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Circadian Misalignment, Sleep Architecture, and Metabolic Health in Urbanizing Youth: A Mechanistic Narrative Review

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Abstract

Rapid global urbanization has fundamentally restructured the photic, acoustic, social, and nutritional environments of children and adolescents, precipitating an epidemic of chronic sleep restriction and circadian misalignment. While epidemiological surveillance consistently links short sleep duration and evening chronotype to pediatric obesity and insulin resistance, the underlying neurobiological and endocrinological mechanisms remain fragmented across sleep medicine, chronobiology, and pediatric endocrinology. This narrative review synthesizes mechanistic and clinical literature published between 2019 and 2024 examining how urban environmental exposures—specifically artificial light at night (ALAN), short-wavelength screen emissions, early school start times, and nocturnal eating—disrupt adolescent circadian phase and alter sleep macro- and micro-architecture. Specifically, suppression of slow-wave sleep (N3) and rapid eye movement (REM) fragmentation impair nocturnal growth hormone pulsatility, elevate sympathetic tone, and uncouple the central suprachiasmatic nucleus (SCN) pacemaker from peripheral metabolic oscillators in the liver, skeletal muscle, and adipose tissue. This internal desynchronization induces hepatic insulin resistance, postprandial dyslipidemia, and hedonic appetite dysregulation. Critical methodological tensions—including reliance on subjective sleep diaries over actigraphy and polysomnography, and the confounding of sleep duration with circadian timing—are interrogated. Finally, multi-level clinical and urban policy interventions, ranging from delayed school start times to spectral lighting reform and chrononutrition, are proposed to safeguard adolescent metabolic trajectories.

Keywords: Circadian misalignment; Sleep architecture; Slow-wave sleep; Urbanization; Adolescents; Insulin resistance; Social jetlag; Peripheral clocks

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Introduction

Over half of the world's youth now reside in urban environments, a demographic shift projected to exceed two-thirds by mid-century. While urbanization expands access to educational and healthcare infrastructure, it simultaneously immerses developing organisms in a sensory and temporal ecology radically divorced from evolutionary solar cycles (Bumgarner et al., 2023; Walker et al., 2020). Urbanizing youth navigate dense concentrations of outdoor artificial light at night (ALAN), pervasive evening exposure to short-wavelength light-emitting diode (LED) screens, ambient traffic noise, late-night caloric availability, and rigid early-morning school schedules (Arora et al., 2020; Jain et al., 2023; Šmotek et al., 2020). Concurrently, pediatric populations across both high-income and rapidly urbanizing low- and middle-income countries (LMICs) exhibit rising rates of visceral adiposity, metabolic syndrome, and early-onset type 2 diabetes mellitus (Magge et al., 2020; Morales-Ghinaglia et al., 2023).

Historically, pediatric metabolic dysfunction was attributed primarily to positive energy balance driven by caloric surplus and sedentary behavior. However, experimental chronobiology and longitudinal cohort research demonstrate that *when* adolescents sleep and eat—and the internal architectural integrity of that sleep—exert profound, independent effects on glucose homeostasis and lipid metabolism (Chaput et al., 2020; Simon et al., 2019). Adolescence represents a uniquely vulnerable developmental window characterized by a normative biological phase delay in the circadian timing system alongside heightened photosensitivity of the developing crystalline lens (Crowley et al., 2020; Hartstein et al., 2022). When this endogenous pubertal phase delay collides with urban evening light pollution and early academic start times, youth experience chronic **circadian misalignment**—a mismatch between endogenous circadian oscillators and externally imposed behavioral sleep–wake and feeding–fasting cycles (Baron & Reid, 2020; Roenneberg et al., 2019).

This mechanistic narrative review synthesizes evidence published between 2019 and 2024 to elucidate the physiological pathways linking urban circadian disruption and altered sleep architecture to metabolic dysregulation in youth. Specifically, it examines: (1) how urban zeitgeber distortion suppresses pineal melatonin and shifts adolescent circadian phase; (2) how circadian misalignment selectively degrades slow-wave sleep (N3) and rapid eye movement (REM) architecture; (3) the molecular and neuroendocrine pathways through which central–peripheral clock desynchronization drives insulin resistance, dyslipidemia, and appetite dysregulation; and (4) methodological limitations and actionable policy levers for clinical and urban health reform.

