The Role of Sleep Disruption During Pregnancy on Maternal Health and Effect on the Nervous System of Fetal Programming

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Abstract

Maternal sleep is a crucial factor affecting the uterine environment. Sleep deprivation, whether in quality or quantity can impair the regulation of metabolic processes, neuroendocrine stress signals, inflammatory pathways essential for placental function, maternal vascular health, and cardiac adaptation. Sleep disturbances are common during pregnancy and often worsen as the pregnancy progresses influenced by physical and hormonal symptoms, as well as pregnancy-related sleep disorders such as sleep apnea. This study combines human observational data and experimental findings on the impact of maternal sleep deprivation (including sleep fragmentation, insomnia, and its symptoms) on early fetal development, particularly during the highly sensitive organogenesis period. Scientific studies support the existence of a neural network through which maternal sleep deprivation leads to circadian rhythm disturbances, pituitary-adrenal axis dysfunction, metabolic and oxidative stress, melatonin imbalances, and in cases of sleep-disordered breathing, intermittent hypoxia. The placenta acts as a key mediator integrating these signals and regulating nutrient and oxygen transport, immune modulation, uteroplacental blood flow, and endocrine functions. Numerous studies have shown that inadequate prenatal sleep is strongly linked to behavioral and developmental outcomes in children, with growing evidence connecting prenatal sleep patterns to changes in the structure and function of the nervous system. To ensure healthy physical and brain development, it is crucial to understand the evolving nature of sleep and its relationship to pregnancy and age, as sleep plays a vital role in preventing prenatal and postnatal disorders.

Keywords: sleep disorder, maternal outcomes, pregnancy, fetal nervous system.

1. Introduction

Sleep is a fundamental biological function for human health and is essential for pregnant women. Previous studies have linked poor sleep quality to negative outcomes. The psychological and physical changes associated with pregnancy increase the risk of different complications like disturbances, fatigue, and lethargy. Problems during pregnancy and childbirth include premature birth, neonatal resuscitation, weak fetuses, cesarean delivery and NICU admission [1,2]. Worldwide, sleep disturbance is the main health problem amongst pregnant women that may have adverse perinatal outcome and morbidity [3]. Sleep needs vary with lifestyle, age, gender and other effects such as stress and work schedules [4]. Poor sleep is linked to metabolic neuroendocrine and inflammatory changes [5]. Studies have also demonstrated tharee sleep disturbances and inadequate sleep duration that may lead to Complications affecting general health [6]. Since most healthcare workers are women, disruption of the circadian rhythm can alter the finely coordinated growth processes, as the onset of specific developmental events is often regulated by a fluctuation in certain hormones, either increasing or decreasing them [7]. During night work, sleep is negatively affected by light exposure, consequently, many women experience heightened concern about the potential adverse effects of night work on their pregnancy and fetal development. The circadian rhythm, or biological clock, regulates bodily functions and behaviors such as sleep, appetite, blood pressure, hormone levels, alertness, and temperature according to a 24-hour day-night cycle. Frequent changes in the mother's circadian rhythm can impact fetal development. [8].

Recent evidence indicates that insufficient maternal sleep during pregnancy affects fetal health and birth outcomes, including intrauterine fetal growth restriction and premature birth [9] Studies have shown that a high sleep quality index (PSQI) in pregnant women during the first trimester may lead to preterm birth. Although the precise relationship between sleep quality in the first trimester and adverse birth outcomes is not fully understood, research suggests that sleep problems during the first 20 weeks of pregnancy may increase systemic inflammation, which is essential for the continuation of early pregnancy but may also exacerbate the inflammatory response that disrupts normal trophoblast inversion. This latter condition can lead to subsequent disruption of maternal vascular remodeling, contributing to preterm birth, preeclampsia, and intrauterine growth restriction. [10,11]. Studies have also reported a link between duration of

