European Journal of Pedagogical Initiatives and Educational Practices
ISSN (E): 2938-3625
Volume 4, Issue 6, June - 2026

THE SIGNIFICANCE OF CIRCADIAN RHYTHM DISRUPTION IN THE DEVELOPMENT OF OBSTETRIC COMPLICATIONS IN PREGNANT WOMEN

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Abstract
Objective: To summarise the significance of circadian rhythm disruption in obstetric complications and preventive prospects.
Materials and methods: analysis of meta-analyses, cohort and experimental studies from 2012–2024.
Results: Shift/night work, sleep deficiency, reduced melatonin and clock-gene dysregulation are associated with preeclampsia, gestational diabetes, preterm birth, fetal growth restriction and pregnancy loss; mechanisms — oxidative stress, inflammation and endothelial dysfunction. Conclusions: it is a modifiable risk factor; sleep correction has preventive value, and melatonin is promising.

Keywords: Circadian rhythm, melatonin, clock genes, preeclampsia, gestational diabetes.

Introduction

HOMILADOR AYOLLARDA AKUSHERLIK ASORATLARI RIVOJLANISHIDA SIRKAD RITMI BUZILISHLARINING AHAMIYATI

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ЗНАЧЕНИЕ НАРУШЕНИЙ ЦИРКАДНЫХ РИТМОВ В РАЗВИТИИ АКУШЕРСКИХ ОСЛОЖНЕНИЙ У БЕРЕМЕННЫХ

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Maqsad:
Sirkad ritm buzilishlarining akusherlik asoratlaridagi ahamiyatini va profilaktik istiqbollarini umumlashtirish. **Materiallar va usullar:** 2012–2024-yillardagi meta-analiz, kogort va eksperimental tadqiqotlar tahlili. **Natijalar:** tungi/smenali ish, uyqu yetishmovchiligi, melatonin pasayishi va soat genlari disregulyatsiyasi preeklampsiya, gestatsion diabet, muddatidan oldingi tug‘ruq, FGR va homila yo‘qotish bilan bog‘liq; mexanizmlar — oksidativ

stress, yallig‘lanish va endotelial disfunksiya. **Xulosa:** bu o‘zgartirilishi mumkin xavf omili; uyqu gigiyenasi va melatonin istiqbolli yo‘nalishdir.

Kalit so‘zlar: sirkad ritm, melatonin, soat genlari, preeklampsiya, gestatsion diabet.

Цель: обобщить значение нарушений циркадных ритмов в развитии акушерских осложнений и профилактические перспективы. **Материалы и методы:** анализ метаанализов, когортных и экспериментальных исследований 2012–2024 гг. **Результаты:** сменная/ночная работа, дефицит сна, снижение мелатонина и дисрегуляция часовых генов ассоциированы с преэклампсией, гестационным диабетом, преждевременными родами, задержкой роста плода и потерей беременности; механизмы — окислительный стресс, воспаление и эндотелиальная дисфункция. **Выводы:** это модифицируемый фактор риска; коррекция сна имеет профилактическое значение, а мелатонин перспективен.

Ключевые слова: циркадный ритм, мелатонин, часовые гены, преэклампсия, гестационный диабет.

INTRODUCTION

Obstetric complications—preeclampsia and other hypertensive disorders, gestational diabetes mellitus, preterm labor, intrauterine growth restriction (IUGR), and fetal loss—remain leading causes of maternal and perinatal morbidity and mortality. Globally, preeclampsia complicates approximately 3–5% of all pregnancies, and a significant proportion of maternal deaths are associated with hypertensive disorders. The pathogenesis of these conditions is multifactorial, and in addition to classical risk factors (maternal age, obesity, chronic diseases, family history), the role of biological rhythms has been actively studied in recent years [12, 14].

Modern lifestyles—night and shift work, prolonged exposure to artificial and blue-spectrum light in the evening, chronic sleep deprivation, irregular eating, and “social jetlag”—disconnect the internal clock from the external environment, leading to circadian disruptions, or chronodisruptions. A large portion of the population, including women of reproductive age and healthcare workers, are regularly exposed to these factors. Increasing evidence suggests that these disruptions negatively impact reproductive function and pregnancy outcomes [5, 15].

The aim of this review article is to systematically summarize the role of circadian rhythm disorders in the development of major obstetric complications, their cellular and systemic mechanisms, as well as their preventive and therapeutic prospects based on modern scientific data. The article first reviews the basic concepts of chronobiology and circadian adaptation in pregnancy, and then analyzes the evidence for individual nosologies.

