Ground water	Water from a subsurface saturated zone that is under a pressure equal to or greater than atmospheric	
	pressure but not under the influence of surface water (open to the atmosphere).	
^a When bottled water comes from a community water system, except when it has been treated to meet the definitions "purified		
water" or "sterilized water" and is labeled as such, the label shall state "from a community water system" or, alternatively, "from		
a municipal source" as appropriate, on the principal display panel or panels.		

4. LIGHT POLLUTION: ADVERSE HEALTH EFFECTS OF NIGHTTIME LIGHTING

Reference committee hearing: see report of <u>Reference Committee D</u>.

HOUSE ACTION: RECOMMENDATIONS ADOPTED AS FOLLOWS AND REMAINDER OF REPORT FILED See Policies H-135.932 and H-135.937

INTRODUCTION

Current AMA Policy H-135.937 (AMA Policy Database) advocates for light pollution control and reduced glare from (electric) artificial light sources to both protect public safety and conserve energy. Lighting the night has become a necessity in many areas of the world to enhance commerce, promote social activity, and enhance public safety. However, an emerging consensus has come to acknowledge the effects of widespread nighttime artificial lighting, including the: 1) impact of artificial lighting on human health, primarily through disruption of circadian biological rhythms or sleep; 2) intersection of ocular physiology, vehicle headlamps, nighttime lighting schemes, and harmful glare; 3) energy cost of wasted and unnecessary electric light; and 4) impact of novel light at night on wildlife and vegetation. In addition to these health and environmental effects, an esthetic deficit is apparent with the progressive loss of the starry night sky and interference with astronomical observations. With the assistance of experts in the field, this report evaluates the effects of pervasive nighttime lighting on human health and performance. Concerns related to energy cost, effects on wildlife and vegetation, and esthetics are also briefly noted.

METHODS

English-language reports in humans were selected from a PubMed search of the literature from 1995 to March 2012 using the MeSH terms "circadian/biological clocks/rhythm," "chronobiology/disorders," "photoperiod," "light/lighting" "sleep," "work schedule," or "adaptation," combined with the terms "physiology," "melatonin," "adverse effects/toxicity," "pathophysiology," "neoplasm," "epidemiology/etiology," "mental disorders," "energy metabolism," and "gene expression." Additional articles were identified by manual review of the references cited in these publications; others were supplied by experts in the field who contributed to this report (see Acknowledgement).

LIGHT AND HUMAN PHYSIOLOGY

The solar cycle of light and dark provides the essential basis for life on Earth. Adaptation to the solar cycle has resulted in fundamental molecular and genetic endogenous processes in virtually all life forms that are aligned with an approximately 24-hour period (circadian biological rhythm). The circadian genetic clock mechanism is intimately involved in many, if not most, facets of cellular and organismal function.¹ Although the circadian system spontaneously generates near-24-hour rhythms, this master clock must be reset daily by the light-dark cycle to maintain proper temporal alignment with the environment. In humans and other mammals, this daily entrainment is achieved primarily by novel photoreceptors that project directly to the site of the circadian clock (suprachiasmatic nuclei (SCN) of the hypothalamus).²⁻⁵ The tandem development of an endogenous rhythm sensitive to light presumably evolved to allow for precise 24-hour regulation of rest and activity, and for adapting to seasonal changes in night-length, while maintaining the advantages of an underlying physiology that anticipates day and night. Understanding the molecular and physiological basis of endogenous rhythms, how light information is communicated, and the health implications of disruptions to this system are topics of intensive study.

ELECTRIC LIGHTING AND HUMAN HEALTH

Biological adaptation to the sun has evolved over billions of years. The power to artificially override the natural cycle of light and dark is a recent event and represents a man-made self-experiment on the effects of exposure to

increasingly bright light during the night as human societies acquire technology and expand industry. At the same time, increasing numbers of people work inside buildings under electric lighting both night and day. Artificial lighting is substantially dimmer than sunlight and provides a very different spectral irradiance. Sunlight is strong at all visible wavelengths, peaking in the yellow region, whereas electric lighting has either extreme characteristic wavelength peaks (fluorescent) or exhibits a monotonic increase in irradiance as wavelength lengthens (incandescent). In contrast to outdoor lighting conditions, much of the modern world now lives and works in relatively dim light throughout the day in isolation from the sun, with often poor contrast between night and day, even for those who live and work in sunny environments.⁶

Extensive nighttime lighting is required for contemporary society and commerce. Therefore, it is imperative to evaluate the unintended adverse health consequences of electric lighting practices in the human environment, and determine their physiological bases so that effective interventions can be developed to mitigate harmful effects of suboptimal light exposure. For example, engineers have already developed less disruptive night lighting technologies, and continued progress in this area is anticipated. That such technologies exist, however, does not guarantee that they will be purchased, installed and properly implemented. The medical community and public can take the lead on advocating a healthier environment, as illustrated by recent changes in public smoking policies worldwide. As the research on the biology of circadian rhythms has advanced, the range of potential disease connections due to disrupted circadian rhythms and sleep has expanded.

