

REPORT 4 OF THE COUNCIL ON SCIENCE AND PUBLIC HEALTH (A-12)

Light Pollution: Adverse Health Effects of Nighttime Lighting

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EXECUTIVE SUMMARY

Objective. To evaluate the impact of artificial lighting on human health, primarily through disruption of circadian biological rhythms or sleep, as well as the impact of headlamps, nighttime lighting schemes, and glare on driving safety. Concerns related to energy cost, effects on wildlife and vegetation, and esthetics also are 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).

Results. 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. In addition to resetting the circadian pacemaker, light also stimulates additional neuroendocrine and neurobehavioral responses including suppression of melatonin release from the pineal gland improving alertness and performance. Low levels of illuminance in the blue or white fluorescent spectrum disrupt melatonin secretion. The primary human concerns with nighttime lighting include disability glare (which affects driving and pedestrian safety) and various health effects. Among the latter are potential carcinogenic effects related to melatonin suppression, especially breast cancer. Other diseases that may be exacerbated by circadian disruption include obesity, diabetes, depression and mood disorders, and reproductive problems.

Conclusion. 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. 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. 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 effects on various chronic diseases

1 INTRODUCTION

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

16 METHODS

17
18 English-language reports in humans were selected from a PubMed search of the literature from
19 1995 to March 2012 using the MeSH terms “circadian/biological clocks/rhythm,”
20 “chronobiology/disorders,” “photoperiod,” “light/lighting” “sleep,” “work schedule,” or
21 “adaptation,” combined with the terms “physiology,” “melatonin,” “adverse effects/toxicity,”
22 “pathophysiology,” “neoplasm,” “epidemiology/etiology,” “mental disorders,” “energy
23 metabolism,” and “gene expression.” Additional articles were identified by manual review of the
24 references cited in these publications; others were supplied by experts in the field who contributed
25 to this report (see Acknowledgement).

26 LIGHT AND HUMAN PHYSIOLOGY

27
28 The solar cycle of light and dark provides the essential basis for life on Earth. Adaptation to the
29 solar cycle has resulted in fundamental molecular and genetic endogenous processes in virtually all
30 life forms that are aligned with an approximately 24-hour period (circadian biological rhythm).
31 The circadian genetic clock mechanism is intimately involved in many, if not most, facets of
32 cellular and organismal function.¹ Although the circadian system spontaneously generates near-24-
33 hour rhythms, this master clock must be reset daily by the light-dark cycle to maintain proper
34 temporal alignment with the environment. In humans and other mammals, this daily entrainment is
35 achieved primarily by novel photoreceptors that project directly to the site of the circadian clock
36 (suprachiasmatic nuclei (SCN) of the hypothalamus).²⁻⁵ The tandem development of an endogenous
37 rhythm sensitive to light presumably evolved to allow for precise 24-hour regulation of rest and
38 activity, and for adapting to seasonal changes in night-length, while maintaining the advantages of
39 an underlying physiology that anticipates day and night. Understanding the molecular and
40
41

1 physiological basis of endogenous rhythms, how light information is communicated, and the health
2 implications of disruptions to this system are topics of intensive study.

3 4 ELECTRIC LIGHTING AND HUMAN HEALTH

5
6 Biological adaptation to the sun has evolved over billions of years. The power to artificially
7 override the natural cycle of light and dark is a recent event and represents a man-made self-
8 experiment on the effects of exposure to increasingly bright light during the night as human
9 societies acquire technology and expand industry. At the same time, increasing numbers of people
10 work inside buildings under electric lighting both night and day. Artificial lighting is substantially
11 dimmer than sunlight and provides a very different spectral irradiance. Sunlight is strong at all
12 visible wavelengths, peaking in the yellow region, whereas electric lighting has either extreme
13 characteristic wavelength peaks (fluorescent) or exhibits a monotonic increase in irradiance as
14 wavelength lengthens (incandescent). In contrast to outdoor lighting conditions, much of the
15 modern world now lives and works in relatively dim light throughout the day in isolation from the
16 sun, with often poor contrast between night and day, even for those who live and work in sunny
17 environments.⁶

18
19 Extensive nighttime lighting is required for contemporary society and commerce. Therefore, it is
20 imperative to evaluate the unintended adverse health consequences of electric lighting practices in
21 the human environment, and determine their physiological bases so that effective interventions can
22 be developed to mitigate harmful effects of suboptimal light exposure. For example, engineers
23 have already developed less disruptive night lighting technologies, and continued progress in this
24 area is anticipated. That such technologies exist, however, does not guarantee that they will be
25 purchased, installed and properly implemented. The medical community and public can take the
26 lead on advocating a healthier environment, as illustrated by recent changes in public smoking
27 policies worldwide. As the research on the biology of circadian rhythms has advanced, the range
28 of potential disease connections due to disrupted circadian rhythms and sleep has expanded.

