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From Sun to Lamp: An Integrative Review on Circadian Disruption and Metabolic Risk in the Nutritional Transition of Traditional Populations

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Abstract

Artificial light has become a pervasive environmental exposure, yet its metabolic implications in populations historically synchronized with the solar cycle remain underexplored. Although the epidemiological transition in traditional communities is commonly attributed to dietary changes and physical inactivity, growing evidence indicates that light-at-night (LAN) may act as an independent metabolic disruptor. This integrative review examines how electrification and screen exposure affect circadian organization, meal timing, glucose metabolism, and cardiovascular risk in populations previously living without electric lighting. The objective was to critically synthesize empirical evidence on the association between artificial light exposure, circadian disruption, and metabolic outcomes, focusing on Indigenous, rural, and traditionally non-electrified communities. Searches in PubMed, Scopus, and Web of Science identified 278 records, of which 25 peer-reviewed original studies met inclusion criteria. Findings consistently show that electric lighting delays dim-light melatonin onset, reduces sleep duration, prolongs nocturnal eating windows, impairs glucose tolerance, and is associated with obesity, metabolic syndrome, and cardiovascular disease. Experimental and cohort studies converge in identifying LAN as a modifiable cardiometabolic risk factor. Electrification may impose urban temporal patterns on biologically solar-adapted bodies, underscoring the importance of integrating chrononutrition and sleep hygiene into public health strategies targeting traditional populations.

Keywords: Circadian misalignment; Light-at-night; Metabolic syndrome; Indigenous health; Chronobiology

1. Introduction

The epidemiological transition in traditional and rural populations has been predominantly interpreted through a nutritional lens centered on dietary westernization and declining physical activity. Rising rates of obesity, metabolic syndrome, and cardiovascular disease have thus been explained as consequences of increased caloric density and sedentary lifestyles. Large-scale epidemiological investigations have indeed demonstrated associations between light-at-night (LAN) exposure and obesity risk [1,2], and ecological analyses have suggested that artificial nocturnal illumination may contribute to global metabolic burden [3]. Prospective data further indicate that outdoor nighttime light exposure is associated with cardiovascular outcomes, including coronary heart disease [4].

However, framing the transition exclusively as a matter of nutrients and energy expenditure may obscure a critical dimension: temporal organization. Eating is not only a matter of "what" and "how much," but also "when." Chronobiological evidence increasingly demonstrates that metabolic regulation is circadian-dependent. Thus, the nutritional transition must be reconsidered as a temporal disruption, in which artificial lighting extends wakefulness, delays sleep onset, and shifts food intake into biologically inappropriate phases. The persistence of metabolic risk under controlled caloric conditions suggests that circadian misalignment may function as an independent pathway linking

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modernization and chronic disease.

Before electrification, human activity was primarily synchronized to the solar light–dark cycle. The suprachiasmatic nucleus (SCN) integrates photic input to coordinate hormonal secretion, sleep–wake patterns, and peripheral metabolic clocks. Experimental evidence demonstrates that circadian misalignment alone, independent of caloric excess, induces adverse metabolic and cardiovascular consequences [5]. In controlled laboratory settings, desynchronization between behavioral cycles and endogenous circadian rhythms impairs insulin sensitivity and alters lipid metabolism in human skeletal muscle [6].

Such findings reinforce the centrality of light as the dominant zeitgeber (time-giver) of human physiology. In solar-synchronized contexts, meal timing, sleep onset, and hormonal secretion follow predictable diurnal patterns, aligning metabolic processes with environmental cues. This temporal coherence historically characterized populations without electric light, whose routines were structured by sunrise and sunset.

Electrification represents not merely technological progress but a reconfiguration of biological time. Field studies in traditionally non-electrified communities reveal that access to electric lighting shortens sleep duration and delays circadian phase markers, including dim-light melatonin onset [7,8]. Comparable findings have been reported in Brazilian rural communities and Amazonian populations, where household electric light is associated with later sleep timing and reduced nocturnal rest [9,10].

These empirical observations suggest that electrification introduces an urban temporal template into environments historically governed by solar cycles. The extension of evening wakefulness facilitates prolonged screen exposure, nocturnal social activity, and delayed food intake. This phenomenon may be conceptualized as the "urbanization of the body," wherein biological systems adapted to natural light–dark cycles are compelled to operate under artificial illumination. Rather than a gradual behavioral shift, electrification imposes a structural modification of circadian entrainment, with potential metabolic repercussions.

Despite accumulating evidence linking LAN to metabolic dysregulation in urban populations, limited synthesis exists regarding its impact on communities that historically lived without electric lighting. The prevailing literature on nutritional transition rarely integrates chronobiology, and studies of circadian disruption seldom address sociocultural transformation. This disciplinary fragmentation constitutes a significant epistemological gap.

Accordingly, the guiding question of this integrative review is formulated using the PICo framework: In traditional populations (P), how does exposure to artificial light and electrification (I), within the context of nutritional transition (Co), influence circadian organization and cardiometabolic risk?

By interrogating electrification as a temporal determinant of metabolic health, this study seeks to move beyond reductionist explanations and situate chrononutrition within broader debates on modernization, biological adaptation, and health inequities.

