

## REVIEW ARTICLE

## Screen time and its Biochemical Effect on Human beings

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## HOW TO CITE THIS ARTICLE:

*Sachin C Narwadiya, Neha Suthar.* Screen time and its Biochemical Effect on Human beings. RFP Jour. of Bio. and Biophy. 2025; 10(2): 61-67.

## ABSTRACT

Excessive screen exposure has become a major public health issue in the modern digital age, with increasing evidence associating long-term exposure to screens with a range of biochemical changes in human subjects. The current review study discusses the physiological and biochemical effects of prolonged exposure to electronic devices, including mechanisms like disruption of circadian rhythm, hormonal imbalance, oxidative stress, neurochemical modification, and metabolic dysregulation. Long-term exposure to blue light from screens is found to inhibit the production of melatonin, disrupting sleep and changing the hypothalamic-pituitary-adrenal (HPA) axis. Sedentary lifestyle from screen time also leads to insulin resistance, higher cortisol levels, higher inflammatory markers (e.g., C-reactive protein, interleukins), and abnormalities in neurotransmitter homeostasis, such as the dopamine and serotonin pathways. These biochemical changes have been linked to sleep disorders, obesity, type 2 diabetes, mood dysregulation, and cognitive performance impairment. By integrating results from more current research, this review stresses the importance of evidence-based guidelines for modulating screen exposure and encouraging healthier digital life to counteract negative biochemical and physiological effects.

## KEYWORDS

- Screen Time • Blue Light • Melatonin Suppression • Circadian Rhythm
- Oxidative Stress • Cortisol • Neurotransmitters

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➤ Received: 15-09-2025   ➤ Accepted: 30-10-2025



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## INTRODUCTION

Screen-based technologies smartphones, tablets, computers, televisions, game consoles are ubiquitous in contemporary life. Screen time, defined broadly as time spent viewing or interacting with any device displaying a screen, and has grown exponentially in the past decades. Although numerous investigations have been conducted into the psychological, behavioral, and social consequences of screen use, mounting evidence for biochemical and molecular consequences exists. This essay discusses what is currently known regarding the biochemical effects of chronic or excessive screen exposure: how it influences hormone control, brain structure and neurotransmission, circadian rhythm, metabolism, gut microbiome, and other physiological systems. Mechanisms are described, and areas of unknowns mentioned.

### Definition and Scope

“Screen time” is time spent viewing screens of electronic devices for recreation, work, study, or social communication. Too much screen time generally implies more than is usual for age or setting, particularly when it replaces sleep or exercise. At-risk populations are children and adolescents (more so because they are still developing), but many findings hold for adults as well. In their research on biochemical effects, they usually look at: Hormonal secretions (melatonin, cortisol, etc.), Neurotransmitter systems (dopamine, GABA, etc.), Brain structure (gray matter, cortical thickness, fronto-striatal connectivity), Metabolic markers (glucose, insulin, lipid profiles), Gut micro biome and metabolite, Inflammatory markers and oxidative stress, Hormonal and Circadian Effects.

Among the most obvious biochemical effects of screen time is on circadian rhythm and hormone control, particularly melatonin and cortisol.

**Melatonin suppression:** Screen-emitted light, especially wavelengths of blue light, can simulate daytime lighting. When screen viewing is done in the evening or nighttime, it suppresses pineal gland melatonin release. Melatonin is a hormone that informs the body of darkness onset and plays a key role in sleep initiation. Delayed melatonin release results in delayed sleep onset as well as circadian dysregulation.<sup>1</sup>

**Sleep disturbance:** Melatonin suppression causes sleep delay, decreased quality and length of sleep, and can break up sleep stages like REM and deep sleep, disrupting sleep structure.<sup>2</sup>

**Cortisol and stress arousal:** Use of screens, particularly engaging or stressful material, may trigger hyperarousal stimulating sympathetic nervous system pathways and elevating secretion of cortisol, the stress hormone. Chronic overestimation of cortisol may have downstream consequences on metabolism, immune response, and even brain structure.<sup>1</sup>

These hormonal derangements tie into much of the subsequent biochemical and physiological impacts outlined below.

### Brain Structure, Neurotransmitters, Cognitive Function

Screen time can influence brain structure and neurotransmitter systems. Some of these changes are measurable via neuroimaging, functional studies, or biochemical proxies.