Biological Impact of Light on Human Physiology

Light is the most powerful stimulus for regulating human circadian rhythms and is the major environmental time cue for synchronizing the circadian clock. In addition to resetting the circadian pacemaker, light also stimulates additional neuroendocrine and neurobehavioral responses, including suppression of melatonin release from the pineal gland, directly alerting the brain, and improving alertness and performance.⁷⁻⁹ Melatonin is one of the most studied biomarkers of the human physiological response to light.¹⁰ This substance is the biochemical correlate of darkness and is only produced at night, regardless of whether an organism is day-active (diurnal) or night-active (nocturnal). Conceptually, melatonin provides an internal representation of the environmental photoperiod, specifically night-length. The synthesis and timing of melatonin production requires an afferent signal from the SCN. Ablation of this pathway, which occurs in some patients from upper cervical spinal damage, completely abolishes melatonin production. Certain other circadian rhythms (e.g., cortisol, body temperature, sleep-wake cycles) do not depend on this pathway and persist if the SCN pathway is damaged.

Light is not required to generate circadian rhythms or pineal melatonin production. In the absence of a light-dark cycle (e.g., totally blind individuals), the circadian pacemaker generates rhythms close to, but not exactly a 24-hour periodicity, reflecting the timing of processes under SCN control.² However, as previously noted, the timing of SCN rhythms and consequently the rhythms controlled by the circadian clock are affected by light, and require daily exposure to the light-dark cycle to be synchronized with the 24-hour day.

When light information fails to reach the SCN to synchronize the clock and its outputs, the pacemaker reverts to its endogenous non-24-hour period (range 23.7-25.0 h). Consequently, the timing of physiology and behavior that is controlled by the circadian system, for example the sleep-wake cycle, alertness and performance patterns, the core body temperature rhythm, and melatonin and cortisol production, becomes desynchronized from the 24-hour day.² The resultant clinical disorder is termed "non-24-hour sleep-wake disorder" and is characterized by alternating episodes of restful sleep, followed by poor night-time sleep and excessive day-time napping, as the non-24-hour circadian pacemaker cycles in and out of phase with the 24-hour social day.¹¹ Another effect of light exposure at night is the immediate suppression of melatonin production. Under natural conditions, organisms would never be exposed to light during the night in substantial amounts and would not experience melatonin suppression. Electric light, however, efficiently suppresses melatonin at intensities commonly experienced in the home at night.¹²

Measures of Illumination

Luminous flux is the measure of the perceived power of light. The lumen is the standard international unit of luminous flux, a measure of the total "amount" of visible light emitted by a source, while illumination is a measure of how much luminous flux is spread over a given area (intensity of illumination). One lux is equal to one lumen/ m^2 . Luminous flux measurements take into account the fact that the human eye and visual system is more sensitive to some wavelengths than others. The peak luminosity function is in the green spectral region; white light sources

produce far fewer lumens. To provide some perspective, the illuminance associated with a full moon is less than 1 lux, versus 50 lux for a typically incandescent lit family room, 80 lux in a narrower hallway, 325-500 lux for office lighting, 1,000 lux for an overcast day, and 32,000-130,000 lux for direct sunlight.

Initially it was thought that bright light of at least 2,500-20,000 lux was needed to suppress nighttime melatonin secretion or phase shift the melatonin rhythm (as in jet lag) in humans.¹³⁻¹⁵ It is now established that when exposure of the human eye is carefully controlled, illuminance as low as 5-17 lux of monochromatic green light or 100 lux of broadband white light can significantly suppress melatonin in normal human volunteers.^{12,16-18} Similarly, circadian phase shifts of the melatonin rhythm can be evoked with an illuminance of 5 lux of monochromatic blue light or <100 lux of white fluorescent light, however, exposure to red light is not disruptive.^{18,19} Typical lighting in bedrooms in the evening after dusk (but before bedtime) can also suppress melatonin and delay its nocturnal surge.¹² Acute enhancement of both subjective and objective measures of alertness can be evoked with as little as 5 lux of monochromatic blue light.²⁰ Dose-response curves for melatonin suppression by night-time light exposure to fluorescent light show that ~100 lux of light induces 50% of the maximal response observed with 1,000-10,000 lux of light.^{18,21}

Ocular Physiology Mediating Photic Effects

Factors that alter the amount and spectral quality of light reaching the retina include gaze behavior relative to a light source, age (of the ocular lens), and pupillary dilation. Once a light stimulus reaches the retina, physiology within the retina and within the nervous system determines the capacity of the stimulus to evoke circadian, neuroendocrine or neurobehavioral responses. This physiology includes: 1) the sensitivity of the operative photopigments and photoreceptors; 2) location of these photoreceptors within the retina; 3) the ability of the nervous system to integrate photic stimuli spatially and temporally; and, 4) the state of photoreceptor adaptation.

