

## REVIEW ARTICLE

## Chronodisruption induced by artificial light exposure: Unraveling its molecular pathways and impacts on cardiometabolic dysfunctions

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## Abstract

Chronodisruption, defined as misalignment between endogenous circadian rhythms and external environmental cues, is increasingly implicated in cardiometabolic dysfunction, which elevates cardiovascular disease risk. Artificial light exposure, particularly during nighttime, disrupts the circadian clock by altering molecular pathways involving clock genes, melatonin secretion, and metabolic regulators. Animal studies have revealed that such disruptions exacerbate insulin resistance, dyslipidemia, and systemic inflammation—key risk factors for cardiovascular disease. Evidence also suggests impaired autonomic regulation, endothelial dysfunction, and altered myocardial metabolism due to circadian misalignment. Artificial light also alters other indices of cardiovascular function by raising blood pressure and the atherogenic index, both of which have been shown to hamper recovery from vascular and cardiovascular diseases. Despite these advances, knowledge gaps remain, including species-specific differences, long-term effects of varying light intensities, durations, and spectra, and translational applicability to humans. This review aims to elucidate several molecular pathways responsible for this disruption, and to examine how they interfere with physiological systems using animal studies and how they translate into clinical contexts. Bridging these gaps could inform novel preventive and therapeutic strategies targeting chronodisruption-induced cardiometabolic risks.

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## 1. Introduction

Cardiometabolic diseases—encompassing obesity, type 2 diabetes, hypertension, and cardiovascular disorders—are a leading cause of global morbidity and mortality, driven by complex genetic, environmental, and lifestyle factors.<sup>1,2</sup> Epidemiological data highlight rising prevalence rates, particularly in populations exposed to modern lifestyle disruptions, including artificial light at night (ALAN).<sup>1</sup> Mechanistically, these disorders stem from dysregulated glucose and lipid metabolism, endothelial dysfunction, chronic low-grade inflammation, and autonomic nervous system

imbalances. Pathophysiological consequences include atherosclerosis, myocardial dysfunction, and an increased risk of major adverse cardiovascular events. Occupational settings with prolonged artificial light exposure, such as shift work, intensify chronodisruption, amplifying cardiometabolic risk via altered sleep-wake cycles and impaired glucose metabolism.<sup>2</sup>

Physiological functions and processes exhibit rhythmic patterns that are governed by a set of proteins known as circadian proteins, located primarily in the suprachiasmatic nucleus (SCN) and present in all other living tissues.<sup>1,2</sup> These proteins include period circadian protein (PER), cryptochromes (CRY), circadian locomotor output cycles protein kaput (CLOCK), and brain and muscle ARNT-like protein 1 (BMAL-1), which are encoded by their respective circadian genes. CLOCK and BMAL-1 are positive regulators of PER and CRY. However, PER dimerizes with CRY to inhibit the transcription of CLOCK and BMAL-1 through a negative feedback loop.<sup>1,3-5</sup> In addition to endogenous regulation, ambient light induces PER expression. This implies that ambient light can entrain circadian proteins to natural cycles of day and night. However, constant light exposure during nighttime disrupts circadian mechanisms and causes desynchronization of internal rhythms, reduced ability to optimize energy utilization, and oxidative stress.<sup>1,3,6,7</sup> Besides being a synchronizer of circadian rhythms, light suppresses melatonin secretion. Melatonin suppression is wavelength-dependent: short-wavelength (blue) light is generally most suppressive, whereas longer wavelengths (e.g., red) tend to be less suppressive.<sup>1,8</sup> As a physiological stressor, light exposure causes perturbations of stress markers.<sup>9,10</sup>

In humans, shifts in the light/dark cycle characterizing shift work and chronic jet lag have been reported to suppress the expression of *PER1* and *PER2* in the SCN, causing delays in the acrophases of circadian expression of *PER1*, *PER2*, *BMAL1*, and D-site binding protein in the liver.<sup>11</sup> It is interesting to note that there is a difference between the expression patterns of circadian genes in the SCN and peripheral tissues. For instance, Yamazaki *et al.*<sup>12</sup> reported that the SCN rapidly adjusts to light shifts, but peripheral tissues shift more slowly. *PER2* in the ovary peaks at light offset, 4–6 h later relative to its expression in the SCN.<sup>13</sup> In addition, the duration of light exposure determines whether there will be shifts in circadian rhythm in both humans<sup>14</sup> and animals.<sup>15</sup>

Both *Per1* and *Per2* are generally involved in generating circadian rhythms in the SCN and affect other oscillations throughout the body. For example, *Per1* knockouts have been shown to affect food-entrainable oscillators and

methamphetamine-sensitive circadian oscillators, whose durations are altered in the absence of *Per1*.<sup>1</sup> In addition, mice with knockouts in both *Per1* and *Per2* genes show no circadian rhythmicity.<sup>16</sup> Light exposure causes increases in *Per1* mRNA, suggesting that the *Per1* gene plays an important role in entrainment of the mammalian biological clock to the light–dark cycle.<sup>1</sup> Expression of the *PER* gene, particularly *PER2*, a circadian protein expressed in all tissues, has been reported to be reduced in mammary cancer.<sup>17</sup>

Although light exposure suppresses melatonin secretion in all mammalian species, increasing evidence shows that the chronodisruptive effects of artificial light depend on light spectra, duration, exposure frequency, and mammalian species. Thus, investigating the interplay between chronodisruption and cardiometabolic disease pathogenesis is clinically relevant, as it may unveil novel preventive and therapeutic strategies targeting circadian alignment to mitigate metabolic and cardiovascular risks.

