MINISTRY OF EDUCATION AND SCIENCE OF UKRAINE
SUMY STATE UNIVERSITY
ACADEMIC AND RESEARCH MEDICAL INSTITUTE

Eastern Ukrainian Medical Journal

116, Kharkivska st., Sumy 40007, Ukraine eumj.med.sumdu.edu.ua
e-mail: eumj@med.sumdu.edu.ua ISSN: 2663-5909 (print)/2664-4231 (online)

How to cite: Garnytska A, Orlyk O, Kochuieva M, Psarova V. EFFECT OF DAYLIGHT EXPOSURE ON GLYCEMIC CONTROL IN ELDERLY PATIENTS WITH 2 TYPE DIABETES. LITERATURE REVIEW. *East Ukr Med J*. 2026;14(2):301-313. DOI: https://doi.org/10.21272/eumj.2026;14(2);301-313

ABSTRACT

Anna Garnytska
https://orcid.org/0000-0002-3554-8864
Shupic National Healthcare University of Ukraine, Kyiv, Ukraine

Olga Orlyk
https://orcid.org/0000-0003-0040-1579
State Scientific Institution "Center for Innovative Medical Technologies of the NAS of Ukraine", Kyiv, Ukraine

Maryna Kochuieva
https://orcid.org/0000-0002-1516-2155
Shupic National Healthcare University of Ukraine, Kyiv, Ukraine

Valentyna Psarova
https://orcid.org/0000-0001-6890-272X
Sumy State University, Sumy, Ukraine

EFFECT OF DAYLIGHT EXPOSURE ON GLYCEMIC CONTROL IN ELDERLY PATIENTS WITH 2 TYPE DIABETES. LITERATURE REVIEW

Type 2 diabetes mellitus (T2DM) is highly prevalent among older adults, and optimal glycemic control remains essential for preventing complications. Increasing evidence highlights the role of circadian rhythms and environmental factors, particularly light exposure, in glucose metabolism. Natural daylight acts as a key regulator of circadian function, whereas disruption of the light–dark cycle, especially artificial light at night, contributes to insulin resistance and impaired glycemic control.

Aim of the study: to analyze current scientific evidence on the impact of natural daylight exposure on glycemic control in elderly patients with type 2 diabetes mellitus and to identify potential mechanisms underlying this effect.

Object of the study: metabolic and circadian mechanisms of glycemic regulation in elderly patients with type 2 diabetes mellitus under conditions of varying exposure to natural and artificial lighting.

Results: This review summarizes current clinical, experimental, and epidemiological data on the impact of light exposure on metabolic regulation in elderly patients with T2DM. Adequate daytime exposure to natural light is associated with improved glycemic control, increased time in target range, and reduced glucose variability. In contrast, nocturnal light exposure disrupts circadian regulation, suppresses melatonin, and increases the risk of metabolic disturbances.

Recent studies, including randomized and large-scale epidemiological analyses, confirm that light environment significantly influences glucose homeostasis.

Conclusions

1. Circadian rhythms and light exposure are important regulators of glucose metabolism.
2. Adequate exposure to natural daylight contributes to improved

glycemic control in patients with type 2 diabetes mellitus.

3. Excessive artificial light exposure during nighttime disrupts circadian regulation and is associated with an increased risk of insulin resistance and diabetes development.

4. Optimization of the light–dark cycle may be considered a promising non-pharmacological component of comprehensive T2DM management in elderly patients.

5. Further clinical studies are required to determine the optimal duration and intensity of light exposure for improving metabolic outcomes.

Keywords: type 2 diabetes mellitus, daylight, circadian rhythms, glycemic control, insulin resistance, light therapy, elderly patients.

Corresponding author: Olga Orlyk, State Scientific Institution "Center for Innovative Medical Technologies of the NAS of Ukraine", Kyiv, Ukraine
e-mail: olgaorlyk1982@gmail.com

РЕЗЮМЕ

Анна Гарницька
https://orcid.org/0000-0002-3554-8864
Національний університет охорони здоров'я України імені П. Л. Шупика, Київ, Україна

Ольга Орлик
https://orcid.org/0000-0003-0040-1579
Державна наукова установа «Центр інноваційних медичних технологій НАН України», Київ, Україна

Марина Кочуєва
https://orcid.org/0000-0002-1516-2155
Національний університет охорони здоров'я України імені П. Л. Шупика, Київ, Україна

Валентина Псарьова
https://orcid.org/0000-0001-6890-272X
Сумський державний університет, Суми, Україна

ВПЛИВ ДЕННОГО СВІТЛА НА ГЛІКЕМІЧНИЙ КОНТРОЛЬ У ПАЦІЄНТІВ ЛІТНЬОГО ВІКУ З ЦУКРОВИМ ДІАБЕТОМ 2 ТИПУ. ОГЛЯД ЛІТЕРАТУРИ

Цукровий діабет 2-го типу (ЦД2) є надзвичайно поширеним серед осіб похилого віку, а досягнення оптимального глікемічного контролю залишається ключовим чинником профілактики ускладнень. Зростає кількість доказів, що підкреслюють роль циркадних ритмів і факторів навколишнього середовища, зокрема світлового впливу, у регуляції глюкозного метаболізму. Природне денне світло виступає основним регулятором циркадної функції, тоді як порушення циклу «світло–темрява», особливо вплив штучного освітлення вночі, сприяє розвитку інсулінорезистентності та погіршенню глікемічного контролю.

Мета дослідження: проаналізувати сучасні наукові дані щодо впливу природного денного освітлення на глікемічний контроль у пацієнтів похилого віку з цукровим діабетом 2 типу та визначити можливі механізми цього впливу.

Об'єкт дослідження: метаболічні та циркадні механізми регуляції глікемії у пацієнтів похилого віку з цукровим діабетом 2 типу в умовах різного рівня впливу природного та штучного освітлення.

Результати. У даному огляді узагальнено сучасні клінічні, експериментальні та епідеміологічні дані щодо впливу світлового середовища на метаболічну регуляцію у пацієнтів похилого віку з ЦД2. Адекватна експозиція природного світла у денний час асоціюється з покращенням глікемічного контролю, збільшенням часу перебування у цільовому діапазоні глікемії та зменшенням її варіабельності. Натомість нічний вплив світла порушує циркадну регуляцію, пригнічує секрецію мелатоніну та підвищує ризик метаболічних порушень.