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Urban Environmental Zeitgebers and Adolescent Circadian Vulnerability

The mammalian circadian system is organized hierarchically, anchored by a master pacemaker in the hypothalamic suprachiasmatic nucleus (SCN) that synchronizes peripheral tissue clocks via neural, humoral, and thermal signals (Hastings et al., 2019; Sinturel et al., 2020). The SCN relies primarily on environmental light–dark cycles transduced by intrinsically photosensitive retinal ganglion cells (ipRGCs) expressing melanopsin, which exhibits peak spectral sensitivity in the short-wavelength blue spectrum (~460–480 nm) (Blume et al., 2019; Prayag et al., 2019).

During puberty, normative neurodevelopmental remodeling slows the accumulation of homeostatic sleep pressure (Process S) during wakefulness while delaying the circadian pacemaker (Process C), shifting dim-light melatonin onset (DLMO) later by approximately 1 to 3 hours compared to prepubertal children (Crowley et al., 2020; Tarokh et al., 2019). Crucially, children and adolescents possess larger pupil diameters and clearer crystalline lenses than adults, transmitting significantly higher levels of short-wavelength irradiance to the retina (Hartstein et al., 2022; Higuchi et al., 2021). Consequently, urban adolescents exhibit heightened biological sensitivity to evening photic stimuli.

Urbanization systematically corrupts natural photic entrainment through two synergistic pathways. First, outdoor urban skyglow, streetlights, and commercial LED signage elevate nocturnal ambient illuminance, while dense urban housing and indoor schooling reduce daytime exposure to high-intensity natural sunlight (Bumgarner et al., 2023; Paksarian et al., 2020). Inadequate daytime bright light exposure blunts circadian amplitude and increases the SCN's phase-delaying sensitivity to dim evening light (Cain et al., 2020; Wright et al., 2020). Second, ubiquitous pre-bedtime engagement with smartphones, tablets, and gaming consoles delivers high-irradiance blue-enriched light directly to the retina during the circadian phase-delay zone, acutely suppressing pineal melatonin synthesis and delaying DLMO by 40 to 90 minutes (Nagare et al., 2019; Šmotek et al., 2020; van der Lely et al., 2021). Comparative epidemiological studies between urban and rural adolescents confirm that urban youth exhibit significantly later sleep onset, shorter nocturnal sleep duration, and elevated fasting insulin levels even when controlling for socioeconomic status (Jain et al., 2023).