## 2. Methodology

A comprehensive literature search was performed using web-based databases, such as PubMed (the National Library of Medicine of the United States, which contains a database of medical and biomedical research literature), the Excerpta Medica Database, Web of Science, Scopus, and Google Scholar. Medical Subject Headings terms used to guide the search strategy were formed using the patient/population, intervention, comparison, and outcome criteria for evidence-based medicine investigations. Search terms included “artificial light” OR “artificial light at night” AND “cardio-metabolic dysfunctions” OR “hypertension” OR “diabetes” OR “hyperlipidemia” AND “circadian rhythm” OR “chronodisruption” OR “clock genes” OR “circadian misalignment.”

The review incorporated evidence from *in vitro* and *in vivo* investigations, as well as clinical trials. No restrictions were applied regarding year of publication or language. Following the removal of duplicate records, studies were initially evaluated through title and abstract screening. Articles deemed potentially relevant underwent full-text assessment, and the reference lists of included studies were examined to capture additional relevant literature. A manuscript review was conducted by two authors and a third reviewer to resolve any disagreements.

## 3. Current evidence

### 3.1. Disruption of normal rhythmic changes in blood pressure and heart rate in rats by artificial light exposure

Blood pressure and heart rate vary across a day in mammals. These rhythmic changes are lost in hypertension,

hypotension, bradycardia, and tachycardia.<sup>18</sup> ALAN exposure causes derangement in indices of cardiovascular function, resulting in hypertension, hypotension, bradycardia, and tachycardia depending on the mammalian species.<sup>19</sup> Unlike humans, who are diurnal (more active during the day), rats are generally nocturnal (more active at night and less active during the day and light-controlled night). Hence, they exhibit rhythms of cardiovascular function that differ from those of humans. Murine blood pressure and pulse rate are higher at night than during the day. Prolonged exposure of rats to light increases physical inactivity and causes sustained reduction in blood pressure (hypotension) and heart rate (bradycardia).<sup>20</sup>

Hilal-Dandan *et al.*<sup>20</sup> conducted a murine study to investigate the mechanism underlying ALAN-induced bradycardia and hypotension. They found that levels of left ventricle endothelin-I, a vasoconstrictor, and sarcoplasmic/endoplasmic reticulum calcium ATPase 2 (SERCA2), which pumps calcium ions from the cytosol into the sarcoplasmic reticulum, were reduced in normotensive rats and spontaneously hypertensive rats following 5 weeks of exposure to ALAN. Angiotensin II receptor type 1 expression was also found to be lower in both normotensive and spontaneously hypertensive rats after 2 weeks of artificial light exposure.<sup>21</sup> These findings suggest that ALAN exposure is associated with lower blood pressure, pulse rate, SERCA2, endothelin-1, and angiotensin II type 1 receptor expression. This implies that ALAN could limit the heart's ability to meet metabolic demands imposed by daily environmental changes, growth, development, and normal cellular activities.

Rumanova *et al.*<sup>22</sup> exposed adult male rats to ALAN at an intensity of 1–2 lux for 2 or 5 weeks. ALAN exposure caused depletion of light–dark rhythms of systolic blood pressure without affecting heart rate. The effect of ALAN on systolic blood pressure rhythms appeared to be more pronounced following 5 weeks of exposure. Using telemetry and other laboratory techniques, Mauer *et al.*<sup>23</sup> investigated the molecular mechanisms underlying ALAN-induced disruption of normal daily rhythms of blood pressure. Specifically, following 3 weeks of exposure to ALAN at an intensity of 1–2 lux, the daily rhythms of pulse pressure, the maximum value of acceleration rate of aortic pressure, and protein expression in the aorta of normotensive male rats were evaluated. ALAN reduced the daily rhythm of pulse pressure and maximum acceleration rate of aortic pressure, but ultradian rhythms of pulse pressure and maximum acceleration rate of aortic pressure were enhanced in a duration-dependent manner. SERCA2 was found to rise both in the dark phase of the day and in the middle of the light phase in rats that were

not exposed to ALAN. However, ALAN exposure did not have a significant effect on daily SERCA2 expression.

Molcan *et al.*<sup>24</sup> studied the effects of ALAN at 1–2 lux on circadian rhythms of blood pressure and heart rate in normotensive rats using telemetry. There was a reduction in circadian rhythms of blood pressure, heart rate, baroreflex sensitivity, and heart rate response to blood pressure changes following 2 weeks of exposure. In their study, ALAN at 1–2 lux for 5 weeks enhanced epinephrine-induced blood pressure response compared to the unchallenged group. Two weeks of exposure to ALAN at 1–2 lux increased endothelial nitric oxide synthase, but melatonin was suppressed following both two- and five-week exposures to ALAN at 1–2 lux. Endothelin levels were unaffected by ALAN at 1–2 lux following 2 and 5 weeks of exposure.