1. BASIC CONCEPTS AND MECHANISMS OF CHRONOBIOLOGY

1.1. Hierarchical structure of the circadian system: The circadian rhythm is an endogenous, approximately 24-hour oscillation that is maintained even in the absence of external signals, but is adjusted to a precise 24-hour period by external synchronizing factors (zeitgebers). The circadian system has a hierarchical structure: a central “clock” is located in the suprachiasmatic

nucleus (SXY) of the hypothalamus, which coordinates billions of peripheral oscillators in various tissues of the body - the liver, pancreas, heart, kidneys, blood vessels and reproductive organs [12, 14].

1.2. Molecular clock mechanism: At the cellular level, the circadian rhythm is driven by a transcriptional-translational feedback loop. The proteins encoding the master clock genes CLOCK and BMAL1 (ARNTL) form heterodimers that bind to E-box sequences in the promoters of PER1/2/3 (Period) and CRY1/2 (Cryptochrome) genes, activating their transcription. The resulting PER and CRY proteins accumulate in the cytoplasm and return to the nucleus, thereby inhibiting the activity of the CLOCK–BMAL1 complex. This process produces an oscillation with a period of approximately 24 hours [6, 12].
An additional control loop is provided by the nuclear receptors NR1D1/2 (REV-ERB α / β) and ROR α : they inhibit or activate BMAL1 expression, respectively, which gives the system stability and precision. This molecular mechanism is present in almost all nucleated cells and regulates the expression of thousands of target genes (related to the cell cycle, metabolism, antioxidant defense, inflammation) in a circadian manner. Disruption of tissue expression of clock genes has been associated with many pathological conditions.

1.3. Melatonin and its biological functions: Melatonin (N-acetyl-5-methoxytryptamine) is an indoleamine hormone synthesized mainly in the pineal gland from tryptophan via serotonin. Its blood levels increase sharply at night and decrease during the day. Melatonin acts through two main membrane receptors, MT1 and MT2, and regulates circadian rhythms and the sleep-wake cycle [2, 14].
However, the biological role of melatonin is not limited to rhythm. It is a powerful antioxidant and free radical scavenger, effectively interacting with reactive oxygen and nitrogen species and organic radicals, activating antioxidant enzymes (glutathione peroxidase, glutathione reductase) and inhibiting prooxidant enzymes (nitric oxide synthase, lipoxygenase). In addition, melatonin modulates the immune system and has a protective effect on tissue damage [1, 2]. These properties make melatonin an important factor in the physiology and pathology of pregnancy.

1.4. Chronodisruption and its sources: These factors disrupt the synchronization between the central and peripheral clocks and reduce melatonin secretion. From a public health perspective, chronodisruption is a recognized risk factor for cardiovascular disease, metabolic syndrome, type 2 diabetes, and some cancers. In women of reproductive age, it has been associated with menstrual dysfunction and pregnancy complications [15].

1.5. Circadian rhythms and reproductive function: The circadian system regulates female reproductive function at several levels. The activity of the hypothalamic-pituitary-ovarian axis, including the preovulatory surge of luteinizing hormone, is under circadian control and coordinated by SXY signals. Clock genes are expressed in the ovary, fallopian tubes, and endometrium and are involved in follicular maturation, ovulation, and endometrial receptivity [12, 14].

Women working night shifts have been reported to have a higher incidence of menstrual irregularities and subfertility; a meta-analysis found that shift work was associated with menstrual dysfunction (OR 1.15) [15]. These data suggest that the effects of circadian disruption may begin before pregnancy, even during the preparation for pregnancy. Therefore, attention to circadian hygiene may be part of pre-pregnancy preparation.

2. ADAPTATION OF THE CIRCULATORY SYSTEM DURING PREGNANCY

2.1. Maternal circadian changes: During pregnancy, the maternal organism undergoes significant circadian adaptation. Many physiological parameters have clear diurnal oscillations: body temperature, arterial pressure, heart rate, leukocyte count, weight gain, intrauterine pressure, and uterine contractions change regularly throughout the day [12, 13]. Melatonin secretion increases with the course of pregnancy, especially in the third trimester, and decreases after delivery, indicating its role in supporting pregnancy [1, 14].

The purpose of these adaptations is to align the needs of the mother and the developing fetus with the circadian rhythm. The mother's circadian system sets the "time frame" for the placenta and fetus. Therefore, disruption of the mother's circadian system can disrupt this delicate alignment, affecting the physiology of both the mother and the fetus.

