a psychological response. This means that the basis of the two responses is fundamentally different and the research into each of the effects is also fundamentally different.

Disability glare is, as the name implies, glare which limits the ability of the driver to see. Disability glare has a direct link to the physiology of the eye and has been researched for many years. The process which causes disability glare was originally discovered by Holladay (1927) and was determined to be light scatter from the ocular media in the eye. As light enters the eye, it collides with components of the ocular media such as the cornea, lens, and the vitreous humor. At each collision, photons scatter and cast a veil of light across the retina. According to Vos *et al.* (1964) and Boyton and Clark (1962) 25–30% of the stray light is from the cornea, approximately the same amount is from the lens, and the rest is scattered in the retina itself. Later measurements showed that much of the scattering also occurs in the vitreous humor. The veil of light has the effect of reducing the contrast of the object which the driver is trying to see which would have the same effect as raising the background luminance of the object.

This veiling light can be modeled and is represented by the term veiling luminance. Many equations have been developed over time, however, the same general form of the equation is used. Originally proposed by Holladay, the relationship is that veiling luminance is directly related to the illuminance of the light source and inversely related to the square of the angle of eccentricity of the light source with an age-dependant multiplier across the entire equation.

Discomfort glare is by far less defined than disability glare. Discomfort glare is defined as a glare source which causes the observer to feel uncomfortable. The definition of discomfort is not precise and some research has shown that a person's response to a glare source is based more on their emotional state than on the light source itself. Discomfort glare is based primarily on the observer's light adaptation level, the size, number, luminance, and location of the light sources in the scene. Models of Discomfort glare have been developed and rely on the illuminance of the light source on the eye. These models continue to need development and the overall impact of discomfort on fatigue and user safety remains an issue. Both discomfort and disability glare have specific impacts on

the user in the nighttime environment. Research has shown that both of these glare effects occur simultaneously. Research also shows that the effects of the glare are cumulative, meaning that the glare from two light sources is the sum of the glare from the individual light sources. As a result, every light source within the field of view has an impact on the comfort and visual capability of the driver.

For overhead roadway lighting, design standards include a methodology for controlling the disability glare through a ratio of the eye adaptation luminance to the veiling luminance caused by the light source. As the veiling luminance is related to the illuminance of the light source at the eye, a roadway luminaire which directs light horizontally has a much greater effect on the driver than a light source that cuts off the horizontal light. A trend towards flat glass luminaires which provide a cut-off of light at horizontal angles provides a lower level of both disability and discomfort glare.

Decorative luminaires such as those called acorn or drop lens luminaires have a high level of horizontal light, and the visible portion of the luminaire provides a different situation. Here, these luminaires are typically used for areas where pedestrians are the primary roadway users. The horizontal light in this situation is useful for facial recognition of a pedestrian but it limits the driver in their ability to perceive other objects in the roadway. As a result, many cities are designing and installing two lighting systems, one for the pedestrian and one for the roadway.

The final issue with glare from overhead lighting is the cyclic nature of the impact. Bennett found that as a driver passes through a roadway, they typically go from one luminaire to another. The glare experience will increase as they approach the luminaire and then fall off as they pass the luminaire. While typically not an issue for disability glare, this is a discomfort issue and can be quite fatiguing.

3. Importance of circadian biological rhythms

From the beginning, the solar cycle of light and dark has provided one of the essential bases for life on Earth. Adaptation to the solar cycle has resulted in fundamental molecular and genetic processes that are aligned to an approximately 24-hour period (the circadian biological rhythm) in virtually all life on the planet. The endogenous nature of this circadian rhythm was realized when researchers studied life forms, plant and animal, in laboratory environments devoid of any time cues. Previously it was assumed that plants and animals only responded to the sun rather than anticipated its cycle. It has now become clear that the circadian genetic clock mechanism is ubiquitous and intimately involved in many, if not most facets of cellular and organism function. It has also become clear that although capable of a self-maintaining rhythm, the master circadian clock in mammals (including humans) responds to light through a novel photoreceptive system in the retina. This tandem development

of an endogenous rhythm and a sensitivity of it to light were presumably designed by nature to allow for a precise 24-hour regulation of rest and activity, and for adapting to seasonal changes in day-length, while maintaining the advantages of a physiology that anticipates day and night. Understanding how these endogenous rhythms work at the molecular and physiological level and how light is communicated to this system have together become one of the hot topics in the life sciences today.