### 3.2. Obesogenic effects of artificial light: Evidence from animal studies

Body weight is the totality of body mass expressed in kilograms. Typically, the body is composed of approximately 60% water, 7% minerals, 15% protein, and 18% fats. Fat is the most dominant solid component of the body, and therefore, it is one of the major determinants of body weight in states of health. Obesity, caused by the accumulation of fat in the body, is a metabolic condition and a risk factor for diseases such as diabetes mellitus, hypertension, and cancer. There is increasing evidence from animal studies that artificial light exposure is a risk factor for obesity.

Fonken *et al.*<sup>25</sup> investigated the relationship between exposure to light at night and obesity using male mice. Mice were raised under a 12-h light/12-h dark regimen (control mice) or under bright and dim night lights. Compared to control mice, animals exposed to bright or dim light at night exhibited higher body mass. It was also reported that the timing of food consumption by control mice differed from that of animals exposed to bright or dim light. Specifically, mice under dim light conditioning consumed more than 50% of their food during the subjective light period. Since mice are nocturnal, active at night, and housed in dark-controlled environments, they normally consume the majority of their calories during the night. In this model, weight gain observed in mice exposed to dim or bright light at night was due to changes in the timing of food consumption, consumption of a larger proportion of food during the light phase, and periods of physical inactivity. When food consumption exceeds utilization, excess glucose is stored as fat in adipose tissue.

In a similar study by Babinec,<sup>26</sup> mice were classified into either control (12-h light/12-h dark), 12-h light/4-h

dim/8-h dark, 12-h light/4-h dark/4-h dim/4-h dark, and 12-h light/8-h dark/4-h dim groups. It was reported that dim light at night increased the percentage of weight gain. In another study by Fonken *et al.*,<sup>27</sup> mice were maintained under dim night light (5 lux). It was reported that a dim night light caused attenuation of *Per1* and *Per2* expression in the hypothalamus. Dim night light also reduced the rhythmic expression of circadian genes in the liver. Specifically, there was attenuation of REV-ERB (nuclear receptor subfamily I group D member 2) expression in the liver and adipose tissues, resulting in feeding pattern changes and weight gain.

Aubrecht *et al.*<sup>28</sup> maintained female mice in 16-h light at 150 lux/8-h dark at 0 lux (control) and 16-h light at 150 lux/8-h dim light at 5 lux (experimental group) for 6 weeks. Compared to the control group, mice exposed to dim light at night exhibited a higher rate of body mass gain. This suggests that dim light exposure at night leads to greater weight gain than bright light exposure.

Meléndez-Fernández *et al.*<sup>29</sup> investigated the effect of indiscriminate light exposure on body weight in male Swiss rodents. Exposure of mice to bright daylight (125 lux) and dark night (0 lux) resulted in lower weight gain compared to mice exposed to bright daylight (125 lux) and dim night light (5 lux), dim daylight with dark night (0 lux), or dim night light (5 lux). It was also found that mice exposed to dim night light shifted their food intake to the resting phase. This suggests that dim night light-induced weight gain is associated with a shift in feeding time, which leads to the accumulation of unused calories and storage as fat.

Kooijman *et al.*<sup>30</sup> reported that when mice were subjected to 16-h light/8-h dark or 24-h light/0-h dark, there was an increase in adipose tissue mass compared to animals in a 12-h light/12-h dark cycle. The increase in adiposity was independent of changes in food intake or physical activity. However, prolonged light exposure caused a reduction in autonomic discharge to brown adipose tissue, a type of adipose tissue that converts energy into heat. Specifically, there was a decrease in the sympathetic  $\beta$ 3-adrenergic pathway to brown adipose tissue. In addition, artificial light suppresses fatty acid uptake from triglyceride-rich lipoproteins, inhibiting the release of fatty acids into adipose tissue.

Sarmiento *et al.*<sup>31</sup> investigated the effect of ALAN on body weight and the underlying mechanisms in mice. Dim light at night was reported to increase body weight compared to mice exposed to a 12-h light/12-h dark regimen. Dim light at night also modulated the taxonomic composition of colonic microbiota. This suggests that changes in microbial metabolic pathways may contribute to artificial light-induced weight gain. Colonic microbiota

are known to play key roles in the synthesis of short-chain fatty acids, which induce insulin secretion, a hormone that stimulates lipogenesis and lipid storage.

Besides changes in the timing of food consumption, reduced uptake of fatty acids from triglyceride transporters, and alterations in colonic microbiota diversity, artificial light-induced body weight gain may be influenced by chemical messengers—including insulin, growth hormone, adrenocorticotrophic hormone, cortisol, leptin, and ghrelin.<sup>32</sup> Ghrelin exhibits rhythmic patterns, typically high during the day and low during the night. Exposure to light at night has been reported to alter nocturnal ghrelin secretion (direction varying across studies) and to reduce leptin secretion, a chemical messenger that suppresses appetite.<sup>33</sup>

Mondal *et al.*<sup>34</sup> evaluated the impact of artificial light on gene expression responsible for appetite using zebrafish. Specifically, the diurnal expression of leptin mRNA was studied in the brain of zebrafish under normal 12-h light/12-h dark, 24-h light/0-h dark, and 0-h light/24-h dark conditions for 72 h. It was reported that continuous illumination caused a reduction in leptin gene expression.