Останні дослідження, включаючи рандомізовані та великі епідеміологічні аналізи, підтверджують, що світлове середовище суттєво впливає на гомеостаз глюкози.

Висновки:

1. Циркадні ритми та світловий вплив є важливими регуляторами метаболізму глюкози.

2. Адекватний вплив природного денного світла сприяє покращенню глікемічного контролю у пацієнтів із цукровим діабетом 2 типу.

3. Надмірний вплив штучного освітлення вночі порушує циркадну регуляцію та пов'язаний із підвищеним ризиком інсулінорезистентності й розвитку діабету.

4. Оптимізація циклу світло–темрява може розглядатися як перспективний немедикаментозний компонент комплексного лікування ЦД2 у пацієнтів похилого віку.

5. Необхідні подальші клінічні дослідження для визначення оптимальної тривалості та інтенсивності світлового впливу з метою покращення метаболічних показників.

***Автор, відповідальний за листування**: Ольга Орлик, Державна наукова установа «Центр інноваційних медичних технологій НАН України», Київ, Україна*
e-mail: olgaorlyk1982@gmail.com

ABBREVIATIONS

T2DM – Type 2 Diabetes Mellitus
SCN – Suprachiasmatic Nucleus
CGM – Continuous Glucose Monitoring
HbA1c – Glycated Hemoglobin
GSP – Glycated Serum Protein
RER (RQ) – Respiratory Exchange Ratio
DLMO – Dim Light Melatonin Onset
PSQI – Pittsburgh Sleep Quality Index
MEQ – Morningness–Eveningness Questionnaire
LAN – Light At Night
RCT – Randomized Controlled Trial
aHR – Adjusted Hazard Ratio
SD – Standard Deviation
PER / CRY / BMAL – Clock genes (circadian regulators)

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is one of the most common chronic diseases among older adults. It is estimated that more than 19% of people aged 65 years and older worldwide suffer from T2DM. Proper glycemic control in this patient population is critically important, as poorly controlled diabetes leads to complications such as cardiovascular disease, nephropathy, retinopathy, and neuropathy, which are particularly dangerous in older individuals.

Traditional approaches to improving glycemic control include pharmacotherapy, dietary modification, and physical activity. However, increasing attention is being paid to lifestyle-related factors such as sleep–wake patterns, circadian rhythms, and environmental influences, including lighting conditions, which may significantly affect glucose metabolism and diabetes progression.

The human body possesses an endogenous circadian system – a biological clock that generates approximately 24-hour rhythms in physiological processes and synchronizes them with the daily light–dark cycle. Circadian rhythm regulation is controlled by the suprachiasmatic nucleus (SCN) of the hypothalamus, which coordinates peripheral clocks in various organs such as the liver and skeletal muscles [1–8].

Light is the primary external zeitgeber, determining the phase of the central circadian clock and enabling the organism to adapt to the daily environmental cycle. During daytime, natural sunlight reaches high intensity and provides a broad spectral composition, effectively synchronizing the biological clock with environmental conditions. At night, darkness maintains normal melatonin secretion, ensuring physiological rest and recovery.

In modern society, lifestyle changes have profoundly affected circadian rhythms. Approximately 80% of the global population is regularly exposed to artificial light at night, while many individuals work night shifts or maintain irregular sleep schedules, resulting in circadian desynchronization. At the same time, most people spend approximately 90% of their time indoors, frequently under artificial lighting, thereby receiving insufficient natural daylight exposure.

Among elderly individuals, this problem may be even more pronounced. Many older adults, particularly residents of long-term care facilities, spend limited time outdoors. Age-related ocular changes, such as lens opacity and reduced retinal sensitivity, further decrease the physiological response to light.

Insufficient daytime light exposure has been associated with sleep disturbances, reduced sleep quality, and depressive symptoms in older adults. Consequently, disruption of the natural light–dark cycle has become a common phenomenon, attracting growing research interest regarding its metabolic consequences.

Increasing evidence suggests that chronic circadian dysregulation contributes to the development of metabolic diseases, including diabetes. Epidemiological studies have demonstrated associations between various forms of circadian disruption – night shift work, late food intake, evening chronotype, and sleep deprivation – and an increased risk of T2DM [1–8].

For example, night shift work and chronic sleep restriction have been linked to higher diabetes incidence. Large cohort studies using wearable light sensors have confirmed that light exposure at inappropriate circadian phases negatively affects metabolic health: individuals exposed to higher levels of artificial light at night have a greater risk of developing T2DM.

Laboratory experiments further demonstrate that artificially induced circadian misalignment impairs glucose tolerance and reduces insulin sensitivity even in healthy individuals. Notably, a single night of sleep under moderate illumination (~100 lux) has been shown to increase heart rate and induce measurable insulin resistance the following morning.

Therefore, optimizing lighting conditions – bright natural light during daytime and minimal light exposure at night – is increasingly considered a potentially underestimated component of diabetes prevention and management [1–8]

In this context, an important question arises: can increased exposure to natural daylight improve glycemic control in elderly patients with T2DM?

Hypothetically, stronger daytime light signals may enhance circadian rhythm amplitude, synchronize peripheral clocks, and optimize diurnal fluctuations of glucose metabolism. This effect may be particularly relevant in elderly individuals, who often exhibit weakened circadian rhythms and sleep disturbances.

This review therefore examines current scientific evidence regarding the impact of natural daylight exposure on glucose metabolism, circadian rhythms, and glycemic control parameters in elderly patients with type 2 diabetes mellitus.

Literature Review

Circadian Rhythms and Glucose Homeostasis

Physiological processes involved in glucose metabolism demonstrate pronounced circadian periodicity. In healthy individuals, the phenomenon of diurnal variation in glucose tolerance has long been established: glucose is metabolized more efficiently in the morning than in the evening. The same meal or glucose load typically results in a greater increase in blood glucose levels in the evening, indicating poorer glucose tolerance compared with the morning.

This phenomenon is explained by both reduced insulin sensitivity in the evening and decreased insulin secretion by pancreatic β-cells later in the day. During normal nocturnal sleep, basal hepatic glucose production generally decreases, while the secretion of counterregulatory hormones changes in a manner that maintains relatively stable glycemia until morning.

In patients with diabetes, however, the so-called "dawn phenomenon" is frequently observed. This refers to an early-morning increase in fasting blood glucose levels associated with circadian fluctuations in hormones such as cortisol, adrenaline, and growth hormone, as well as increased insulin resistance at the beginning of the active phase of the day [1–8].