In particular, both short and long-term photoreceptor adaptation can significantly modify the biological and behavioral responses to light and acutely suppress melatonin in humans.²² For example, a full week of daytime exposure to bright light (by daylight and/or indoor light boxes at ~ 5,000 lux) or a three-day period of exposure to moderate indoor lighting (200 lux) reduces an individual's sensitivity to light suppression of nighttime melatonin compared with exposure to dim indoor lighting (0.5 lux); similar dim light conditions also enhance circadian phase shifting.²³⁻²⁵ Two hours of exposure to 18 lux of white incandescent light versus full dark exposure in a single evening modifies the sensitivity of an individual for light-induced melatonin suppression later that same night.²⁶ Hence, photoreceptor adaptation, like the other ocular and neural elements noted above, can significantly modify the biological and behavioral responses to light.¹⁶

In general, photobiological responses to light are not all-or-none phenomena. In the case of acutely suppressing high nightime levels of melatonin or phase-shifting the entire melatonin rhythm, light works in a dose-response fashion. Once threshold is exceeded, increasing irradiances of light elicit increasing acute plasma melatonin suppression or longer-term phase-shifts of the melatonin rhythm in healthy individuals.^{16,18,27} All humans, however, are not equally sensitive to light; significant individual differences exist in sensitivity to light for both neuroendocrine and circadian regulation.^{16,18} For a detailed description of the molecular and cellular basis for how photoreceptive input regulates circadian and neuroendocrine system function, see the Addendum.

HUMAN CONCERNS-DISABILITY AND DISCOMFORT GLARE

Glare from nighttime lighting can create hazards ranging from discomfort to frank visual disability. 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. Discomfort glare is less well-defined and more subjective as it is not based on a physical response per se but rather a psychological response. Accordingly, the respective bases of (and research into) these two responses are fundamentally different.

Disability Glare

Disability glare is unwanted and poorly directed light that temporarily blinds, causes poor vision by decreasing contrast, and creates an unsafe viewing condition, especially at night, by limiting the ability of the person to see. There are natural causes of disability glare, such as solar glare at sunset on a dirty windshield which can be lessened by cleaning the windshield. Unfortunately, nighttime glare while driving is not easily remedied. It is caused by the

misapplication of luminaires that comprise the lighting design which are generally overly bright and unshielded, and/or sources of poorly directed light that enters the eye and scatters among ocular structures resulting in diminished contrast and impeded vision. Such effects dramatically worsen as the human eye ages, contributing to poor night vision and difficulty in driving at night for older drivers.

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Disability glare is caused by light scatter from ocular media.²⁸ As light enters the eye, it collides with cornea, lens, and vitreous humor, scattering photons and casting a veil of light across the retina²⁹⁻³¹ (see Figure 1). The veil of light reduces the contrast of the object that the driver is trying to see, having the same effect as increasing the background luminance of the object. This veiling light is represented by the term veiling luminance. 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 dependent multiplier across the entire equation.²⁸ This means that the disability from a light source is lessened the farther the source is from the line of sight.^{α}

Accordingly, proper design techniques and consideration for the glare caused by lighting systems need to be considered. One of the primary difficulties, especially for roadways, is that the lighting is not governed by a single jurisdiction. Roadway lighting may be designed properly and provide a low level of glare; however lighting can emanate from adjacent properties, spilling out into the roadway thus affecting the driver and overall performance and suitability of a lighting system. Control over all environmental sources of nighttime lighting is therefore critical for the overall control of disability glare.

Discomfort Glare

Discomfort glare is less well defined but emanates from a glare source that 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 his/her emotional state than on the light source itself. Discomfort glare may be based primarily on the observer's light adaptation level, the size, number, luminance and location of the light sources in the scene.³²

Both overhead roadway lighting and opposing headlamps are involved with discomfort glare in the driver. A numerical rating scale based on the dynamic nature of glare in simulations is available to measure the discomfort level experienced by drivers (Appendix).³³ The overall impact of discomfort glare on fatigue and driver safety remains an issue.