The likelihood of conscious exposure to ALAN is high during sleep deprivation. Schmid *et al.*<sup>35</sup> reported that plasma levels of ghrelin were elevated following a single night of sleep deprivation, which was associated with increased feelings of hunger. Schüssler *et al.*<sup>36</sup> investigated nighttime secretions of ghrelin, adrenocorticotrophic hormone, growth hormone, and cortisol before and after a night of sleep deprivation. Peak ghrelin secretion was observed during the first half of sleep and following sleep deprivation. Growth hormone levels were high after a night of sleep deprivation, while adrenocorticotrophic hormone and cortisol levels were high during the second half of the night.

Besides sleep deprivation, night workers are susceptible to artificial light exposure. Wan *et al.*<sup>37</sup> reported that early growth response 3 is expressed in human and mouse tissues. However, the protein is downregulated in adipose tissues of obese individuals and experimental mice that were fed with a high-fat diet. Glucocorticoid administration was shown to negatively modulate early growth response 3 protein. Glucocorticoids are known to induce weight gain by stimulating salt and water retention through mineralocorticoid activity. This finding suggests an additional pathway through which glucocorticoids may promote weight gain.

Fonken *et al.*<sup>38</sup> evaluated the effect of dim light exposure on experimental high-fat diet-induced obesity in male mice. In their study, mice were kept under a natural 12-h



light/12-h dark, dim light, or a high-fat diet for 4 weeks. Mice exposed to both dim light at night and a high-fat diet showed the highest weight gain, accompanied by altered insulin secretion, reduced glucose tolerance, changes in the timing of food consumption, increased tumor necrosis factor  $\alpha$  (TNF- $\alpha$ ), and elevated expression of the macrophage 1 (*Mac1*) gene in white adipose tissue. These findings suggest that dim light exposure enhances high-fat diet-induced body weight gain through these metabolic and inflammatory mechanisms.

Borniger *et al.*<sup>39</sup> investigated the effects of nighttime dim light exposure (5 lux) for 2 weeks on mice. Mice exposed to dim light during the night gained more body weight. They also exhibited reduced energy expenditure, increased carbohydrate oxidation relative to fat, and disrupted circadian rhythm of body temperature. These alterations in energy metabolism and circadian regulation are all potential mechanisms underlying artificial light-induced body weight gain. Fonken *et al.*<sup>38</sup> further hypothesized that dim night light, rather than complete darkness, affects body weight in mice. Indeed, mice exposed to dim nights, instead of completely dark nights, showed weight gain.

Kumar *et al.*<sup>40</sup> investigated the effect of dim night light on fat metabolism in adult male zebra finch birds (*Taeniopygia guttata*) that were hatched and raised under different lighting conditions. Exposure of birds to dim night light disrupted diurnal behavioral activity and feeding patterns, including increased nocturnal feeding, resulting in body weight gain and elevated nocturnal glucose levels.

Yonis *et al.*<sup>41</sup> examined the impact of artificial light exposure during the night on body weight. Body weight gain was higher in rats exposed to acute bright light at night compared to controls, and feeding and drinking behavior, as well as insulin and glucose levels, were altered in light-exposed animals.

In a study by Luo *et al.*,<sup>42</sup> high-fat diet-fed mice with null peroxisome proliferator-activated receptor (PPAR)  $\alpha$  and wild-type mice were exposed to neon light at night for 6 weeks. High-fat diet-fed wild-type mice exposed to neon light at night exhibited greater weight gain compared to high-fat diet-fed knockout mice exposed to neon light. The increase in weight was associated with larger adipocyte size, increased adipose tissue mass, elevated lipid accumulation in the liver, and enhanced lipid transfer from the liver to peripheral tissues. Expression of *Clock* genes in adipose tissues and liver was increased in wild-type mice exposed to neon light.

In a study conducted by Russart *et al.*,<sup>43</sup> male mice were subjected to ALAN, and metabolic symptoms of experimental type 2 diabetes mellitus were evaluated.

Exposure of seven-week-old male mice to nighttime light caused increased body weight compared to animals maintained under a normal 12-h daylight/12-h dark cycle.

The effect of ALAN on energy metabolism in male C57BL6/J mice was evaluated by Borck *et al.*<sup>44</sup> Eight weeks of exposure to ALAN resulted in elevated body weight, increased fat pad mass, and disruption of hepatic fibroblast growth factor 21 expression.

Okuliarova *et al.*<sup>45</sup> evaluated the role of dim light at 2 lux during the night on lipid metabolism in the epididymal fat pad and liver. Exposure to dim night light at night caused triacylglycerol accumulation in the liver, upregulation of liver genes responsible for endogenous fatty acid synthesis, and increased fatty acid uptake. Dim night light exposure also increased expression of PPAR family members  $\alpha$  and  $\gamma$  in adipose tissue and liver, representing one of the mechanisms underlying the obesogenic effects of artificial light (Figure 1).