The natural alternation of daytime light and nighttime darkness is essential for maintaining the appropriate structure of these metabolic rhythms. Daylight acts as a strong physiological signal that promotes wakefulness, activates the sympathetic nervous system, increases morning cortisol secretion, and prepares the body for daytime activity. Conversely, darkness at night stimulates melatonin secretion by the pineal gland, facilitating physiological rest and recovery, increasing parasympathetic tone, and slowing both heart rate and metabolic activity.

Melatonin and circadian signaling also regulate the metabolic state associated with overnight fasting. At night, glucose tolerance decreases, and the body normally relies less on glucose and more on lipid oxidation for energy. Melatonin has been shown to suppress insulin secretion by pancreatic β-cells; therefore, food intake late in the evening – when melatonin levels are elevated – can lead to a more pronounced rise in blood glucose levels. These mechanisms represent evolutionarily conserved processes that synchronize energy metabolism with the natural day–night cycle [1–8].

Circadian Dysregulation and the Development of Diabetes

Disruption of the daily light–dark cycle – such as exposure to artificial light at night, irregular sleep–wake schedules, or insufficient daytime light exposure – can impair circadian regulation of metabolism. As a result, disturbances occur in the secretion of key metabolic hormones (including insulin, incretins, ghrelin, and leptin), tissue insulin sensitivity becomes misaligned with metabolic demands, and alterations in appetite regulation and energy intake may develop.

Experimental studies in both animals and humans have demonstrated that nocturnal light exposure, which shifts or suppresses normal circadian rhythms, may lead to hyperglycemia, impaired insulin secretion, dyslipidemia, and weight gain. Consequently, chronic disruption of the natural light–dark cycle is increasingly recognized as a risk factor for insulin resistance and the development of type 2 diabetes [1–8].

Large-scale observational studies provide further support for this association. In the UK Biobank study, approximately 85,000 middle-aged and older participants wore wrist-based light sensors for one week to measure personal light exposure. The results demonstrated a clear association between higher nighttime light exposure and an increased risk of developing diabetes during approximately eight years of follow-up.

Individuals exposed to the highest levels of nighttime light (upper quartile of exposure) had a 53% higher risk of developing type 2 diabetes compared with those who slept in near-complete darkness. Moreover, individuals with low circadian light amplitude – that is, those who did not receive sufficient daylight or lacked a clear contrast between day and night – also exhibited an increased risk of diabetes.

Importantly, the detrimental effect of nighttime light exposure appeared to be independent of genetic susceptibility. The difference in diabetes risk between individuals sleeping in bright light versus darkness was comparable to the difference observed between individuals with low and moderate genetic risk for diabetes. These findings suggest that reducing nighttime light exposure may represent a simple and cost-effective strategy for lowering diabetes risk, even among individuals with increased genetic predisposition.

The Role of Daytime Light Exposure

If nighttime light exposure has negative metabolic consequences, an important question arises: can increased exposure to daylight improve metabolic health?

Several mechanisms support this hypothesis. First, a pronounced contrast between bright daytime light and dark nights helps maintain a robust circadian rhythm characterized by adequate amplitude of hormonal fluctuations and proper timing of metabolic processes.

Second, daylight exposure may influence behavioral and lifestyle factors. Brighter daytime environments increase alertness and activity levels and may encourage greater physical activity among older adults, who tend to remain more awake and active under brighter conditions and nap less during the day. Daylight exposure may also improve mood and sleep quality, indirectly contributing to improved glycemic control.

Third, several clinical studies have investigated light therapy interventions, in which controlled bright light exposure during the daytime was administered to patients with diabetes or individuals at metabolic risk in order to evaluate potential metabolic benefits.

In the following section, “Analysis of Contemporary Studies,” we examine the findings of recently published research – ranging from clinical trials involving elderly patients to controlled laboratory experiments – that provide scientific evidence addressing these questions.

METHODOLOGY

This study represents a narrative literature review with elements of analytical synthesis of contemporary research findings.

A systematic search of peer-reviewed publications was conducted in the PubMed, Scopus, and Google Scholar databases using keywords in both Ukrainian and English, including: *daylight, natural light exposure, circadian rhythms, glucose metabolism, type 2 diabetes, elderly patients, light therapy*, and related terms.

Particular emphasis was placed on studies published between 2015 and 2025 in high-impact medical journals such as Cell Metabolism, JAMA, Diabetes Care, Lancet Diabetes & Endocrinology, and others that investigated the relationship between lighting conditions – particularly natural daylight exposure – and markers of glycemic control.

The analysis included:

Randomized controlled trials (RCTs) evaluating the effects of different lighting conditions (natural versus artificial) on metabolic outcomes in patients with type 2 diabetes. This included a recent crossover RCT assessing the impact of workplace daylight exposure on glycemic control in older adults, as well as RCTs investigating light therapy interventions in long-term care facilities.

Observational (epidemiological) studies assessing the association between long-term exposure to natural or artificial lighting (e.g., nighttime light exposure) and the incidence of diabetes or measures of glycemic control.

Experimental studies investigating the acute metabolic effects of light and darkness, including sleep studies conducted under different lighting conditions and controlled circadian laboratory protocols assessing insulin resistance.

Review articles and meta-analyses focusing on circadian biology and metabolic regulation, which provide mechanistic insights and a broader synthesis of current evidence.

Priority was given to studies involving middle-aged and older populations, particularly those reporting clinical endpoints related to glycemic regulation, including blood glucose levels, glycated hemoglobin (HbA1c), time in target glucose range, indices of insulin resistance, or indirect biomarkers such as melatonin levels and circadian metabolic profiles.

Animal studies and isolated case reports were excluded to ensure a focus on human clinical evidence.

The information obtained from selected sources was organized into thematic sections. Summary tables present a concise comparative overview of key studies.

Analysis and Results of Randomized Studies on Light Exposure

Several randomized and controlled experimental studies have investigated the influence of lighting conditions on glycemic control in adults with type 2 diabetes (T2D), particularly in older populations.

One of the most informative studies was a crossover randomized controlled trial published in *Cell Metabolism* in 2025. The study included 13 patients with T2D (mean age approximately 70 years; mean HbA1c 6.8%). Each participant underwent two experimental conditions lasting 4.5 days: exposure to natural daylight during daytime hours (08:00–17:00) in a room with large windows allowing sunlight exposure, and artificial lighting, during which participants remained in a comparable room illuminated only by standard office electric lighting without access to daylight [1, 9–13]. Evening and nighttime conditions were identical for both interventions. Daily schedules, dietary intake, and physical activity were standardized to isolate the effect of lighting conditions. The primary outcome was glycemic control assessed using continuous glucose monitoring (CGM).