29 30 *Biological Impact of Light on Human Physiology*

31
32 Light is the most powerful stimulus for regulating human circadian rhythms and is the major
33 environmental time cue for synchronizing the circadian clock. In addition to resetting the circadian
34 pacemaker, light also stimulates additional neuroendocrine and neurobehavioral responses,
35 including suppression of melatonin release from the pineal gland, directly alerting the brain, and
36 improving alertness and performance.⁷⁻⁹ Melatonin is one of the most studied biomarkers of the
37 human physiological response to light.¹⁰ This substance is the biochemical correlate of darkness
38 and is only produced at night, regardless of whether an organism is day-active (diurnal) or night-
39 active (nocturnal). Conceptually, melatonin provides an internal representation of the
40 environmental photoperiod, specifically night-length. The synthesis and timing of melatonin
41 production requires an afferent signal from the SCN. Ablation of this pathway, which occurs in
42 some patients from upper cervical spinal damage, completely abolishes melatonin production.
43 Certain other circadian rhythms (e.g., cortisol, body temperature, sleep-wake cycles) do not depend
44 on this pathway and persist if the SCN pathway is damaged.

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

1
2 When light information fails to reach the SCN to synchronize the clock and its outputs, the
3 pacemaker reverts to its endogenous non-24-hour period (range 23.7-25.0 h). Consequently, the
4 timing of physiology and behavior that is controlled by the circadian system, for example the sleep-
5 wake cycle, alertness and performance patterns, the core body temperature rhythm, and melatonin
6 and cortisol production, becomes desynchronized from the 24-hour day.² The resultant clinical
7 disorder is termed “non-24-hour sleep-wake disorder” and is characterized by alternating episodes
8 of restful sleep, followed by poor night-time sleep and excessive day-time napping, as the non-24-
9 hour circadian pacemaker cycles in and out of phase with the 24-hour social day.¹¹ Another effect
10 of light exposure at night is the immediate suppression of melatonin production. Under natural
11 conditions, organisms would never be exposed to light during the night in substantial amounts and
12 would not experience melatonin suppression. Electric light, however, efficiently suppresses
13 melatonin at intensities commonly experienced in the home at night.¹²

14 15 *Measures of Illumination*

16
17 Luminous flux is the measure of the perceived power of light. The lumen is the standard
18 international unit of luminous flux, a measure of the total “amount” of visible light emitted by a
19 source, while illumination is a measure of how much luminous flux is spread over a given area
20 (intensity of illumination). One lux is equal to one lumen/m². Luminous flux measurements take
21 into account the fact that the human eye and visual system is more sensitive to some wavelengths
22 than others. The peak luminosity function is in the green spectral region; white light sources
23 produce far fewer lumens. To provide some perspective, the illuminance associated with a full
24 moon is less than 1 lux, versus 50 lux for a typically incandescent lit family room, 80 lux in a
25 narrower hallway, 325-500 lux for office lighting, 1,000 lux for an overcast day, and 32,000-
26 130,000 lux for direct sunlight.

27
28 Initially it was thought that bright light of at least 2,500-20,000 lux was needed to suppress
29 nighttime melatonin secretion or phase shift the melatonin rhythm (as in jet lag) in humans.¹³⁻¹⁵ It
30 is now established that when exposure of the human eye is carefully controlled, illuminance as low
31 as 5–17 lux of monochromatic green light or 100 lux of broadband white light can significantly
32 suppress melatonin in normal human volunteers.^{12,16-18} Similarly, circadian phase shifts of the
33 melatonin rhythm can be evoked with an illuminance of 5 lux of monochromatic blue light or <100
34 lux of white fluorescent light, however, exposure to red light is not disruptive.^{18,19} Typical lighting
35 in bedrooms in the evening after dusk (but before bedtime) can also suppress melatonin and delay
36 its nocturnal surge.¹² Acute enhancement of both subjective and objective measures of alertness
37 can be evoked with as little as 5 lux of monochromatic blue light.²⁰ Dose-response curves for
38 melatonin suppression by night-time light exposure to fluorescent light show that ~100 lux of light
39 induces 50% of the maximal response observed with 1,000-10,000 lux of light.^{18,21}

40 41 *Ocular Physiology Mediating Photic Effects*

42
43 Factors that alter the amount and spectral quality of light reaching the retina include gaze behavior
44 relative to a light source, age (of the ocular lens), and pupillary dilation. Once a light stimulus
45 reaches the retina, physiology within the retina and within the nervous system determines the
46 capacity of the stimulus to evoke circadian, neuroendocrine or neurobehavioral responses. This
47 physiology includes: 1) the sensitivity of the operative photopigments and photoreceptors; 2)
48 location of these photoreceptors within the retina; 3) the ability of the nervous system to integrate
49 photic stimuli spatially and temporally; and, 4) the state of photoreceptor adaptation.
50 In particular, both short and long-term photoreceptor adaptation can significantly modify the
51 biological and behavioral responses to light and acutely suppress melatonin in humans.²² For

