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Review Article

A Systematic Review on Health Disorders Due to Excessive Usage of Smartphones

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ABSTRACT

Smartphone use has become nearly ubiquitous worldwide, paralleling a rise in problematic and excessive usage patterns that pose emerging public health concerns. While behavioral and psychological consequences are well documented, the underlying pathophysiological mechanisms remain insufficiently integrated. This systematic review synthesizes current evidence on the biological pathways through which excessive smartphone use disrupts physiological homeostasis and contributes to multisystem health disorders. A systematic search of major scientific databases was conducted in accordance with Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines, identifying 63 studies that explicitly examined mechanistic links between smartphone use and health outcomes. Excessive smartphone use was consistently associated with neuropsychological alterations, including impaired executive control and reward-system dysregulation, supported by neuroimaging evidence of structural and functional brain changes. Circadian rhythm disruption emerged as a central mechanism, driven by nighttime screen exposure and cognitive hyperarousal, leading to melatonin suppression, hormonal imbalance, impaired glucose metabolism, and increased cardiometabolic risk. Ocular effects were mediated by reduced blink rate, tear film instability, accommodative stress, and blue light-induced oxidative damage. Additional outcomes included noise-induced cochlear injury, cumulative musculoskeletal strain, autonomic imbalance, inflammation, and systemic oxidative stress. Collectively, the evidence indicates that excessive smartphone usage functions as a multisystem physiological stressor that disrupts homeostatic regulation through interconnected neuroendocrine, circadian, oxidative, and autonomic pathways. Recognizing smartphone overuse as a modifiable public health exposure is essential for guiding prevention, clinical evaluation, and evidence-based intervention strategies.

Keywords: Excessive Smartphone use, Homeostasis, Neuroendocrine dysfunction, Oxidative stress, Pathophysiology, Public health.

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INTRODUCTION

Smartphones have become indispensable tools for communication, work, education, and social interaction. As of 2024, approximately 4.9 billion individuals worldwide use smartphones, representing nearly 60% of the global population, with more than 7 billion devices in active use due to multiple-device ownership. China remains the largest smartphone market, with close to one billion users.[1–3]

Parallel to this widespread adoption, problematic smartphone use-often referred to as smartphone addiction-has emerged as a growing public health concern. Excessive and compulsive smartphone engagement has been associated with disturbances in physiological regulation and an increased risk of multisystem health disorders.[1,4]

The concept of homeostasis, first articulated by Walter Bradford Cannon in 1929 as the dynamic maintenance of internal equilibrium, provides a useful framework for understanding these effects.[5] Persistent behavioural stressors, including excessive digital device use, can disrupt homeostatic control across interconnected biological systems. Increasing evidence links excessive smartphone use to neuropsychological, ocular, vestibulo-cochlear, musculoskeletal, endocrine, metabolic, and cardiovascular abnormalities.

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While the behavioral and psychological consequences of smartphone overuse are well documented, the underlying pathophysiological mechanisms remain insufficiently synthesized. This systematic review therefore aims to integrate current evidence on the biological pathways through which excessive smartphone use affects human health, with the goal of informing prevention strategies, clinical assessment, and public health interventions.

METHODOLOGY

A systematic literature search was conducted in Scopus, MEDLINE, Web of Science, PubMed, ProQuest Central, and PsycINFO using relevant Medical Subject Headings (MeSH) terms related to smartphone use, excessive usage, addiction, effects of electromagnetic radiations emitted by smartphones and physiological outcomes. Publications from database inception through December 2025 were eligible. The review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.

Studies were included if they examined health outcomes associated with smartphone use and explicitly described underlying pathophysiological mechanisms. Both human as well as animal studies were included. Following title and abstract screening, full-text assessment was performed. Extracted data included study design, population characteristics, exposure assessment, outcomes, mechanistic findings, and limitations.

RESULTS

Study Selection and Characteristics

The initial search identified 564 records. After duplicate removal and screening, 440 articles were excluded due to non-relevant outcomes, lack of mechanistic focus, or inadequate exposure definition. Sixty-three studies met inclusion criteria and were included in the qualitative synthesis.

Included studies comprised cross-sectional, case-control, cohort studies, and systematic reviews. Most involved adolescents and young adults, although several included middle-aged and occupational cohorts, and a few were animal studies. Smartphone exposure was assessed using self-reported screen time, validated addiction scales, or objective usage metrics. Reported outcomes spanned neuropsychological, ocular, vestibulo-cochlear, musculoskeletal, endocrine, metabolic, and cardiovascular domains (Figure 1 and Table 1).