**Cortical thinning and gray matter alterations:** High screen exposure, particularly among young adults, has been shown to be linked with cerebral cortex thinning, which is involved in memory, decision-making, and higher-order cognitive processes. Adults with high passive screen exposure or those with problematic screen/smartphone use exhibit reduced gray matter volume.<sup>3</sup>

**Fronto-striatal circuitry/inhibitory control:** Prolonged daily screen time has been linked to diminished fronto-striatal connectivity strength, which is involved in inhibitory control, regulation of impulses, and executive function. This indicates that certain components of self-regulation and impulse inhibition brain networks are structurally or biochemically affected.<sup>4</sup>

**Dopamine/reward pathways:** Screen use, especially engaging material (games, social media etc.), prompts release of dopamine, supporting usage and perhaps desensitizing reward pathways. Regularly, increasing amounts of stimulation (more/longer screen time) will be required to yield similar dopamine-motivated reward, diminishing baseline sensitivity, perhaps contributing to mood dysregulation or addiction-like behavior. Although direct biochemical evidence in humans is less available, behavioral and neuroimaging findings suggest this potential.<sup>1</sup>

**Neurotransmitter metabolism:** Current molecular research indicates that screen time is associated with changed metabolomic profiles, such as amino acid levels of alanine, tyrosine, proline, etc. They are substrates or end-products in neurotransmitter biosynthesis and mitochondrial energy metabolism.<sup>5</sup>

#### Metabolic Effects and Cardio-metabolic Risk

Overuse of screens has a strong link with adverse metabolic and cardiovascular markers, through a variety of mechanisms.

**Obesity/Overweight:** Sedentary time replaces physical activity and contributes to more sitting; screen use is often in conjunction with snacking or unhealthy food. Epidemiological surveys correlate greater screen time with increased adiposity in youth and children.<sup>2</sup>

**Glucose metabolism, insulin sensitivity:** Evidence (from metabolome studies) that excessive screen time correlates with metabolic profiles indicative of impaired insulin sensitivity (risk of Type 1 & Type 2 diabetes), increased markers of risk of obesity & cardiovascular disease. (ASU News)<sup>6</sup>

**Cardio-metabolic risk in young people:** A Danish study recently discovered that in 10-year-olds and 18-year-olds, every additional hour of recreational screen time was linked to greater cardio-metabolic risk scores (including waist circumference, blood pressure, HDL cholesterol, triglycerides, blood sugar). Sleep duration and timing explained some of the risk (brief sleep, delayed sleep exacerbated the relationship).<sup>7</sup>

**Mitochondrial dysfunction & energy metabolism:** The molecular analysis of high vs. low screen time in college students detected anticipated changes in enzyme abundances, metabolic pathways, and mitochondrial function, including such examples as different amino acid metabolism (alanine, tyrosine etc.), energy harvesting in the gut micro biome, etc. These indicate potential mitochondrial stress or inefficiency.<sup>5</sup>

#### Gut Micro biome and Metabolome

One of the growing fields is the relationship between screen time and the composition of gut micro biota, metabolites generated, and potential downstream health effects.

**Micro biome structure:** The elevated screen time group in one study demonstrated gut bacterial taxa divergence from low screen

time e.g., lower abundance of taxa typically found with healthier metabolic profiles (Akermansia, Ruminococcaceae etc.), and higher abundance of others like Prevotella, Veillonella etc.<sup>5</sup>

**Metabolomic alterations:** Five metabolites were significantly lower in high screen time group, according to the same study: 1-methylhistidine, alanine, proline, picolinic acid, and tyrosine. Several of them play essential roles in amino acid metabolism and neurotransmitter or immune function. For instance, tyrosine is a biosynthetic precursor of dopamine and norepinephrine; alanine is engaged in gluconeogenesis as well as in metabolic processes. (Nature)<sup>5</sup>

**Pathway enrichment:** There were changes in pathways such as phenylalanine, tyrosine, tryptophan biosynthesis; alanine, aspartate, glutamate metabolism; and glutamate/glutamine metabolism. These pathways are important to neurotransmission, energy metabolism, and immune modulation.<sup>5</sup>

**Disease correlations:** The microbiome/metabolome signatures for the high screen time group were expected to correlate with different disease signatures: inflammatory bowel disease, celiac disease, chronic fatigue syndrome, obesity etc. While these are not evidence of causation, they indicate potential downstream health impacts through changes in the microbiome.<sup>5</sup>

**Inflammatory, Oxidative, and Immune System Effects** Screen time has the potential to also affect inflammatory and oxidative stress markers and immune function through both direct and indirect means.