sleep-in pregnant women with inflammation and postpartum depression. Poor sleep will increase levels of inflammatory markers such as, interleukin-1 (IL-1), IL-6, IL-2, tumor necrosis factor (TNF) α and C-reactive protein and women with postpartum depression have been observed to have elevated levels of pro-inflammatory cytokines [12]. The results of a study in Shanghai in 2020 showed an increased risk of respiratory allergies in children, linked to short maternal sleep duration, excessive screen exposure, and lack of physical activity, but this was only found in boys [13]. Several studies of further investigations revealed a sex-specific effect, with a marked increase in risk for males compared to females, based on the quality and quantity of sleep during early pregnancy and in relation to small fetal size for gestational age. This sex-specific effect can be explained by the different impact of maternal sleep disturbances on the placenta and yolk sac ,Similar to other studies found that mothers pregnant with male fetuses who experienced sleep disturbances had significantly thicker and less homogeneous placentas and yolk sacs to compensate for the unfavorable uterine environment [14]. As is well known, melatonin plays a key role in reducing oxidative stress and has anti-inflammatory properties. Its levels increase in pregnant women, and it also works to eliminate free radicals and contains antioxidants [15]. The pineal gland produces and secretes melatonin during the dark period of the daily cycle (biological night). Exposure to light during pregnancy and sleep disturbances can lead to decreased melatonin levels, especially if this continues for several consecutive nights. [16].

A review study on mice found that melatonin may prevent premature birth associated with inflammation [17]. Melatonin has been reported to modulate progesterone secretion and contribute to the maintenance of pregnancy [18]. The first stages of fetal development in pregnancy are crucial, as organs and body systems develop during this period and are particularly vulnerable to harmful factors. Organogenesis is a stage of accelerated cell differentiation, tissue formation, and morphogenesis and disturbances during this period can affect both the long-term functional programming of the fetus and structural development [19]. This review aims to consolidate the available evidence on the relationship between maternal sleep deprivation (including insomnia, insufficient sleep duration, and fragmented sleep) and it's related to early fetal growth and organ development; to identify proposed biological mechanisms, particularly the role of placenta, inflammatory pathways, hormonal redox pathways, and circadian rhythm disruption to summarize its impact on the development of key nervous system organs. This

review will integrate molecular insights from experimental models and human observational studies, considering critical gestational periods when the mother is most vulnerable, and will identify important knowledge gaps to inform future applied research and preventive efforts in antenatal care. [20].

1.1 Role of placenta as a central mediator between embryonic development and maternal sleep disturbance

The placenta is the bridge that transmits functions between the maternal and fetal environments. Placental implantation and remodeling of the uterine spiral arteries by the trophoblast transform the uterine circulation into a high-flow, low-resistance system capable of organogenesis and supporting rapid fetal growth. This remodeling process is essential to ensure adequate uteroplacental oxygenation and perfusion, as its disruption is closely associated with major obstetric complications and fetal growth retardation in the early stages of pregnancy [21]. Stress-related neuroendocrine signals, sympathetic nervous system activity, and inflammatory balance are affected by maternal sleep deprivation. It has also an impact on the immune and vascular state essential for optimal placental development, thus indirectly affecting fetal growth through placental dysfunction. The placenta also performs other vital functions according to fetal needs, such as regulating nutrients and oxygen delivery. The mechanisms for transporting lipids, amino acids, and glucose in the placenta are subject to precise and balanced regulation and can change in response to maternal hormonal and metabolic signals. Alterations in transport have a direct impact on fetal development and growth. Several studies have likewise indicated that the placenta contains genes essential for the biological clock (such as PER, BMAL1, and CLOCK) and may demonstrate the functional organization of the biological clock. It has been observed that circadian biology provides another pathway through which sleep disturbances may affect fetal development via the placenta. Human placental tissue can synthesize melatonin, a chemical important for circadian signaling and an antioxidant [22].