Neuropsychological Outcomes

Excessive smartphone use was consistently associated with higher prevalence of depression, anxiety, attentional deficits, and impaired impulse control.[1,6–8] Neuroimaging studies demonstrated reduced cortical Gamma-*Aminobutyric Acid* (GABA) concentrations in regions involved in emotional regulation and inhibitory control.[9]

Structural MRI studies reported reduced gray matter volume in the orbitofrontal cortex, anterior cingulate cortex, and prefrontal regions.[10] Functional imaging revealed altered resting-state connectivity and hyperactivation of reward-related circuits, particularly within the striatum, suggesting enhanced reward sensitivity coupled with diminished executive control.[9–12] These patterns resemble neurobiological changes observed in behavioral addictions.

Sleep and Circadian Rhythm Disruption

Late-night and prolonged smartphone use was strongly associated with delayed sleep onset, reduced total sleep time, and impaired sleep quality.[13,14] Experimental and observational studies demonstrated suppression of nocturnal melatonin secretion following evening exposure to smartphone-emitted blue light, resulting

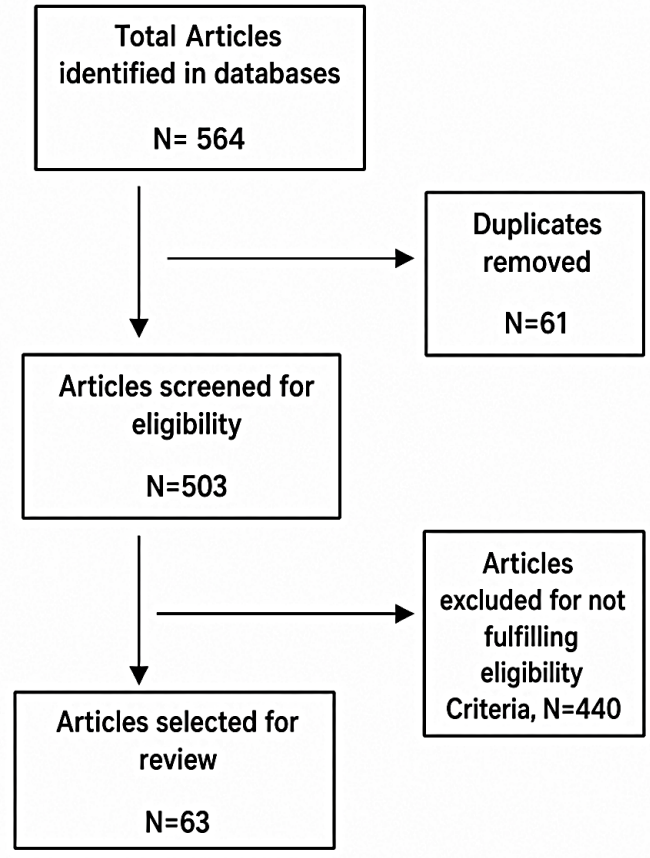

Figure 1: PRISMA flow chart of Search Strategy.

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Table 1: Summary of Pathophysiological Mechanisms Linking Excessive Smartphone Use to Multisystem Health Disorders.