1 example, a full week of daytime exposure to bright light (by daylight and/or indoor light boxes at ~
 2 5,000 lux) or a three-day period of exposure to moderate indoor lighting (200 lux) reduces an
 3 individual's sensitivity to light suppression of nighttime melatonin compared with exposure to dim
 4 indoor lighting (0.5 lux); similar dim light conditions also enhance circadian phase shifting.²³⁻²⁵
 5 Two hours of exposure to 18 lux of white incandescent light versus full dark exposure in a single
 6 evening modifies the sensitivity of an individual for light-induced melatonin suppression later that
 7 same night.²⁶ Hence, photoreceptor adaptation, like the other ocular and neural elements noted
 8 above, can significantly modify the biological and behavioral responses to light.¹⁶

9
 10 In general, photobiological responses to light are not all-or-none phenomena. In the case of acutely
 11 suppressing high nighttime levels of melatonin or phase-shifting the entire melatonin rhythm, light
 12 works in a dose-response fashion. Once threshold is exceeded, increasing irradiances of light elicit
 13 increasing acute plasma melatonin suppression or longer-term phase-shifts of the melatonin rhythm
 14 in healthy individuals.^{16,18,27} All humans, however, are not equally sensitive to light; significant
 15 individual differences exist in sensitivity to light for both neuroendocrine and circadian
 16 regulation.^{16,18} For a detailed description of the molecular and cellular basis for how
 17 photoreceptive input regulates circadian and neuroendocrine system function, see the Addendum.

18 19 HUMAN CONCERNS-DISABILITY AND DISCOMFORT GLARE

20
 21 Glare from nighttime lighting can create hazards ranging from discomfort to frank visual disability.
 22 Disability glare has been fairly well-defined based on the physiology of the human eye and
 23 behavior of light as it enters the ocular media. Discomfort glare is less well-defined and more
 24 subjective as it is not based on a physical response per se but rather a psychological response.
 25 Accordingly, the respective bases of (and research into) these two responses are fundamentally
 26 different.

27 28 *Disability Glare*

29
 30 Disability glare is unwanted and poorly directed light that temporarily blinds, causes poor vision by
 31 decreasing contrast, and creates an unsafe viewing condition, especially at night, by limiting the
 32 ability of the person to see. There are natural causes of disability glare, such as solar glare at sunset
 33 on a dirty windshield which can be lessened by cleaning the windshield. Unfortunately, nighttime
 34 glare while driving is not easily remedied. It is caused by the misapplication of luminaires that
 35 comprise the lighting design which are generally overly bright and unshielded, and/or sources of
 36 poorly directed light that enters the eye and scatters among ocular structures resulting in
 37 diminished contrast and impeded vision. Such effects dramatically worsen as the human eye ages,
 38 contributing to poor night vision and difficulty in driving at night for older drivers.

39
 40 Disability glare is caused by light scatter from ocular media.²⁸ As light enters the eye, it collides
 41 with cornea, lens, and vitreous humor, scattering photons and casting a veil of light across the
 42 retina²⁹⁻³¹ (see Figure 1). The veil of light reduces the contrast of the object that the driver is trying
 43 to see, having the same effect as increasing the background luminance of the object. This veiling
 44 light is represented by the term veiling luminance. Veiling luminance is directly related to the
 45 illuminance of the light source and inversely related to the square of the angle of eccentricity of the
 46 light source with an age dependent multiplier across the entire equation.²⁸ This means that the
 47 disability from a light source is lessened the farther the source is from the line of sight.^a

^a 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

1
2 Accordingly, proper design techniques and consideration for the glare caused by lighting systems
3 need to be considered. One of the primary difficulties, especially for roadways, is that the lighting
4 is not governed by a single jurisdiction. Roadway lighting may be designed properly and provide a
5 low level of glare; however lighting can emanate from adjacent properties, spilling out into the
6 roadway thus affecting the driver and overall performance and suitability of a lighting system.
7 Control over all environmental sources of nighttime lighting is therefore critical for the overall
8 control of disability glare.

9 10 *Discomfort Glare*

11
12 Discomfort glare is less well defined but emanates from a glare source that causes the observer to
13 feel uncomfortable. The definition of discomfort is not precise, and some research has shown that
14 a person's response to a glare source is based more on his/her emotional state than on the light
15 source itself. Discomfort glare may be based primarily on the observer's light adaptation level, the
16 size, number, luminance and location of the light sources in the scene.³²

17
18 Both overhead roadway lighting and opposing headlamps are involved with discomfort glare in the
19 driver. A numerical rating scale based on the dynamic nature of glare in simulations is available to
20 measure the discomfort level experienced by drivers (Appendix).³³ The overall impact of
21 discomfort glare on fatigue and driver safety remains an issue.

22
23 Lighting and Glare. Both discomfort and disability glare have specific impacts on the user in the
24 nighttime environment. Research has shown that both of these glare effects occur simultaneously.
25 Research also shows that the effects of the glare are cumulative, meaning that the glare from two
26 light sources is the sum of the glare from the individual light sources. As a result, every light
27 source within the field of view has an impact on the comfort and visual capability of the driver.