Exposure to natural daylight was associated with improved glycemic stability. Although the mean 24-hour glucose level did not differ significantly between conditions (approximately 7.4 mmol/L under daylight vs. 7.8 mmol/L under artificial lighting; $p = 0.37$), time within the target glycemic range (3.9–10 mmol/L) was significantly greater during daylight exposure (50.9% vs. 43.3% of the day; $p = 0.036$) [1, 9–13]. In addition, glycemic variability decreased, as indicated by a significant reduction in the amplitude of glucose fluctuations during the daylight condition ($p = 0.010$). These findings suggest that daytime natural light exposure may improve overall glycemic stability in individuals with T2D.

Metabolic substrate utilization was also affected by lighting conditions. Participants exposed to natural daylight demonstrated a lower respiratory quotient (RQ/RER) after lunch and during the remainder of the day, indicating increased fatty acid oxidation and reduced carbohydrate utilization compared with artificial lighting conditions ($p < 0.05$) [14–33]. Similar metabolic patterns were observed during a standardized mixed-meal tolerance test performed on the final study day.

Hormonal analyses revealed differences in circadian endocrine responses. Evening melatonin secretion, assessed as the area under the curve, was significantly higher following the daylight exposure period compared with the artificial lighting condition ($p = 0.029$), although the timing of dim light melatonin onset (DLMO) remained unchanged [14–33]. These findings suggest that daytime natural light exposure increased circadian amplitude, strengthening the contrast between daytime and nighttime biological signals. Enhanced nocturnal melatonin secretion may support metabolic recovery processes and potentially improve insulin sensitivity.

Molecular analyses provided additional mechanistic insights. Skeletal muscle biopsies demonstrated increased expression of several core circadian clock genes, including *PER1*, *PER2*, and *CRY1*, following daytime daylight exposure (p = 0.01; 0.066; and 0.021, respectively) [14–33]. These results indicate improved synchronization of peripheral metabolic clocks with the central circadian pacemaker. Multi-omics analyses further revealed alterations in metabolites and gene expression pathways related to glucose metabolism.

Overall, this study provided direct experimental evidence that daytime exposure to natural light may positively influence metabolic regulation in individuals with T2D. Even short-term exposure to daylight resulted in increased time in normoglycemia, reduced glycemic variability, and metabolic shifts toward greater lipid oxidation [9, 13–24].

Additional evidence comes from a randomized trial conducted in long-term care facilities in China involving elderly patients with T2D and sleep disturbances [14-33]. In this study, 45 participants (mean age approximately 85 years) were randomized to receive either four weeks of bright light therapy or standard care. The intervention consisted of daily exposure to high-intensity light, most likely administered during morning hours. Primary outcomes included sleep quality (Pittsburgh Sleep Quality Index and wearable sleep monitoring) and chronotype assessed using the Morningness–Eveningness Questionnaire (MEQ). The secondary outcome was glycated serum protein (GSP), reflecting average glycemia over approximately two weeks.

After four weeks of treatment, participants in the light therapy group demonstrated significant improvements in sleep quality, including lower PSQI scores and increased objective sleep duration compared with controls [23–33]. Chronotype also shifted toward a more morning-oriented circadian pattern, indicating improved circadian alignment.

However, no statistically significant differences in glycemic markers were observed between groups. GSP levels did not differ significantly immediately after the intervention or at four-week follow-up [23–33]. Nevertheless, a non-significant trend toward reduced GSP was observed in the intervention group, whereas values slightly increased in the control group. The authors suggested that longer intervention periods or inclusion of patients with poorer baseline glycemic control may be required to detect metabolic effects.

Importantly, the observed improvements in sleep and circadian regulation may indirectly contribute to long-term metabolic benefits.

Another randomized trial evaluated the effects of morning bright light therapy in patients with T2D and comorbid major depressive disorder [15–33]. In this study, 83 participants received either 10,000 lux white light exposure for 30 minutes each morning or placebo light for four weeks. The primary outcomes were changes in depressive symptoms (Inventory of Depressive Symptomatology) and insulin sensitivity measured using the hyperinsulinemic–euglycemic clamp technique.

Light therapy did not significantly outperform placebo in reducing depressive symptoms or improving insulin sensitivity in the overall cohort [15–33]. However, subgroup analyses suggested that patients with higher baseline insulin resistance experienced greater improvements in depressive symptoms following light therapy compared with individuals with better metabolic status. These findings highlight the complex interaction between circadian regulation, mood disorders, and metabolic processes.

Interim conclusion. Evidence from randomized studies suggests that increased exposure to natural or high-intensity light during daytime hours may contribute to improved metabolic stability in patients with T2D. The strongest evidence comes from experimental studies demonstrating increased time in normoglycemia and reduced glycemic variability during daytime natural light exposure [23–33]. Other trials have primarily shown improvements in circadian alignment and sleep quality, which may indirectly support metabolic health. Collectively, these findings indicate that optimization of daytime light exposure represents a potentially important, non-pharmacological factor in diabetes management.

Observational Evidence and Circadian Factors

In addition to interventional studies, observational evidence should also be considered, as it reflects real-life conditions and the long-term impact of the light environment on diabetes risk. The previously mentioned study from the United Kingdom (UK Biobank) demonstrated that older adults who sleep with artificial light exposure have a significantly higher risk of subsequently developing type 2 diabetes (T2D).

Another large epidemiological study conducted in China and published in *Diabetologia* (2022) used satellite-based mapping of nighttime illumination. The study found that regions and cities with the highest levels of outdoor artificial light at night exhibited a higher prevalence and increased risk of diabetes among the population. Importantly, this association persisted after adjustment for urbanization, socioeconomic status, and other potential confounding factors. These findings suggest that urban light pollution may represent a novel environmental contributor to the global diabetes epidemic.

Cohort studies from several countries indicate that sleeping in complete darkness is associated with better metabolic health. For example, a study published in *JAMA Internal Medicine* followed more than 43,000 women in the United States over a five-year period. Women who reported sleeping with a television or night light on were more likely to experience weight gain and had a higher risk of developing obesity compared with those who slept without artificial light. Because obesity is closely associated with insulin resistance and diabetes, these findings support the hypothesis that nighttime light exposure may negatively affect metabolic health.