A study by Ross *et al*. 2025 on individual births showed that obstructive sleep apnea and insomnia are associated with an increased risk of postpartum syndrome and Parkinson's disease, Compared to individuals without sleep disorders and insomnia, obstructive sleep apnea is associated with an increased risk of certain complications, including low birth weight, placental

abruption, preterm birth before 32 weeks of gestation, and some serious complications such as stroke, hysterectomy and disseminated intravascular coagulation [23].

1.2 Effects of Maternal Sleep Loss in Embryogenesis

The fetus is affected when the mother is deprived of sleep through a series of interconnected biological processes that intersect at the level of the mother-placenta-fetus axis. Effects of sleep disorders or insomnia are not limited to one aspect alone, but also include physiological changes in the mother, characterized by stress-axis activation, oxidative stress, metabolic imbalance and disruption of the circadian rhythm. These factors combined can disrupt the delicate balance of the fetal environment, the normal function of the placenta and critical periods of fetal growth and organogenesis [24]. Melatonin performs biological clock and antioxidant functions; therefore, irregular or inhibited melatonin secretion during sleep may weaken antioxidant defenses and disrupt rhythmic hormonal signals that regulate placental function and fetal tissue growth [25]. Irregular routines such as late-night work, shift work, and irregular sleep patterns indicate disruption of the biological clock and melatonin secretion. The placenta exhibits a genetic mechanism for the biological clock, and studies have examined gene expression of melatonin signals and placental clocks as elements of communication between mother and fetus [26]. Experimental and applied evidence indicates that long-term maternal exposure to disruption of the biological clock exposes the offspring to significant metabolic stress and affects the growth characteristics of the placenta and the newborn [27].

1.3 The effect of disruption of circadian rhythms on the development of the fetal nervous system

Nervous systems are mainly sensitive through early development due to the formation of autonomic circuits and hypothalamic pituitary within severely restricted timeframes and appearance of neuronal migration, synaptogenesis, and neurogenesis. Not with standing direct evidence in human concerning fetal brain development is limited by practical consideration and ethical, converge data from human cohorts' studies (sleep quality/period, insomnia signs, sleep disturbance breathing) and regulated animal models (sleep restriction/deprivation during definite pregnancy days) demonstrate a relation between modified neurodevelopmental outcomes and maternal sleep deprivation, encompassing behavior and cognition. Many investigations recorded that inadequate prenatal sleep health constantly relation with behavioral and developmental

outcomes in offspring, with growing indication connecting antenatal sleep patterns with alteration in offspring brain function and structure [28]. The poor sleep effects on eye movement by reducing rapid eye movement (REM) sleep, restriction of REM sleep during late gestation results in depression like traits in newborns which continuous until middle age [29]. Experimental investigations using animal models provide the strongest mechanistic evidence. In rats, motherly sleep deficiency during different phases of pregnancy is associated with decrease in hippocampus-dependent memory and learning, raise disquiet and depressive attitude, and reduce adult hippocampal neurogenesis in offspring, as shown by reduce BrdU-positive cells counts [30]. Maternal sleep deficiency led to cognitive-behavioral impairments in progeny correlation neuroinflammation markers and diminished neurogenesis, confirm the hippocampus as a vulnerable neural aim that indicate with Zhao et al. [31]. The studies show that prenatal sleep deficiency can "program" developing neural circuits alternation in the ways that persist Postnatally, with consistent involvement of the hippocampus and limbic networks