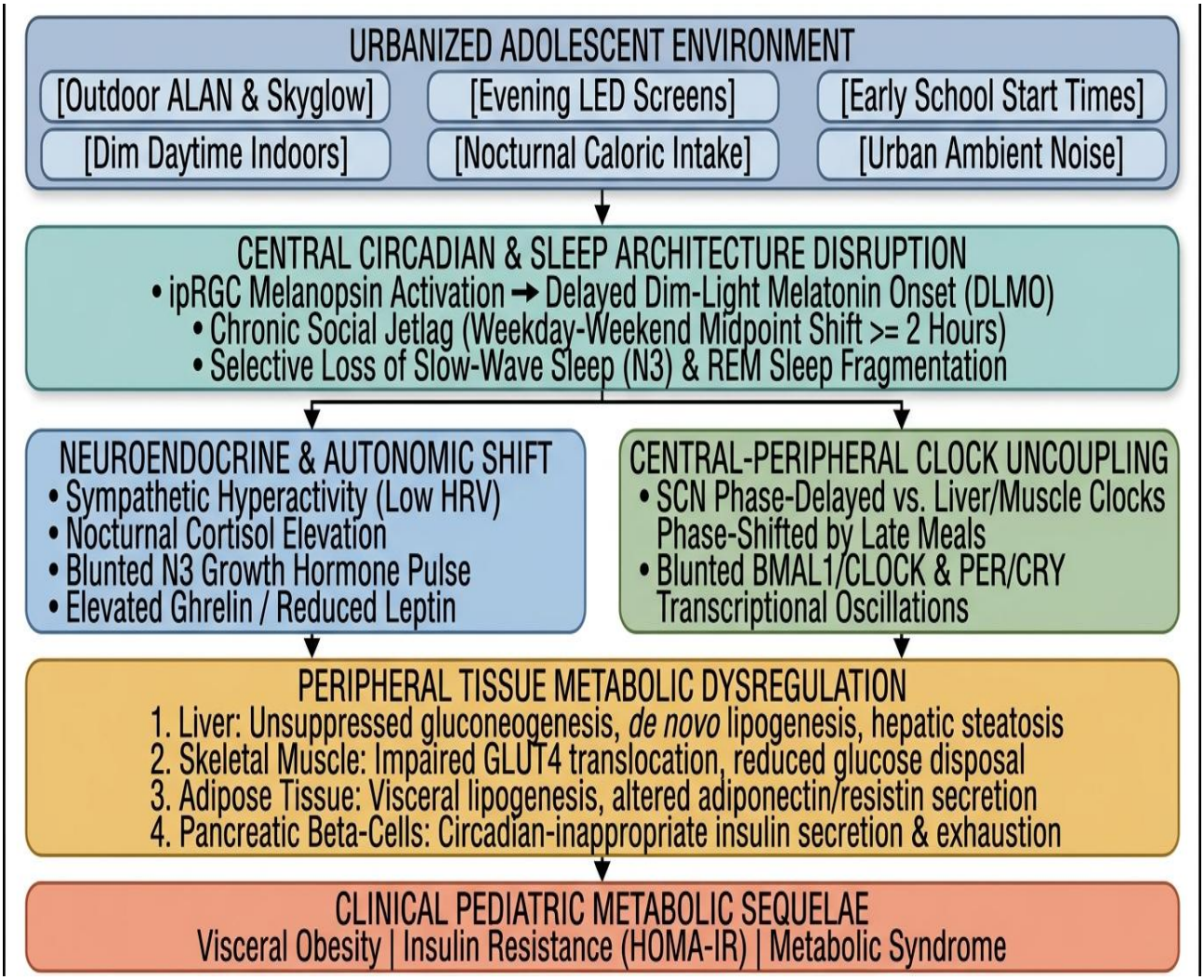


Figure 1. Mechanistic Pathway Linking Urban Environmental Zeitgeber Distortion to Circadian Misalignment, Altered Sleep Architecture, and Peripheral Metabolic Pathology in Youth.

When biologically phase-delayed adolescents are forced to wake early for school start times, they incur severe weekday sleep debt followed by compensatory weekend catch-up sleep—a phenomenon termed **social jetlag** (Roenneberg et al., 2019; Henderson et al., 2019). Quantified as the absolute difference between midsleep time on free days and school days, social jetlag exceeding 2 hours affects over 40% of urban adolescents and functions as a chronic, weekly transmeridian flight that prevents stable circadian entrainment (Cespedes Feliciano et al., 2019; Mathew et al., 2022).

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Sleep Architecture Remodeling Under Circadian Misalignment

Metabolic health depends not merely on total sleep duration, but on the preserved ultradian cycling and structural integrity of non-rapid eye movement (NREM) and rapid eye movement (REM) sleep stages (Dutil et al., 2022; Simon et al., 2019). Polysomnographic investigations reveal that urban circadian misalignment and sleep restriction remodel sleep macro- and micro-architecture in specific, metabolically deleterious ways.

