Invited Commentary

Invited Commentary: Nighttime Light Exposure as a Risk Factor for Obesity Through Disruption of Circadian and Circannual Rhythms

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In this issue of the American Journal of Epidemiology, McFadden et al. (Am J Epidemiol. 2014;180(3):245–250) report findings on the relationship between light exposure at night and obesity from a cross-sectional study of United Kingdom women. Their research extends findings from a previous study with elderly participants by including a larger sample size of over 100,000 women and a broader age range of 16 years or older. The findings are consistent with animal studies showing that prolonged light exposure leads to weight gain. Humans’ circadian, circannual, and metabolic regulatory systems evolved to be adaptive in environments that were quite different from those faced in modern industrial society. Technology has allowed exposures to levels and timing of light, nutrient intake, and physical activity never before possible. This commentary discusses how nighttime light exposure can increase the risk of obesity and the metabolic syndrome by disrupting circadian and circannual rhythms.

circadian rhythms; circannual rhythms; light at night; metabolic syndrome; obesity; sleep

Animals have evolved over millions of years to adapt to the solar day, which at the equator includes 12 hours of daylight and 12 hours of darkness (1). As the distance from the equator increases, the seasonal variation in light exposure increases to the extremes (at the poles) of continuous daylight in summer and continuous darkness in winter. Endogenous biological clocks evolved to synchronize activity, consumption, and rest to the circadian and circannual cycles using hormones and the autonomic nervous system. The suprachiasmatic nucleus, a tiny region in the hypothalamus, acts as the central biological clock, synchronizing multiple peripheral oscillators to optimally time metabolism, physiology, and behavior. Light exposure is the primary environmental signal that entrains circadian rhythms, but other periodical cues such as nutrient intake and physical activity also influence clock signaling (2). For our hunter-gatherer ancestors, the timing and periodicity of light exposure was tied to the rising and setting of the sun, which occurred in a precise and predictable fashion. Changes in the duration of daylight provided accurate signals of the impending seasons, with each season posing different environmental challenges requiring unique adaptive responses. The seasonal variations in light exposure, temperature, and food availability became more extreme as the distance from the equator increased. Summertime was synonymous with longer photoperiods, higher availability of food and simple sugars, shorter sleep durations, and more physical activity. These summertime environmental cues are theorized to trigger the seasonal expression of the “thrifty genotype” to facilitate the deposition of fat reserves through insulin resistance and other metabolic changes in preparation for wintertime food scarcity (3).

The ability to cycle insulin resistance and obesity in response to seasonal food availability is pervasive in the animal kingdom (4). In the extreme case of hibernating animals, large amounts of weight are gained in the late summer and fall, mainly through the accumulation of stores of triglycerides in adipose tissue (5). Since weight gain does not typically occur during the period of maximal food intake, variations in energy expenditure are likely to appreciably mediate the changes in body fat (6). The fat-storing phase is characterized by hyperinsulinemia and insulin insensitivity, but these changes are transitory and reverse themselves once hibernation begins. In winter there is a seasonal switch from carbohydrate-based metabolism to fat-based metabolism, with the stored fat, including large abdominal depots, providing the primary metabolic fuel for the entire winter hibernation season (5).

Although humans do not hibernate, genes have been identified in humans that are homologous to hibernation genes in
true hibernators (4, 7). Human responses to seasonal changes in the natural photoperiod were more robust prior to the Industrial Revolution and have since been increasingly suppressed by man-made alterations in the environment (4, 8). Seasonal changes in mood, with seasonal affective disorder being on the extreme end of the spectrum, have been theorized to have evolved to facilitate weight gain and the conservation of energy to endure winter food shortages and cold temperatures (9). Seasonal affective disorder typically begins in the fall when light exposure dwindles and is characterized by increased appetite, carbohydrate cravings, hypersomnia, lethargy, psychomotor retardation, and anhedonia. Increased appetite and carbohydrate cravings would increase the motivation to forage for and consume the remaining seasonally available fruits and carbohydrates. Hypersomnia, fatigue, psychomotor retardation, and anhedonia would reduce physical activity, slow metabolism, and decrease caloric expenditure. Seasonally depressed mood and behavior have been found to be significantly associated in humans with seasonal increases in body weight and waist circumference and diagnosis of the metabolic syndrome (10). The wintertime diet in colder climates for our hunter-gatherer ancestors would have been largely ketogenic, comprising more wild game, virtually no simple sugars, and a dearth of carbohydrates (when available, they would have had a low glycemic index). The herbivores that our ancestors hunted had wintertime fat reserves high in ω-3 fatty acids from grazing on foliage during warmer months (11). Our circadian, circannual, and metabolic regulatory systems evolved to be adaptive in environments that were quite different from those faced in modern industrial society.