<u>Lighting and Glare</u>. 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.

Overhead lighting

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 the light source produces at the eye, a roadway luminaire that 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 (e.g., acorn or drop lens) have a high level of horizontal light and typically are used in 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's 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.

^{α} As an example, high mast lighting systems where the roadway lighting is over 100 feet in the air have significantly less glare than traditional systems, which are typically located 30–50 feet in the air. Because of the inverse squared relationship, a high mast system reduces glare by 75% compared with a traditional system.

Luminaires employing solid state technologies and light-emitting diodes (LED) provide light from an array of small sources rather than a single large source. These designs either rely on each small source to provide a component of the light distribution, or the components of the lighting array provide individual luminating fields of the light distribution. In the first instance, the arrays are typically flat and have an optic to provide the light distribution; if a single LED fails, the others still provide the light distribution. In the second method, the components of the array are aimed to different areas of the beam distribution. This approach typically results in light aimed at the driver and pedestrians causing a higher glare impact. The other issue with the multiple sources used in LED luminaires is that each of the sources typically has a very high luminance itself as the source is very small and very bright; in the absence of sufficient diffusion, they cause significant glare. Accordingly, solid state lighting systems typically have a higher glare impact than traditional sources.

The final issue with glare from overhead lighting is the cyclic nature of the impact. As drivers course along a roadway, they pass from one luminaire to another. The glare experience increases as they approach the luminaire and then diminishes as they pass beyond. While typically not an issue for disability glare, this repetitive process can cause discomfort and fatigue.³⁴

Opposing vehicle headlamps

Vehicle headlamps are aimed at the opposing driver eye level resulting in very high ocular illuminance and significant disability glare. The impact of opposing headlamps on the ability of the oncoming driver to observe beyond the headlamps is significant. For example, the visibility of a pedestrian standing behind a vehicle can be reduced by as much as 50%.³⁵

In order to minimize the glare impact, headlamps are designed with lower left side light intensity than the right side. This reduces the glare to an opposing vehicle but does not eliminate it. New technologies such as turning headlamps and headlamps that hide part of the headlamp beam when a vehicle passes are possible solutions for this issue. With the advent of high intensity discharge Xenon headlamps and LED-based technologies, the glare issue has become more serious. While the intensity towards a driver is limited, the small but brighter source generates a much higher impression of glare than traditional technologies. These "blue" headlamp sources have a higher complaint rate for glare than for any other light source.

Effects of Lighting Design on Traffic Accidents

Adult, and especially elderly drivers, experience increased glare sensitivity, and elderly drivers may not be able to sufficiently fulfill the criteria for night driving ability because of contrast and glare sensitivity.³⁶ Prospective studies indicate that reduction in the useful field of view, visual field loss, and glare sensitivity increase crash risk in older drivers.^{37,38} Crash risk begins to increase around age 50 years of age and continues to increase with aging.³⁹ No studies have explicitly compared traffic accident rates under different highway lighting conditions.

HEALTH EFFECTS OF DISRUPTED CIRCADIAN RHYTHMS

Epidemiological studies are a critical component of the evidence base required to assess whether or not light exposure at night affects disease risk, including cancer. These studies, however, are necessarily observational and can rarely provide mechanistic understanding of the associations observed. Carefully designed and controlled basic laboratory studies in experimental animal models have the potential to provide the empiric support for a causal nexus between light exposure at night and biological/health effects and to help establish plausible mechanisms. One area of considerable study on the possible effects of nighttime light exposure involves cancer.

CANCER

Light at Night, Melatonin and Circadian Influences on Carcinogenesis

Experimental Evidence. The majority of earlier studies in experimental models of either spontaneous or chemicallyinduced mammary carcinogenesis in mice and rats demonstrated an accelerated onset of mammary tumor development accompanied by increased tumor incidence and number in animals exposed to constant bright fluorescent light during the night as compared with control animals maintained on a strict 12 hours light/12 hours dark cycle.⁴⁰⁻⁵¹ More recent work has focused on the ability of light at night to promote the growth progression and metabolism in human breast cancer xenografts. Nocturnal melatonin suppresses the growth of both estrogen receptor negative (ER-) and estrogen receptor positive (ER+) human breast cancer xenografts; the essential polyunsaturated fatty acid, linoleic acid is necessary for the growth of such (ER-) tumors, and its metabolism can be used as a biomarker of cellular growth.⁵²⁻⁵⁵ Exposure of rats with such cancer xenografts to increasing intensities of white, fluorescent polychromatic light during the 12 hour dark phase of each daily cycle results in a dose-dependent suppression of peak nocturnal serum melatonin levels and a corresponding marked increase in tumor metabolism of linoleic acid and the rate of tumor growth. Exposure to even the very dimmest intensity of light during the night (0.2 lux) suppressed the nocturnal peak of circulating melatonin by 65% and was associated with marked stimulation in the rates of tumor growth and linoleic acid metabolic activity. In this model, measurable effects on xenograft growth and linoleic acid metabolism were apparent with 15% suppression in nocturnal melatonin levels.