**Chronic inflammation:** Vandalized sleep, physical inactivity, metabolic derangement, and compromised gut permeability or microbiome dysbiosis may cause low-grade systemic inflammation. For example, research correlates excessive screen use (and related obesity) with increased inflammatory markers.<sup>6</sup>

**Oxidative stress:** While fewer human studies have quantified oxidative markers directly with screen exposure, some animal and in vitro evidence indicates that blue light and artificial light exposure can cause oxidative stress in retinal and neural tissues. This could contribute to cellular injury if exposure is prolonged. (More human molecular studies are needed.)

Immune function: Sleep deprivation, disrupted circadian rhythms, and metabolic stress may influence immune cell behavior. For example, melatonin has immuno-regulatory functions (antioxidant, anti-inflammatory), and therefore its suppression can compromise immune regulation. Dysbiosis (imbalance of the gut microbiome) may contribute to atypical immune responses, enhanced susceptibility to GI inflammation, etc.<sup>5</sup>

#### Mental Health, Mood, and Neurochemical Effects

Although purely psychological consequences lie outside the "biochemical" realm, most mental problems have biochemical causes, and screen time seems to modulate them.

**Anxiety, depression:** Screen time is associated with increased prevalence of anxiety and depressive symptoms in children and teenagers. That is partly mediated through sleep disturbance, but also through neurotransmitter imbalance, reward system desensitization, and possibly inflammatory/neuroimmune pathways.<sup>2</sup>

**ADHD/attention issues:** High levels of screen use, particularly interactive and high-speed content, have been associated with decreased executive function, attention control, impulsivity. This map to biochemical alterations in the brain (dopamine pathways, cortical connectivity).<sup>2</sup>

**Reward desensitization, addiction-like behavior:** The repeated use of rewarding content (social media, gaming) has the potential to repeatedly activate dopaminergic circuits, potentially resulting in down-regulation of sensitivity (tolerance). As more intense stimulation is required, mood will suffer when screen time is out of reach. This is comparable in some ways to substance addiction or other behavioral addictions.<sup>1</sup>

**Mechanisms of Biochemical Change:** Converging evidence, various mechanistic pathways are involved in how screen time produces these biochemical effects.

**1. Exposure to light (particularly blue light):** It result in Inhibition of melatonin; phase-shifts circadian and also modifies retinal and neural physiology (potentially oxidative stress).

**2. Disruption of sleep:** It can reduced sleep, poorer quality sleep results in reduced recovery, hormonal dysregulation (e.g., ghrelin/leptin

ratio, cortisol), abnormal glucose metabolism, and immune suppression.

**3. Sedentary displacement:** More sedentary behavior relates to lower expenditure of energy, worse mitochondrial function, and lower exercise benefits (which typically enhance metabolic, cardiovascular, and even neural function).

**4. Dietary correlates:** Screen time tends to be associated with snacking or ingestion of high-calorie, low-nutrient foods, which further stress the metabolism.

**5. Alteration of gut microbiome:** With changes in diet, activity changes, circadian disruption, etc., the gut microbiota population changes; metabolites from the microbiome affect immune, metabolic and neurological functions.

**6. Disturbance of neurotransmitter and enzyme pathway:** Abnormal amino acid availability/metabolism (e.g., phenylalanine, tyrosine) potentially alters neurotransmitter production (dopamine, norepinephrine, and serotonin).

Altered energy metabolism enzymatic changes (mitochondrial enzymes, etc.).

**7. Inflammation and oxidative stress:** Through sleep deprivation, metabolic dysregulation, gut barrier dysfunction, etc., resulting in systemic inflammation.

**Evidence from Recent Molecular/Cohort Studies:** Other recent studies provide more direct biochemical / molecular evidence, beyond correlation of association.