28 29 *Overhead lighting*

30
31 For overhead roadway lighting, design standards include a methodology for controlling the
32 disability glare through a ratio of the eye adaptation luminance to the veiling luminance caused by
33 the light source. As the veiling luminance is related to the illuminance the light source produces at
34 the eye, a roadway luminaire that directs light horizontally has a much greater effect on the driver
35 than a light source that cuts off the horizontal light. A trend towards flat glass luminaires, which
36 provide a cut off of light at horizontal angles, provides a lower level of both disability and
37 discomfort glare.

38
39 Decorative luminaires (e.g., acorn or drop lens) have a high level of horizontal light and typically
40 are used in areas where pedestrians are the primary roadway users. The horizontal light in this
41 situation is useful for facial recognition of a pedestrian, but it limits the driver's ability to perceive
42 other objects in the roadway. As a result, many cities are designing and installing two lighting
43 systems, one for the pedestrian and one for the roadway.

44 Luminaires employing solid state technologies and light-emitting diodes (LED) provide light from
45 an array of small sources rather than a single large source. These designs either rely on each small
46 source to provide a component of the light distribution, or the components of the lighting array
47 provide individual luminating fields of the light distribution. In the first instance, the arrays are

the inverse squared relationship, a high mast system reduces glare by 75% compared with a traditional system.

1 typically flat and have an optic to provide the light distribution; if a single LED fails, the others still
 2 provide the light distribution. In the second method, the components of the array are aimed to
 3 different areas of the beam distribution. This approach typically results in light aimed at the driver
 4 and pedestrians causing a higher glare impact. The other issue with the multiple sources used in
 5 LED luminaires is that each of the sources typically has a very high luminance itself as the source
 6 is very small and very bright; in the absence of sufficient diffusion, they cause significant glare.
 7 Accordingly, solid state lighting systems typically have a higher glare impact than traditional
 8 sources.

9
 10 The final issue with glare from overhead lighting is the cyclic nature of the impact. As drivers
 11 course along a roadway, they pass from one luminaire to another. The glare experience increases
 12 as they approach the luminaire and then diminishes as they pass beyond. While typically not an
 13 issue for disability glare, this repetitive process can cause discomfort and fatigue.³⁴

14
 15 Opposing vehicle headlamps

16
 17 Vehicle headlamps are aimed at the opposing driver eye level resulting in very high ocular
 18 illuminance and significant disability glare. The impact of opposing headlamps on the ability of
 19 the oncoming driver to observe beyond the headlamps is significant. For example, the visibility of
 20 a pedestrian standing behind a vehicle can be reduced by as much as 50%.³⁵

21
 22 In order to minimize the glare impact, headlamps are designed with lower left side light intensity
 23 than the right side. This reduces the glare to an opposing vehicle but does not eliminate it. New
 24 technologies such as turning headlamps and headlamps that hide part of the headlamp beam when a
 25 vehicle passes are possible solutions for this issue. With the advent of high intensity discharge
 26 Xenon headlamps and LED-based technologies, the glare issue has become more serious. While
 27 the intensity towards a driver is limited, the small but brighter source generates a much higher
 28 impression of glare than traditional technologies. These “blue” headlamp sources have a higher
 29 complaint rate for glare than for any other light source.

30
 31 *Effects of Lighting Design on Traffic Accidents*

32
 33 Adult, and especially elderly drivers, experience increased glare sensitivity, and elderly drivers
 34 may not be able to sufficiently fulfill the criteria for night driving ability because of contrast and
 35 glare sensitivity.³⁶ Prospective studies indicate that reduction in the useful field of view, visual
 36 field loss, and glare sensitivity increase crash risk in older drivers.^{37,38} Crash risk begins to increase
 37 around age 50 years of age and continues to increase with aging.³⁹ No studies have explicitly
 38 compared traffic accident rates under different highway lighting conditions.

39
 40 **HEALTH EFFECTS OF DISRUPTED CIRCADIAN RHYTHMS**

41
 42 Epidemiological studies are a critical component of the evidence base required to assess whether or
 43 not light exposure at night affects disease risk, including cancer. These studies, however, are
 44 necessarily observational and can rarely provide mechanistic understanding of the associations
 45 observed. Carefully designed and controlled basic laboratory studies in experimental animal
 46 models have the potential to provide the empiric support for a causal nexus between light exposure
 47 at night and biological/health effects and to help establish plausible mechanisms. One area of
 48 considerable study on the possible effects of nighttime light exposure involves cancer.