The ability of light exposure at night to stimulate tumor growth (including dim exposures) has been replicated in rat hepatoma models.^{54,56-58} The reverse also is true; gradually restoring circulating melatonin by reducing initial exposure to light at night (24.5 lux) is accompanied by a marked reduction in tumor growth and linoleic acid metabolic activity to baseline rates in the breast cancer and hepatoma models.⁵⁹

The important role of melatonin as a nocturnal anticancer signal is further supported by the growth responses of human breast cancer xenografts perfused with human whole blood collected from young, healthy premenopausal female subjects exposed to complete darkness at night (e.g., high melatonin), compared with xenografts that were perfused with blood collected from the same subjects during the daytime (e.g., low melatonin).⁵⁴ The growth of xenografts perfused with blood collected during the dark was markedly reduced. Addition of a physiological nocturnal concentration of melatonin to blood collected from light-treated subjects restored the tumor inhibitory activity to a level comparable to that observed in the melatonin-rich blood collected during darkness. Moreover, the addition of a melatonin receptor antagonist to the blood collected during darkness (i.e., high melatonin) eliminated the ability of the blood to inhibit the growth and metabolic activity of perfused tumors. Some evidence also exists that circadian disruption by chronic phase advancement (e.g., simulating jet lag) may increase cancer growth in laboratory animals.^{60,61}

Potential Anticancer Mechanisms of Melatonin

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.^{62,63} It has been postulated that disruption in the phasing/timing of the central circadian pacemaker in the SCN, in general, and the suppression of circadian nocturnal production of melatonin, in particular, by light at night, may be an important biological explanation for the observed epidemiological associations of cancer risk and surrogates for nocturnal light exposure (such as night shift work, blindness, reported hours of sleep, etc.) (see below).⁶⁴

Melatonin exerts several cellular effects that may be relevant in this regard. It exhibits antiproliferative and antioxidant properties, modulates both cellular and humoral responses, and regulates epigenetic responses.⁶⁵⁻⁶⁷ Melatonin also may play a role in cancer cell apoptosis and in inhibiting tumor angiogenesis.^{68,69}

Human Studies

While the experimental evidence from rodent cancer models links disruption of circadian rhythms and circulating melatonin concentrations (inversely) with progression of disease, the human evidence is indirect and based on epidemiological studies. Breast cancer has received the most study.

The hypothesis that the increasing use of electricity to light the night might be related to the high breast cancer risk in the industrialized world, and the increasing incidence and mortality in the developing world was first articulated in 1987.⁷⁰ Potential pathways include suppression of the normal nocturnal rise in circulating melatonin and circadian gene function.^{54,71,72} Conceptually, this theory would predict 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 population nighttime light level would co-distribute with breast cancer incidence worldwide.^{72,73} Only the first hypothesis has been systematically evaluated. 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" (Recommendation Level 2A).⁷⁴ A detailed review of the individual studies supporting this conclusion is available.⁷⁵

Since the IARC evaluation was conducted, several new studies of breast cancer and nighttime light have been published with mixed results.⁷⁶⁻⁷⁹ Two found no significant association between shift work and risk of breast cancer.^{76,77} A large case-control study of nurses in Norway⁷⁸ found a significantly elevated risk in subjects with a history of regularly working five or more consecutive nights between days off, and another found that as the type of shift (e.g., evening, night, rotating) became more disruptive, the risk increased.^{79,80} In the Nurses Health Study cohort, increased urinary excretion of melatonin metabolites also was associated with a lower risk of breast cancer.⁸¹ Each of these studies has strengths and limitations common to epidemiology, particularly in exposure assessment and appropriate comparison groups (e.g., no woman in the modern world is unexposed to light-at-night, but quantifying that exposure is difficult).

Although shiftwork represents the most extreme example of exposure to light at night and circadian disruption, perturbation of circadian rhythms and the melatonin signal is also experienced by non- shift workers with a normal sleep/wake-cycle.¹² Anyone exposing themselves to light after dusk or before dawn is overriding the natural light-dark exposure pattern as noted in the earlier discussion on measures of illumination.