Technology has allowed exposures to levels and timing of light, nutrient intake, and physical activity never before possible. Human exposure to artificial light at night began approximately 250,000 years ago when we discovered how to use fire. Light at night became more pervasive as oil lamps were introduced about 6,500 years ago, candles about 5,000 years ago, gas street lighting about 250 years ago, and electric lighting about 130 years ago (1). Electric lighting has gradually changed since the 1960s from an incandescent-bulb form consisting of mainly low-level yellow wavelengths to high-intensity discharge forms that contain blue wavelengths, with blue wavelengths having a more suppressive effect on circulating melatonin levels (12). Even with the lights off, our bedrooms are often aglow at night from luminous clocks, light-emitting diodes from electronic devices, and outside lighting that seeps through porous curtains and shades. We now have year-round access to tropical fruits and highly processed sweets anywhere on the globe. Modern conveniences have drastically diminished our need to engage in physical activity, but if we choose, we can vigorously exercise at night while basking in artificial light.

At first blush these common aspects of modern life seem innocuous or even beneficial, but these exposures can disrupt circadian and circannual rhythms that control metabolism and induce the expression of a summer phenotype favoring weight gain. Mice exposed to bright continuous lighting have been shown to have significantly decreased glucose tolerance and increased body mass compared with mice exposed to a standard light/dark cycle, despite equivalent levels of caloric intake and total daily activity (13). Exposing broiler chickens to constant light shifts metabolic efficiency, with chickens reared in constant light gaining significantly more weight (12, 14). The size, timing, and composition of nutrients have been shown to help entrain circadian rhythms in mice (15, 16). Mice that are active and consume meals during daylight, their usual resting period, have been shown to gain weight even with the same caloric intake (13). Glucose has been found to be a potent entrainer of the suprachiasmatic nucleus (17) and peripheral clocks (16) in rats. Fat loading that is short-lived in seasonally responsive animals is beneficial, but among humans in modern society, where the availability of food and simple sugars does not diminish in wintertime, the prolonged fat-loading response can be detrimental.

In this issue of the American Journal of Epidemiology, McFadden et al. (18) report findings from a cross-sectional study of United Kingdom women on the relationship between light exposure at night and obesity. They found a significant association between increasing levels of light at night and obesity as measured by body mass index, waist:height ratio, and waist circumference. Their study replicates the findings of an earlier cross-sectional study with elderly participants (19) that found the intensity of light at night to be significantly associated with obesity and dyslipidemia after controlling for covariates. The elderly participants with high exposure to light at night also had elevated, yet not statistically significant, odds of suffering from diabetes in multivariate models, with the lack of significance likely due to inadequate statistical power (19). One of the strengths of this earlier study was the use of objective measures of light exposure at night, as opposed to the current study, which relied upon self-reported light exposure. However, the earlier study had a relatively small sample size of 528 participants. The present study extends previous findings related to obesity by including a larger sample size of over 100,000 women and a broader age range of 16 years or older. The authors were able to control for a number of potential mediating and confounding variables in their multivariate models.

Variables theorized to mediate the relationship between nighttime light exposure and obesity include sleep duration, sleep quality, physical activity, and diet. Increased light intensity and prolonged light exposure could shorten sleep, and short sleep duration has been shown to be associated with obesity in children, adolescents, and adults. Experimental sleep restriction has been shown to decrease glucose tolerance, compromise insulin sensitivity (20), raise blood pressure (21), increase levels of total and low-density lipoprotein cholesterol (22), decrease leptin levels, increase ghrelin levels, and elevate appetite, with particular cravings for sweet, starchy, and salty snacks (23). Lack of sleep or poor-quality sleep could result in daytime fatigue that could decrease physical activity. Dietary variables, such as caloric and macronutrient content, and the timing of meals are likely to act as mediators in the relationship between nighttime light exposure and obesity. McFadden et al. were not able to control for poor sleep quality, sleep disturbances, timing of meals, or total caloric intake, but they were able to control for sleep duration and physical activity (18). The association between nighttime light exposure and obesity was not

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control, appetite ratings, dietary variables, sleep parameters, and metabolic measures such as glucose tolerance, insulin sensitivity, blood pressure, and cholesterol.

The results from McFadden et al.’s study (18) suggest that light exposure at night could play a role in the etiology of obe-
sity in some individuals. If metabolic changes resulting from nighttime light exposure are contributing to the obesity epide-
mic, it may be beneficial to take measures to limit this ex-
posure. Efforts are currently under way to decrease levels of urban sky glow through the use of light shields, reduction in the number of lights, and adjustment of the color spectrum produced by external lighting towards low-level red lighting and away from highly disruptive high-energy blue lighting (12). At an individual level, we can take measures to ensure that our bedrooms are dark at night by hanging blackout curtains and hiding or covering up light-emitting diodes and luminous clocks. Maintaining a dark sleeping environ-
ment is a commonly recommended sleep hygiene technique. Could the same advice help with maintaining or losing weight?

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