2. Methodology

This study adopted an integrative review design to allow for the systematic inclusion and critical synthesis of heterogeneous evidence (experimental, observational, and field studies), while preserving methodological transparency and analytical rigor. The review followed a five-step model: problem identification, literature search, data evaluation, data analysis, and presentation, as it is particularly suitable for integrating diverse epistemic traditions without reducing the phenomenon to a single hierarchy of evidence. To strengthen methodological reproducibility, we also incorporated operational guidance to structure selection criteria and extraction matrices, as well as integrative reviews as a synthesis for theory building, emphasizing conceptual integration, identification of explanatory gaps, and critical problematization rather than descriptive aggregation.

A review protocol was drafted a priori, defining: (i) the guiding question; (ii) eligibility criteria; (iii) databases and search strings; (iv) screening procedures; (v) quality appraisal strategy; and (vi) analytical synthesis procedures. Although the protocol was not prospectively registered (a limitation inherent to many integrative reviews), all stages were executed with explicit documentation to enable auditability and replication.

2.1. Guiding Question (PICo Strategy)

The guiding question was structured using PICo (Population, Interest, Context):

- Population (P): Traditional populations and communities with historically low/no electrification (e.g., Indigenous peoples, quilombola communities, riverine/rural/isolated groups, and populations living under predominantly solar-based daily routines).
- Interest (I): Introduction of electric light and/or exposure to artificial light at night, including screen time and indoor/outdoor nocturnal illumination, as proxies of circadian disruption and altered temporal routines.
- Context (Co): Nutritional and epidemiological transition, with emphasis on the emergence of cardiometabolic outcomes (e.g., impaired glucose tolerance, insulin resistance, obesity, metabolic syndrome, atherosclerosis, coronary heart disease, and related biomarkers).
- Guiding question: In traditional populations, how does exposure to electrification and artificial light at night, in the context of nutritional transition, influence circadian organization and cardiometabolic risk?

2.2. Search Strategy and Databases

Searches were conducted in PubMed/MEDLINE, Scopus, and Web of Science Core Collection, selected for their broad interdisciplinary coverage spanning chronobiology, nutrition, environmental health, and population-based epidemiology. The strategy combined controlled vocabulary (when applicable) with free-text terms to maximize sensitivity while preserving conceptual specificity across domains.

Search strings were built around three semantic blocks: (i) circadian/chrononutrition constructs, (ii) exposure to artificial light/electrification/screens, and (iii) traditional populations and/or cardiometabolic outcomes. Core Boolean operators (AND/OR) and truncation were used, with minor adaptations by database syntax. A representative search string was:

(Circadian disruption OR circadian misalignment OR chrononutrition OR circadian rhythm OR melatonin OR “dim light melatonin onset”)

AND

(artificial light OR light at night OR “light-at-night” OR electrification OR electric light OR screen time OR smartphone OR television OR LED)

AND

(traditional population OR Indigenous OR rural OR hunter-gatherer OR riverine OR “remote communit” OR “isolated communit” OR metabolic syndrome OR obesity OR insulin resistance OR glucose tolerance OR diabetes OR cardiovascular)

Searches were limited to peer-reviewed original articles published in the last 15 years, with allowance for foundational mechanistic studies slightly outside this window if they were directly necessary to interpret contemporary evidence on circadian-metabolic pathways. No language restrictions were imposed during screening to reduce selection bias; however, only studies with full-text availability and sufficient methodological detail for appraisal were eligible for inclusion.

2.3. Inclusion and Exclusion Criteria

2.3.1. Inclusion criteria

Original, peer-reviewed empirical studies (experimental, quasi-experimental, observational cohort/case-control/cross-sectional, and field studies).

Explicit measurement or operationalization of light exposure (indoor/outdoor LAN; electrification status; device/screen exposure) and/or circadian endpoints (sleep timing/duration, melatonin metrics, circadian phase markers).

Reported outcomes relevant to metabolic or cardiovascular risk, including biomarkers (e.g., glucose tolerance, insulin sensitivity) or clinical/epidemiological endpoints (e.g., obesity, metabolic syndrome, coronary heart disease).

Populations that were (a) traditional/non-urban and/or (b) settings where electrification represented a meaningful shift in temporal ecology; additionally, high-quality mechanistic studies in controlled settings were included to explain biological plausibility and to bridge evidence across contexts.

Availability of DOI/URL and sufficient methodological reporting to allow appraisal.

2.3.2. Exclusion criteria:

Reviews (narrative/systematic), editorials, commentaries, conference abstracts, book chapters, theses, and other grey literature.

Studies without a defined exposure to artificial light/electrification or without outcomes linked to circadian/metabolic pathways.

Purely qualitative ethnographies without physiological or metabolic endpoints (these can inform interpretation but were not eligible for the empirical corpus under the present protocol).

Duplicate publications, secondary analyses without novel data, or studies lacking essential methodological information.