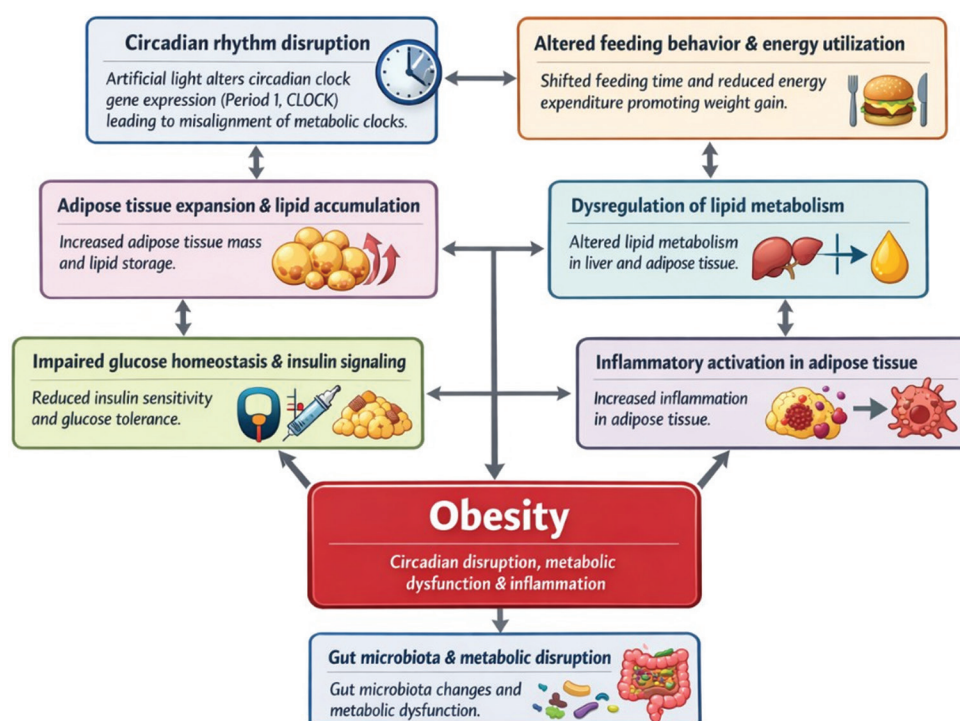
Coomans *et al.*<sup>46</sup> reported that exposure of C57BL/6J mice to continuous light increased feeding rate and decreased energy expenditure, culminating in body weight gain compared with mice maintained under a normal 12-h daylight/12-h dark cycle. In nestling songbirds, Raap *et al.*<sup>47</sup> demonstrated the impact of nighttime artificial light exposure on body mass and oxidative stress. Body weight increased in nestlings subjected to artificial night light without changes in the oxidant–antioxidant balance.

### 3.3. Artificial light-induced hyperglycemia and insulin resistance: Evidence from animal studies

Hyperglycemia is a metabolic disorder characterized by a sustained increase in blood glucose, indicating derangements in glucose homeostasis, the inability to maintain balance between glucose appearance in the blood (mediated by hyperglycemic hormones) and its removal (orchestrated by insulin, a hypoglycemic hormone). Insulin resistance is a reduced response of tissues to insulin.

In animal studies, the impact of artificial light on blood glucose has been documented. Fonken *et al.*<sup>25</sup> reported that mice maintained under bright or dim night-light conditions exhibited reduced glucose tolerance compared to animals maintained under a normal 12-h light/12-h dark cycle. The glucose tolerance test assesses the rate at which tissues take up glucose from the blood, a process facilitated by insulin. Therefore, these findings suggest that bright or dim night light impairs glucose homeostasis in mice.

Cheung *et al.*<sup>48</sup> examined the short-term impact of three-h morning or evening exposure to blue-enriched artificial light on metabolic parameters. The results



**Figure 1.** Mechanisms of artificial light-induced obesity. Image created by the authors.  
Abbreviation: CLOCK: Circadian locomotor oscillation cycles kaput.

indicated that peak glucose levels occurred when mice were exposed to blue-enriched artificial light in the evening. Moreover, Fonken *et al.*<sup>49</sup> reported that dim night light exposure in mice reduced glucose tolerance and altered insulin secretion.

Opperhuizen *et al.*<sup>50</sup> evaluated the effect of acute exposure to ALAN on glucose metabolism in rats. Exposure to ALAN caused an immediate reduction in glucose tolerance. It was also shown that exposure to green light at night induced glucose intolerance, whereas red and blue lights had no effect on glucose metabolism. The finding contradicts that of Cheung *et al.*,<sup>48</sup> likely due to differences in methodology.

Photopollution is a physiological stressor.<sup>51</sup> Stress-induced sympathetic activation is widely known to stimulate gluconeogenesis in the liver and alter the ability of beta islet cells to regulate glucose secretion. Russart *et al.*<sup>43</sup> hypothesized that exposure to ALAN would not worsen metabolic symptoms of experimental type 2 diabetes mellitus in mice. Seven-week-old male mice exposed to light at night (14-h daylight [150 lux]/10-h night dim light [40 lux] cycle) for 8 weeks exhibited reversibly exacerbated insulin resistance and glucose intolerance compared to animals in the control group (14-h daylight [150 lux]/10-h darkness [0 lux]).

In another study, Borck *et al.*<sup>44</sup> investigated the impact of ALAN exposure on energy metabolism in male C57BL/6J mice. Exposure to ALAN for 8 weeks resulted in glucose intolerance and increased fasting glucose levels. There was also a rise in phosphoenolpyruvate carboxykinase expression. Okuliarova *et al.*<sup>45</sup> examined the effect of dim light at night (2 lux) on metabolic pathways in the liver. Exposure to dim night light increased glucose uptake in the liver.