Sleep Architectural Parameter	Normative Adolescent Profile	Alteration Under Urban Circadian Misalignment	Primary Neuroendocrine & Metabolic Consequence
Slow-Wave Sleep (NREM Stage N3)	High proportion (20–25% of total sleep); concentrated in first third of nocturnal sleep period	Truncated by late sleep onset and fragmented by nocturnal noise/light; reduced delta power (0.5–4 Hz)	Blunted nocturnal growth hormone (GH) surge; heightened sympathetic tone; 20–30% drop in whole-body insulin sensitivity
REM Sleep	Concentrated in final third of nocturnal sleep cycle; critical for autonomic stabilization	Truncated by early school wake times; fragmented by elevated nocturnal core body temperature	Unstable sympathovagal balance; impaired emotional regulation and heightened hedonic drive for energy-dense foods
Sleep Continuity & Efficiency	Sleep efficiency > 90%; minimal wake after sleep onset (WASO)	Prolonged sleep onset latency (SOL); frequent micro-arousals from urban traffic noise and device alerts	Tonic hypothalamic-pituitary-adrenal (HPA) axis activation; elevated nocturnal free fatty acids (FFAs)

Table 1. Alterations in Adolescent Sleep Macro- and Micro-Architecture Under Urban Circadian Misalignment and Associated Metabolic Sequelae.

Slow-wave sleep (N3), characterized by high-amplitude, low-frequency delta oscillations (0.5–4 Hz), is the most metabolically restorative phase of human sleep (Dutil et al., 2022; Tasali et al., 2020). During N3 sleep, cerebral glucose utilization declines by up to 40%, parasympathetic (vagal) activity peaks, sympathetic nervous system outflow reaches its nadir, and the anterior pituitary releases its largest pulsatile surge of growth hormone (GH) (van Cauter & Tasali, 2023). Experimental suppression of N3 sleep in adolescents and young adults—even without reducing total sleep duration—rapidly decreases insulin sensitivity by 20% to 30% and impairs acute insulin response to glucose (Simon et al., 2019; Tasali et al., 2020). In urban youth, late bedtimes compress early-night N3 opportunities, while nocturnal ambient noise from road traffic and late-night digital notifications induce cortical micro-arousals that fragment delta wave continuity (Basner & McGuire, 2018; Rudzik et al., 2020).

Conversely, early school start times selectively amputate the final hours of the nocturnal sleep period, which are predominantly composed of **REM sleep** (Crowley et al., 2020; Wheaton et al., 2020). REM sleep loss disrupts nocturnal autonomic recalibration, leaving adolescents in a state of persistent morning sympathetic dominance characterized by reduced heart rate variability (HRV) and elevated resting heart rate (Castro-Diehl et al., 2021; Morales-Ghinaglia et al., 2023). Moreover, neuroimaging studies demonstrate that REM truncation impairs prefrontal cortical top-down inhibition of the amygdala and ventral striatum, amplifying neural reward reactivity to hyper-palatable, high-glycemic foods during daytime waking hours (Duraccio et al., 2021; Jensen et al., 2019).

Molecular and Neuroendocrine Mechanisms of Metabolic Dysregulation

The transition from circadian and sleep architectural disruption to overt clinical metabolic pathology is mediated by four interlocking physiological mechanisms: central–peripheral clock desynchronization, impaired insulin signaling and glucose disposal, nocturnal lipolysis and hepatic steatosis, and neuroendocrine appetite dysregulation.

Central–Peripheral Clock Desynchronization

Virtually every nucleated cell in metabolic organs—including hepatocytes, skeletal myocytes, white and brown adipocytes, and pancreatic β-cells—harbors an autonomous molecular circadian clock driven by a transcription–translation feedback loop (TTFL) involving core clock proteins **BMAL1**, **CLOCK**, **PER1/2**, and **CRY1/2** (Panda, 2019; Poggiogalle et al., 2020). These peripheral clocks rhythmically regulate up to 15%

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of the local transcriptome, gating key rate-limiting enzymes in glycolysis, gluconeogenesis, lipogenesis, and mitochondrial oxidative phosphorylation to specific times of day (Reinke & Asher, 2019).