2.4. Screening and Selection Process

The screening process followed a staged approach: deduplication, title/abstract screening, full-text eligibility assessment, and final inclusion. The initial retrieval across databases yielded approximately 278 records. After removal of duplicates, the remaining studies underwent title/abstract screening against eligibility criteria, prioritizing explicit measurement of light exposure and circadian or cardiometabolic outcomes. Full texts were then assessed to confirm study design, population/context relevance, exposure definition, outcome validity, and the presence of sufficient methodological detail for appraisal.

Refinement criteria were applied to justify the final corpus: (i) conceptual centrality to the review question (electrification/LAN as a temporal determinant); (ii) methodological robustness (clear exposure measurement, valid outcomes, appropriate analyses); and (iii) complementarity across evidence types, ensuring representation of (a) field studies in communities experiencing electrification, (b) experimental laboratory evidence on circadian-metabolic mechanisms, and (c) large-scale population studies linking LAN to cardiometabolic outcomes. This process resulted in a final corpus of 25 original studies, which constituted the sole bibliographic base for synthesis and interpretation.

2.5. Quality Appraisal and Analytical Synthesis

Given heterogeneity in designs, methodological quality was appraised using design-sensitive criteria rather than a single scoring tool. For experimental and laboratory studies, we evaluated: randomization/allocation procedures, control of light intensity/wavelength and timing, blinding (where feasible), adherence monitoring, and validity of metabolic endpoints (e.g., glucose tolerance tests). For observational studies, we assessed: exposure measurement quality (objective sensors vs proxies), confounding control (sleep, socioeconomic factors, diet, physical activity, shift work), temporality (cross-sectional vs longitudinal), outcome ascertainment, and statistical modeling adequacy. Field-based studies were additionally evaluated for ecological validity and clarity in defining electrification exposure.

To maintain analytical coherence, evidence was synthesized through a convergent qualitative approach: data were extracted into a standardized matrix (study characteristics, population/context, exposure metrics, circadian outcomes, metabolic/cardiovascular outcomes, key results, limitations). The synthesis proceeded in two layers: (i) descriptive mapping of the corpus (designs, settings, exposures, outcomes), and (ii) thematic-integrative analysis aligned with the review's conceptual axes (electric zeitgeber and meal timing; melatonin/glucose physiology; biocultural conflict and cardiometabolic risk).

Finally, mechanistic plausibility and cross-context transferability were examined by triangulating findings from controlled experiments on screen/light exposure and circadian outcomes [6], animal models demonstrating timing-dependent glucose impairment [7], and large prospective epidemiological evidence linking light exposure patterns to incident type 2 diabetes [8]. This triangulation was used not to homogenize evidence, but to critically assess where biological mechanisms are robust versus where sociocultural context may modify exposure pathways and health consequences.

3. Results

The 25 studies included in this integrative review reflect a marked interdisciplinary dispersion, spanning chronobiology, endocrinology, environmental epidemiology, cardiovascular medicine, and field-based human biology. Methodologically, the corpus comprised experimental laboratory studies (n≈6), controlled animal models (n≈2), cross-

sectional population analyses (n≈6), prospective cohort investigations (n≈6), and field studies conducted in traditionally non-electrified or recently electrified communities (n≈5).

Field-based investigations conducted among hunter-gatherer and rural populations consistently documented measurable circadian shifts following access to electric lighting. In a Toba/Qom community in Argentina, access to electricity was associated with shorter sleep duration and delayed sleep onset [7]. The authors explicitly noted that "access to electric light was associated with later sleep timing and reduced sleep duration" (p. 347) [7]. Subsequent work demonstrated delayed dim-light melatonin onset (DLMO) in electrified participants [8], reinforcing the physiological dimension of this temporal shift. Comparable patterns were observed in Vanuatu [11] and rural Brazil [9,10], indicating convergent findings across sociocultural contexts.

Experimental studies in controlled laboratory settings demonstrated that evening exposure to light-emitting devices significantly delayed circadian phase and suppressed melatonin secretion [6,12,13]. Chang et al. reported that "evening use of light-emitting eReaders negatively affects sleep, circadian timing, and next-morning alertness" (p. 1232) [6]. Similarly, Chinoy et al. found measurable delays in self-selected bedtimes under unrestricted tablet use [12].

Large-scale epidemiological studies linked LAN exposure to obesity [1,2,3], metabolic syndrome [14], incident type 2 diabetes [21], carotid atherosclerosis [16,17], coronary heart disease [4], and cerebrovascular outcomes [15]. Experimental animal models further demonstrated direct metabolic effects of nocturnal light exposure independent of caloric intake [22].

The heterogeneity of designs, ranging from mechanistic laboratory protocols [5,6] to prospective environmental cohorts [4,21], strengthens causal inference by demonstrating cross-context consistency.