Many essential biological methods link maternal sleep deficiency to effect on the embryonic neural system the main pathway is oxidative stress and maternal–placental irritation that affected fetal neural development by convert vascular function, oxidative damage and cytokine exposure.
The relation of sleep deficiency with miscarriage and intrauterine growth limited with high indices of oxidative damage and pro-inflammatory changes in placenta and maternal serum, along with reduced melatonin signaling components (MT1/MT2, AANAT) in chorionic tissue, this demonstration by (Li *et al*.) who utilized a rat model [25]. This was found to be relevant to brain development as inflammation and oxidative stress can impact placental perfusion and the transfer of nutrients and oxygen necessary for embryonic neural development, in addition to altering endocrine signals that regulate neural differentiation. Sleep deficiency, mainly when collective with circadian disorder, may reduce maternal melatonin signals and potentially deteriorate antioxidant resistance during critical brain developmental duration [32].
For development of body and brain in a healthy way it's important to understand the changing nature of sleep in correlation to state of pregnancy and age plays a crucial role in preventing the conditions of prenatal disorder. Even though sleep is important for a dynamic brain (work during day) it still an undervalued pattern for a development of body and brain in a healthy way it's important to understand the changing nature of sleep in correlation to state of pregnancy and age

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1.4 Clinical Relevance and Applications

Sleep should be recognized as a routine component of maternal health "vital sign" in prenatal cure. Considering the susceptibility for sleep quality to disturbers as development progresses and explain the high prevalence of insomnia manifestations during gestation, Healthcare providers could systematically integrate concise screening tools of sleep duration and quality into routine prenatal evaluations, excessive daytime sleepiness insomnia symptoms, snoring or observed apneas, restless legs syndrome, and into standard prenatal care. This is demonstrated by healthcare assessments indicating that sleep disorders are common and affect obstetric outcomes [33]. Physicians should exercise caution when assessing pregnant women at elevated risk of sleep-disturbance breathing/obstructive sleep apnea, and the clinical approach should focus on targeted rather than comprehensive screening (hypertension, obesity, marked apnea, loud snoring, and excessive sleepiness), since the sympathetic nervous system activation associated with OSA (obstructive sleep apnea) and intermittent hypoxia may plays significant role to placental impairment and fetal risks [34].

Clinicians and public health practitioners must consider sleep as a critical aspect of health equity. Sleep deprivation is often shaped by modifiable social determinants, namely extended working hours; alter work, housing conditions, caregiving duties and psychosocial stressors. Identifying these influences during prenatal assessment support practical interventions (including organized sleep therapy, asses to social support resources, work schedule adjustments and mental health assistance) mainly for patients with an elevated baseline threat of adverse outcomes, Guidance and management should demonstrate the relation of sleep to maternal issues that affect fetal growth. Meta-analytic records links negative pregnancy outcomes with shortened and extended sleep periods with, particularly GDM, therefore endorsing the obligation for regular counseling on good sleep habits, symptom recognition, and prompt treatment escalation, such as assessment and continuous positive airway pressure (CPAP) for OSA when appropriate and cognitive behavioral therapy for insomnia (CBT-I) [35].

1.5 Future research directions

Patterns that continuously evaluate sleep patterns and systemic health indicators during the begging stages of gestations are useful for accurate differentiation between the effects of uterine implantation/ placental formation and fetal development and growth. A helpful next step would be to standardize the reporting of "sleep health" (timing, duration, regularity, quality, and fatigue) regularly throughout all three trimesters and the postpartum period, especially given the dynamic changes in sleep patterns during the perinatal period. To link sleep exposure to fetal organ development, research should correlate sleep measurements with developmental stages rather than relying on "overall averages for each stage of pregnancy. Further investigation should incorporate objective assessment of sleep alongside detailed placental characterization including evaluation of (oxidative stress and inflammatory biomarkers, perfusion/vascular indices, endocrine outputs and expression of circadian regulatory gene) and examine their association with early developmental organ phenotypes. This provides the most direct translational evidence of relationships between maternal sleep patterns and the multi-organ programming on the embryo. Subsequent studies should examine the environmental exposures (obesity, diabetes, stress, smoking, mood disorder, irregular work schedule), evaluate fetal-sex specific prenatal effect, and analyze the role of social and economic condition on sleep deprivation. This reinforced causal validity and improved the applicability of the findings for prenatal care implementation and public health policy.