49
 50 **CANCER**

51

1 *Light at Night, Melatonin and Circadian Influences on Carcinogenesis*

2
3 Experimental Evidence. The majority of earlier studies in experimental models of either
4 spontaneous or chemically-induced mammary carcinogenesis in mice and rats demonstrated an
5 accelerated onset of mammary tumor development accompanied by increased tumor incidence and
6 number in animals exposed to constant bright fluorescent light during the night as compared with
7 control animals maintained on a strict 12 hours light/12 hours dark cycle.⁴⁰⁻⁵¹

8
9 More recent work has focused on the ability of light at night to promote the growth progression and
10 metabolism in human breast cancer xenografts. Nocturnal melatonin suppresses the growth of both
11 estrogen receptor negative (ER-) and estrogen receptor positive (ER+) human breast cancer
12 xenografts; the essential polyunsaturated fatty acid, linoleic acid is necessary for the growth of such
13 (ER-) tumors, and its metabolism can be used as a biomarker of cellular growth.⁵²⁻⁵⁵ Exposure of
14 rats with such cancer xenografts to increasing intensities of white, fluorescent polychromatic light
15 during the 12 hour dark phase of each daily cycle results in a dose-dependent suppression of peak
16 nocturnal serum melatonin levels and a corresponding marked increase in tumor metabolism of
17 linoleic acid and the rate of tumor growth. Exposure to even the very dimmest intensity of light
18 during the night (0.2 lux) suppressed the nocturnal peak of circulating melatonin by 65% and was
19 associated with marked stimulation in the rates of tumor growth and linoleic acid metabolic
20 activity. In this model, measurable effects on xenograft growth and linoleic acid metabolism were
21 apparent with 15% suppression in nocturnal melatonin levels.

22
23 The ability of light exposure at night to stimulate tumor growth (including dim exposures) has been
24 replicated in rat hepatoma models.^{54,56-58} The reverse also is true; gradually restoring circulating
25 melatonin by reducing initial exposure to light at night (24.5 lux) is accompanied by a marked
26 reduction in tumor growth and linoleic acid metabolic activity to baseline rates in the breast cancer
27 and hepatoma models.⁵⁹

28
29 The important role of melatonin as a nocturnal anticancer signal is further supported by the growth
30 responses of human breast cancer xenografts perfused with human whole blood collected from
31 young, healthy premenopausal female subjects exposed to complete darkness at night (e.g., high
32 melatonin), compared with xenografts that were perfused with blood collected from the same
33 subjects during the daytime (e.g., low melatonin).⁵⁴ The growth of xenografts perfused with blood
34 collected during the dark was markedly reduced. Addition of a physiological nocturnal
35 concentration of melatonin to blood collected from light-treated subjects restored the tumor
36 inhibitory activity to a level comparable to that observed in the melatonin-rich blood collected at
37 night during total darkness. Moreover, the addition of a melatonin receptor antagonist to the blood
38 collected during darkness (i.e., high melatonin) eliminated the ability of the blood to inhibit the
39 growth and metabolic activity of perfused tumors. Some evidence also exists that circadian
40 disruption by chronic phase advancement (e.g., simulating jet lag) may increase cancer growth in
41 laboratory animals.^{60,61}

42

1 *Potential Anticancer Mechanisms of Melatonin*

2
3 The preponderance of experimental evidence supports the hypothesis that under the conditions of
4 complete darkness, high circulating levels of melatonin during the night not only provide a potent
5 circadian anticancer signal to established cancer cells but help protect normal cells from the
6 initiation of the carcinogenic process in the first place.^{62,63} It has been postulated that disruption in
7 the phasing/timing of the central circadian pacemaker in the SCN, in general, and the suppression
8 of circadian nocturnal production of melatonin, in particular, by light at night, may be an important
9 biological explanation for the observed epidemiological associations of cancer risk and surrogates
10 for nocturnal light exposure (such as night shift work, blindness, reported hours of sleep, etc.) (see
11 below).⁶⁴

12
13 Melatonin exerts several cellular effects that may be relevant in this regard. It exhibits
14 antiproliferative and antioxidant properties, modulates both cellular and humoral responses, and
15 regulates epigenetic responses.⁶⁵⁻⁶⁷ Melatonin also may play a role in cancer cell apoptosis and in
16 inhibiting tumor angiogenesis.^{68,69}

17
18 *Human Studies*

19
20 While the experimental evidence from rodent cancer models links disruption of circadian rhythms
21 and circulating melatonin concentrations (inversely) with progression of disease, the human
22 evidence is indirect and based on epidemiological studies. Breast cancer has received the most
23 study.