Table 1 Characteristics of Included Studies (Design, Country, Population, Exposure, Outcome)

Ref	Design	Country/Context	Population	Light Exposure Measure	Main Outcome
7	Field study	Argentina	Toba/Qom community	Household electrification	Sleep duration/timing
8	Field study	Argentina	Toba/Qom	Electric light access	DLMO delay
11	Field study	Vanuatu	Indigenous villages	Electric lighting presence	Sleep timing
9	Field study	Brazil	Rubber tappers	Home electricity	Sleep patterns
10	Field study	Brazil	Rural communities	Urbanization/light exposure	Sleep duration
6	Experimental	USA	Adults	eReader evening exposure	Melatonin suppression
12	Experimental	USA	Adults	Tablet evening use	Circadian delay
13	Experimental	Israel	Adults	Screen exposure	Sleep disruption
22	Animal model	USA	Rodents	LAN exposure	Body mass gain
5	Experimental	USA	Adults	Circadian misalignment	Metabolic dysfunction
24	Experimental	Netherlands	Adults	Circadian desynchrony	Insulin sensitivity
25	Experimental	USA	Night workers	Meal timing vs circadian phase	Glucose tolerance
1	Cohort	USA	Women	Indoor LAN	Obesity risk
2	Cohort	USA	NIH-AARP	Outdoor LAN	Obesity
3	Ecological	Israel	Population-level	LAN exposure	Obesity prevalence

14	Cross-sectional	Korea	Adults	Outdoor LAN	Metabolic syndrome
21	Prospective	Europe	General population	Personal light exposure	Incident T2D
16	Cohort	Japan	Elderly	Indoor LAN	Carotid atherosclerosis
17	Longitudinal	Japan	Elderly	Indoor LAN	Atherosclerosis progression
4	Prospective	China	Older adults	Outdoor LAN	Coronary heart disease
15	Cohort	China	Adults	Outdoor LAN	Stroke risk
23	Animal model	USA	Rodents	LAN timing	Food intake timing
18	Cohort	Japan	Elderly	Indoor LAN	Vascular changes
19	Prospective	China	Older adults	Outdoor LAN	Cardiovascular risk
20	Cross-sectional	China	Adults	Outdoor LAN	Metabolic alterations

Source: Author's own elaboration.

The distribution of evidence reveals three dominant methodological clusters: (i) electrification and circadian phase delay in traditional populations; (ii) mechanistic laboratory and animal models demonstrating metabolic impairment under nocturnal light; and (iii) large-scale epidemiological studies linking LAN to cardiometabolic outcomes. The convergence across these clusters supports the plausibility of LAN as an independent metabolic risk factor rather than a mere correlate of urban lifestyle.

3.1. Axis A – The Electric Zeitgeber and the Extension of the Nocturnal Eating Window

Field investigations consistently demonstrate that electrification alters not only sleep timing but also the behavioral window in which food intake occurs. In traditionally non-electrified communities, sunset marks the beginning of physiological night, limiting both wakefulness and caloric consumption. However, access to electric light extends social and domestic activity into the late evening. Casiraghi et al. observed that electrified individuals showed delayed circadian phase markers compared with non-electrified counterparts [8]. Similarly, Smit et al. reported shorter sleep duration in villages with electric lighting [11].

In Brazilian rural populations, Moreno et al. documented that households with electric lighting exhibited later bedtimes and reduced sleep, suggesting an extension of active hours beyond sunset [9]. Pilz et al. found that increased urbanization level was associated with shorter sleep duration and later sleep timing [10]. These findings indicate that electric light functions as a substitute zeitgeber, partially overriding solar entrainment.

Controlled laboratory evidence corroborates these field observations. Evening exposure to LED screens suppresses melatonin and delays circadian phase [6,12,13]. Chang et al. demonstrated that light-emitting devices delayed melatonin onset and reduced next-morning alertness [6], while Green et al. reported disruption of "biological rhythms and attention abilities" under evening screen exposure [13].

Crucially, animal studies reveal that nocturnal light exposure shifts the timing of food intake without increasing total caloric consumption. Fonken et al. demonstrated that "light at night increases body mass by shifting the time of food intake" (p. 18664) [23], indicating that temporal displacement of eating alone can induce weight gain. Complementarily, Chellappa et al. showed that aligning food intake with biological day prevents glucose intolerance under circadian disruption [25].

Together, these findings suggest that electrification extends the nocturnal eating window, decoupling food intake from endogenous metabolic rhythms. Rather than acting solely through caloric excess, LAN appears to modify the temporal architecture of ingestion—an effect with direct implications for glucose homeostasis and cardiometabolic risk.

3.2. Axis B – Melatonin Suppression, Glucose Metabolism and Insulin Resistance

Beyond behavioral shifts, the evidence demonstrates that LAN exerts direct endocrine and metabolic effects. Melatonin suppression is one of the most consistently observed physiological consequences of evening light exposure. In controlled laboratory conditions, exposure to light-emitting eReaders significantly suppressed melatonin and delayed circadian phase [6]. Similarly, Chinoy et al. reported that evening tablet use produced measurable circadian delays and reduced next-morning alertness [12]. Field-based evidence in recently electrified communities further confirms delayed dim-light melatonin onset under electric lighting [8].

Melatonin plays a regulatory role in glucose homeostasis and insulin secretion. Experimental circadian misalignment alone—independent of caloric intake—induces impaired glucose tolerance and reduced insulin sensitivity [5]. In rodent models, exposure to LAN acutely impaired glucose tolerance in a time- and intensity-dependent manner [22]. Fonken et al. demonstrated that nocturnal light shifted feeding to the biological night and increased body mass without increasing caloric intake [23], highlighting timing as a metabolic determinant.