While the central SCN clock is entrained primarily by light, peripheral clocks in the liver and gastrointestinal tract are powerfully entrained by **feeding–fasting cycles** and nutrient-sensing pathways (AMPK, mTOR, SIRT1) (Chaix et al., 2019; Manoogian et al., 2022). Urban youth experiencing circadian misalignment routinely shift caloric intake into the biological night—skipping breakfast due to rushed morning school commutes and consuming energy-dense dinners and snacks late at night when endogenous melatonin levels are rising (Beebe et al., 2020; Zerón-Rugerio et al., 2020). Late-night food intake phase-shifts hepatic and adipose clocks independently of the light-entrained SCN, creating **internal desynchronization** between central and peripheral oscillators (Chellappa et al., 2021; Mason et al., 2020).

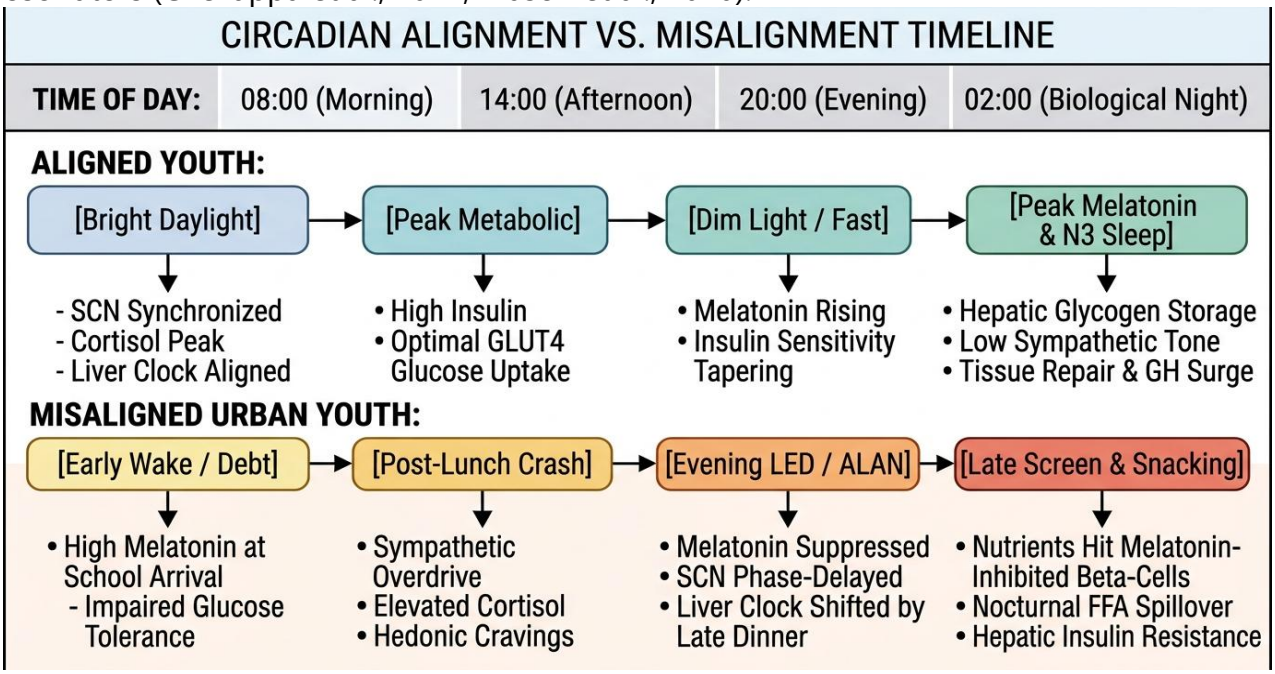


Figure 2. Diurnal Temporal Architecture Comparing Circadian-Aligned Physiology Versus Internal Central–Peripheral Desynchronization in Urban Youth.