24
25 The hypothesis that the increasing use of electricity to light the night might be related to the high
26 breast cancer risk in the industrialized world, and the increasing incidence and mortality in the
27 developing world was first articulated in 1987.⁷⁰ Potential pathways include suppression of the
28 normal nocturnal rise in circulating melatonin and circadian gene function.^{54,71,72} Conceptually,
29 this theory would predict that non-day shift work would raise risk, blind women would be at lower
30 risk, reported sleep duration (as a surrogate for hours of dark) would be inversely associated with
31 risk, and population nighttime light level would co-distribute with breast cancer incidence
32 worldwide.^{72,73} Only the first hypothesis has been systematically evaluated. Based on studies of
33 non-day shift occupation and cancer (mostly breast cancer) published through 2007, the
34 International Agency for Research on Cancer (IARC) concluded “shift-work that involves
35 circadian disruption is *probably carcinogenic* to humans” (Recommendation Level 2A).⁷⁴ A
36 detailed review of the individual studies supporting this conclusion is available.⁷⁵

37
38 Since the IARC evaluation was conducted, several new studies of breast cancer and nighttime light
39 have been published with mixed results.⁷⁶⁻⁷⁹ Two found no significant association between shift
40 work and risk of breast cancer.^{76,77} A large case-control study of nurses in Norway⁷⁸ found a
41 significantly elevated risk in subjects with a history of regularly working five or more consecutive
42 nights between days off, and another found that as the type of shift (e.g., evening, night, rotating)
43 became more disruptive, the risk increased.^{79,80} In the Nurses Health Study cohort, increased
44 urinary excretion of melatonin metabolites also was associated with a lower risk of breast cancer.⁸¹
45 Each of these studies has strengths and limitations common to epidemiology, particularly in
46 exposure assessment and appropriate comparison groups (e.g., no woman in the modern world is
47 unexposed to light-at-night, but quantifying that exposure is difficult).

48
49 Although shiftwork represents the most extreme example of exposure to light at night and circadian
50 disruption, perturbation of circadian rhythms and the melatonin signal is also experienced by non-
51 shift workers with a normal sleep/wake-cycle.¹² Anyone exposing themselves to light after dusk or

1 before dawn is overriding the natural light-dark exposure pattern as noted in the earlier discussion
2 on measures of illumination.

3
4 After lights out for bedtime, it is not yet clear whether the ambient background light from weak
5 sources in the bedroom or outside light coming through the window could influence the circadian
6 system; a brief exposure at these levels may not have a detectable impact in a laboratory setting,
7 although long-term chronic exposure might. Four case-control studies have now reported an
8 association of some aspect of nighttime light level in the bedroom with breast cancer risk.⁸²⁻⁸⁵ The
9 elevated risk estimate was statistically significant in two of them.^{83,85} As case-control designs, in
10 addition to the limitation of recall error, there is also the potentially significant limitation of recall
11 bias.

12
13 Despite the difficulty of gathering reliable information on bedroom light level at night, the
14 possibility that even a very low luminance over a long period of time might have an impact is
15 important. The lower limit of light intensity that could, over a long time period, affect the
16 circadian system is not established. In the modern world few people sleep in total darkness. When
17 eyelids are shut during sleep, only very bright light can penetrate to lower melatonin and only in
18 some individuals.⁸⁶ Frequent awakenings with low level light exposure in the bedroom and certain
19 nighttime activities (e.g., bathroom visits) may disrupt the circadian system, but any related health
20 effects are unknown.⁸⁷

21 22 *Other Cancers*

23
24 Light-at-night and circadian disruptions have been suggested to play a role in other cancers
25 including endometrial, ovarian, prostate, colorectal, and non-Hodgkins lymphoma but evidence
26 comparable to that obtained for breast cancer has not yet been developed.⁸⁸ On the other hand,
27 engaging in night shift work may protect against skin cancer and cutaneous melanoma.⁸⁹

28 29 *Other Diseases*

30
31 Obesity, Diabetes, and Metabolic Syndrome. The modern world has an epidemic of obesity and
32 diabetes that may be influenced by lack of sleep, lack of dark, and/or circadian disruption.⁹⁰ Non-
33 day shift workers have a higher incidence of diabetes and obesity.⁹¹ Epidemiological studies also
34 show associations of reported sleep duration and risk of obesity and diabetes.⁹² Circadian
35 disruption may be a common mechanism for these outcomes and potential links between the
36 circadian rhythm and metabolism.⁹³⁻⁹⁵

37
38 Other Disorders. Although in the early stage of development, emerging evidence suggests that
39 other chronic conditions also may be exacerbated by light at night exposure and ongoing disruption
40 of circadian rhythms, including depression and mood disorders, gastrointestinal and digestive
41 problems, and reproductive functions.⁸⁸

42 43 DARK VERSUS SLEEP

44
45 The circadian rhythm and sleep are intimately related but not the same thing. Adequate daily sleep
46 is required for maintenance of cognitive function and for a vast array of other capabilities that are
47 only partially understood. Sleep is not required to synchronize the endogenous circadian rhythm,
48 whereas a stable 24-hour light-dark cycle is required. The epidemiological and laboratory research
49 on sleep and health cannot entirely separate effects of sleep duration from duration of exposure to
50 dark, because the sleep-wake cycle partitions light-dark exposure to the SCN and pineal gland.⁹⁶
51 The distinction is important because a requirement for a daily and lengthy period of dark to

1 maintain optimal circadian health has different implications than a requirement that one must be
 2 asleep during this entire period of dark; many individuals normally experience a wakeful episode in
 3 the middle of a dark night.⁸⁷