Biological adaptation to the Sun has evolved for a very long time. In the last hundred years, however, bright light increasingly invades the night as human societies gain industry, technology, and wealth; lighting of the night took a major turn for the brighter as the world began to use electricity. At the same time, ever greater numbers of people work inside buildings under electric lighting. This lighting is vastly dimmer than sunlight, and it provides a very different spectral irradiance; whereas daylight is strong at all visible wavelengths peaking in the blue region, electric lighting has extreme characteristic wavelength peaks (fluorescent) or monotonic increases in irradiance as wavelength lengthens (incandescent). Much of the modern world now lives in a murky cloud of dim light throughout the day and night in isolation from the sun; it is remarkable how little sun people get even in such sunny environments as San Diego.

It is imperative to determine if there are adverse health consequences of our electric lighting practices in the human environment, and if so, the mechanisms underlying these effects. Effective interventions could then be identified that would mitigate the downsides of suboptimal light exposure. Society will not go back to life before electricity, and light during the night is required for our way of life. However, there are undoubtedly ways of lighting the night that are less disruptive than others to our well-being. Considerations in this regard are light spectrum, intensity, duration, and timing during the day and night, all of which determine the effect of light on physiology.

As the research on the biology of circadian rhythms has advanced, the range of potential disease connections has expanded. The first focus was breast cancer, but many more are now being pursued as well such as other cancers (such as prostate cancer), obesity, and diabetes.

4. Light at night, cancer, and importance of melatonin

The first step in determining whether electric lighting affects human health is to understand the impact of light on human physiology. The endogenous human circadian rhythm is complex. Perhaps the most studied aspect of that rhythm is the hormone melatonin, both as a marker of the rhythm and also as an important modulator of the rhythm. There is scientific evidence in humans that support the following features of the impact on electric light exposure on melatonin production, and by extension to the circadian rhythm. The biology of phototransduction continues to be unraveled, and will undoubtedly yield further insights into potential health impacts of electric lighting.

Epidemiological studies are a critical component of the evidence base required to assess whether or not light-at-night (LAN) affects disease risk, including cancer. However, these studies are necessarily observational and can rarely provide mechanistic understanding of the associations observed. For this reason, a robust body of basic scientific studies is also needed before causal inference can be pursued. Only carefully designed and controlled basic laboratory studies in experimental animal models of cancer have the potential to provide the empirical support for a causal nexus between light at night and elevated cancer risk as well as for a plausible biological mechanism to explain such a connection.

The preponderance of experimental evidence supports the hypothesis that under the conditions of complete darkness, high circulating levels of melatonin during the night not only provide a potent circadian anticancer signal to established cancer cells but help protect normal cells from the initiation of the carcinogenic process in the first place. It has been postulated that disruption in the phasing/timing of the central circadian pacemaker in the brain's suprachiasmatic nucleus (SCN), in general, and the suppression of circadian nocturnal production of melatonin, in particular, by light at night (LAN), may be an important biological explanation for the observed epidemiological associations of cancer risk and surrogates for LAN (such as night shift work, blindness, reported hours of sleep, and so on).

The majority of earlier studies in experimental models of either spontaneous or chemically-induced mammary carcinogenesis in mice and rats, respectively, demonstrated an accelerated onset of mammary tumor development accompanied by increased tumor incidence and number in animals exposed to constant bright fluorescent LAN as compared with control animals maintained on a strict LD12:12 light/dark.