Furthermore, sleeping in illuminated environments has been associated with higher fasting glucose levels and poorer glycemic control in several population studies, including research conducted among older adults in the United States. These observations further support the potential role of nighttime light exposure as a metabolic risk factor.

Several mechanisms may explain the adverse effects of nighttime light exposure. Even moderate illumination (~100 lux), which corresponds to dim indoor lighting or television light, can activate the sympathetic nervous system, increase heart rate, and prevent the body from fully entering the physiological nocturnal resting state. As a result, elevated fasting insulin levels and signs of insulin resistance may be observed the following morning. This effect was clearly demonstrated in an experimental study conducted at Northwestern University (USA), where even a single night of sleep under moderate light exposure increased markers of insulin resistance in healthy volunteers compared with sleep in complete darkness.

In essence, the human body may interpret nocturnal light exposure as a signal of daytime activity, thereby disrupting the normal nighttime metabolic transition and contributing to dysglycemia. Another potential mechanism involves suppression of melatonin secretion. Light exposure, particularly in the blue spectrum, is known to delay or reduce melatonin production. Reduced nocturnal melatonin levels may negatively affect pancreatic β-cell function and other metabolic processes, although these mechanisms remain under investigation.

It should also be noted that individuals who sleep with artificial light exposure may exhibit other unfavorable lifestyle habits, such as late evening meals or reduced total sleep duration, which may further contribute to impaired glycemic regulation.

Conversely, daytime light exposure – particularly natural daylight – appears to be associated with favorable health outcomes. Observational studies indicate that individuals working in offices with access to natural daylight report higher productivity, improved sleep quality, and potentially a lower risk of chronic diseases. One study demonstrated that employees with workplace access to natural light slept, on average, 46 minutes longer per night and reported better overall quality of life compared with individuals working in windowless environments. Although these findings do not directly measure glycemic outcomes, they highlight the importance of daylight exposure as a component of healthy circadian regulation.

In the context of older adults with diabetes, observations from long-term care facilities are particularly informative. Facilities that provide higher daytime light intensity – approaching natural daylight conditions – have reported better circadian rhythm stability and improved sleep quality among residents. These findings suggest that optimizing daytime lighting conditions may help restore circadian alignment in elderly individuals, potentially contributing to improved metabolic regulation. Table 1 summarizes key contemporary studies examining the relationship between light exposure and glycemic control, with particular emphasis on middle-aged and older adults with type 2 diabetes [1, 2, 10, 11, 16] .

Table 1 **– Brief overview of selected studies on the effects of light exposure patterns on glycemic control**

Study (Year)	Population and Study Design	Lighting Conditions	Main Outcomes for Glycemic Control
Harmsen et al., 2025 (Cell Metabolism) – crossover RCT	13 patients with T2D, mean age ~70 years; two experimental periods of 4.5 days each	1. Natural daylight exposure (windowed environment) 08:00–17:00; 2. Artificial office lighting 08:00–17:00 (control)	Time in the target glycemic range increased during daylight exposure (50.9% vs 43.3%, p = 0.036). Daily glucose variability decreased (p = 0.01). Metabolic shifts toward greater lipid oxidation (lower RER) were observed during daylight exposure, accompanied by higher evening melatonin levels and increased expression of circadian clock genes in skeletal muscle.
Wang et al., 2024 (Frontiers in Endocrinology) – RCT	45 elderly patients with T2D residing in a long-term care facility (mean age ~85 years); 4-week intervention	1. Daily bright light therapy during daytime (4 weeks); 2. Standard lighting conditions (control)	Sleep quality improved in the light therapy group (↓PSQI, ↑sleep duration). Circadian rhythm shifted toward a more morning-oriented chronotype (MEQ). No significant differences in glycated serum protein (GSP) between groups; however, a non-significant trend toward decreased GSP in the light therapy group and increased levels in the control group was observed. Conclusion: four weeks of light therapy did not significantly change glycemic markers, although longer exposure may produce metabolic benefits.
Phillips et al., 2024 (Lancet Regional Health – Europe) – prospective cohort	~85,000 adults (UK Biobank), mean age 62 years; follow-up ~7.9 years	Personal light exposure measured using wrist-worn sensors for 7 days; analysis of daytime and nighttime light patterns (intensity, circadian amplitude)	Higher nighttime light exposure was associated with an increased risk of T2D. Participants with the brightest nighttime exposure had a 53% higher diabetes risk compared with those sleeping in darkness (aHR ≈1.53). Low circadian light amplitude (weak contrast between day and night) was also associated with increased risk (HR ≈1.07 per 1 SD decrease). Conclusion: nighttime light exposure and disrupted circadian light rhythms represent independent risk factors for diabetes.
Zee et al., 2022 (PNAS / Scientific Reports) – laboratory experiment	20 healthy adults, mean age ~37 years; crossover protocol with two nights in a sleep laboratory	1. Nighttime sleep under moderate light (~100 lux; simulated night light/television); 2. Sleep under dim light (<3 lux; near darkness)	After one night under 100 lux: increased nighttime heart rate and reduced heart rate variability (indicative of increased sympathetic activation). In the morning, participants demonstrated reduced insulin sensitivity, higher glucose levels, and markers of insulin resistance. These changes were absent under dark conditions. Conclusion: even acute exposure to moderate nighttime light can impair glucose regulation and cardiometabolic parameters.
Brouwer et al., 2019 (Diabetes Care) – RCT (LiDiA trial)	83 patients with T2D and major depressive disorder, mean age ~57 years; 4-week intervention	1. Morning bright light therapy (10,000 lux) for 30 min daily; 2. Placebo light (<500 lux green light) for 30 min daily	Depression outcomes: bright light therapy was not superior to placebo in reducing depressive symptoms. Metabolic outcomes: no significant improvement in insulin sensitivity (hyperinsulinemic–euglycemic clamp). No major adverse effects were reported. Subgroup analysis suggested greater antidepressant effects in patients with higher baseline insulin resistance, indicating potential benefits in metabolically vulnerable subgroups.

DISCUSSION OF RESULTS

The evidence presented above confirms that circadian light exposure represents a significant determinant of metabolic health and has important implications for the management of older adults with type 2 diabetes (T2D). Natural daylight exposure appears to exert beneficial metabolic effects: improved glycemic control (increased time in range and reduced glucose variability), optimization of substrate utilization between lipids and carbohydrates, and reinforcement of physiological hormonal rhythms, including melatonin secretion. In contrast, excessive nighttime light exposure is associated with disruption of these beneficial rhythms and with an increased risk of hyperglycemia and diabetes.