Physiological System	Observed Health Outcomes	Key Pathophysiological Mechanisms	Level of evidence
Neuropsychological	Depression, anxiety, attentional deficits, impaired impulse control, addictive behaviors.	Reward-system dysregulation (striatal hyperactivation), reduced executive control (prefrontal cortex and ACC), decreased GABA concentrations, altered resting-state connectivity, maladaptive neuroplasticity resembling behavioral addiction.	Moderate
Sleep & Circadian System	Delayed sleep onset, reduced sleep duration, poor sleep quality, daytime fatigue, cognitive impairment.	Evening blue-light exposure suppresses melatonin; circadian phase delay; HPA-axis dysregulation; elevated evening cortisol; autonomic hyperarousal.	Moderate
Ocular (Visual System)	Digital eye strain, dry eye disease, blurred vision, transient myopia, asthenopia.	Reduced blink rate → tear film instability; tear hyperosmolarity; ocular surface inflammation (↑ IL-1, TNF-α, MMPs); accommodative stress; blue-light-induced retinal oxidative stress and mitochondrial dysfunction.	Moderate
Auditory System	Elevated hearing thresholds, tinnitus, sensorineural hearing loss.	High-volume earphone use (>100 dB) causing cochlear hair cell damage via mechanical stress, oxidative injury, metabolic exhaustion; maladaptive cortical neuroplasticity contributing to tinnitus.	Moderate
Vestibular System	Dizziness, imbalance, vestibular discomfort	Possible EMR-related changes in neuronal excitability, ionic balance, and vestibulo-ocular integration; visual-vestibular mismatch during prolonged screen use.	Limited
Musculoskeletal	Neck pain, shoulder pain, wrist/thumb disorders, De Quervain's tenosynovitis, carpal tunnel syndrome.	Forward head posture increases cervical load; sustained isometric contraction → ischemia; inflammatory mediator release (IL-6, TNF-α); repetitive microtrauma; tendon compression and nerve entrapment.	Moderate
Endocrine - Appetite Regulation	Increased hunger, weight gain, obesity risk	Circadian disruption alters leptin (↓) and ghrelin (↑); sleep deprivation impairs appetite regulation and energy balance	Moderate
Endocrine - Glucose Metabolism	*Insulin* resistance, impaired glucose tolerance, metabolic syndrome risk.	Circadian misalignment disrupts CLOCK/BMAL1 gene expression; reduced *insulin* sensitivity; altered glucose homeostasis; sedentary behaviour.	Limited
Endocrine - Stress Hormones	Elevated stress, burnout, mood disturbances	Chronic digital stimulation elevates evening cortisol; flattened diurnal cortisol rhythm; HPA-axis dysregulation.	Moderate
Endocrine - Reproductive Hormones	Reduced testosterone and semen quality (males); menstrual irregularities and reduced fecundity (females).	Circadian disruption of HPG axis; oxidative stress; altered gonadotropin and sex hormone secretion timing.	Limited
Endocrine - Thyroid	Altered TSH, T3, T4 levels (inconsistent findings).	EMR-induced oxidative stress; altered hypothalamic signaling; compensatory changes within the HPT axis.	Limited
Cardiovascular & Autonomic	Reduced heart rate variability, hypertension risk, endothelial dysfunction.	Sympathetic overactivation; parasympathetic withdrawal; oxidative stress reduces nitric oxide bioavailability; circadian disruption worsens vascular repair and lipid metabolism.	Moderate

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Table 1: (Continued)

Physiological System	Observed Health Outcomes	Key Pathophysiological Mechanisms	Level of evidence
Systemic Oxidative & Inflammatory Pathways	Low-grade inflammation, endothelial dysfunction, increased chronic disease risk.	Increased reactive oxygen species from sleep loss, psychological stress, sedentary behavior; activation of inflammatory cytokines; impaired antioxidant defenses.	Limited

in circadian phase delay and increased sleep latency. Chronic sleep disruption was associated with daytime fatigue, impaired cognitive performance, dysregulated cortisol rhythms, and increased cardiometabolic risk markers, positioning circadian misalignment as a central mechanistic pathway.[13–22]

Ocular Outcomes

Prolonged smartphone use was associated with digital eye strain, dry eye disease, blurred vision, and transient myopic shifts.[23–28] Reduced blink frequency during screen use destabilizes the tear film, leading to tear hyperosmolarity, ocular surface inflammation, and epithelial damage mediated by cytokines such as IL-1, Tumor Necrosis Factor-Alpha (TNF-α), and matrix metalloproteinases.[26,27] Sustained near-focus activity induces accommodative stress and ciliary muscle fatigue, contributing to asthenopia and transient myopia. Chronic near-work strain may promote axial elongation of the eyeball, increasing long-term myopia risk, particularly in younger populations.[23,24]

Smartphone-emitted blue light (400-490 nm) induces oxidative stress in retinal pigment epithelial cells, leading to mitochondrial dysfunction and apoptosis in experimental models. Although definitive evidence of permanent retinal injury in humans remains limited, biomarkers of oxidative damage are consistently elevated.[29,30]

Auditory and Vestibular Outcomes

Use of smartphone-connected earphones at high volume levels was associated with elevated hearing thresholds, tinnitus, and cochlear synaptopathy. Chronic exposure to sound levels exceeding 100 dB damages cochlear hair cells via mechanical stress, oxidative injury, and metabolic exhaustion, resulting in irreversible sensorineural hearing loss.[31–39]

