

MATERNAL CHRONODISRUPTION

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THERE'S A CARCINOGEN PRESENT IN EVERY PREGNANT WOMAN'S HOME THAT NONE OF HER PRENATAL CARE PROVIDERS ARE GOING TO WARN HER ABOUT.

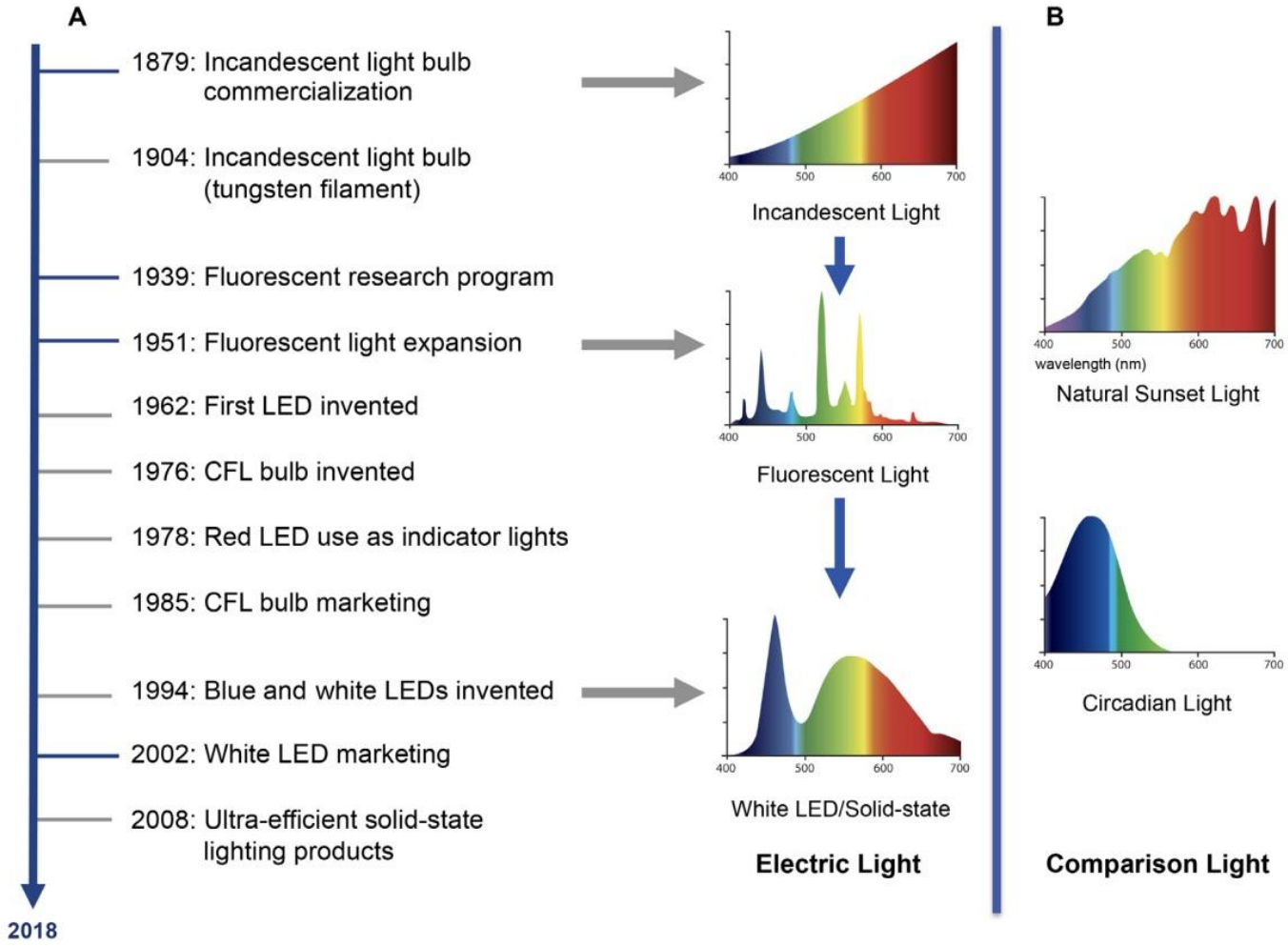
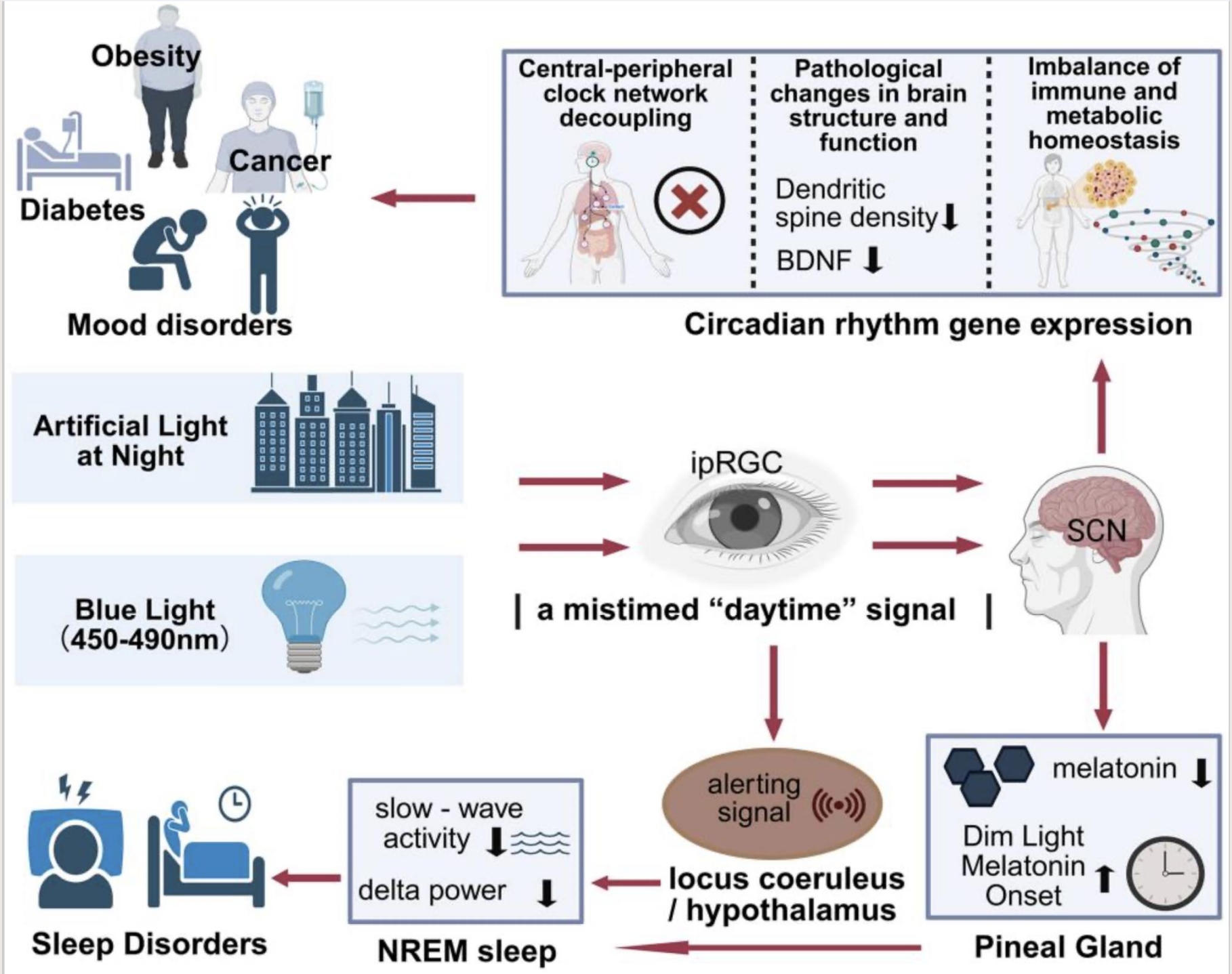
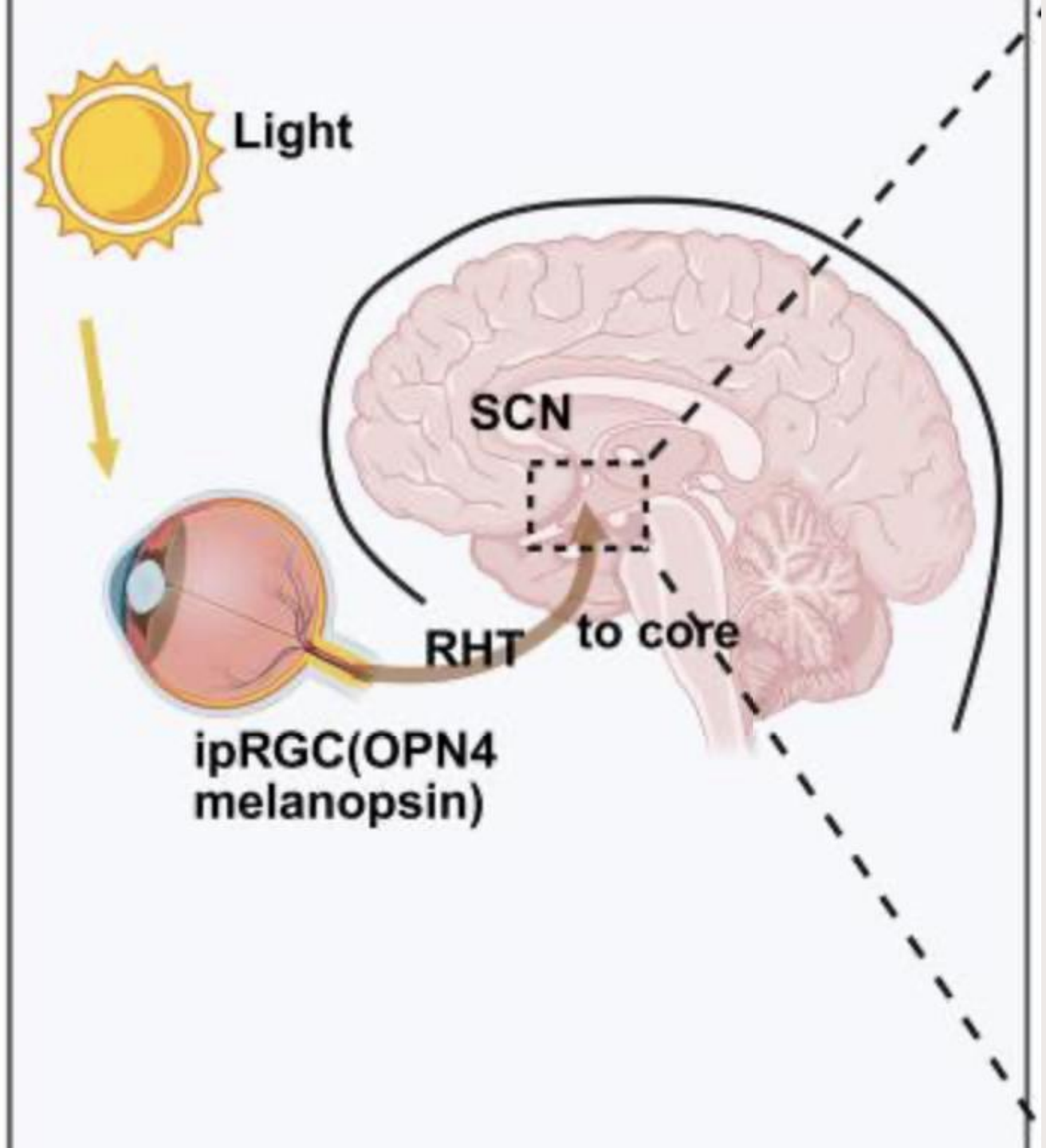