Overall, findings from different types of studies converge into a unified concept of circadian regulation of glycemic metabolism.

First, bright light during daytime is necessary to effectively activate the organism's daytime physiological mode. It promotes wakefulness, stimulates morning secretion of stress-related hormones, and synchronizes central and peripheral circadian clocks. In older adults, several factors may interfere with this process, including reduced mobility, prolonged indoor stay, and age-related decline in retinal light sensitivity. Therefore, strategies aimed at increasing daytime light exposure – such as outdoor walks, access to natural daylight through windows, or light therapy – may partially compensate for insufficient natural circadian signals.

Second, darkness in the evening and nighttime is equally important. Darkness effectively "switches off" daytime physiological processes, promotes melatonin secretion, and initiates the nocturnal metabolic state associated with recovery and restoration. Consequently, minimizing light exposure in the bedroom (e.g., reducing screen exposure and ensuring a dark sleeping environment) represents a simple recommendation that may indirectly improve glycemic control by enhancing sleep quality and increasing morning insulin sensitivity. This is particularly relevant for older adults who often keep night lights on because of fear of falls. In such cases, sleep medicine specialists recommend very dim, warm-colored lights positioned close to the floor, or other safety solutions that maintain visibility while minimizing circadian disruption.

Mechanisms Linking Light Exposure and Glycemic Regulation

The effects of light on glycemic control can be explained by the interaction between the circadian system and metabolic regulation. Daytime light activates neurons in the suprachiasmatic nucleus, which suppress melatonin secretion during the day and stimulate the release of cortisol and catecholamines. This results in transient increases in circulating glucose and free fatty acids, providing energy substrates for skeletal muscle activity.

More importantly, regular daylight exposure maintains the stability of the circadian oscillatory system. Under the influence of appropriate light signals, peripheral clock genes (including PER, CRY, and BMAL) exhibit rhythmic expression patterns. As a result, metabolically active organs such as the liver, skeletal muscles, and adipose tissue cyclically adjust insulin sensitivity and other metabolic processes in accordance with the daily activity schedule.

When daytime light exposure is insufficient, circadian rhythms may become attenuated, potentially leading to internal circadian desynchronization. In such conditions, metabolic organs may operate out of phase – for example, hepatic glucose production and pancreatic insulin secretion may become temporally mismatched – resulting in dysregulated glucose homeostasis.

Conversely, nighttime light exposure activates inappropriate signaling pathways. Blue-light–sensitive retinal ganglion cells containing melanopsin transmit signals to the brain indicating that daytime conditions persist, thereby suppressing or delaying melatonin secretion. Importantly, melatonin is not only a sleep-regulating hormone but also acts through receptors distributed throughout the body and plays a role in metabolic regulation.

When melatonin secretion is suppressed at night, pancreatic β-cells and adipocytes may remain metabolically active instead of entering their normal nocturnal state of reduced activity. Consequently, even during sleep, the organism remains in a partially activated physiological state, as evidenced by elevated nighttime heart rate and sustained sympathetic nervous system activity. This state interferes with metabolic recovery processes and may increase insulin resistance by morning. Over the long term, chronically elevated fasting glucose and compensatory hyperinsulinemia may accelerate β-cell dysfunction and promote the clinical manifestation of diabetes.

Limitations of the Evidence

Despite promising findings, several limitations should be considered. The randomized crossover study examining daylight exposure (Cell Metabolism, 2025) involved a small sample size (n = 13) and short experimental duration. Therefore, although the observed effects appear robust, they require confirmation in larger cohorts and longer-term studies.

Additionally, seasonal and geographical factors may influence results, as natural daylight intensity and duration vary considerably across seasons and latitudes. The study conducted in long-term care facilities (Wang et al.) did not demonstrate significant changes in glycemic markers, possibly because a relatively coarse metabolic indicator (glycated serum protein) was measured over a short observation period.

Longer interventions – potentially several months – may be necessary for metabolic effects to accumulate. Furthermore, many participants in that study had relatively well-controlled glycemia at baseline, leaving limited room for improvement. Consequently, light-based interventions may be more effective in individuals with poorer glycemic control or pronounced insulin resistance.

This interpretation is indirectly supported by findings from Brouwer et al., where patients with greater baseline insulin resistance experienced more pronounced antidepressant effects from light therapy, suggesting that metabolically vulnerable subgroups may derive greater benefits.

Combined interventions may also produce stronger effects. For example, protocols integrating morning light therapy with time-restricted feeding (particularly early time-restricted eating) are currently being investigated to evaluate potential synergistic effects on glycemic regulation.

Spectral Composition of Light

The spectral composition of light may also influence circadian and metabolic responses. Natural sunlight contains a full spectrum of wavelengths, including blue light, which is particularly effective in synchronizing circadian rhythms but can suppress melatonin if present during evening hours.

While blue light exposure during daytime is beneficial for circadian alignment, exposure during evening hours – particularly from electronic screens – may be detrimental. Older adults with impaired vision may require higher light intensity or longer outdoor exposure to achieve circadian effects comparable to those observed in younger individuals.

Lens transparency also plays a role. In individuals with cataracts, reduced transmission of light – especially in the blue spectrum – limits circadian photoreception. Cataract surgery has been shown to improve sleep quality and circadian rhythm stability, likely because more daylight reaches the retina. Therefore, appropriate management of visual impairments may indirectly enhance circadian synchronization and metabolic regulation.

Practical Implications

For clinicians managing older adults with T2D, these findings highlight an additional dimension of non-pharmacological therapy. Alongside diet, physical activity, and weight management, the light–dark cycle represents a modifiable lifestyle factor.

Simple recommendations may include:

- daily outdoor exposure to natural daylight (30–60 minutes when possible),
- opening curtains during daytime,
- positioning workspaces near windows,
- ensuring adequate lighting in daytime areas of hospitals and care facilities.

In institutional environments, installing bright full-spectrum lighting systems that mimic natural daylight – particularly during morning hours – may help reinforce circadian rhythms.

Conversely, nighttime environments should be kept dark by using blackout curtains, avoiding bright night lights, and limiting screen exposure. If lighting is necessary, dim warm-colored lights should be used.