**Microbiome/Metabolome analysis in university students:** A cross-sectional comparison of ~60 university students with high screen time ( $\geq 75$  min/day) vs low screen time identified significantly different fecal microbiome taxa and metabolome profiles. Important findings included decreased taxa of beneficial bacteria, decreased amino acids (alanine, tyrosine etc.), were predictive of mitochondrial dysfunction and disrupted metabolic pathways. These were in line with obesity risk signatures, type I diabetes, inflammatory bowel disease, chronic fatigue syndrome.<sup>5</sup>

**Study:** Excessive screen time is toxic at molecular level: Besides behavioral/epidemiologic correlations, this study revealed internal metabolic process connections Type I

diabetes, obesity, chronic fatigue, inflammatory bowel disease etc.<sup>6</sup>

**Cardio-metabolic risk in young children/adolescents:** Danish cohorts found that more discretionary screen time is related to poorer cardio metabolic profiles (waist circumference, BP, lipids, glucose), with sleep duration/timing as mediator. That gives a mechanistic chain of screen time → sleep disruption → metabolic disturbance.<sup>7</sup>

**Brain structural and functional changes:** A number of studies indicate that excessive screen exposure is linked with worse inhibitory control as well as fronto-striatal connectivity and cortical thinning in adults. These are structural correlates and can potentially be indicative of reconfigured synaptic pruning, neural plasticity etc.<sup>4</sup>

**Health Consequences and Long-Term Effects:** The biochemical changes mentioned above can add up and result in significant health consequences if screen use is high for many years.

**Metabolic disorders:** Higher risk of obesity, insulin resistance, type II diabetes, dyslipidemia, metabolic syndrome.

**Cardiovascular disease:** Unfavorable cardio-metabolic profiles in children can set the stage for cardiovascular disease in adulthood. Higher blood pressure, worse lipid profiles, more visceral fat etc.

**Gastrointestinal/inflammatory disorders:** Dysbiosis and metabolomic changes could enhance risk of inflammatory bowel disease, gut permeability, maybe even autoimmunity in susceptible individuals.

**Cognitive decline:** If structural changes in the brain pile up (cortical thinning, decreased connectivity), possibilities include reduced executive function, memory, maybe vulnerability to neurodegenerative disease in later life.

**Mental health conditions:** Long-term sleep disruption, dysregulation of the reward system, inflammation et al. is associated with depression, anxiety, ADHD et al.

**Accelerated aging phenotypes:** It is hypothesized in some work that long-term screen-based leisure time activity is linked to biomarkers or phenotypes of biological aging.<sup>8</sup>

**Moderating and Mediating Factors:** The effects of screen time are not equal. There

are several modulators or mediators of the strength of the biochemical effects.

**Duration and timing:** Screens viewed later at night (particularly closer to bedtime) have more effect on suppression of melatonin, sleep disturbance, and secondary downstream effects. Total amount of screen time is also related in a dose-response fashion.<sup>2</sup>

**Type of screen experience:** Passive viewing (e.g., TV) versus interactive (social media, gaming) does count; content type (violent, stressful vs soothing) also counts.

**Age:** Kids and teens more vulnerable, as many systems (hormonal, brain, metabolic) are still in development. Adult brains more resilient but not immune.

**Physical activity:** Greater physical activity levels offset many of the metabolic risks. Screen time displacing exercise is a key issue.

**Sleep health:** The extent of sleep disturbance (short duration, poor quality, inconsistent sleep timing) is a central mediator for most of the downstream consequences.

**Diet and lifestyle:** Unhealthy diet, snacking, inconsistent meal timing, low physical activity, and sedentary posture enhance adverse outcomes.

**Genetic/inter-individual variability:** Genetic susceptibility to metabolic disorders, susceptibility to anxiety or mood disorder, difference in micro-biome baseline etc.

**Biochemical Pathways:** The following are more specific biochemical pathways that research indicates are impacted by screen time through the processes outlined above.

**1. Melatonin and Circadian Signaling:** Exposure to blue wavelengths of light (approx. 460-480 nm) causes activation of retinal ganglion cells (melanopsin pathway) → signal to the suprachiasmatic nucleus (SCN) → inhibition of melatonin synthesis in the pineal gland.

Outcome: Delayed onset of sleep, shift of circadian rhythms.

**2. Hypothalamic-pituitary-adrenal (HPA) axis:** Stressful or stimulating screen content stimulates sympathetic nervous system and HPA axis → rise in cortisol.

Elevated cortisol levels chronically may cause insulin resistance, changes in lipolysis, immune suppression.

**3. Neurotransmitters and Amino Acid Metabolism:** Tyrosine → Dopamine/Norepinephrine; Tryptophan → Serotonin. Alteration in precursor levels (e.g., decreased tyrosine, etc.) can decrease catecholamine synthesis.<sup>5</sup>

Disturbance in glutamate/glutamine system (observed in pathway analyses) could affect excitatory/inhibitory balance of brain.