Figure 4. Technological advances in lighting over time have led to lighting with higher levels of short wavelengths

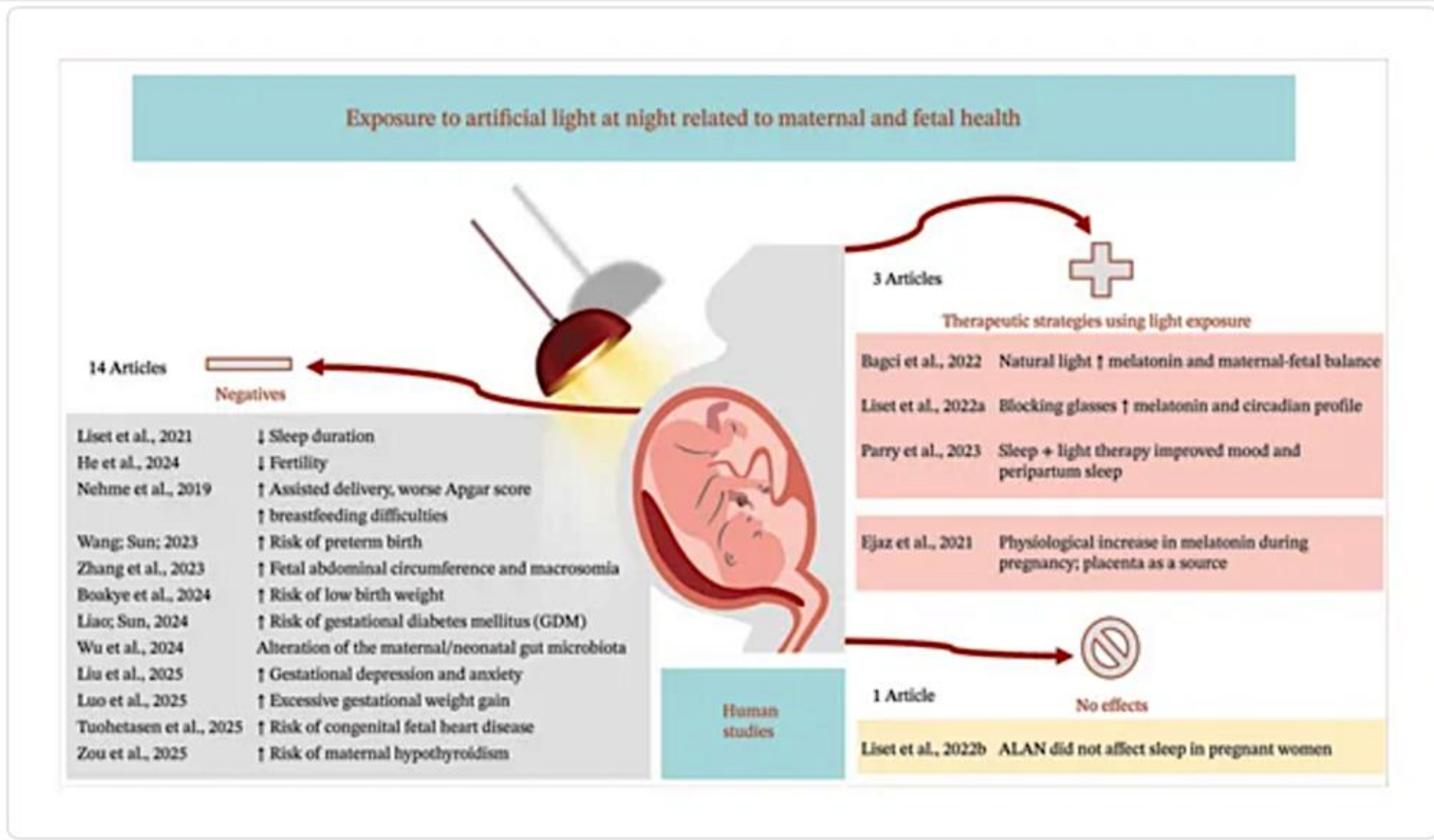

Obesity
Cancer
Diabetes
Mood disorders
Central-peripheral clock network decoupling
Pathological changes in brain structure and function
Dendritic spine density
BDNF
Imbalance of immune and metabolic homeostasis
Circadian rhythm gene expression
Artificial Light at Night
Blue Light (450-490nm)
ipRGC
SCN
a mistimed "daytime" signal
alerting signal
locus coeruleus / hypothalamus
melatonin
Dim Light Melatonin Onset
Pineal Gland
slow - wave activity
delta power
NREM sleep
Sleep Disorders

"At night, SCN activity wanes, disinhibiting the pathway and allowing for robust melatonin production. Light exposure at night, detected by retinal ipRGCs, rapidly reactivates the SCN, which clamps this pathway shut, acutely suppressing melatonin synthesis. The spectral sensitivity of this suppression reflex mirrors that of melanopsin, with short-wavelength blue light being the most potent stimulus." (Yuan et al).



"EVENING EXPOSURE TO LONG-WAVELENGTH RED LIGHT, WHICH MINIMALLY STIMULATES IPRGCS, HAS LITTLE TO NO EFFECT ON MELATONIN OR CIRCADIAN PHASE AND PRESERVES SLEEP ARCHITECTURE. THIS PRINCIPLE UNDERPINS THE STRATEGY OF USING 'CIRCADIAN LIGHTING' OR BLUE-BLOCKING FILTERS TO CREATE A TRANSITION TO SLEEP" (YUAN, ET AL).

EFFECTS OF ARTIFICIAL LIGHT AT NIGHT (ALAN) ON MATERNAL AND FETAL OUTCOMES IN HUMAN STUDIES.