Circadian hormonal rhythms are of particular importance in pregnancy. Cortisol levels are normally highest in the early morning and lowest in the evening; although total cortisol levels increase during pregnancy, their circadian oscillations are maintained. Disturbances in cortisol rhythms (e.g., due to chronic stress or sleep deprivation) affect metabolic and immune processes and may play a role in the programming of fetal development. The coordinated rhythms of melatonin, cortisol, prolactin, and other hormones form a complex “time orchestra” that ensures the normal course of pregnancy [12, 14].

2. 2. Fetal circadian system and developmental programming: In the fetus, independent circadian oscillators are not fully developed before birth, so the fetus relies directly on maternal signals—melatonin, cortisol levels, body temperature, and feeding rhythms. Maternal melatonin freely crosses the placenta, providing a “time signal” to the fetus and participating in the proper regulation of peripheral clocks in fetal organs [14, 16].

When the mother's circadian rhythm is disrupted, the timing signals reaching the fetus are also disrupted. In animal models, such maternal circadian desynchronization has been shown to “program” the offspring to develop metabolic (impaired glucose tolerance, obesity) and cardiovascular disease [16]. These data suggest that the consequences of circadian disruption are not limited to the gestational period, but may also affect the long-term health of the child.

3. GENERAL PATHOGENETIC MECHANISMS OF CIRCADIAL DISORDERS: The association of circadian disorders with various obstetric complications is explained by several interrelated mechanisms. A separate consideration of these mechanisms will help to understand their occurrence and points of prevention.

3.1. Melatonin deficiency: Exposure to light in the evening and irregular sleep patterns reduce pineal secretion of melatonin. This reduces its antioxidant defenses, immunomodulation, and progesterone-supporting effects. Melatonin stimulates progesterone production, which is

essential for maintaining pregnancy; therefore, melatonin deficiency may impair the mechanisms of pregnancy maintenance [1, 14].

Melatonin freely crosses the placenta and reaches fetal tissues, where it also functions as an antioxidant defense and developmental signal. The physiological increase in maternal melatonin levels during pregnancy is an adaptation to support placental function and fetal development. If this increase is suppressed by exposure to nighttime light or sleep deprivation, the maternal-placental-fetal system is vulnerable. This mechanism suggests that melatonin deficiency is a common pathogenetic link linking many obstetric complications [11, 14].

3.2. Tissue dysregulation of the molecular clock: Dysregulation of CLOCK, BMAL1, PER, and CRY gene expression in the placenta and endometrium disrupts trophoblast invasion, placentation, and interorgan metabolic coordination. Loss of circadian expression of clock genes in placental cells has been shown in preeclampsia [6]. Because clock genes control cell cycle and metabolism, their dysregulation has broad implications for placental function.

3.3. Oxidative stress and chronic inflammation: Circadian dysregulation and melatonin deficiency enhance prooxidant and proinflammatory states. Oxidative stress and chronic low-grade inflammation lead to endothelial dysfunction, which is central to the pathogenesis of preeclampsia, gestational diabetes, and FGR. Melatonin, as an antioxidant, can inhibit these processes, while its deficiency weakens the defenses [2, 11].

3.4. Neuroendocrine and autonomic dysregulation : Circadian disruption alters the rhythm of cortisol and catecholamines (including noradrenaline). Elevated noradrenaline levels may increase uterine contractility, increasing the risk of preterm labor and miscarriage. Increased sympathetic activity of the autonomic system increases blood pressure and reduces the physiological nocturnal fall in arterial pressure.

3.5. Metabolic disorders: Sleep deprivation and circadian imbalance increase insulin resistance, impair glucose tolerance, and disrupt the balance of appetite hormones (leptin, ghrelin). Since physiological insulin resistance already exists during pregnancy, this additional effect creates a predisposition to the development of gestational diabetes [7, 10].

The five mechanisms listed — melatonin deficiency, molecular clock dysregulation, oxidative stress and inflammation, neuroendocrine-vegetative disorders, and metabolic changes — do not operate in isolation, but as a single pathophysiological network. For example, melatonin deficiency enhances both oxidative stress and inflammation; dysregulation of clock genes disrupts the timing of metabolic and immune processes. The final common pathway of these mechanisms is often endothelial dysfunction and placental insufficiency, which constitute the common pathogenetic basis of various obstetric complications. It is this commonality that explains the simultaneous increase in the risk of several complications of circadian disorders and allows us to consider the circadian system as a single preventive target.