More recent work, however, has focused on the ability of light at night to promote the growth progression and metabolism in human breast cancer xenografts. Blask and co-workers (2005) assessed the dose-response effects of light exposure during darkness on the growth of tissue-isolated human breast cancer xenografts in nude female rats; these human tumors are estrogen receptor negative (ER–) and depend on the essential polyunsaturated fatty acid linoleic acid for their growth. Both ER– and ER+ human breast cancer xenografts are highly sensitive to the direct growth and linoleic acid metabolic inhibitory effects of nocturnal concentrations of melatonin. Five different groups of xenograft-bearing rats were each exposed to one of five increasing intensities of white, fluorescent polychromatic light, ranging from very dim to very bright, during the dark phase of each LD 12:12 cycle beginning two weeks before tumor implantation and continuing thereafter until the end of the tumor growth experiment; a sixth control group of tumor-bearing rats was exposed to complete darkness during the dark phase of each LD 12:12 cycle. Following several weeks of exposure of rats to increasingly brighter light during the dark phase there was a dose-dependent increase in the percent suppression of peak

nocturnal serum melatonin levels. There was also an accompanying marked, dose-related increase in tumor metabolism of linoleic acid and the rate of tumor growth as light intensity during the night increased and the nocturnal amplitude of blood melatonin levels decreased. A particularly important aspect of this study was that exposure to even the very dimmest intensity of LAN (0.2 lux), which induced approximately a 65% suppression of the nocturnal peak of circulating melatonin levels, resulted in a marked stimulation in the rates of tumor growth and linoleic acid metabolic activity that was nearly equivalent to that observed in constant bright light-exposed tumor-bearing rats in which there was complete melatonin suppression. Furthermore, as little as a 15% suppression of nocturnal melatonin levels, in response to an extremely low intensity of LAN, was required to elicit a small but significant increase in xenograft growth and linoleic acid metabolism. This finding suggested that even a seemingly marginal suppression of the nocturnal circadian melatonin signal induced by exposure to extremely dim LAN could translate into a significant stimulation of human breast cancer growth and metabolism.

Similar findings were also obtained by this group on the growth and linoleic metabolism of a highly melatonin-sensitive rat hepatoma in male rats exposed to the same increasing intensities of light at night. The stimulatory effects of dim LAN (0.2 lux) observed in rat hepatoma and human breast cancer xenograft growth were subsequently and independently corroborated with respect to the growth of DMBA-induced mammary carcinomas in female rats by Cos *et al.* (2006). They documented a significant decrease in nighttime urinary excretion of the main liver metabolite of melatonin, 6-sulfatoxymelatonin, in animals exposed to dim LAN as well as a marked increase in serum estradiol. More recently, Dauchy *et al.* (1997) reported high tumor growth and linoleic acid metabolic rates and completely suppressed nocturnal melatonin levels in rats bearing human breast cancer xenografts or rat hepatomas as a result of their initial exposure to LAN (24.5 lux). However, as the amount of LAN exposure was subsequently and sequentially reduced to zero, there was a gradual restoration of circulating melatonin concentrations to high nocturnal peak levels accompanied by a marked reduction in tumor growth and linoleic acid metabolic activity to baseline rates.

In the same investigation by Blask and colleagues cited above, important new relationships between circadian biology, the endogenous nocturnal melatonin signal, and its suppression by LAN, relative to human breast cancer risk were uncovered using a unique experimental strategy that combined blood collection from human subjects, exposed to either complete darkness or light at night, and the direct perfusion of human breast cancer xenografts with the resulting blood samples. The linoleic acid metabolic and growth activity of ER– (and ER+) human breast cancer xenografts (growing in nude rats) directly perfused in situ with whole blood collected during completely dark nights from young, healthy premenopausal female subjects (high melatonin), was markedly reduced as compared to when the xenografts were perfused with

blood collected during the daytime (low melatonin). Following dark exposure, the exposure of the same subjects to bright (such as 2800 lux), polychromatic, white fluorescent light during the night reduced their melatonin levels almost to daytime concentrations and extinguished the tumor and metabolic inhibitory activity of their blood.