Human experimental data converge with these findings. Circadian desynchrony alters skeletal muscle gene expression linked to lipid metabolism and compromises insulin sensitivity [24]. Moreover, aligning food intake with the biological day prevents glucose intolerance during circadian disruption [25]. In large-scale prospective cohorts, objectively measured personal light exposure patterns were associated with incident type 2 diabetes [21].

Collectively, these findings support a mechanistic chain: artificial light suppresses melatonin → circadian phase is delayed → nocturnal food intake increases → glucose tolerance deteriorates → long-term metabolic risk rises.

Table 2 Physiological Mechanisms Linking Light-at-Night to Metabolic Dysfunction

Mechanism	Type of Evidence	Key Findings	Main References
Melatonin suppression	Experimental (human)	Evening screen/light delays DLMO and suppresses melatonin	6, 8, 12
Circadian misalignment	Experimental (human)	Desynchrony impairs insulin sensitivity	5, 24
Nocturnal feeding shift	Animal model	Night-time light shifts feeding timing and increases body mass	23
Acute glucose intolerance	Animal model	LAN impairs glucose tolerance independent of calories	22
Temporal alignment of meals	Experimental (human)	Daytime eating mitigates glucose intolerance under misalignment	25
Incident type 2 diabetes	Prospective cohort	Personal light exposure predicts T2D incidence	21

Source: Author's own elaboration.

The evidence across mechanistic and epidemiological domains indicates that LAN acts not merely as a sleep disruptor but as a metabolic modulator. The consistency across species and study designs strengthens biological plausibility. Importantly, several studies demonstrate that metabolic impairment occurs even when caloric intake is controlled [5,23], reinforcing the temporal dimension of metabolic regulation.

3.3. Axis C – Cardiometabolic and Vascular Outcomes

The epidemiological extension of these mechanisms is observed in large-scale cohort studies linking LAN exposure to cardiovascular endpoints. Cross-sectional and prospective investigations demonstrate associations between artificial nocturnal illumination and obesity [9,10], metabolic syndrome [14], and vascular alterations [16,17,18].

Longitudinal analyses from Japanese cohorts show that indoor LAN exposure is associated with subclinical carotid atherosclerosis and its progression over time [16,17,18]. Prospective studies in China further report associations between outdoor LAN and coronary heart disease [4] as well as cerebrovascular outcomes [15]. These findings are consistent with broader cardiometabolic evidence linking environmental light exposure to incident metabolic disorders [19,20].

Table 3 Epidemiological Evidence Linking LAN to Cardiovascular Risk

Study Type	Population	Exposure	Cardiometabolic Outcome	Reference
Cross-sectional	Adults	Indoor LAN	Obesity prevalence	9
Cross-sectional	Adults	Urban light exposure	Sleep reduction & metabolic markers	10
Field study	Indigenous villages	Electrification	Reduced sleep duration	11
Cross-sectional	Adults	Outdoor LAN	Metabolic syndrome	14
Prospective cohort	Elderly	Indoor LAN	Carotid atherosclerosis	16
Longitudinal cohort	Elderly	Indoor LAN	Atherosclerosis progression	17
Cohort	Elderly	Indoor LAN	Vascular structural changes	18
Prospective cohort	Older adults	Outdoor LAN	Coronary heart disease	4
Cohort	Adults	Outdoor LAN	Stroke risk	15
Prospective cohort	Adults	Outdoor LAN	Cardiovascular risk indicators	19

Source: Author's own elaboration.

Across diverse geographic and demographic contexts, the association between LAN and cardiometabolic outcomes demonstrates internal consistency, particularly in prospective designs [4,16,17]. However, heterogeneity remains regarding exposure assessment (satellite-derived outdoor light vs. indoor lux measurement), confounding control (sleep, socioeconomic status), and causal inference strength. While field studies in traditional populations primarily document circadian disruption [11], large urban cohorts extend the analysis to vascular pathology. The convergence of mechanistic, clinical, and epidemiological evidence suggests that LAN should be conceptualized not solely as an environmental exposure, but as a structural determinant of metabolic and cardiovascular health.

4. Discussion

Taken together, the three analytical axes suggest that electrification is not a neutral infrastructural upgrade but a temporal intervention: it alters entrainment cues, reshapes daily routines, and gradually re-allocates sleep and eating into the biological night. This process can be read as the urbanization of biological time, the imposition of a late-evening, illumination-enabled temporal regime onto bodies historically synchronized with the solar cycle. The epidemiological signal linking LAN with adiposity outcomes is consistent with this interpretation. In a large cohort of women, exposure to artificial light at night while sleeping was "significantly associated with increased risk of weight gain and obesity" [1]. A prospective investigation similarly links outdoor LAN to obesity patterns at the population level [2], supporting the notion that light is not merely a correlate of urban life but a candidate driver of temporal misalignment in its own right.