2. Conclusion

Problems from distributed maternal sleep throughout gestation are common and progressively recognized as a prominent physiological influence during embryonic growth and development. The accumulating evidence suggests a integrated multi-pathway model found disrupt or inadequate sleep is often related with circadian disorder and in certain instances, sleep-disturbances respiratory influences maternal neuroendocrine regulation, inflammatory-oxidative conditions, autonomic balance and metabolic equilibrium. Alteration in the maternal condition can be transferred to the embryo essentially via the placenta, which integrate maternal signals and regulate oxygen transfer, immune system regulation, uteroplacental perfusion, function of endocrine system and nutrition during fetal organ development. Experimental study offers causal evidence that disorder in pregnancy sleep can cause persistent changes in child

neurocognitive and behavioral profile, energy hemostatsis set level, liver susceptibility to steatotic injury, blood pressure control, reproductive consequences renal physiology and modified immunological. Human evidence, even though frequently limited by subjective sleep measures and confounders, generally support these mechanisms linked to inadequate sleep with gestation complications (e.g., hypertensive disorders and pregnancy diabetes) and later-life child outcomes reflecting early developmental risk.

These results suggest the need to integrate sleep health into routine prenatal nurturing through early diagnosis of sleep-disturbance respiratory and insomnia, along with the application of safe and widely available treatments, such as protocols for treating suspected obstructive sleep apnea, cognitive behavioral treatment for accurate diagnosis and insomnia. From a public health perspective, mitigating circadian rhythm disruption and promoting sleep, particularly for pregnant women experiencing high levels of stress, shift work, or socioeconomic challenges, is a modifiable factor in promoting placental health and reducing the risk of fetal abnormalities.

References

1. R. B. Bangsgaard, S. Høgh, C. Borgsted, et al., “Sleep Quality in Late Pregnancy Is Associated With Maternal Mental Health in the Early Postpartum Period,” European Journal of Obstetrics, Gynecology, and Reproductive Biology 311 (2025): 113980.

2. S. Celik and E. Nazik, “The Effect of Aromatherapy Applied to Preg nant Women on Sleep Quality and Fatigue Level: A Randomized Clini cal Trial,” Explorer 21, no. 3 (2025): 103157

3. Tsega SS, Kiflu M, Wubante SM, Mekonnen BD, Tarekegn YA. Sleep disturbance and its associated factors among pregnant women in Ethiopia: systematic review and meta-analysis. BMC Psychiatry. 2024 Jan 15;24(1):51. doi: 10.1186/s12888-023-05456-7. PMID: 38225632; PMCID: PMC10789060.

4. Fatima, Y.; Doi, S.A.; Najman, J.M.; Mamun, A.A. Exploring Gender Difference in Sleep Quality of Young Adults: Findings from a

Large Population Study. Clin. Med. Res. 2016, 14, 138–144. [CrossRef]

5. Nodine, P.M.; Matthews, E.E. Common Sleep Disorders: Management Strategies and Pregnancy Outcomes. J. Midwifery Womens

Health 2013, 58, 368–377. [CrossRef]

6. Yannakoulia, M.; Anastasiou, C.A.; Karfopoulou, E.; Pehlivanidis, A.; Panagiotakos, D.B.; Vgontzas, A. Sleep Quality Is Associated

with Weight Loss Maintenance Status: The MedWeight Study. Sleep Med. 2017, 34, 242–245. [CrossRef]

7. Magar V, Gerecke M, Dhillon I, and Campbell J. Women's contribution to sustainable development through work in health: using a gender lens to advance a transformative 2030 agenda. 2016;1–18

View Article Google Scholar

8. Mark P. J., Crew R. C., Wharfe M. D., Waddell B. J. Rhythmic Three-Part Harmony: The Complex Interaction of Maternal, Placental and Fetal Circadian Systems. J.Biol.Rhythms. 2017;32:534–549 pmid:28920512

View ArticlePubMed/NCBIGoogle Scholar

9. Hajipour M, Soltani M, Safari-Faramani R, Khazaei S, Etemad K, Rahmani S, Valadbeigi T, Yaghoobi H and Rezaeian S. Maternal sleep and related pregnancy outcomes: a multicenter cross-sectional study in 11 provinces of Iran. J Family Reprod Health 2021; 15: 53-60.