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, although long-term chronic exposure might. Four case-control studies have now reported an association of some aspect of nighttime light level in the bedroom with breast cancer risk.⁸²⁻⁸⁵ The elevated risk estimate was statistically significant in two of them.^{83,85} As case-control designs, in addition to the limitation of recall error, there is also the potentially significant 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 is important. The lower limit of light intensity that could, over a long time period, affect the circadian system is not established. 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 only in some individuals.⁸⁶ Frequent awakenings with low level light exposure in the bedroom and certain nighttime activities (e.g., bathroom visits) may disrupt the circadian system, but any related health effects are unknown.⁸⁷

Other Cancers

Light-at-night and circadian disruptions have been suggested to play a role in other cancers including endometrial, ovarian, prostate, colorectal, and non-Hodgkins lymphoma but evidence comparable to that obtained for breast cancer has not yet been developed.⁸⁸ On the other hand, engaging in night shift work may protect against skin cancer and cutaneous melanoma.⁸⁹

Other Diseases

<u>Obesity</u>, <u>Diabetes</u>, and <u>Metabolic Syndrome</u>. The modern world has an epidemic of obesity and diabetes that may be influenced by lack of sleep, lack of dark, and/or circadian disruption.⁹⁰ Non-day shift workers have a higher incidence of diabetes and obesity.⁹¹ Epidemiological studies also show associations of reported sleep duration and risk of obesity and diabetes.⁹² Circadian disruption may be a common mechanism for these outcomes and potential links between the circadian rhythm and metabolism.^{93,95}

<u>Other Disorders</u>. Although in the early stage of development, emerging evidence suggests that other chronic conditions also may be exacerbated by light at night exposure and ongoing disruption of circadian rhythms, including depression and mood disorders, gastrointestinal and digestive problems, and reproductive functions.⁸⁸

DARK VERSUS SLEEP

The circadian rhythm and sleep are intimately related but not the same thing. 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 required to synchronize the endogenous circadian rhythm, whereas a stable 24-hour light-dark cycle is

required. The epidemiological and laboratory research on sleep and health cannot entirely separate effects of sleep duration from duration of exposure to dark, because the sleep-wake cycle partitions light-dark exposure to the SCN and pineal gland.⁹⁶ The distinction is 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; many individuals normally experience a wakeful episode in the middle of a dark night.⁸⁷

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 being intensively investigated.⁹⁷ A growing number of observational and clinical studies on sleep and metabolism suggest short sleep periods have substantial harmful effects on health; however, it is not yet clear that sleep and dark have been entirely disentangled in these studies.^{97,98} For example, in one study, sleep duration (verified by polysomnography) was associated with morning blood levels of leptin, a hormone that plays a key role in energy expenditure and appetite.⁹⁹ However, the duration of typical sleep reported by each subject was more strongly associated with leptin concentrations. Mean verified sleep was 6.2 hours, whereas mean reported sleep was 7.2 hours. Reported "sleep duration" probably reflects the time from when a person turns out their light for bed and falls asleep and when they get up in the morning (i.e., actual hours of dark exposure). An important question is to determine what portion of the 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.

Media use at night (i.e., televisions, computer monitors, cell phone screens) negatively affects the sleep patterns of children and adolescents and suppresses melatonin concentrations. ¹⁰⁰⁻¹⁰² The American Academy of Pediatrics recommends removing televisions and computers from bedrooms to assist in limiting total "screen time" on a daily basis.¹⁰¹ This action also may help in improving sleep patterns.

ENERGY COST

Electric lighting accounts for about 19% of electricity consumption worldwide and costs about \$360 billion.¹⁰³ Much of the light that is produced is wasted, for example, by radiating light 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.¹⁰⁴ Such a percentage worldwide would account for an annual cost of about \$100 billion.

ENVIRONMENTAL ISSUES

Although not directly under the purview of human health and disease, the following considerations are indirectly related to human well-being.

Esthetics

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 of the innumerable stars from Earth's surface.¹⁰⁵ This has been carefully documented in a cover story by National Geographic Magazine in November 2008, which includes extensive visual documentation on its website.¹⁰⁶ Though the major impact of electric light at night is in major metropolitan areas, even the once pristine nights of the US National Parks are beginning to be degraded, more rapidly in the East but also in parks in the West as well.¹⁰⁷

Impact on Wildlife

Life on the planet has evolved to accommodate the 24-hour solar cycle of light and dark. Human imposition of light at night and disruption of the natural dark-light cycle represents a dramatic change to the environment.¹⁰⁸ Study of the effects of light at night on animal and plant life is in the early stages, but the entire spectrum of life, including animal, plant, insect, and aquatic species, may be affected.