4.CIRCADIAN DISORDERS AND HYPERTENSIVE DISORDERS AND PREECLAMATION

4.1. Epidemiological evidence: Hypertensive disorders—gestational hypertension, preeclampsia, and eclampsia—are among the most dangerous complications of pregnancy and are among the leading causes of maternal mortality worldwide. They also increase the risk of FGR, preterm birth, and perinatal death. Identifying modifiable risk factors for these conditions is of great practical importance, as they may serve as points of prevention.

A large systematic review and meta-analysis showed that shift work, compared with fixed daytime shifts, increased the risk of preeclampsia by approximately 75% (OR 1.75; 95% CI 1.01–3.01) and gestational hypertension by 19% (OR 1.19; 95% CI 1.10–1.29) [5]. This epidemiological association supports a role for circadian disruption in the pathogenesis of hypertensive disorders.

4.2. Diurnal profile of blood pressure: In healthy pregnancy, blood pressure has a clear diurnal rhythm: during the night it decreases by 10–20% compared to the daytime (physiological "dipping"). In preeclampsia, this nocturnal decrease is reduced or disappears ("non-dipping" profile), sometimes the pressure even increases at night. This phenomenon indicates that circadian rhythm disturbances are closely related to hypertensive disorders and emphasizes the predictive value of ambulatory blood pressure monitoring (ABPM) [12].

4.3. Placental clock genes and melatonin: Molecular data support this link. Expression of clock genes (BMAL1, CLOCK, PER2, CRY2, NR1D1) and melatonin synthesis are impaired in the placenta of women with preeclampsia; the circadian rhythm of placental macrophages is lost and is not restored even when co-cultured with healthy trophoblasts [6]. In addition, blood melatonin levels are significantly reduced in severe preeclampsia [1, 11].

These events are associated with inadequate spiral artery remodeling, impaired trophoblast invasion, oxidative stress, and endothelial dysfunction. Whether circadian disruption is a cause of preeclampsia or an early marker (or both) is still not fully resolved and requires prospective studies. Nevertheless, the protective role of melatonin is being actively investigated in this area.

5. CIRCULATION DISORDERS AND GESTATIONAL DIABETES

Physiological insulin resistance increases during pregnancy; sleep and circadian rhythm disruptions further impair glucose metabolism and contribute to the development of gestational diabetes mellitus (GDM) [10]. The association between sleep duration and GDM is U-shaped: a meta-analysis showed that both short (OR 1.50; 95% CI 1.07–2.10) and long sleep (OR 1.28; 95% CI 1.13–1.46) increased the risk of GDM, with the effect of short sleep being stronger [10].

This association has been consistent across populations: cohort and cross-sectional studies in different countries have shown that short sleep duration, poor sleep quality, and sleep-disordered breathing are associated with the incidence of GSD. Sleep deprivation impairs glucose metabolism in several ways: it reduces insulin sensitivity, increases cortisol and sympathetic activity, and disrupts appetite and energy balance. These effects, combined with the natural insulin resistance of pregnancy, facilitate the progression to GSD. Therefore,

adequate and quality sleep is an important but often overlooked aspect of GSD prevention [7, 10].

6. SLEEP DISORDERS AND OBSTRUCTIVE SLEEP APNEA: Sleep disorders are very common in late pregnancy and are often overlooked as a physiological condition. Weight gain, the growing uterus, the diaphragm moving, and hormonal changes can reduce the patency of the upper airway, predisposing to the development of obstructive sleep apnea (OSA).

OSA is characterized by repeated intermittent hypoxia, sleep fragmentation, and increased sympathetic activity; this leads to oxidative stress, endothelial dysfunction, and insulin resistance. In a large national cohort, OSA was associated with an increased risk of preeclampsia (aOR 2.22; 95% CI 1.94–2.54), eclampsia (aOR 2.95), and gestational diabetes (aOR 1.51; 95% CI 1.34–1.72), even after adjustment for obesity [4]. OSA is also associated with an increased incidence of FGR, preterm labor, and cesarean section.

These data suggest the need to consider sleep disorders, including OSA, as an obstetric risk factor and to screen women at risk. Continuous positive airway pressure (CPAP) therapy is the most effective treatment, but adherence is often poor [4].

OSA and circadian disruption are closely linked: sleep fragmentation disrupts circadian rhythms, and circadian disruption impairs sleep quality, resulting in a vicious cycle. Mechanistically, repeated intermittent hypoxia-reoxygenation cycles induce severe oxidative stress and sympathetic activity, leading to endothelial dysfunction and elevated blood pressure—a finding that is directly consistent with the pathophysiology of preeclampsia. Increased insulin resistance, in turn, increases the risk of gestational diabetes. Thus, OSA is an important but often overlooked link connecting circadian-metabolic-vascular disruptions.