10. Okun ML, Roberts JM, Marsland AL and Hall M. How disturbed sleep may be a risk factor for adverse pregnancy outcomes. Obstet Gynecol Surv 2009; 64: 273-280.

11. Okun ML, Schetter CD and Glynn LM. Poor sleep quality is associated with preterm birth. Sleep 2011; 34: 1493-1498.

12. Irwin MR, Wang M, Campomayor CO, Collado-Hidalgo A, Cole S. Sleep deprivation and activation of morning levels of cellular and genomic markers of inflammation. Arch Intern Med. 2006;166(16):1756–62. doi: 10.1001/archinte.166.16.1756. [DOI] [PubMed] [Google Scholar]

13. Chen, Y.; Lyu, J.; Xia, Y.; Zhu, J.; Tong, S.; Ying, Y.; Qu, J.; Li, S. Effect of maternal sleep, physical activity and screen time during pregnancy on the risk of childhood respiratory allergies: A sex-specific study. Respir. Res. 2020, 21, 230. [Google Scholar] [CrossRef] [PubMed

14.Vietheer A., Kiserud T., Haaland Ø.A., Lie R.T., Kessler J. Effect of maternal sleep on embryonic development. Sci. Rep. 2022; 12:17099. doi: 10.1038/s41598-022-21516-6. [DOI] [PMC free article] [PubMed] [Google Scholar]

15. Reiter R. J., Tan D. X., Korkmaz A., Rosales-Corral S. A. Melatonin and stable circadian rhythms optimize maternal, placental and fetal physiology. Hum.Reprod.Update. 2014; 20:293–307 pmid:24132226

16.Verteramo R, Pierdomenico M, Greco P, Milano C.The role of melatonin in pregnancy and the health benefits for the newborn,Biomedicines. 2022;10(12):3252. doi:10.3390/biomedicines10123252.

17. Dominguez Rubio AP, Sordelli MS, Salazar AI, Aisemberg J, Bariani MV, Rosenstein RE, Franchi AM. Melatonin prevents experimental preterm labor and increases offspring survival. J Pineal Res. 2014;56(2):154–162. doi:10.1111/jpi.12108

18. Di Renzo GC, Giardina I, Clerici G, Brillo E, Gerli S. Progesterone in normal and pathological pregnancy. Horm Mol Biol Clin Investig. 2016;27(1):35–48. doi:10.1515/hmbci-2016-0038

19. Donovan MF, Cascella M.Embryology, Weeks 6–8. In: StatPearls [Internet].

Treasure Island (FL): StatPearls Publishing; 2022. Available from: https://www.ncbi.nlm.nih.gov/books/NBK563181/%E2%81%A0%EF%BF%BD

20. Astiz M, Oster H. Feto-Maternal Crosstalk in the Development of the Circadian Clock System. Front Neurosci. 2021;14:631687. Published 2021 Jan 12. doi:10.3389/fnins.2020.631687

21. Schiffer V, Evers L, de Haas S, Ghossein-Doha C, Al-Nasiry S, Spaanderman M. Spiral artery blood flow during pregnancy: a systematic review and meta-analysis. *BMC Pregnancy Childbirth*. 2020;20(1):680.

22. Villafuerte G, Miguel-Puga A, Rodríguez EM, Machado S, Manjarrez E, Arias-Carrión O. Sleep deprivation and oxidative stress in animal models: a systematic review. Oxid Med Cell Longev. 2015;2015:234952. doi:10.1155/2015/234952

23.Ross, N., Baer, R. J., Oltman, S. P., Gossett, D. R., Jelliffe-Pawlowski, L., Brandt, J. S., et al. (2025). Ischemic Placental Disease and Severe Morbidity in Pregnant Patients With Sleep Disorders. JAMA Network Open, 8(9), e2532189. doi:10.1001/jamanetworkopen.2025.32189.