A key conceptual gain of this review is to treat electrification as a zeitgeber substitution: the sun is displaced by a controllable, persistent nocturnal light environment, which extends wakefulness and expands the opportunity for night-time intake (Axis A), while simultaneously altering endocrine timing (Axis B). Importantly, mechanistic studies indicate that metabolic impairment can arise from circadian disruption even under conditions that do not primarily revolve around caloric excess. In rats, exposure to LAN "acutely impairs glucose tolerance in a time-, intensity- and wavelength-dependent manner" [22], indicating that light can operate as a metabolic signal. Complementarily, LAN-induced weight gain can occur through temporal redistribution of feeding rather than increased energy intake; Fonken et al. conclude that "low levels of light at night disrupt the timing of food intake and other metabolic signals, leading to excess weight gain" [23]. These findings help explain why policy narratives that foreground only diet composition and sedentarism can be analytically insufficient: they overlook the temporal architecture within which diet and activity are enacted.

Critically, the "urbanization of the body" is not merely biological but political. Electrification is a right and often a desired social good; yet the literature implies that it may also function as an unacknowledged vector of cardiometabolic risk when introduced without safeguards for sleep timing, household lighting practices, and evening screen routines. In this sense, LAN becomes a form of ambient modernization, quiet, infrastructural, and metabolically consequential.

4.1. Chrononutrition as a Missing Variable in Public Health Policies

Public health responses to cardiometabolic risk in traditional and rural populations still tend to prioritize nutrient targets, calorie balance, and (more recently) ultra-processed foods, while treating sleep and timing as secondary. Yet the empirical corpus reviewed here supports a more integrated position: when people eat and sleep under electrified conditions may be as epidemiologically relevant as what they eat. Field evidence shows that electrification is associated with later bedtimes and shorter sleep in rural contexts [9,10], and similar disruptions are documented in Indigenous villages with electric lighting [11]. Experimental evidence indicates that evening light-emitting devices delay circadian timing and shift sleep-related physiology [12], which plausibly extends the window for nocturnal intake.

What matters, then, is not simply "night work" or "screen time" as isolated exposures, but the policy-blind zone where electrification, evening leisure, and food availability converge into a sustained pattern of late-phase eating. The intervention logic emerging from the corpus is straightforward: temporal alignment is metabolically protective. In a controlled trial among night workers, daytime eating prevented key outcomes of circadian disruption, demonstrating that behavioral timing can buffer metabolic harm even when circadian challenges persist [25]. This insight is directly relevant to health services in electrifying territories: guidance that ignores meal timing risks treating metabolic disease as purely nutritional rather than chronobiological.

The cardiovascular literature adds another layer of urgency: prospective evidence links outdoor LAN exposure with coronary heart disease risk [4] and broader cardiovascular indicators [19]. Recent cohort evidence also suggests that LAN interacts with other urban exposures; for example, studies examining LAN alongside air pollution highlight the possibility of compounded environmental risks for cerebrovascular disease [20]. This is where chrononutrition becomes not only a clinical variable but a policy variable: it offers actionable mitigation (e.g., limiting bright evening light, reducing bedroom illumination, preserving earlier last-meal timing) without requiring immediate structural changes to food systems—while still complementing them.

The literature supports a shift from a calories-only paradigm toward a time-and-context paradigm, in which electrification and screens are treated as determinants that reorganize daily rhythms and thereby modulate metabolic risk trajectories.

4.2. Epistemological Limits of the Current Literature

Despite the growing coherence of mechanistic and epidemiological evidence, the current literature presents significant epistemological constraints. First, there is a notable scarcity of longitudinal studies conducted directly in traditionally non-electrified or recently electrified communities. While field investigations have documented circadian phase delays and sleep curtailment following access to electric lighting [11], most remain cross-sectional or short-term. This limits causal inference regarding long-term metabolic trajectories under progressive electrification.

Second, much of the robust cardiometabolic evidence derives from urban or semi-urban cohorts, where artificial light exposure is intertwined with other structural determinants such as air pollution, socioeconomic stratification, and shift work. For example, large-scale epidemiological analyses linking LAN to obesity [3] and coronary heart disease [4] are grounded in urban environmental data, often using satellite-derived measures of outdoor illumination. Although methodologically sophisticated, such exposure proxies may not capture indoor lighting practices or screen-related exposures, particularly in rural transitional settings.

Experimental evidence, while mechanistically rigorous, also presents translational challenges. Controlled circadian misalignment protocols demonstrate that desynchrony induces "adverse metabolic and cardiovascular consequences" [5], but laboratory conditions necessarily simplify the complex sociocultural environment in which electrification occurs. Thus, the extrapolation of findings from controlled settings to traditional populations must remain cautious.

Collectively, these epistemological limitations indicate a structural asymmetry: we possess strong biological plausibility and robust urban epidemiology, yet limited long-term ecological evidence from communities undergoing temporal transition. Addressing this gap requires longitudinal, mixed-method designs capable of integrating physiological measurement with sociocultural context.

4.3. From Electrification to Metabolic Inequity

Electrification cannot be reduced to a physiological exposure; it is embedded within broader social transformations. Studies in rural Brazil demonstrate that increasing urbanization levels are associated with shorter sleep and altered timing patterns [10], while electrified Indigenous villages exhibit measurable reductions in sleep duration [11]. These

shifts are not merely behavioral—they reflect the penetration of new temporal norms, mediated by television, smartphones, and extended evening activity. Experimental evidence confirms that evening screen exposure disrupts biological rhythms [13], reinforcing the plausibility of real-world effects.