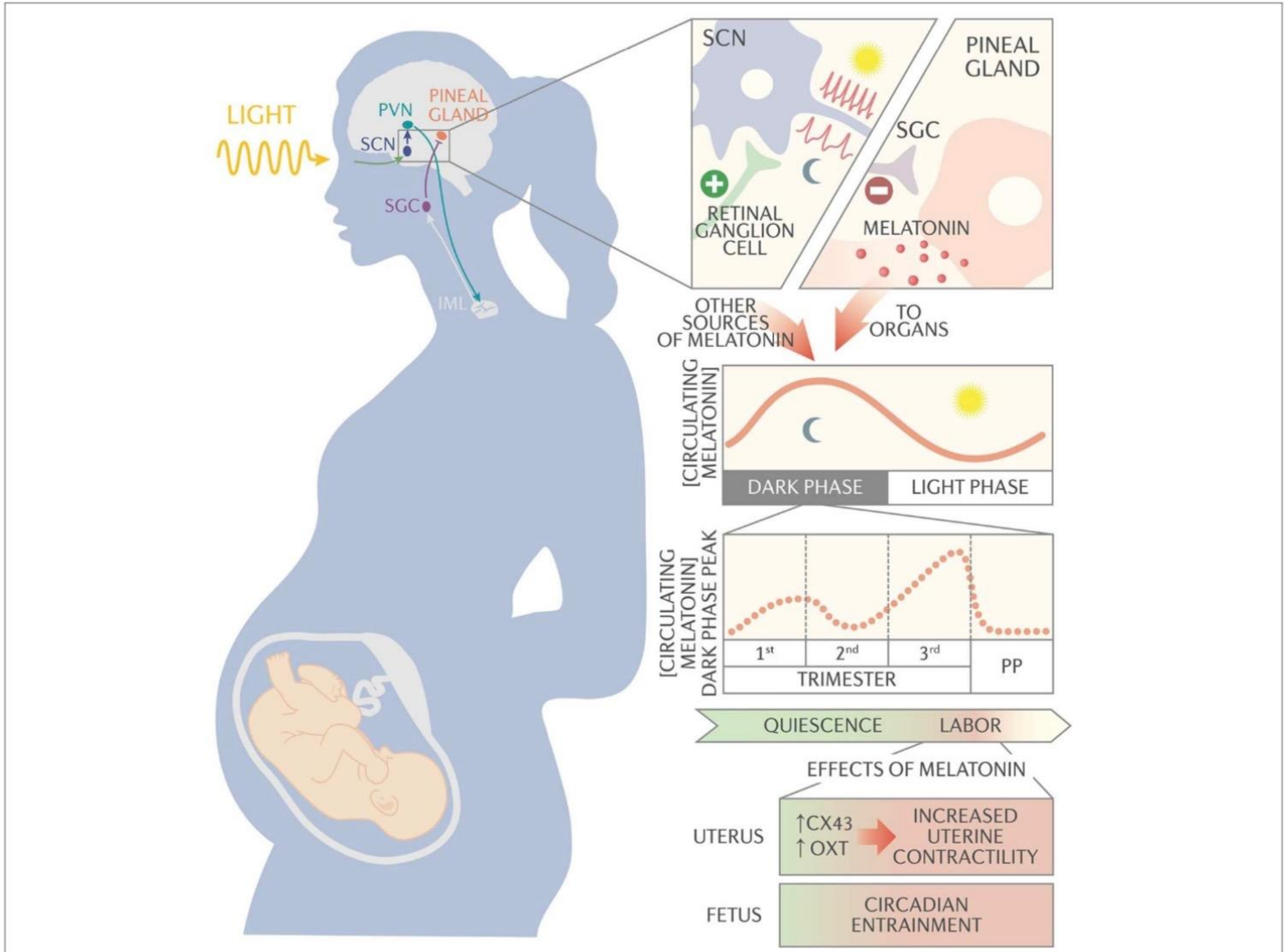


FIGURE 1 | Schematic of melatonin circadian regulation and action during pregnancy. The suprachiasmatic nucleus (SCN) synchronizes to the external light/dark cycles via signals received from the melanopsin-containing retinal ganglion cells. The SCN neurons have a high firing rate during the light period and a slow firing rate during the dark period. The SCN projects to the paraventricular nucleus (PVN), which connects to the intermediolateral cell column (IML). The IML signals to the superior cervical ganglion (SCG), which signals to the pineal gland to synthesize and secrete melatonin into circulation. Melatonin, along with other neurotransmitters, synchronizes circadian rhythms throughout the body. During pregnancy, night time peak serum melatonin concentration decreases slightly between 1st and 2nd trimester, then begins to increase after 24 weeks of gestation until it reaches maximum concentration at the end of pregnancy. Serum melatonin acts synergistically with oxytocin via melatonin receptor on the uterus to activate membrane-bound phospholipase C and protein kinase C pathways. These pathways promote expression of the gap junction protein connexin-43 and increase uterine sensitivity to oxytocin, increasing uterine contractility. In addition, melatonin passes unaltered through the placenta and appears to be important for entraining fetal circadian rhythms.