4
 5 Light during the night will disrupt circadian function as well as sleep, and the health consequences
 6 of short sleep and of chronic circadian disruption are being intensively investigated.⁹⁷ A growing
 7 number of observational and clinical studies on sleep and metabolism suggest short sleep periods
 8 have substantial harmful effects on health; however, it is not yet clear that sleep and dark have been
 9 entirely disentangled in these studies.^{97,98} For example, in one study, sleep duration (verified by
 10 polysomnography) was associated with morning blood levels of leptin, a hormone that plays a key
 11 role in energy expenditure and appetite.⁹⁹ However, the duration of typical sleep reported by each
 12 subject was more strongly associated with leptin concentrations. Mean verified sleep was 6.2
 13 hours, whereas mean reported sleep was 7.2 hours. Reported “sleep duration” probably reflects the
 14 time from when a person turns out their light for bed and falls asleep and when they get up in the
 15 morning (i.e., actual hours of dark exposure). An important question is to determine what portion
 16 of the health effects of dark disruption is due to sleep disruption and what portion is due directly to
 17 circadian impact of electric light intrusion on the dark of night.

18
 19 Media use at night (i.e., televisions, computer monitors, cell phone screens) negatively affects the
 20 sleep patterns of children and adolescents and suppresses melatonin concentrations.¹⁰⁰⁻¹⁰² The
 21 American Academy of Pediatrics recommends removing televisions and computers from bedrooms
 22 to assist in limiting total “screen time” on a daily basis.¹⁰¹ This action also may help in improving
 23 sleep patterns.

24 25 ENERGY COST

26
 27 Electric lighting accounts for about 19% of electricity consumption worldwide and costs about
 28 \$360 billion.¹⁰³ Much of the light that is produced is wasted, for example, by radiating light into
 29 space away from the task or environment intended to be illuminated. Estimates of how much is
 30 wasted vary; one estimate from the International Dark-Sky Association is 30% in the United
 31 States.¹⁰⁴ Such a percentage worldwide would account for an annual cost of about \$100 billion.

32 33 ENVIRONMENTAL ISSUES

34
 35 Although not directly under the purview of human health and disease, the following considerations
 36 are indirectly related to human well-being.

37 38 *Esthetics*

39
 40 The Milky Way is no longer visible to the majority of people in the modern world. As societies
 41 have increasingly used electricity to light the night, it has become difficult to see more than a few
 42 of the innumerable stars from Earth's surface.¹⁰⁵ This has been carefully documented in a cover
 43 story by National Geographic Magazine in November 2008, which includes extensive visual
 44 documentation on its website.¹⁰⁶ Though the major impact of electric light at night is in major
 45 metropolitan areas, even the once pristine nights of the U.S. National Parks are beginning to be
 46 degraded, more rapidly in the East but also in parks in the West as well.¹⁰⁷

47 48 *Impact on Wildlife*

49
 50 Life on the planet has evolved to accommodate the 24-hour solar cycle of light and dark. Human
 51 imposition of light at night and disruption of the natural dark-light cycle represents a dramatic

1 change to the environment.¹⁰⁸ Study of the effects of light at night on animal and plant life is in the
2 early stages, but the entire spectrum of life, including animal, plant, insect, and aquatic species,
3 may be affected.

4
5 About 30% of all vertebrate species and 60% of invertebrate species on Earth are nocturnal and
6 depend on dark for foraging and mating.¹⁰⁸ Documented wildlife destruction by light at night has
7 been evident in bird species, which fly into lit buildings at night in enormous numbers when
8 migrating, and in the disruption of migration and breeding cycles in amphibians.¹⁰⁸⁻¹¹¹ The most
9 studied case in reptiles involves sea turtle hatchlings on the coast of Florida, which historically
10 have scurried from their nest directly to the ocean. With increased development along the coast,
11 and attendant increased electric lighting at night, these hatchlings become confused and often
12 migrate away from shore to the lights. Hundreds of thousands of hatchlings are believed to have
13 been lost as a result of this stray electric lighting at night in Florida.¹⁰⁹ Furthermore, many billions
14 of insects are lost to electric light annually, which reduces food availability for other species in
15 addition to unnecessarily reducing living biomass. It is concerning that light at night also may be
16 vector attractant for diseases such as malaria.¹¹²

17
18 The circadian biology of plants is as robust as animals, and the impact of light at night on plant life
19 may also be considerable due to the role of light in photosynthesis and the fact that many plants are
20 pollinated at night.^{113,114}

21 22 POLICY AND PUBLIC HEALTH IMPLICATIONS OF LIGHT AT NIGHT

23
24 Some responses to public health concerns associated with light-at-night exposures are readily
25 apparent, such as developing and implementing technologies to reduce glare from vehicle
26 headlamps and roadway lighting schemes, and developing lighting technologies at home and at
27 work that minimize circadian disruption, while maintaining visual efficiency and aesthetics.
28 Additionally, clinical studies support efforts to reduce child and adolescent night-time exposure
29 from exogenous light derived from various media sources, especially in the bedroom environment.
30 Recommendations to use dim lighting in residences at night raise issues for elderly patients. The
31 American Geriatrics Society recommends ensuring well lit pathways within households to reduce
32 the incidence of falls in elderly patients.¹¹⁵