These interventions are inexpensive, safe, and can be incorporated into routine lifestyle recommendations for patients with diabetes. Some endocrinologists and sleep medicine specialists already advise patients to sleep in complete darkness and spend more time in daylight as part of a healthy lifestyle strategy for diabetes management.

CONCLUSIONS

Circadian rhythms and environmental light cycles play a substantial role in glucose metabolism, particularly in older individuals with type 2 diabetes. Current evidence suggests that increasing exposure to natural daylight may improve glycemic control by stabilizing blood glucose levels and potentially enhancing insulin sensitivity through restoration of normal circadian metabolic dynamics.

Conversely, excessive artificial light exposure – especially during nighttime – has been associated with impaired metabolic regulation and increased diabetes risk.

Ensuring an appropriate light–dark cycle therefore represents a promising non-pharmacological strategy for improving disease management in older adults with T2D. Daylight exposure acts as a natural synchronizer of biological rhythms and may partially counteract the negative effects of aging and sedentary indoor lifestyles on circadian regulation. Light therapy may be particularly beneficial in institutional care settings where patients have limited access to outdoor environments.

Equally important is patient education regarding sleep hygiene. Nighttime sleep in darkness is essential for metabolic recovery; even simple behavioral changes, such as turning off televisions or lights at night, may improve sleep quality and morning glycemic control.

PROSPECTS FOR FUTURE RESEARCH

Although the current findings are encouraging, several questions remain unresolved. Larger and longer-term studies are needed to confirm the metabolic benefits of daylight exposure. For instance, it would be valuable to determine whether architectural redesign of hospital environments to maximize natural daylight could reduce HbA1c levels over time, or whether daily outdoor exposure could decrease the need for glucose-lowering medications in older adults with diabetes.

Further research is also needed to clarify underlying mechanisms, including the roles of melatonin, cortisol, and autonomic nervous system activity. Another promising area involves investigating interactions between light-based interventions and pharmacological therapies such as metformin or SGLT2 inhibitors.

A personalized approach may also be required. Individuals differ in chronotype and light sensitivity, and genetic variants – such as those in the melatonin receptor gene MTNR1B – have been associated with both diabetes risk and circadian responses to light.

Final Perspective

Light, an ordinary and ubiquitous element of daily life, exerts profound biological effects on metabolic regulation. For older adults with type 2 diabetes, returning to a more natural daily rhythm – characterized by greater daylight exposure and darkness at night – represents a simple yet promising strategy for improving health outcomes.

Although not a substitute for standard medical therapy, circadian optimization may become an important component of comprehensive diabetes management. Future studies will help determine the optimal timing, intensity, and duration of light exposure needed to achieve clinically meaningful benefits.

Current scientific evidence already suggests that architectural design, environmental lighting, and daily behavioral habits – such as spending time outdoors or turning off nighttime lights – may influence the course of complex metabolic diseases such as diabetes. This insight encourages an interdisciplinary approach integrating medicine, chronobiology, and environmental design to create living environments that support both circadian health and metabolic well-being.

AUTHOR CONTRIBUTIONS

Anna Garnytska: work concept and design, data collection and analysis, responsibility for statistical analysis, writing (not revising) sections of the manuscript, final approval of the article.

Olga Orlyk: work concept and design, writing (not revising) sections of the manuscript, data collection and analysis, critical review, final approval of the article.

Maryna Kochuieva: collection of data, writing (not revising) sections of the manuscript.

Valentyna Psarova: data collection and analysis, writing (not revising) sections of the manuscript.

FUNDING

None.

CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

ETHICAL CONSIDERATIONS

The study was conducted without involving human subjects.

REFERENCES

1. Harmsen JF, Habets I, Biancolin AD, et al. Natural daylight during office hours improves glucose control and whole-body substrate metabolism. Cell Metab. 2025;38(1):65-81.e10. doi: https://doi.org/10.1016/j.cmet.2025.11.006
2. Mason IC, Grimaldi D, Reid KJ, et al. Light exposure during sleep impairs cardiometabolic function. Proc Natl Acad Sci. 2022;119(12):e2113290119. doi: https://doi.org/10.1073/pnas.2113290119
3. Morris CJ, Yang JN, Garcia JI, et al. Endogenous circadian system and circadian misalignment impact glucose tolerance via separate mechanisms in humans. Proc Natl Acad Sci. 2015;112(17):E2225-E2234. doi: https://doi.org/10.1073/pnas.1418955112
4. Poggiogalle E, Jamshed H, Peterson CM. Circadian regulation of glucose, lipid, and energy metabolism in humans. Metabolism. 2018;84:11-27. doi: https://doi.org/10.1016/j.metabol.2017.11.017