**4. Mitochondrial Function/Energy Metabolism:** Enzyme enrichment analysis in microbiome/metabolome research reveals disrupted function of enzymes such as glutamate dehydrogenase, etc. These are related to energy generation (mitochondrial respiration, NAD/NADP cycles) and breakdown of amino acids. Deficiencies can result in decreased cellular energy and enhanced oxidative stress.<sup>5</sup>

**5. Inflammatory Pathways and Immune Signaling:** Altered gut microbiota may produce metabolites (short chain fatty acids, lipopolysaccharides) that regulate immune signaling. Dysbiosis can cause enhanced gut permeability ("leaky gut"), leakage of endotoxins, systemic activation of the immune system.

**6. Reactive Oxygen Species / Oxidative Damage:** Elevated metabolic rates, inefficient sleep repair, exposure to high-energy visible light, inflammation together because elevated reactive oxygen species (ROS) that damage lipids, proteins, DNA.

## CHALLENGES, LIMITATIONS, AND GAPS

Although there is large and growing evidence, some limitations and gaps need to be mentioned.

**Causality:** Most studies are cross-sectional. They report associations but cannot necessarily show that high screen time leads to the biochemical alterations. Reverse causation can occur (e.g., individuals with specific metabolic or mood disorders may use screens more).

**Measurement error:** Self-reported screen time are typical; objective measurement (software logs etc.) less typical. Self-report could under or over estimate actual exposure.

**Heterogeneity of screen content and use:** Screen use is diverse leisure vs work, passive vs interactive; content can be relaxing or stressful. These distinctions are important but not routinely disaggregated.

**Population biases:** Many molecular studies have small sample sizes, are in college students or limited geographic/ethnic populations. Less is known in very young children, older adults, or non-Western populations.

**Confounders:** Diet, socioeconomic factors, physical activity, sleep hygiene etc., may confound associations. Some studies try to control or statistically adjust; but residual confounding remains a concern.

**Lack of longitudinal molecular studies:** More prospective studies are needed to track biochemical markers over time with changes in screen time, to see what is reversible or persistent.

## IMPLICATIONS AND RECOMMENDATIONS

Given the evidence, there are implications for public health, clinical practice, and individual behavior.

**Guideline formation:** Better guidelines are needed for safe screen use, especially in children. Recommendations around timing (avoid screens before bedtime), duration (limiting unnecessary screen time), and content (calming, educational vs stimulating or stressful).

**Sleep hygiene:** Interventions that ensure sufficient sleep duration and quality may buffer many of the negative effects. Blue light filters, nighttime screen curfew, reducing exposure in the hour(s) before sleep.

**Physical activity promotion:** Encouraging regular exercise to offset metabolic and neural risks.

**Dietary moderation:** Reducing high-sugar, high-fat snacks during screen time, ensuring nutrient-rich diet to support neurotransmitter synthesis, gut health.

**Mindful screen use:** Encouraging awareness of how screen time feels, regulating content, balancing with non-screen time (nature, social interaction).

**Monitoring vulnerable populations:** Children, adolescents, people with metabolic risk, mood disorders. Clinicians might consider screen time as a factor when evaluating disorders of sleep, mood, metabolic health.

**Further research:** Longitudinal, larger, more diverse cohorts; objective measures; integration of molecular, neuroimaging, behavioral data to clarify causality, thresholds, reversibility.

## CONCLUSION

In summary, while screen time is an integral part of modern life, a growing body of evidence suggests that excessive or poorly timed screen exposure has measurable biochemical effects on human beings. These include hormonal disruptions (melatonin, cortisol), altered neurotransmitter metabolism, changes in brain structure and connectivity, metabolic dysfunction, gut microbiome alterations, increased inflammation and oxidative stress. Many of these effects are interlinked sleep disturbance often lies at the center, mediating downstream consequences. The long-term health risks are substantial, especially for young people whose biological systems are still developing.

To mitigate these risks, behavioral, policy, and technological actions are warranted limiting screen time, especially in the evening; promoting sleep health; encouraging physical activity; improving diet; and mindful screen usage. More research is needed to define safe thresholds, understand individual differences, and develop interventions.

## DECLARATIONS

**Funding:** This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

**Conflicts of Interest:** The authors declare no conflicts of interest regarding the publication of this manuscript.

**Ethical Declaration:** This article is a review based on previously published literature. No new studies involving human participants or animals were conducted by the authors.

Hence, ethical approval and informed consent were not required.

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