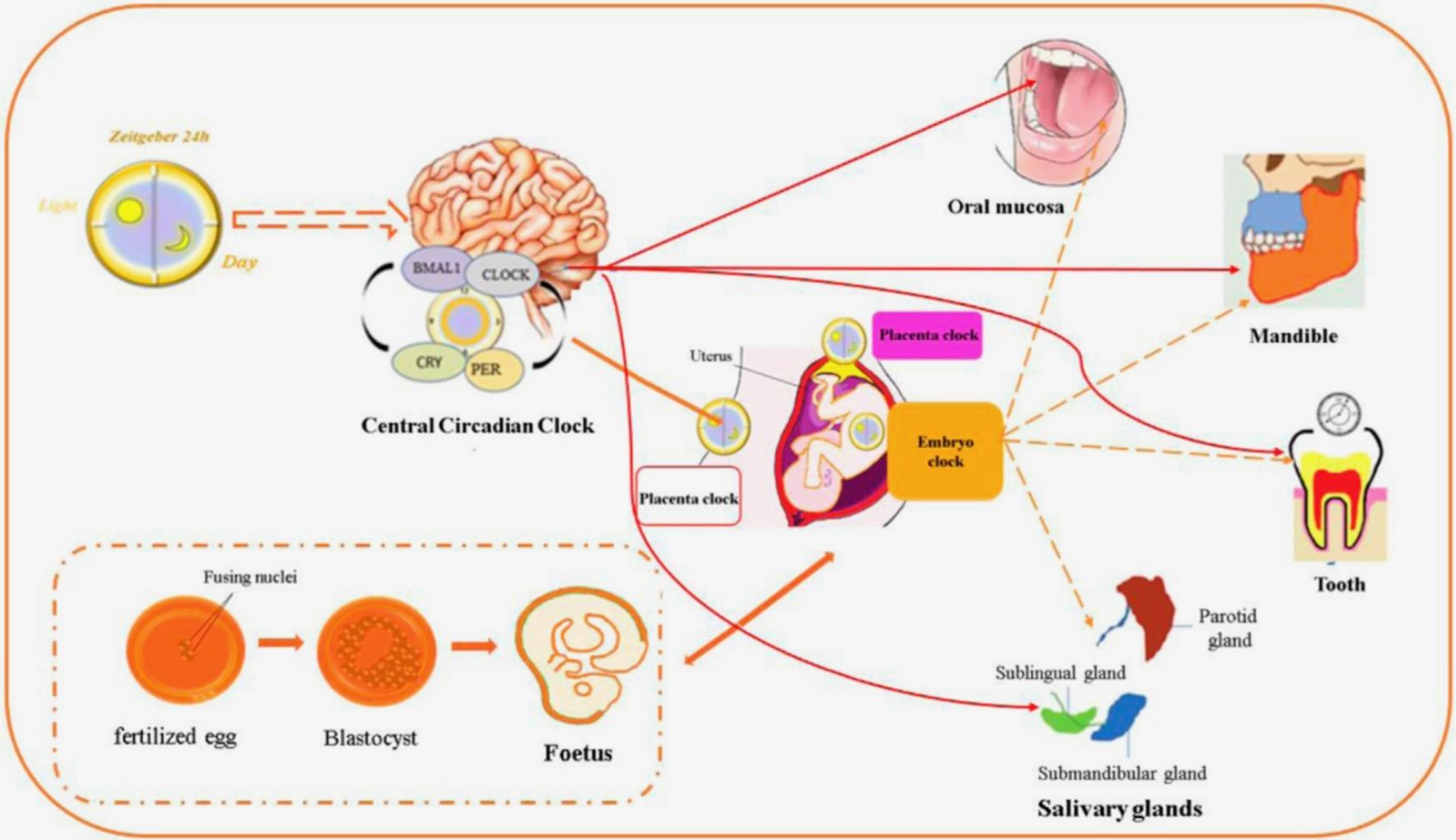


FIGURE 1
Circadian clock genes are expressed in the oral and maxillofacial region. The circadian clock regulates development in different stages of the embryonic period and postnatal stage. Circadian clock gene expression was detected in the maxillomandibular region, teeth, salivary glands, oral epithelium and periodontal tissue, which comprise the oral and maxillofacial regions.