Coomans *et al.*<sup>46</sup> exposed male C57BL/6J mice to constant light and investigated its effect on insulin sensitivity and energy metabolism. Constant light exposure caused a complete loss of normal circadian variation in insulin sensitivity. Masís-Vargas *et al.*<sup>52</sup> investigated the immediate impact of blue light on glucose metabolism and feeding rate in high-fat-/high-sucrose-fed male and female Sudanian grass mice. One hour of exposure to artificial blue light during the dark period increased sucrose feeding, raised random glucose levels, and reduced plasma insulin levels.

Adeniyi *et al.*<sup>6</sup> reported that female rats exposed to a long lighting period (20-h light [120–150 lux]/4-h dark [3–20 lux]) exhibited elevated plasma glucose levels compared to controls (Wistar rats maintained under a natural 12-h light/12-h dark cycle). Kumar *et al.*<sup>40</sup> showed that exposure of adult male zebra finches to night dim light

(12-h daylight [150–200 lux]/12-h dim night light [4 lux]) resulted in higher nocturnal glucose levels compared to controls (12-h daylight [150–200 lux]/12-h darkness [0 lux]).

Zhang *et al.*<sup>53</sup> investigated whether the metabolic status of normal and high-fat diet-induced obese mice was affected by specific light wavelength. It was observed that exposure to green light induced glucose intolerance compared to mice maintained under white light. Zhu *et al.*<sup>54</sup> evaluated the blood glucose of zebra finch birds following brief exposure to light at night for 3 and 6 days. While blood glucose was reduced after 3 days of light-at-night exposure, glucose levels and the expression of genes associated with hyperglycemia were elevated after 6 days of exposure.

Rumanova *et al.*<sup>22</sup> investigated the influence of artificial light exposure on spontaneously hypertensive rats. There was an elevated expression of PPAR $\alpha$  and PPAR $\gamma$ , transcription factors involved in lipid metabolism. There was also an increase in epididymal fat and a reduction in glucose transporter 4 production in the heart.

The effect of dim ALAN (2 lux) on energy metabolism and gene expression in male Wistar rats was further assessed by Rumanova *et al.*<sup>22,32</sup> The animals were maintained under a 12-h daylight/12-h dark cycle or ALAN. Exposure to ALAN caused phase advancement of the glucose transporter 2 rhythm and loss of forkhead box O1 rhythm.

### 3.4. Dyslipidemic effect of artificial light

Lipids are important molecules for living cells. They form cell membranes, provide heat insulation, act as transmitters and chemical mediators, serve as energy substrates, and act as a reserve pool for excess glucose, fatty acids, and glycerol. Lipids are stored primarily in adipose tissues, particularly white adipose tissue, and are also present in steroidogenic tissues (such as gonads and adrenal glands) and in smaller quantities in other body tissues, including the liver. Lipids are transported through the blood by various transporters.<sup>55</sup>

Fatty acids with shorter carbon lengths are transported through facilitated diffusion, while fatty acids with longer carbon chains and cholesterol are transported by chylomicrons, very low-density lipoproteins, high-density lipoproteins (HDLs), intermediate-density lipoproteins, and low-density lipoproteins (LDLs).

As lipids constitute the highest percentage of solid components of the body, increases in lipid content lead to an enlargement of adipose tissue and elevated levels of very

low-density lipoproteins, LDLs, and intermediate-density lipoproteins, along with reduced levels of HDL.

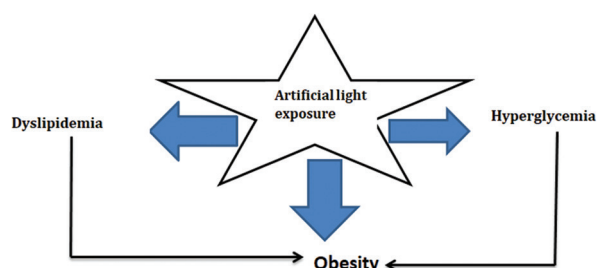
Exposure to artificial light has been reported to alter body lipid contents. In a cross-sectional study by Han *et al.*<sup>55</sup> involving 10,894 participants, the relationship between prolonged ALAN exposure (measured using satellite imaging data) and dyslipidemia prevalence was investigated. Prolonged exposure to ambient ALAN was associated with increased dyslipidemia prevalence.

In animal studies, Zhang *et al.*<sup>53</sup> evaluated whether the metabolic status of normal and high-fat diet-induced obese mice would be affected by artificial light at specific light wavelengths. It was reported that exposure to green light caused metabolic impairments and elevated liver and serum lipid levels compared to those maintained under white light. In addition, genes related to lipid metabolism in the liver were downregulated. In a study, APOE\*3-Leiden CETP female mice, a model for studying hyperlipidemia, were maintained under either a normal 12-h daylight/12-h dark cycle or 24-h bright light (100 lux) for 14 weeks.<sup>56</sup> Although there was no significant change in the extent and size of atherosclerotic plaques in the root of the aorta, constant light exposure caused elevated plasma levels of pro-atherogenic non-HDL cholesterol.