24. Bublitz MH, Bourjeily G, D'Angelo C, Stroud LR. Maternal Sleep Quality and Diurnal Cortisol Regulation Over Pregnancy. Behav Sleep Med. 2018;16(3):282-293.

25.Li J, Peng X, Zhong H, Liu S, Shi J, Zhou X, Li B. Sleep deprivation during pregnancy leads to poor fetal outcomes in Sprague-Dawley rats. J Reprod Immunol. 2023 Dec;160:104166. doi: 10.1016/j.jri.2023.104166.

26.Valenzuela FJ, Vera J, Venegas C, Pino F, Lagunas C. Circadian System and Melatonin Hormone: Risk Factors for Complications during Pregnancy. Obstet Gynecol Int. 2015;2015:825802. doi:10.1155/2015/825802

27.Yao N, Kinouchi K, Katoh M, Ashtiani KC, Abdelkarim S, Morimoto H, et al. Maternal circadian rhythms during pregnancy dictate metabolic plasticity in offspring. Cell Metab. 2025 Feb 4;37(2):395-412.e6. doi: 10.1016/j.cmet.2024.12.002.

28.Nevarez-Brewster M, Han D, Todd EL, Keim P, Doom JR, Davis EP. Sleep During Pregnancy and Offspring Outcomes From Infancy to Childhood: A Systematic Review. Biopsychosoc Sci Med. 2025;87(1):7-32. doi:10.1097/PSY.0000000000001352)

29.Kamalesh K. Gulia Effect of Sleep Restriction during Pregnancy on Fetal Brain Programming and Neurocognitive Development of Offspring: A Review. 2023, ational Academy of Medical Sciences, 59(3);129-138doi:10.1055/s-0043-1770157.

30. Wei R-M, Zhang Y-M, Li Y, Wu Q-T, Wang Y-T, Li X-Y, Li X-W and Chen G-H (2022) Altered cognition and anxiety in adolescent offspring whose mothers underwent different-pattern maternal sleep deprivation, and cognition link to hippocampal expressions of Bdnf and Syt-1. Front. Behav. Neurosci. 16:1066725. doi: 10.3389/fnbeh.2022.1066725

31.Zhao Q, Peng C, Wu X, Chen Y, Wang C, You Z. Maternal sleep deprivation inhibits hippocampal neurogenesis associated with inflammatory response in young offspring rats. Neurobiol Dis. 2014 Aug;68:57-65. doi: 10.1016/j.nbd.2014.04.008.

32. Feybesse C, Chokron S, Tordjman S. Melatonin in Neurodevelopmental Disorders: A Critical Literature Review. Antioxidants (Basel). 2023 Nov 20;12(11):2017. doi: 10.3390/antiox12112017. PMID: 38001870; PMCID: PMC10669594.

33.Yang C, Fu R, Wang H, Jiang Y, Zhang S, Ji X. Evaluating the global prevalence of insomnia during pregnancy through standardized questionnaires and diagnostic criteria: a systematic review and meta-analysis. Front Psychiatry. 2024;15:1427255.

34. Dominguez J, Cantrell SML, Habib AS, Izci-Balserak B, Lockhart E, Louis JM, et al. Society of Anesthesia and Sleep Medicine and the Society for Obstetric Anesthesia and Perinatology consensus guideline on the screening, diagnosis, and treatment of obstructive sleep apnea in pregnancy. Obstet Gynecol. 2023 Aug;142(2):403–423. doi:10.1097/AOG.0000000000005261

35. Bonzini M, Palmer KT, Coggon D, Carugno M, Cromi A, Ferrario MM. Shift work and pregnancy outcomes: a systematic review with meta-analysis of currently available epidemiological studies. BJOG. 2011;118(12):1429-1437. doi:10.1111/j.1471-0528.2011.03066.x]