Circadian misalignment directly impairs whole-body glucose homeostasis through dual pancreatic and peripheral mechanisms. First, pancreatic β-cells express melatonin membrane receptors (**MT1** and **MT2**); binding of endogenous melatonin during the biological night inhibits adenylate cyclase and cAMP signaling, physiologically suppressing glucose-stimulated insulin secretion while the organism is meant to be

fasting (Garaulet et al., 2020; Mason et al., 2020). When circadian-misaligned adolescents consume carbohydrates late at night—or wake early for breakfast while morning melatonin remains elevated due to a delayed DLMO—circulating melatonin directly blunts β-cell insulin release, producing exaggerated postprandial hyperglycemia (Eckel et al., 2019; Garaulet et al., 2020).

Second, skeletal muscle—which accounts for ~80% of insulin-stimulated glucose disposal—exhibits intrinsic circadian rhythmicity in insulin sensitivity, peaking in the biological morning and declining toward evening (Hansen et al., 2023; Wefers et al., 2020). Experimental circadian misalignment dampens skeletal muscle *BMAL1* expression, impairs insulin-receptor substrate-1 (IRS-1) phosphorylation, and reduces GLUT4 glucose transporter translocation to the sarcolemma (Wefers et al., 2020). Concurrently, elevated evening and nocturnal cortisol levels stimulate hepatic phosphoenolpyruvate carboxykinase (PEPCK) and glucose-6-phosphatase, driving unrestrained hepatic gluconeogenesis despite elevated circulating insulin (Simon et al., 2019). Longitudinal cohort studies confirm that higher social jetlag and shorter sleep duration in adolescence predict significantly elevated Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) and glycated hemoglobin (HbA1c) trajectories independent of body mass index (BMI) (Cespedes Feliciano et al., 2019; Higgins et al., 2021).Circadian misalignment profoundly disrupts lipid trafficking across adipose and hepatic tissues. Under aligned conditions, insulin suppresses hormone-sensitive lipase in white adipose tissue (WAT) during postprandial periods, while nocturnal fasting gently mobilizes free fatty acids (FFAs) for oxidative metabolism. In sleep-restricted, circadian-misaligned youth, elevated nocturnal catecholamines and cortisol stimulate excessive lipolysis, flooding the portal circulation with non-esterified fatty acids (NEFAs) during the biological night (Broussard et al., 2020; Ness et al., 2022). Excess hepatic NEFA influx promotes ectopic triglyceride accumulation (hepatic steatosis) and stimulates very-low-density lipoprotein (VLDL) secretion, elevating fasting triglycerides and lowering high-density lipoprotein (HDL) cholesterol (Morales-Ghinaglia et al., 2023). Furthermore, exposure to artificial light at night suppresses sympathetic β_3-adrenergic signaling in **brown adipose tissue (BAT)**, impairing non-shivering thermogenesis and reducing fatty acid clearance, thereby favoring positive energy storage and visceral adiposity (Borniger et al., 2019; Fleury et al., 2020).Sleep architecture truncation and circadian misalignment alter peripheral satiety hormones that signal hypothalamic arcuate nucleus circuits. Short sleep and reduced N3 duration suppress anorexigenic **leptin** secretion from adipocytes while elevating orexigenic **ghrelin** release from gastric oxyntic cells, increasing subjective hunger and caloric intake by 250 to 500 kcal/day in

adolescents (Beebe et al., 2020; Duraccio et al., 2021; Lin et al., 2020). Simultaneously, circadian misalignment elevates circulating endocannabinoids (2-arachidonoylglycerol) during afternoon and evening hours, amplifying hedonic reward signaling and driving preferential consumption of ultra-processed, high-sugar, and high-fat snacks during evening screen use (Hanlon et al., 2020; Jensen et al., 2019).

Methodological Critique and Translational Interventions

Despite compelling mechanistic convergence, several methodological limitations in the 2019–2024 pediatric literature warrant critical scrutiny before translating findings into clinical guidelines.