33
34 Individuals who are subject to shift work experience disrupted circadian rhythms, fatigue, and
35 cognitive dysfunction. Many industries, including hospitals, require a 24-hour workforce. The
36 American College of Occupational and Environmental Medicine has established guidelines to
37 address fatigue risk management in the workplace.¹¹⁶ In healthcare workers, such as nurses who
38 experience rapidly rotating shifts, brief morning light exposure improves subjective symptoms and
39 performance.¹¹⁷ The judicious use of bright light and/or melatonin supplements can improve
40 adaptation to permanent, long-term night work.¹¹⁸

41 42 SUMMARY AND CONCLUSIONS

43
44 The natural 24-hour cycle of light and dark helps maintain precise alignment of circadian
45 biological rhythms, the general activation of the central nervous system and various biological and
46 cellular processes, and entrainment of melatonin release from the pineal gland. Pervasive use of
47 nighttime lighting disrupts these endogenous processes and creates potentially harmful health
48 effects and/or hazardous situations with varying degrees of harm. The latter includes the
49 generation of glare from roadway, property, and other artificial lighting sources that can create
50 unsafe driving conditions, especially for older drivers. Current AMA policy advocates that all
51 future outdoor lighting be of energy efficient designs to reduce energy use and waste. Future

1 streetlights should incorporate fully shielded or similar non-glare design to improve the safety of
2 our roadways for all, but especially vision impaired and older drivers.

3
4 More direct health effects of nighttime lighting may be attributable to disruption of the sleep-wake
5 cycle and suppression of melatonin release. Even low intensity nighttime light has the capability of
6 suppressing melatonin release. In various laboratory models of cancer, melatonin serves as a
7 circulating anticancer signal and suppresses tumor growth. Limited epidemiological studies
8 support the hypothesis that nighttime lighting and/or repetitive disruption of circadian rhythms
9 increases cancer risk; most attention in this arena has been devoted to breast cancer. The quality
10 and duration of sleep and/or period of darkness affect many biological processes that are currently
11 under investigation. Further information is required to evaluate the relative role of sleep versus the
12 period of darkness in certain diseases or on mediators of certain chronic diseases or conditions
13 including obesity. Due to the nearly ubiquitous exposure to light at inappropriate times relative to
14 endogenous circadian rhythms, a need exists for further multidisciplinary research on occupational
15 and environmental exposure to light-at-night, the risk of cancer, and exacerbation of chronic
16 diseases.

17 18 RECOMMENDATIONS

19
20 The Council on Science and Public Health recommends that the following statements be adopted
21 and the remainder of the report be filed:

22
23 That our American Medical Association:

- 24
25 1. Supports the need for developing and implementing technologies to reduce glare from vehicle
26 headlamps and roadway lighting schemes, and developing lighting technologies at home and at
27 work that minimize circadian disruption, while maintaining visual efficiency. (New HOD
28 Policy)
- 29
30 2. Recognizes that exposure to excessive light at night, including extended use of various
31 electronic media, can disrupt sleep or exacerbate sleep disorders, especially in children and
32 adolescents. This effect can be minimized by using dim red lighting in the nighttime bedroom
33 environment. (New HOD Policy)
- 34
35 3. Supports the need for further multidisciplinary research on the risks and benefits of
36 occupational and environmental exposure to light-at-night. (New HOD Policy)
- 37
38 4. That work environments operating in a 24/7 hour fashion have an employee fatigue risk
39 management plan in place. (New HOD Policy)
- 40
41 5. That Policy H-135.937 be reaffirmed. (Reaffirm HOD Policy)

Fiscal Note: Less than \$500

Acknowledgements

The Council gratefully acknowledges the following national experts who contributed to the content and development of this report: David Blask, PhD, MD (Tulane University School of Medicine); George Brainard, PhD (Jefferson Medical College); Ronald Gibbons, PhD (Virginia Tech); Steven Lockley, PhD (Brigham and Women's Hospital, Harvard Medical School); Richard

Stevens, PhD (University Connecticut Health Center); and Mario Motta, MD (CSAPH, Tufts Medical School).

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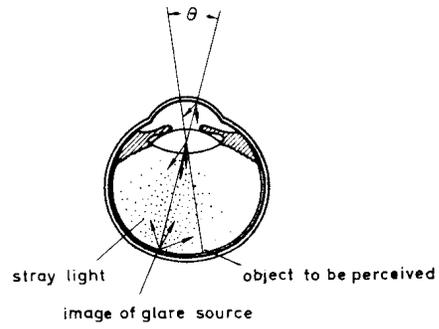
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Figure 1. Stray light in the ocular media



Appendix

DeBoer Scale

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

Addendum

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.