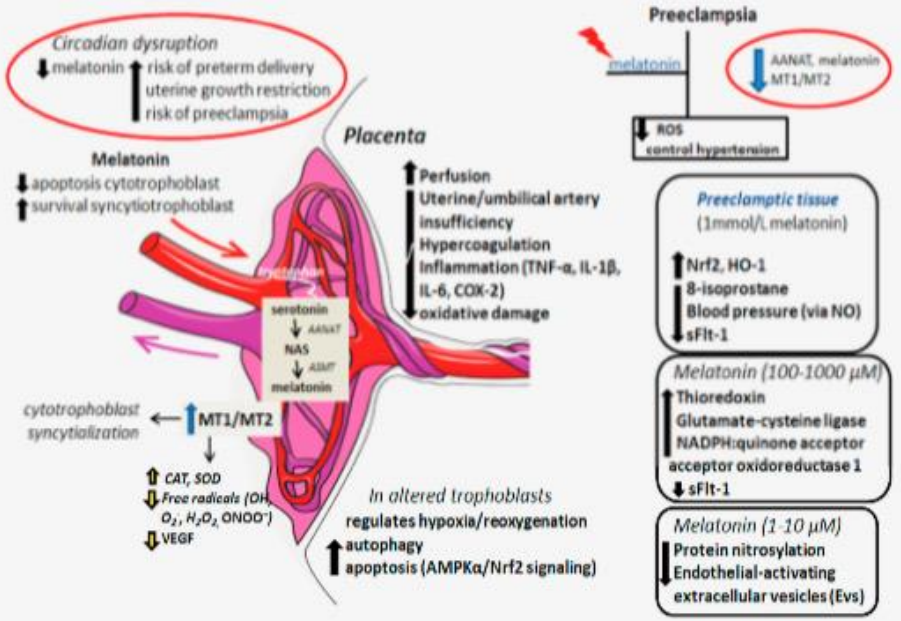
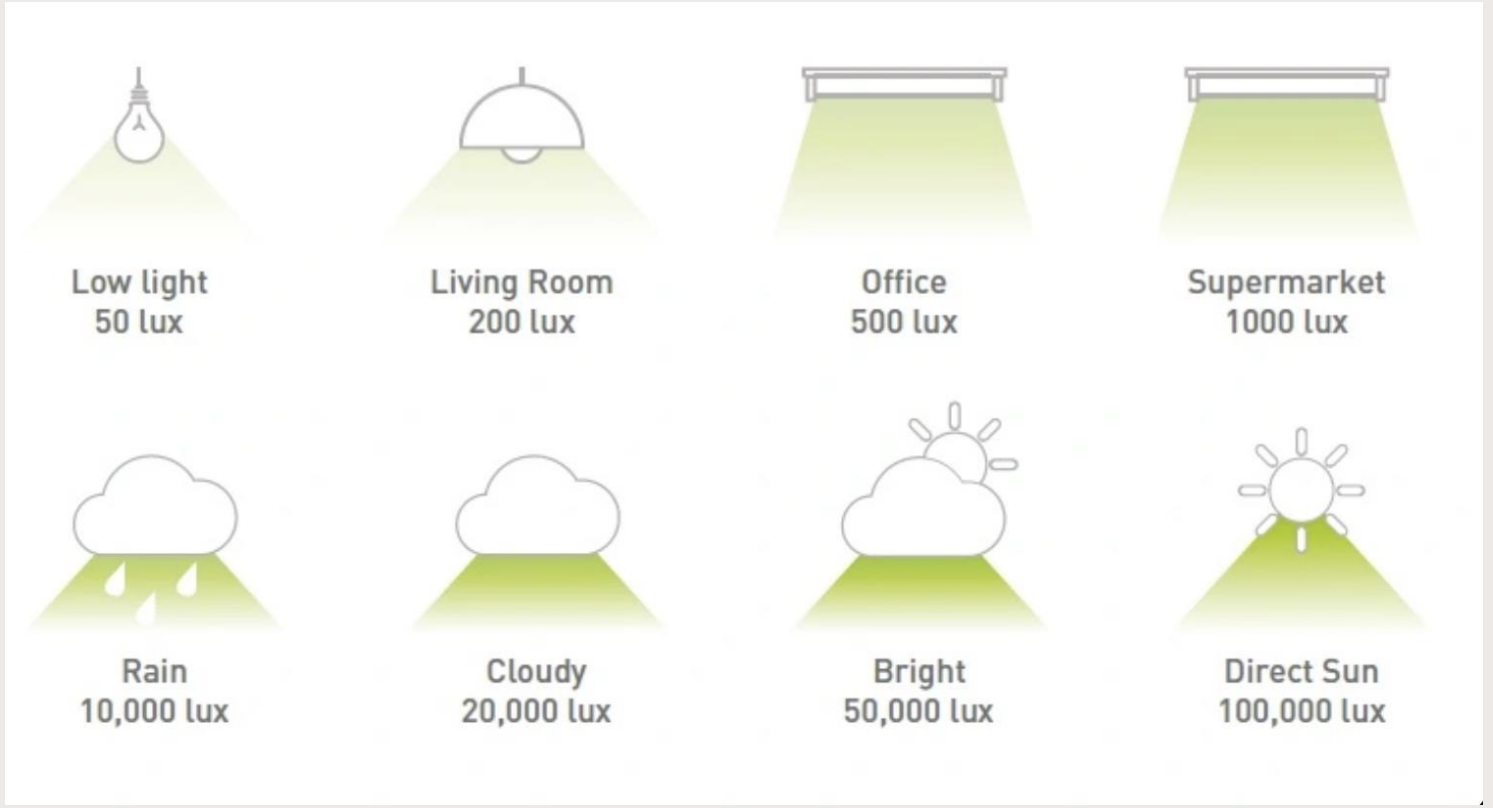