That this effect was achieved via LAN-induced melatonin suppression was supported by the fact that addition of a physiological nocturnal concentration of melatonin to blood collected from light-treated subjects (low melatonin) restored the tumor inhibitory activity to a level comparable to that observed in the melatonin-rich blood collected at night during total darkness. Moreover, the addition of a melatonin receptor antagonist to the blood collected during darkness (high melatonin) completely eliminated the ability of the blood to inhibit the growth and metabolic activity of perfused tumors. Therefore, melatonin is the first soluble, nocturnal anticancer signal to be identified in humans that directly links the central circadian clock with some of the important mechanisms regulating breast carcinogenesis.

These findings provide the first definitive nexus between the exposure of healthy premenopausal female human subjects to bright, white LAN and the enhancement of human breast oncogenesis via circadian disruption (that is, suppression) of the nocturnal, anticancer melatonin signal.

5. Light-at-night and sleep disruption

As alluded to above, in addition to light-at-night suppression of the nocturnal melatonin signal, another type of circadian disruption can be caused by chronically advancing the phasing of light exposure (chronic jet lag). Filipski and co-workers (2004, 2005) maintained male mice in either an alternating LD12:12 light/dark cycle or exposed them to experimental chronic jet lag (through serial eight-hour advances of LD12:12 cycles every two days) in order to disrupt the rest-activity circadian rhythm. Ten days after the start of the light-dark cycle advances, animals in both groups were implanted with mouse osteosarcoma. In the mice undergoing "jet-lag" via repeated advances in circadian phase, tumor growth progression, during a narrow window of time between days 8 and 11 after tumor transplantation, was modestly but significantly faster as compared with that in mice kept in LD12:12. Although circulating melatonin levels were not assessed in the two groups, it is important to note that the specialized strain of mice used in this study exhibits an abnormal melatonin profile with highest levels of melatonin occurring during the light phase rather than during the dark phase. Nevertheless, this study indicated that in addition to light-at-night-induced melatonin suppression, circadian disruption induced by chronic phase advances in light exposure may represent another biological mechanism for increased cancer growth.

The human evidence, primarily from epidemiological studies, is indirect. The greatest amount of evidence so far is on cancer risk, particularly of breast.

Among the potential health effects of circadian disruption from electric lighting, the development of breast cancer has received the most attention. The idea that the increasing use of electricity to light the night might explain a portion of the high breast cancer risk in the industrialized world, and the increasing incidence and mortality in the developing world, was first articulated in 1987 by Stevens. The idea was originally based on suppression of the normal nocturnal rise in circulating melatonin, but has since expanded to include impact on circadian gene function. From this theory came a series of predictions, including that non-day shift work would raise risk, blind women would be at lower risk, reported sleep duration (as a surrogate for hours of dark) would be inversely associated with risk, and that population nighttime light level would co-distribute with breast cancer incidence worldwide. The most studied of these predictions is that non-day shift work would be associated with increased risk. Based on studies of non-day shift occupation and cancer (mostly breast cancer) published through 2007, the International Agency for Research on Cancer (IARC) concluded "shift-work that involves circadian disruption is probably carcinogenic to humans (Group 2A [level of confidence of carcinogenic potential])." The detailed review of the individual studies is also available (IARC 2010).

Since the IARC evaluation was conducted, several new studies of breast cancer have been published. Lie *et al.* (2011) conducted a large case-control study of nurses in Norway and found a significantly elevated risk in subjects with a history of regularly working five or more consecutive nights between days off. Hansen and Stevens (2011) evaluated the impact of type of shift (such as evening, night, rotating) and found a roughly increasing risk as the expected disruptiveness of the shift increased. Each of these studies has strengths and limitations common to epidemiology, particularly in exposure assessment and appropriate comparison groups; that is to say, no woman in the modern world is unexposed to light-at-night but quantifying that exposure is difficult. However, on balance the new evidence is consistent with the elevated risk from the previous studies evaluated by IARC.