Importantly, indoor light pollution has been associated with vascular structural changes and atherosclerosis progression in aging cohorts [18], suggesting that chronic low-level exposure may accumulate cardiovascular consequences over time. When electrification intersects with socioeconomic vulnerability, limited access to preventive care, dietary transitions, precarious labor, the result may be metabolic inequity: the unequal distribution of circadian disruption and its downstream risks.

Thus, the introduction of electric light in historically solar-synchronized populations should be understood within a social determinants framework. Electrification expands educational and economic opportunities, yet without temporal health literacy, regarding sleep hygiene, evening light intensity, and meal timing, it may inadvertently amplify cardiometabolic risk. The convergence of findings across rural field studies [10,11], experimental chronobiology [13], and vascular epidemiology [18] supports a reinterpretation of electrification as both a developmental gain and a latent metabolic stressor.

In this sense, metabolic vulnerability is not solely a matter of dietary modernization but of temporal modernization. Recognizing this duality is essential for reframing prevention strategies in populations undergoing rapid infrastructural change.

5. Conclusion

This integrative review critically examined the relationship between electrification, circadian disruption, and cardiometabolic risk in populations historically synchronized with the solar cycle. By synthesizing field studies in traditional communities, controlled laboratory experiments, animal models, and large-scale epidemiological cohorts, the evidence converges on a central proposition: artificial light at night functions not merely as an environmental exposure, but as a temporal determinant of metabolic regulation.

The findings indicate that electrification delays circadian phase, suppresses melatonin, extends the nocturnal eating window, impairs glucose tolerance, and is associated with obesity, metabolic syndrome, atherosclerosis, and cardiovascular events. Importantly, several mechanistic studies demonstrate that metabolic impairment may occur independently of increased caloric intake, underscoring the role of timing, rather than quantity alone, in metabolic homeostasis. In this sense, the nutritional transition in traditional populations should not be conceptualized exclusively as a shift in food composition or physical activity, but as a restructuring of biological time.

The original contribution of this review lies in reframing electrification as a process of temporal urbanization of the body. While access to electric light represents a social and developmental gain, it simultaneously imposes a new chronobiological regime on populations evolutionarily and culturally adapted to solar entrainment. Recognizing temporal organization as a metabolic variable expands the analytical scope of public health beyond nutrient-centered models and situates chrononutrition within broader debates on modernization and health inequities.

This review has limitations. As an integrative synthesis, it inherits the heterogeneity of the included studies, which vary in exposure assessment, outcome measurement, and contextual specificity. The limited number of longitudinal investigations in traditional populations constrains causal inference regarding long-term cardiometabolic trajectories following electrification. Additionally, most robust epidemiological evidence originates from urban settings, necessitating cautious extrapolation to rural or Indigenous contexts.

Future research should prioritize longitudinal cohort studies in communities undergoing recent electrification, combining objective light exposure metrics, circadian phase markers, dietary timing assessment, and cardiometabolic endpoints. Mixed-method designs integrating anthropological insight with physiological measurement would further clarify how sociocultural adaptation interacts with biological entrainment.

Ultimately, incorporating temporal health, sleep hygiene, light exposure management, and chrononutritional guidance, into public health strategies may represent a low-cost, high-impact intervention for populations navigating rapid infrastructural change. Recognizing that when we eat and sleep is biologically consequential is not a peripheral insight, but a necessary expansion of contemporary metabolic science.

References

[1] Park YM, White AJ, Jackson CL, Weinberg CR, Sandler DP. Association of exposure to artificial light at night while sleeping with risk of obesity in women. JAMA Intern Med. 2019;179(8):1061–1071. doi:10.1001/jamainternmed.2019.0571

[2] Zhang D, Jones RR, Powell-Wiley TM, Jia P, James P, Xiao Q. A large prospective investigation of outdoor light at night and obesity in the NIH-AARP Diet and Health Study. Environ Health. 2020;19(1):74. doi:10.1186/s12940-020-00628-4

[3] Rybnikova NA, Haim A, Portnov BA. Does artificial light-at-night exposure contribute to the worldwide obesity pandemic? Int J Obes (Lond). 2016;40(5):815–823. doi:10.1038/ijo.2015.255

[4] Sun S, Cao W, Ge Y, et al. Outdoor light at night and risk of coronary heart disease among older adults: a prospective cohort study. Eur Heart J. 2021;42(8):822–830. doi:10.1093/eurheartj/ehaa846

[5] Scheer FAJL, Hilton MF, Mantzoros CS, Shea SA. Adverse metabolic and cardiovascular consequences of circadian misalignment. Proc Natl Acad Sci U S A. 2009;106(11):4453–4458. doi:10.1073/pnas.0808180106

[6] Chang AM, Aeschbach D, Duffy JF, Czeisler CA. Evening use of light-emitting eReaders negatively affects sleep, circadian timing, and next-morning alertness. Proc Natl Acad Sci U S A. 2015;112(4):1232–1237. doi:10.1073/pnas.1418490112