5. Xue P, Nôga DA, Benedict C. The dark side of light: light at night may raise the risk of type 2 diabetes. Lancet Reg Health Eur. 2024;42:100955. doi: https://doi.org/10.1016/j.lanepe.2024.100955
6. Stenvers DJ, Scheer FAJL, Schrauwen P, la Fleur SE, Kalsbeek A. Circadian clocks and insulin resistance. Nat Rev Endocrinol. 2019;15(2):75-89. doi: https://doi.org/10.1038/s41574-018-0122-1
7. Cedernaes J, Waldeck N, Bass J. Neurogenetic basis for circadian regulation of metabolism by the hypothalamus. Genes Dev. 2019;33(17-18):1136-1158. doi: https://doi.org/10.1101/gad.328633.119
8. Huang W, Ramsey KM, Marcheva B, Bass J. Circadian rhythms, sleep, and metabolism. J Clin Invest. 2011;121(6):2133-2141. doi: https://doi.org/10.1172/JCI46043
9. Windred DP, Burns AC, Rutter MK, et al. Personal light exposure patterns and incidence of type 2 diabetes: analysis of 13 million hours of light sensor data and 670,000 person-years of prospective observation. Lancet Reg Health Eur. 2024;42:100943. doi: https://doi.org/10.1016/j.lanepe.2024.100943
10. Wang Q, Wu S, Luo Z, et al. Effects of light therapy on sleep and circadian rhythm in older type 2 diabetics living in long-term care facilities: a randomized controlled trial. Front Endocrinol. 2024;15:1307537. doi: https://doi.org/10.3389/fendo.2024.1307537
11. Brouwer A, van Raalte DH, Nguyen HT, et al. Effects of light therapy on mood and insulin sensitivity in patients with type 2 diabetes and depression: results from a randomized placebo-controlled trial. Diabetes Care. 2019;42(4):529-538. doi: https://doi.org/10.2337/dc18-1732
12. Blume C, Garbazza C, Spitschan M. Effects of light on human circadian rhythms, sleep and mood. Somnologie. 2019;23(3):147-156. doi: https://doi.org/10.1007/s11818-019-00215-x
13. Brown TM, Brainard GC, Cajochen C, et al. Recommendations for daytime, evening, and nighttime indoor light exposure to best support physiology, sleep, and wakefulness in healthy adults. PLoS Biol. 2022 Mar 17;20(3):e3001571. doi: https://doi.org/10.1371/journal.pbio.3001571
14. Sun H, Saeedi P, Karuranga S, et al. IDF Diabetes Atlas: global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045. Diabetes Res Clin Pract. 2022;183:109119. doi: https://doi.org/10.1016/j.diabres.2021.109119
15. International Diabetes Federation. IDF Diabetes Atlas. 10th ed. Brussels, Belgium: International Diabetes Federation; 2021.
16. Phillips AJK, Clerx WM, O'Brien CS, et al. Irregular sleep/wake patterns are associated with poorer academic performance and delayed circadian and sleep/wake timing. Scientific Reports. 2017;7:3216. doi: https://doi.org/10.1038/s41598-017-03171-4
17. Wright KP, McHill AW, Birks BR, Griffin BR, Rusterholz T, Chinoy ED. Entrainment of the human circadian clock to the natural light-dark cycle. Curr Biol. 2013;23(16):1554-1558. doi: https://doi.org/10.1016/j.cub.2013.06.039
18. McHill AW, Wright KP. Role of sleep and circadian disruption on energy expenditure and in metabolic predisposition to human obesity and metabolic disease. Obes Rev. 2017;18:15-24. doi: https://doi.org/10.1111/obr.12503
19. Scheer FA, Hilton MF, Mantzoros CS, Shea SA. Adverse metabolic and cardiovascular consequences of circadian misalignment. Proc Natl Acad Sci. 2009;106(11):4453-4458. doi: https://doi.org/10.1073/pnas.0808180106
20. Obayashi K, Saeki K, Iwamoto J, Ikada Y, Kurumatani N. Association between light exposure at night and nighttime blood pressure in the elderly independent of nocturnal urinary melatonin excretion. Chronobiol Int. 2014;31(6):779-786. doi: https://doi.org/10.3109/07420528.2014.900501
21. Lunn RM, Blask DE, Coogan AN, et al. Health consequences of electric lighting practices in the modern world: A report on the National Toxicology Program's workshop on shift work at night, artificial light at night, and circadian disruption. Sci Total Environ. 2017;607-608:1073-1084. doi: https://doi.org/10.1016/j.scitotenv.2017.07.056
22. Duffy JF, Wright KP Jr. Entrainment of the human circadian system by light. J Biol Rhythms. 2005;20(4):326-338. doi: https://doi.org/10.1177/0748730405277983
23. Spitschan M, Santhi N. Individual differences and diversity in human physiological responses to light. EBioMedicine. 2022;75:103640. doi: https://doi.org/10.1016/j.ebiom.2021.103640
24. Huang T, Mariani S, Redline S. Sleep irregularity and risk of cardiovascular events: the multi-ethnic study of atherosclerosis. J Am Coll Cardiol. 2020;75(9):991-999. doi: https://doi.org/10.1016/j.jacc.2019.12.054
25. Shan Z, Ma H, Xie M, et al. Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies. Care. 2015;38(3):529-537. doi: https://doi.org/10.2337/dc14-2073
26. Reutrakul S, Van Cauter E. Sleep influences on obesity, insulin resistance, and risk of type 2 diabetes. Metabolism: clinical and experimental. 2018;84:56-66. doi: https://doi.org/10.1016/j.metabol.2018.02.010
27. Kalsbeek A, la Fleur S, Fliers E. Circadian control of glucose metabolism. Mol Metab. 2014;3(4):372-83. doi: https://doi.org/10.1016/j.molmet.2014.03.002
28. Qian J, Scheer FAJL. Circadian system and glucose metabolism: implications for physiology and disease. Trends Endocrinol Metab. 2016;27(5):282-293. doi: https://doi.org/10.1016/j.tem.2016.03.005
29. Depner CM, Stothard ER, Wright KP Jr. Metabolic consequences of sleep and circadian disorders. Curr

Diab Rep. 2014;14(7):507. doi: https://doi.org/10.1007/s11892-014-0507-z
30. Wirth MD, Burch J, Shivappa N, et al. Association of a dietary inflammatory index with inflammatory indices and metabolic syndrome among police officers. J Occup Environ Med. 2014;56(9):986-989. doi: https://doi.org/10.1097/jom.0000000000000213
31. Chellappa SL, Vujovic N, Williams JS, Scheer FA. Impact of circadian disruption on cardiovascular function and disease. Trends in endocrinology and metabolism. 2019;30(10):767-779. doi: https://doi.org/10.1016/j.tem.2019.07.008
32. Dashti HS, Scheer FA, Jacques PF, Lamon-Fava S, Ordovás JM. Short sleep duration and dietary intake: epidemiologic evidence, mechanisms, and health implications. Adv Nutr. 2015;6(6):648-659. doi: https://doi.org/10.3945/an.115.008623
33. Skene DJ, Skornyakov E, Chowdhury NR, et al. Separation of circadian- and behavior-driven metabolite rhythms in humans provides a window on peripheral oscillators and metabolism. Proc Natl Acad Sci. 2018;115(30):7825-7830. doi: https://doi.org/10.1073/pnas.1801183115

INFORMATION ABOUT THE AUTHORS

Anna Garnytska, PhD, Associate Professor, Department of Therapy, Age-Associated Diseases and Diabetology of the Shupic National Healthcare University of Ukraine

Olga Orlyk, PhD, leading researcher of the Department of Diagnostics and Treatment of Metabolic Diseases State Scientific Institution “CENTER FOR INNOVATIVE MEDICAL TECHNOLOGIES OF THE NAS OF UKRAINE”

Maryna Kochuieva, Doctor of Medical Sciences, MD, PhD, Professor of the Shupyk National Healthcare University of Ukraine

Valentyna Psarova, Doctor of Medical Sciences, MD, PhD, Professor of the Sumy State University

Received: 09.04.2026

Accepted for publication: 20.05.2026

Published: 23.06.2026