Zhu *et al.*<sup>54</sup> evaluated the effect of brief exposure to two different night lighting periods (3 days and 6 days) on triglyceride in zebra finch birds. After 3 days of exposure to night light, the expression of genes involved in fat synthesis was increased, while the expression of genes responsible for triglyceride breakdown and fatty acid oxidation was reduced. On the other hand, after 6 days of exposure, genes responsible for fatty acid catabolism were upregulated, and genes associated with fat synthesis were downregulated.

Using high-fat diet-fed mice with *Ppara*<sup>-/-</sup> and wild-type mice, Luo *et al.*<sup>42</sup> demonstrated the effects of exposure to neon light at night for 6 weeks. High-fat diet-fed wild-type mice exposed to night neon light demonstrated lipid accumulation in the liver and enhanced lipid transfer to peripheral tissues from the liver. Expression of *Clock* genes in adipose tissues and liver was increased in wild-type mice exposed to neon light.

The effect of dim ALAN (2 lux) on energy metabolism and gene expression in male Wistar rats was investigated by Rumanova *et al.*<sup>32</sup> The animals were maintained under a 12-h daylight/12-h dark cycle or exposed to ALAN. Exposure to ALAN caused an increase in the hepatic and plasma cholesterol levels. Hyperglycemia and dyslipidemia are important contributors to the development of obesity (Figure 2). Artificial light-exposed animals also showed



**Figure 2.** Metabolic dysfunctions induced by artificial light exposure. Exposure to artificial light elicits dyslipidemia, hyperglycemia/insulin resistance, and obesity. Hyperlipidemia and hyperglycemia are key contributors to the development of obesity. Image created by the authors.

phase advancement in the expression of hepatic PPAR $\alpha$ , nicotinamide phosphoribosyltransferase, and PPAR $\gamma$  coactivator 1 $\alpha$ , as well as arrhythmic expression of sirtuin 1 and liver X receptor  $\alpha$ . In the adipose tissue, nocturnin was arrhythmic.

A plethora of evidence from animal studies suggests that artificial light exposure increases the likelihood of developing metabolic dysfunctions, especially hyperlipidemia, hyperglycemia, and obesity. Obesity induced by artificial light is mediated by several mechanisms, including changes in feeding time, decreased energy utilization, lipid accumulation, increased adipose tissue size, increased expression of PPARs in both the liver and adipose tissues, altered insulin secretion, reduced glucose tolerance, altered *Per1* and *Clock* gene expression in the liver, increased expression of *Tnfa* and *Mac1* in adipose tissues, reduced fatty acid uptake from lipoproteins, disruption of microflora metabolic pathways, and enhanced transfer of lipid to adipose tissue.

Hyperglycemia/insulin resistance and hyperlipidemia are important factors in the development of obesity. Although the effects of artificial light exposure observed in animal studies may not fully translate to humans, it has been shown to impair blood pressure and heart rate. Dim night light exposure appears to induce more adverse metabolic outcomes than bright night light. Public health policies may be required at the governmental and institutional levels to regulate human and animal exposure to artificial light, including bright and dim night lights.

#### 4. Species-specific cardiometabolic dysfunctions orchestrated by artificial light exposure

Nocturnal mammals are night-active organisms. The evolution of nocturnal activity in these organisms appears to be a strategy to escape attacks and onslaughts from diurnal mammals, particularly humans. Although exposure to light suppresses circulating melatonin

secretion, the biological impacts of exposure to ALAN may differ between diurnal and nocturnal mammals.

Evidence from murine studies indicates that artificial light exposure causes a decrease in blood pressure and bradycardia.<sup>19</sup> In contrast, empirical human studies have shown that artificial light increases systolic and diastolic blood pressure. A Chinese study reported a 1.31-fold increase in the likelihood of hypertension in individuals within the highest quartile of outdoor artificial light intensity.<sup>57</sup> In humans, the tendency of ALAN to raise blood pressure has previously been attributed to the blood pressure-lowering properties of melatonin.<sup>58</sup> In rats, increased nocturnal melatonin causes blood pressure-lowering effects and bradycardia.<sup>59,60</sup>

The physiological actions of melatonin via G protein-coupled MT1 and MT2 receptors (coupled to inhibitory G-proteins [G0/1]) are orchestrated primarily through a decrease in cyclic adenosine monophosphate, leading to vasodilation.<sup>60</sup> While melatonin is known for its vasodilatory tendency, studies have also reported vasoconstrictive effects in certain tissues.<sup>61</sup> It is worth noting that these effects are mediated by the  $\beta$  and  $\gamma$  subunits rather than the  $\alpha$  subunit. The  $\beta$  and  $\gamma$  subunits are known to bind to certain  $\beta$ -isoforms of phospholipase C, particularly  $\beta 3$  and  $\beta 2$  isoforms, to elicit the opening of voltage-gated calcium channels.<sup>62</sup> However, the  $\beta$  and  $\gamma$  subunits have relatively lower affinity and potency than the  $\alpha$  subunit of G-proteins.<sup>63</sup> Conversely, exposure to ALAN causes a reduction in blood pressure and bradycardia<sup>20</sup> and reduces suprachiasmatic neural activity, resulting in suppression of physical and metabolic activity in nocturnal rats,<sup>64</sup> with attendant reduction in blood pressure and heart rate, because these indices are low when physical and metabolic activities are reduced.