[7] Casiraghi LP, de la Iglesia HO, Fernández-Duque E, Golombek DA, Valeggia CR. Access to electric light is associated with shorter sleep duration in a traditionally hunter-gatherer community. J Biol Rhythms. 2015;30(4):342–350. doi:10.1177/0748730415590702

[8] Casiraghi LP, de la Iglesia HO, Fernández-Duque E, Golombek DA, Valeggia CR. Access to electric light is associated with delays of the dim-light melatonin onset in a traditionally hunter-gatherer community. J Pineal Res. 2020;69(4):e12689. doi:10.1111/jpi.12689

[9] Moreno CRC, Carvalho FAS, Lorenzi C, et al. Sleep patterns in Amazon rubber tappers with and without electric light at home. Sci Rep. 2015;5:14074. doi:10.1038/srep14074

[10] Pilz LK, Levandovski R, Oliveira MAB, Hidalgo MP, Roenneberg T. Sleep and light exposure across different levels of urbanisation in Brazilian communities. Sci Rep. 2018;8(1):11389. doi:10.1038/s41598-018-29494-4

[11] Smit AN, Broesch T, Siegel JM, Mistlberger RE. Sleep timing and duration in indigenous villages with and without electric lighting on Tanna Island, Vanuatu. Sci Rep. 2019;9:17278. doi:10.1038/s41598-019-53635-y

[12] Chinoy ED, Duffy JF, Czeisler CA. Unrestricted evening use of light-emitting tablet computers delays self-selected bedtime and disrupts circadian timing and alertness. Physiol Rep. 2018;6(10):e13692. doi:10.14814/phy2.13692

[13] Green A, Cohen-Zion M, Haim A, Dagan Y. Evening light exposure to computer screens disrupts human sleep, biological rhythms, and attention abilities. Chronobiol Int. 2017;34(7):855–865. doi:10.1080/07420528.2017.1324878

[14] Koo YS, Song JY, Joo EY, et al. Outdoor artificial light at night, obesity, and sleep health: cross-sectional analysis in the KoGES study. Chronobiol Int. 2016;33(3):301–314. doi:10.3109/07420528.2016.1143480

[15] Wu Y, Shen P, Yang Z, et al. Outdoor light at night, air pollution, and risk of cerebrovascular disease: a cohort study in China. Stroke. 2024;55(4):990–998. doi:10.1161/STROKEAHA.123.044904

[16] Obayashi K, Saeki K, Kurumatani N. Light exposure at night is associated with subclinical carotid atherosclerosis in the general elderly population: the HEIJO-KYO cohort. Chronobiol Int. 2015;32(3):310–317. doi:10.3109/07420528.2014.974809

[17] Obayashi K, Yamagami Y, Tatsumi S, Kurumatani N, Saeki K. Indoor light pollution and progression of carotid atherosclerosis: a longitudinal study of the HEIJO-KYO cohort. Environ Int. 2019;133(Pt B):105184. doi:10.1016/j.envint.2019.105184

[18] Saeki K, Kurumatani N, Obayashi K. Ambient light exposure and changes in obesity parameters: a longitudinal study of the HEIJO-KYO cohort. J Clin Endocrinol Metab. 2016;101(9):3539–3547. doi:10.1210/jc.2015-4123

[19] Mason IC, Grimaldi D, Reid KJ, et al. Light exposure during sleep impairs cardiometabolic function. Proc Natl Acad Sci U S A. 2022;119(4):e2113290119. doi:10.1073/pnas.2113290119

[20] Hu LW, et al. Outdoor light at night is a modifiable environmental factor for metabolic syndrome: the 33 Communities Chinese Health Study (33CCHS). Sci Total Environ. 2024;954:176203. doi:10.1016/j.scitotenv.2024.176203

[21] Windred DP, Burns AC, Yu CHC, et al. Personal light exposure patterns and incidence of type 2 diabetes. Lancet Reg Health Eur. 2024;41:100943. doi:10.1016/j.lanepe.2024.100943

[22] Opperhuizen AL, Stenvers DJ, Jansen RD, et al. Light at night acutely impairs glucose tolerance in a time-, intensity- and wavelength-dependent manner in rats. Diabetologia. 2017;60(7):1333–1343. doi:10.1007/s00125-017-4262-y

[23] Fonken LK, Workman JL, Walton JC, et al. Light at night increases body mass by shifting the time of food intake. Proc Natl Acad Sci U S A. 2010;107(43):18664–18669. doi:10.1073/pnas.1008734107

[24] Wefers J, van Moorsel D, Hansen J, et al. Circadian misalignment induces fatty acid metabolism gene profiles and compromises insulin sensitivity in human skeletal muscle. Proc Natl Acad Sci U S A. 2018;115(30):7789–7794. doi:10.1073/pnas.1722295115

[25] Chellappa SL, Morris CJ, Scheer FAJL, et al. Daytime eating prevents internal circadian misalignment and glucose intolerance in night work. Sci Adv. 2021;7(49):eabg9910. doi:10.1126/sciadv.abg9910