7. CIRCULATION DISORDERS, PREMATURE BIRTH AND FETAL GROWTH RESTRICTION

7.1. Epidemiological evidence: Night shift work is associated with an increased risk of preterm birth (PTB). In a cohort study of Swedish health care workers, women who worked frequent (>25) night shifts in the first trimester had a 1.62-fold increased risk of PTB (aOR 1.62; 95% CI 1.03–2.53) [9]. Meta-analyses have shown that rotating shifts increase the risk of PTB by approximately 13% and fixed night shifts by 21%, and that there is also an increased risk of small-for-gestational-age (SGA) infants [5].

7.2. Conflicting data: However, some large registry-based cohort studies, especially those using objective data (based on work hours rather than self-report), have not found a reliable association between night shift work and PTB. These conflicting results are likely due to methodological differences—in the methods of measuring exposure, shift definition, observation period, and the extent to which confounding factors were taken into account. Standardized, prospective studies are needed in this area.

7.3. Possible mechanisms: Possible mechanisms of the relationship include: decreased melatonin secretion due to exposure to light at night, which reduces progesterone production and affects myometrial activity; increased noradrenaline levels due to sleep deprivation and

stress, which increase uterine contractility; and the role of melatonin and placental clock genes in trophoblast function and fetal growth [5, 9, 13, 14].

Of note is the dose-dependent nature of the effect: the risk increases with the number and duration of night shifts. It has been reported that working night shifts in the first trimester with a short rest period (rapid return) is associated with a several-fold increase in the risk of preterm birth. This dose-dependent relationship provides further evidence in favor of a causal relationship and suggests that the risk may be reduced by adjusting the work schedule [9].

7.4. Fetal growth restriction: Intrauterine growth restriction (FGR) is often caused by placental insufficiency and exposes the fetus to hypoxia and oxidative stress. Severe FGR with early onset is associated with a high risk of perinatal mortality and neurological impairment. Circadian disruption and melatonin deficiency may contribute to this process by increasing placental oxidative stress; conversely, the antioxidant protection of melatonin is considered a protective factor [11, 14].

8. CIRCADIAN DISORDERS AND MISSION: Night shift work and circadian disruption have also been associated with an increased risk of miscarriage. A systematic review and meta-analysis found that fixed night shift work was associated with a 23% increased risk of spontaneous abortion (OR 1.23; 95% CI 1.03–1.47) [5]. Another meta-analysis found that night shift work was associated with an increased risk of miscarriage (OR 1.29; 95% CI 1.11–1.50; OR 1.41 after adjustment for confounders) and menstrual dysfunction [15].

At the molecular level, this association is explained by the importance of the endometrial and placental molecular clock: women with recurrent miscarriage have reduced endometrial BMAL1 expression, which leads to impaired trophoblast invasion and decidualization [6]. Furthermore, the lack of antioxidant and progesterone-promoting effects of melatonin may increase oxidative stress in early pregnancy, making implantation and pregnancy maintenance more difficult [1, 14].

It is important to note that miscarriage is a multifactorial condition, and circadian disruption is not the sole cause, but rather one of the possible contributing factors. The associations in the available meta-analyses are relatively modest and may be partially explained by confounding factors (stress, exercise, socioeconomic status). Nevertheless, the consistency of the evidence and the existence of biologically plausible mechanisms suggest that circadian hygiene should be considered, especially in women with a history of recurrent miscarriage. Further research is needed to make definitive recommendations in this area [3, 15].

CONCLUSION:

Accumulating data suggest that circadian rhythm disturbances—night and shift work, sleep deprivation and sleep timing disorders, obstructive sleep apnea, decreased melatonin secretion, and dysregulation of placental and endometrial clock genes—are important, modifiable risk factors for the development of obstetric complications such as preeclampsia and gestational hypertension, gestational diabetes mellitus, preterm birth, fetal growth restriction, and fetal loss.

The main pathogenetic mechanisms include melatonin deficiency, molecular clock dysregulation, oxidative stress and inflammation, endothelial dysfunction, and metabolic and hemodynamic disturbances. In clinical practice, assessment of sleep and circadian hygiene in pregnant women, regulation of the circadian rhythm, and restriction of night shifts are promising approaches to improve perinatal outcomes. The protective role of melatonin is being actively studied, but further evidence is needed for its clinical application. In general, large prospective and controlled studies are needed to clarify causal relationships and to justify intervention strategies.

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