Some effects of ALAN exposure appear similar in both diurnal and nocturnal organisms. Table 1 summarizes how ALAN exposure affects cardiometabolic parameters in selected species. As described in earlier sections, studies in mice and zebra finches have shown that dim night light increases body weight,<sup>26,28</sup> elevates nocturnal glucose levels,<sup>40</sup> and exacerbates insulin resistance and glucose intolerance.<sup>43</sup> In addition, ALAN exposure elevates plasma cholesterol levels and promotes pro-atherogenic lipid profiles, as observed in mice and rats.<sup>32,56</sup> These findings collectively indicate that ALAN negatively impacts glucose metabolism and lipid homeostasis across species.

Similarly, in humans, a Chinese national survey comprising 129,500 children and adolescents aged between 10 and 18 years revealed that exposure to ALAN has a direct association with overweight and obesity.<sup>65</sup> This indicates that with each unit increase in ALAN exposure,



**Table 1. Species-specific cardiometabolic effects of artificial light at night**

Species	Amount of artificial light at night	Health parameter	Cardiometabolic effects
Mice <sup>28</sup>	16-h light at 150 lux/8-h dim light at 5 lux for six weeks	Body weight	Increased rate of body mass change compared to the control group (16-h light at 150 lux/8-h dark at 0 lux)
Humans <sup>66</sup>	The highest quartile of artificial light at night exposure		Increased odds ratio of being overweight
Zebra finch birds <sup>40</sup>	12-h daylight (150–200 lux)/12-h dim night light (4 lux)	Glucose profile	Elevated nocturnal glucose levels
Humans <sup>68</sup>	Bright light (>500 lux)		Elevated plasma glucose and insulin secretion
Mice <sup>56</sup>	Exposure to 100-lux lighting under either a 12 h: 12 h light–dark cycle or a 12 h: 12 h light–light cycle for 14 weeks	Lipid profile	Increased plasma levels of pro-atherogenic non-HDL cholesterol
Humans <sup>55</sup>	Per-quintile nighttime artificial light exposure		Positive correlation with the prevalence of dyslipidemia, high triglycerides, high LDL cholesterol, and low HDL cholesterol

Abbreviations: HDL: High-density lipoprotein; LDL: Low-density lipoprotein.

the likelihood of overweight and obesity increases. A cross-sectional study by Fan *et al.*<sup>66</sup> involving 11,729 individuals indicated that the odds ratios of overweight and dyslipidemia increased in individuals with the highest quartile of ALAN exposure.

Another study showed that when 17 healthy individuals were exposed to bright light (>500 lux), plasma glucose and insulin levels were elevated compared to those exposed to dim light (<5 lux).<sup>67</sup> Qi *et al.*<sup>68</sup> showed that each standard deviation increase in bedroom ALAN was associated with a 1.8  $\mu$ U/mL in insulin secretion and a 0.61-unit increase in Homeostatic Model Assessment of Insulin Resistance.

In addition, a cross-sectional study by Han *et al.*<sup>55</sup> recruited 10,894 individuals aged 45 years and above. They reported that for every incremental quintile of ALAN exposure, there was a positive correlation with the prevalence of dyslipidemia, high triglycerides, high LDL cholesterol, and low HDL cholesterol. Specifically, when compared to the first quintile of exposure, the fifth quintile of ALAN exposure was associated with an elevated prevalence of dyslipidemia.

Exposure to ALAN affects a large proportion of the global population and ecosystems. With the current increase in public acceptance and utilization, as well as the proliferation of artificial light sources, control measures, policies, and individual behavioral interventions could be overwhelmed, potentially leading to substantial adverse health consequences. Suppression of melatonin secretion, disruption of circadian rhythms, reduced vitamin D synthesis, potential genotoxic effects, and alterations in the normal microbiota, together with increases in body weight, hyperglycemia, insulin resistance, dyslipidemia, and derangements in normal cardiovascular rhythmicity,

position ALAN as a pervasive environmental risk factor for cardiometabolic disease and a significant threat to ecosystem health.

## 5. Conclusion

Chronodisruption induced by artificial light exposure is increasingly recognized as a significant risk factor for cardiometabolic dysfunction, with evidence from animal studies demonstrating altered circadian gene expression, metabolic dysregulation, and cardiovascular impairments. Translation of these findings to human health is supported by epidemiological studies linking night-shift work and excessive nocturnal light exposure to obesity, insulin resistance, hypertension, and cardiovascular disease. Occupational health policies should incorporate circadian-friendly lighting, optimized shift scheduling, and melatonin preservation strategies to mitigate adverse health effects. Public health interventions, such as limiting blue-light exposure before sleep, promoting natural daylight exposure, and implementing urban lighting regulations, are supported by current evidence as approaches to reducing chronodisruption. Preventive strategies, including personalized chronotherapy and behavioral modifications, may further optimize circadian alignment and metabolic homeostasis. Future interdisciplinary research integrating chronobiology, endocrinology, and occupational medicine remains essential for developing targeted interventions to address this emerging public health concern.

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The authors declare they have no competing interests.

## Author contributions

*Conceptualization:* All authors

*Visualization:* All authors

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