Figure 2. Circadian disruption with a decline in melatonin levels has been associated with placental disturbances possibly affecting maternal and fetal health. Melatonin and its receptors MT1 and MT2 are constantly synthesized by the placental cells to promote survival and syncytialization of the cytotrophoblast while reducing free radicals and angiogenesis. Through still via unknown mechanisms, melatonin enhances placental perfusion and prevents artery insufficiency, hypercoagulation, and inflammatory and oxidative damage; melatonin also protects altered placental trophoblasts from hypoxia/reoxygenation by regulating apoptosis and autophagy. In the preeclampsia condition, enzymatic machinery for melatonin synthesis is inhibited in addition to the low expression of MT1 and MT2 receptors; exogenous administration of melatonin has a prominent role in controlling ROS and hypertension. Experimentally, the treatment of preeclamptic tissues with different doses of melatonin has evidenced positive responses against various deleterious parameters; in general, melatonin attenuates ROS, restores the antioxidant status of tissue, recovers endothelial monolayer integrity and mitigates vascular cell adhesion. ROS: reactive oxygen species, AANAT: aralkylamine N-acetyltransferase, NAS: N-acetylserotonin, ASMT: acetylserotonin O-methyltransferase; MT1 and MT2: melatonin receptors 1 and 2, CAT: catalase, SOD: superoxide dismutase, VEGF: vascular endothelial growth factor, AMPKα: AMP-activated protein kinase α subunit, Nrf2: nuclear factor erythroid 2-related factor 2, HO-1: heme oxygenase-1, NO: nitric oxide, NADPH: reduced nicotinamide adenine dinucleotide phosphate, TNF-α: tumor necrosis factor alpha, IL-1β: interleukin 1β, IL-6: interleukin 6, COX-2: cyclooxygenase 2, sFlt-1: soluble fms-like tyrosine kinase 1.

MATERNAL SHORT AND LONG-TERM COMPLICATIONS	FETAL SHORT AND LONG-TERM COMPLICATIONS
↓ Sleep duration ↓Fertility ↑Assisted delivery, worse Apgar score ↑ Breastfeeding difficulties ↑ Risk of maternal hyperglycemia ↑ Risk of gestational diabetes mellitus (GDM) Further increases risk of later developing type 2 diabetes, cardiovascular disease, and metabolic syndrome) ↑ Risk of hypertensive disorders of pregnancy Alteration of the maternal/neonatal gut microbiota ↑ Gestational depression and anxiety ↑ Excessive gestational weight gain ↑ Risk of maternal hypothyroidism ↑ Maternal stress	↑ Risk of preterm birth ↑ Fetal abdominal circumference and macrosomia ↑ Risk of low birth weight or intrauterine growth restriction (IUGR) ↑ Risk of low birth weight ↑ Risk of congenital fetal heart disease ↑ Risk of insulin resistance, obesity, and diabetes Alteration of the maternal/neonatal gut microbiota ↑ Risk of metabolic dysfunction ↑ Risk of altered cardiovascular health ↑ Risk of altered mental health ↑ Risk of hypertension Increased adiposity ↑ Risk of poor glucose tolerance Altered corticosterone rhythms ↑ Risk of brain immaturity ↑ Risk of high BMI ↑ Risk of altered fetal size and thyroid hormones Altered neurologic systems ↑ Risk of endocrine dysfunction

LIGHT IS MORE THAN A FLIP OF A SWITCH.

Research continues to evidently show our physiology needs **bright light during the day**, **dim lighting in the evening**, and **dark environments at night** for optimal health. Bright light means light that is potent enough to have a meaningful non-visual effect, given its timing, spectrum, duration, and direction. **Expert recommendations propose indoor lux to be above 250 during the day, below 10 lux in the evening, and below 1 lux during sleep** (Piper, M et al).



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