Translational Domain	Current Empirical & Clinical Gap	Mechanistic Target	Recommended Intervention & Policy Strategy
Measurement & Phenotyping	Over-reliance on self-reported sleep duration and subjective chronotype questionnaires; conflation of sleep debt with circadian phase	Objective circadian phase (DLMO), ultradian sleep micro-architecture (N3 delta power), and glycemic variability	Integrate multi-week wrist actigraphy, continuous glucose monitoring (CGM), and salivary DLMO assays into pediatric cohort studies
Educational & Structural Policy	Early high-school start times (< 08:00 AM) enforce chronic weekday REM truncation and >= 2 hr social jetlag	Alignment of external social schedules with pubertal biological phase delay	Mandate secondary school start times at 08:30 AM or later to expand morning REM sleep and reduce weekday–weekend midsleep drift
Urban & Domestic Lighting	Ubiquitous evening blue-enriched LED	ipRGC melanopsin signaling, pineal melatonin	Implement spectral shifting (warm < 2700 K evening lighting,

Translational Domain	Current Empirical & Clinical Gap	Mechanistic Target	Recommended Intervention & Policy Strategy
	screens and outdoor urban ALAN alongside dim daytime classrooms	amplitude, and SCN phase entrainment	blue-light filters), bedroom screen curfews 60 min pre-bed, and bright daytime school lighting
Chrononutrition & Meal Timing	Late-night snacking during biological night; breakfast skipping	Peripheral hepatic/muscle clock alignment; avoidance of melatonin–insulin antagonism	Establish consistent daytime eating windows (e.g., 10–12 hr time-restricted eating), prioritizing morning protein/complex carbohydrates and fasting >= 3 hr pre-bed

Table 2. *Methodological Gaps in Pediatric Circadian Research and Multi-Level Mechanistic Interventions.*

First, a substantial proportion of large-scale urban youth studies relies on single-item self-reported sleep duration or subjective chronotype scales (e.g., the Munich ChronoType Questionnaire), which correlate only moderately with objective dim-light melatonin onset and polysomnographic sleep staging (Crowley et al., 2020; Meltzer et al., 2021). Second, epidemiological models frequently treat sleep duration, social jetlag, and evening screen exposure as independent covariates without accounting for their collinearity and bidirectional mediation. Third, research in rapidly urbanizing LMICs remains underrepresented relative to North American and European cohorts, despite urban youth in South Asia, East Asia, and Latin America experiencing distinct combinations of high academic night-study pressure, dense air pollution, and rapid nutritional transitions (Jain et al., 2023; Morales-Ghinaglia et al., 2023).

Addressing circadian-mediated metabolic risk requires moving beyond individual sleep-hygiene counseling toward structural and environmental interventions (Chaput et al., 2020). Delaying secondary school start times to 08:30 AM or later represents one of the most empirically validated population-level interventions, consistently increasing adolescent sleep duration by 30 to 60 minutes, reducing social jetlag, and improving daytime alertness and metabolic markers (Dunster et al., 2021; Wheaton et al., 2020). Complementing schedule reform, architectural and municipal lighting policies—such as shielding outdoor LED streetlights, increasing daytime classroom illuminance, and enforcing pre-bedtime digital curfews—protect endogenous melatonin rhythmicity (Bumgarner et al., 2023; Nagare et al., 2019). Finally, clinical chrononutrition strategies that consolidate caloric intake into the biological day prevent nutrient-induced uncoupling of hepatic and muscle clocks from the central SCN pacemaker (Manoogian et al., 2022; Zerón-Rugerio et al., 2020).

Conclusion

Urbanization exposes developing youth to a chronic mismatch between endogenous pubertal circadian biology and modern photic, social, and nutritional schedules. Far from being a benign lifestyle inconvenience, circadian misalignment remodels sleep architecture—truncating restorative slow-wave and REM sleep—while uncoupling central hypothalamic pacemakers from peripheral metabolic oscillators. Through melatonin–insulin antagonism, nocturnal sympathetic and glucocorticoid elevation, hepatic lipogenesis, and hedonic appetite dysregulation, chronic circadian disruption accelerates insulin resistance and visceral adiposity during a critical window of somatic development. Protecting adolescent metabolic health in an urbanizing world requires recognizing circadian alignment as a foundational pillar of pediatric preventive medicine, supported by delayed school start times, circadian-informed urban lighting, and structured daytime chrononutrition.

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