Four case-control studies have now reported an association of some aspect of nighttime light level in the bedroom and breast cancer risk. The elevated risk estimate was statistically significant in two of them. As case-control designs, in addition to the limitation of recall error there is also the potentially severe limitation of recall bias. Despite the difficulty of gathering reliable information on bedroom light level at night, the possibility that even a very low luminance over a long period of time might have an impact on cancer risk is important. It is not yet clear what is the lower limit of light level that could, over a long time period, affect the circadian system.

Until 1980 when Lewy and colleagues (1980) published in *Science* magazine that bright light can suppress circulating melatonin concentration, it was thought that humans were insensitive to light. Since that time, the intensity believed to be adequate to inhibit melatonin production has steadily declined. In pioneering work, Brainard and colleagues (1988) have shown that very low

monochromatic photon flux density can have an effect which is wavelength dependent. And it has now been reported that the lighting typical in bedrooms in the evening after dusk (but before bedtime) can also suppress melatonin and delay its nocturnal surge.

After lights out for bedtime, it is not yet clear whether the ambient background light from weak sources in the bedroom or outside light coming through the window could influence the circadian system; a brief exposure at these levels may not have a detectable impact in a laboratory setting though long-term chronic exposure might. In the modern world few people sleep in total darkness. When eyelids are shut during sleep, only very bright light can penetrate to lower melatonin, and this in only some people. However, it is normal to awaken in the middle of the night as most people do, and there is often the need to use the facilities, which increases as people age. Therefore the potential for low level light in the bedroom to affect human health by disrupting the circadian system should be a research priority.

The modern world has an epidemic of obesity and diabetes that may in part be due to lack of sleep, lack of dark, and/or circadian disruption. The circadian rhythm and sleep are intimately related but not the same thing. It has long been known that non-day shift workers are at greater risk of diabetes and obesity. Epidemiological studies also show associations of reported sleep duration and risk of obesity and diabetes. Circadian disruption may be a common mechanism for these outcomes. This is based on the rapidly emerging understanding of the link between the circadian rhythm and metabolism.

Adequate daily sleep is required for maintenance of cognitive function, and for a vast array of other capabilities that are only partially understood. Sleep is not, however, required for maintenance of the endogenous circadian rhythm (such as melatonin cycling), whereas dark is required. The epidemiological and laboratory research on sleep and health cannot entirely separate effects of sleep duration from duration of exposure to dark, so that this work can be difficult to interpret. The distinction is quite important because a requirement for a daily and lengthy period of dark to maintain optimal circadian health has different implications than a requirement that one must be asleep during this entire period of dark; it may be normal to experience a wakeful period in the middle of a dark night.

Light during the night will disrupt circadian function as well as sleep, and the health consequences of short sleep and of chronic circadian disruption are both now the subject of intense research. There is a growing number of both observational and clinical studies of sleep and metabolism which suggest an alarming and important impact of short sleep on health; however it is not yet clear that sleep and dark have been entirely disentangled in these studies. An example of the difficulty of interpretation of the "sleep" studies is the carefully conducted study of Taheri *et al.* (2004), who reported that sleep duration, as verified by polysomnography, was associated with morning blood levels of leptin in a sample of 1,024 adults in Wisconsin. However, in the same analysis,

the duration of typical sleep reported by each subject was more strongly associated with leptin level. Mean verified sleep was 6.2 hours, whereas mean reported sleep was 7.2 hours, a full hour different. Reported "sleep" duration probably means the difference in time when a person turns out their light for bed, and when they get up in the morning; or actual hours of dark. An important question is: what portion of health effects of dark disruption is due to sleep disruption and what portion is due directly to circadian impact of electric light intrusion on the dark of night?

6. Conclusion

It is clear now that light-at-night has profound effects on human physiology, and many of these effects are just now becoming known. These effects have spawned a burgeoning field of research that shows how profoundly both humans and life on earth are affected by the loss of the dark at night. Light-at-night affects more than just our views of the night sky.

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