About 30% of all vertebrate species and 60% of invertebrate species on Earth are nocturnal and depend on dark for foraging and mating.¹⁰⁸ Documented wildlife destruction by light at night has been evident in bird species, which fly into lit buildings at night in enormous numbers when migrating, and in the disruption of migration and breeding cycles in amphibians.¹⁰⁸⁻¹¹¹ The most studied case in reptiles involves 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.¹⁰⁹ Furthermore, many billions of insects are lost to electric light annually, which reduces food availability for other species in addition to unnecessarily reducing living biomass. It is concerning that light at night also may be vector attractant for diseases such as malaria.¹¹²

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.^{113,114}

POLICY AND PUBLIC HEALTH IMPLICATIONS OF LIGHT AT NIGHT

Some responses to public health concerns associated with light-at-night exposures are readily apparent, such as developing and implementing technologies to reduce glare from vehicle headlamps and roadway lighting schemes, and developing lighting technologies at home and at work that minimize circadian disruption, while maintaining visual efficiency and aesthetics. Additionally, clinical studies support efforts to reduce child and adolescent night-time exposure from exogenous light derived from various media sources, especially in the bedroom environment. Recommendations to use dim lighting in residences at night raise issues for elderly patients. The American Geriatrics Society recommends ensuring well lit pathways within households to reduce the incidence of falls in elderly patients.

Individuals who are subject to shift work experience disrupted circadian rhythms, fatigue, and cognitive dysfunction. Many industries, including hospitals, require a 24-hour workforce. The American College of Occupational and Environmental Medicine has established guidelines to address fatigue risk management in the workplace.¹¹⁶ In healthcare workers, such as nurses who experience rapidly rotating shifts, brief morning light exposure improves subjective symptoms and performance.¹¹⁷ The judicious use of bright light and/or melatonin supplements can improve adaptation to permanent, long-term night work.¹¹⁸

SUMMARY AND CONCLUSIONS

The natural 24-hour cycle of light and dark helps maintain precise alignment of circadian biological rhythms, the general activation of the central nervous system and various biological and cellular processes, and entrainment of melatonin release from the pineal gland. Pervasive use of nighttime lighting disrupts these endogenous processes and creates potentially harmful health effects and/or hazardous situations with varying degrees of harm. The latter includes the generation of glare from roadway, property, and other artificial lighting sources that can create unsafe driving conditions, especially for older drivers. Current AMA policy advocates that all future outdoor lighting be of energy efficient designs to reduce energy use and waste. Future streetlights should incorporate fully shielded or similar non-glare design to improve the safety of our roadways for all, but especially vision impaired and older drivers.

More direct health effects of nighttime lighting may be attributable to disruption of the sleep-wake cycle and suppression of melatonin release. Even low intensity nighttime light has the capability of suppressing melatonin release. In various laboratory models of cancer, melatonin serves as a circulating anticancer signal and suppresses tumor growth. Limited epidemiological studies support the hypothesis that nighttime lighting and/or repetitive disruption of circadian rhythms increases cancer risk; most attention in this arena has been devoted to breast cancer. The quality and duration of sleep and/or period of darkness affect many biological processes that are currently under investigation. Further information is required to evaluate the relative role of sleep versus the period of darkness in certain diseases or on mediators of certain chronic diseases or conditions including obesity. Due to the nearly ubiquitous exposure to light at inappropriate times relative to endogenous circadian rhythms, a need exists for further multidisciplinary research on occupational and environmental exposure to light-at-night, the risk of cancer, and exacerbation of chronic diseases.

RECOMMENDATIONS

The Council on Science and Public Health recommends that the following statements be adopted and the remainder of the report be filed:

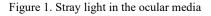
That our American Medical Association:

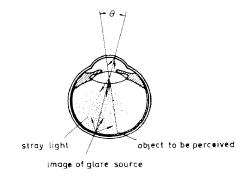
- 1. Supports the need for developing and implementing technologies to reduce glare from vehicle headlamps and roadway lighting schemes, and developing lighting technologies at home and at work that minimize circadian disruption, while maintaining visual efficiency.
- 2. Recognizes that exposure to excessive light at night, including extended use of various electronic media, can disrupt sleep or exacerbate sleep disorders, especially in children and adolescents. This effect can be minimized by using dim red lighting in the nighttime bedroom environment.
- 3. Supports the need for further multidisciplinary research on the risks and benefits of occupational and environmental exposure to light-at-night.
- 4. That work environments operating in a 24/7 hour fashion have an employee fatigue risk management plan in place.
- 5. That Policy H-135.937 be reaffirmed.

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APPENDIX





DeBoer Scale		
DeBoer Numerical Rating	Glare Intensity	
1	Unbearable	
3	Disturbing	
5	Just Admissible	
7	Satisfactory	
9	Unnoticeable	

Molecular and Cellular Basis for Photoreceptive Regulation of Circadian and Neuroendocrine System Function

In the past decade, there has been an upheaval in the understanding of photoreceptive input to the human circadian and neuroendocrine systems. A study on healthy human subjects confirmed that the three-cone system that mediates human vision during the daytime is not the primary photoreceptor system that transduces light stimuli for acute melatonin suppression.¹¹⁹ That discovery was rapidly followed by the elucidation of two action spectra in healthy human subjects that identified 446-477 nm as the most potent wavelength region for melatonin suppression.^{3,4} To date, ten published action spectra have examined neuroendocrine, circadian, and neurobehavioral responses in humans, monkeys, and rodents. The action spectra demonstrate peak sensitivities in the blue region of the visible spectrum, with calculated peak photosensitivities ranging from 459 nm to 484 nm.¹²⁰⁻¹²² Further, a set of studies has confirmed that shorter wavelength, monochromatic light is more potent than equal photon

densities of longer wavelength light for evoking circadian phase shifts, suppressing melatonin, enhancing subjective and objective correlates of alertness, increasing heart rate, increasing body temperature, and inducing expression of the circadian clock gene Per2 in humans.^{19,20,123-126}

Studies using both animal and human models are clarifying the neuroanatomy and neurophysiology of the photosensory system that provides input for circadian, neuroendocrine, and neurobehavioral regulation. A recently discovered photopigment, named melanopsin, has been localized both in the retinas of rodents and humans.¹²⁷ More specifically, melanopsin is found in a subtype of intrinsically photoreceptive retinal ganglion cells (ipRGCs).^{128,129} These light sensitive ganglion cells project to nuclei and regions of the central nervous system that mediate the biological and behavioral effects of light.^{130,131} Although ipRGCs provide the strongest input for regulation of biology and behavior, studies on genetically manipulated rodents, normal monkeys, and humans demonstrate that the visual rod and cone photoreceptors integrate into this physiology.^{5,132-134} Continued advances in understanding the physiology of this phototransduction will undoubtedly yield further insights into potential health impacts of electric lighting.

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5. TAXES ON BEVERAGES WITH ADDED SWEETENERS (RESOLUTION 417-A-11)

Reference committee hearing: see report of <u>Reference Committee D</u>.

HOUSE ACTION: RECOMMENDATIONS ADOPTED IN LIEU OF RESOLUTIONS 417-A-11 AND 407 AND REMAINDER OF REPORT FILED See Policy H-150.933

INTRODUCTION

Resolution 417-A-11, "Taxes on Beverages with Added Sweeteners," introduced by the Oklahoma Delegation at the 2011 American Medical Association (AMA) Annual Meeting and referred to the Board of Trustees, asks:

That our AMA: (1) support the adoption of a state tax on sugar-sweetened soft drinks with a substantial portion of the revenue from these taxes to be earmarked for the prevention and treatment of obesity; (2) work for and encourage all levels of the Federation and other interested groups to pass a tax on sugar sweetened beverages at the municipal and state levels; and, (3) work with its national partners and Federation members on developing and implementing a national strategy to pass municipal and state taxes on sugar sweetened beverages.

Two reports by the Board of Trustees previously addressed this issue at the 2006 Annual and Interim Meetings. Both reports recommended that the AMA support adoption of small local, state, and federal taxes on soft drinks sweetened with caloric sugars, with a substantial portion of the revenue from these taxes being earmarked for the prevention and treatment of obesity, as well as public health and medical programs that serve vulnerable populations. However, these recommendations were not adopted.

This report examines literature that has emerged in the last six years, as well as some earlier studies, to determine if limiting consumption of beverages with added sweeteners is likely to improve health outcomes; and, if so, whether taxation of sweetened beverages would be an effective public health strategy to help reduce consumption.

CURRENT AMA POLICY RELATED TO TAXES AND BEVERAGES WITH ADDED SWEETENERS

Current AMA policy does not directly address taxes on food or beverages, other than alcohol. However, several AMA policies support public health efforts to promote the consumption of naturally nutritious beverages and to discourage consumption of added sweeteners and of beverages high in calories and naturally low in other nutrients, particularly as obesity reduction strategies (Appendix). AMA policies also support taxes as a public health strategy to discourage consumption of alcohol and tobacco products and to use the resultant funds for health education, disease treatment, and counter-advertising efforts.