Tinnitus appears to arise from maladaptive neuroplasticity within the auditory cortex following cochlear injury.[40,41] Evidence regarding vestibular dysfunction is limited and heterogeneous; proposed mechanisms include electromagnetic radiation-related alterations in ionic balance and neuronal excitability within vestibular pathways.[41–43]

Musculoskeletal Outcomes

Excessive smartphone use was consistently associated with cervical spine, shoulder, wrist, and thumb disorders.[44] Forward head posture increases cervical spine loading and static muscle contraction, reducing blood flow and triggering ischemic pain. This condition activates inflammatory mediators such as Interleukin-6 (IL-6) and Tumor Necrosis Factor-Alpha (TNF-α), perpetuating stiffness and myofascial discomfort. Prolonged strain may lead to fibrosis and restricted mobility.[44–49]

Repetitive thumb and finger movements during texting or gaming predispose users to De Quervain's tenosynovitis and carpal tunnel syndrome. Continuous flexion and extension increase tendon pressure and nerve compression, resulting in pain, tingling, and reduced grip strength. Small device size and awkward hand postures further aggravate ligament strain and microtrauma in the wrist and thumb joints.[50]

Endocrine and Metabolic Outcomes

Excessive smartphone use was associated with sedentary behavior, sleep disruption, and hormonal dysregulation

a. Alterations in leptin and ghrelin: Late-night screen use exposes individuals to blue light, which suppresses nocturnal melatonin secretion and delays circadian rhythms. This circadian misalignment and reduced sleep duration disrupt normal hormonal regulation, resulting in increased ghrelin secretion and decreased leptin levels. Because leptin typically rises at night to suppress appetite, its reduction alongside elevated ghrelin promotes increased hunger and potential overeating.[14,16,17,51]

b. *Insulin*: Meo *et al.* Demonstrated an association between long-term exposure to activated mobile phones and increase in fasting blood glucose and serum *insulin* in Albino rats.[52] Nighttime exposure to screen-based light has been shown to reduce *insulin* sensitivity and impair glucose tolerance in laboratory studies. At the systemic level, circadian misalignment impairs glucose homeostasis, alters the expression of core clock genes (e.g., CLOCK, BMAL1, PER, CRY), and promotes metabolic dysregulation, thereby increasing the risk of obesity and metabolic syndrome.[14,16,17,51–53]

c. Cortisol: Excessive exposure to stimulating digital media elevates evening cortisol and disrupts its normal diurnal rhythm, reflecting Hypothalamic-Pituitary-Adrenal (HPA) axis

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dysregulation associated with hyperarousal, sleep disturbance, and increased risk for mood disorders and burnout.[54]

d. Melatonin: A key circadian hormone, is suppressed by evening exposure to blue-enriched screen light, resulting in delayed secretion onset and altered timing.[55] Experimental and observational studies link this suppression to prolonged sleep onset latency, reductions in Rapid Eye Movement (REM) and deep Non-Rapid Eye Movement (NREM) sleep, and poorer overall sleep quality, with potential long-term consequences for circadian alignment and metabolic and mood regulation.[14–17,22,54,55]

e. Reproductive hormones: In males, circadian disruption has been associated with reduced circulating testosterone concentrations, impaired spermatogenesis, and decreased semen quality, likely mediated through dysregulation of the Hypothalamic-Pituitary-Gonadal (HPG) axis.[56–58] In females, circadian misalignment-particularly resulting from nocturnal light exposure and nighttime screen use-may disrupt the temporal regulation of estrogen and progesterone secretion, leading to menstrual cycle irregularities, altered ovulatory function, and reduced fecundity.[59]

f. Thyroid hormones: Electromagnetic Radiation (EMR) exposure may influence thyroid hormone homeostasis via central and peripheral pathways. Proposed mechanisms include altered hypothalamic neuroendocrine signaling through changes in neuronal excitability, calcium channel activity, and redox balance, as well as EMR-induced oxidative stress affecting thyroid follicular cell function and hormone synthesis.[60–62] Reported alterations in Thyroid-Stimulating Hormone (TSH), Triiodothyronine (T3), and Thyroxine (T4) in animal models are inconsistent and likely reflect compensatory adaptations within the hypothalamic-pituitary-thyroid axis rather than direct thyroid toxicity.[63]

Cardiovascular Outcomes

Excessive smartphone use is linked to cardiovascular disturbances mainly through sympathetic overactivation, oxidative stress, endothelial dysfunction, and impaired autonomic regulation.[64,65] Continuous digital stimulation activates the HPA axis, increasing cortisol and catecholamine release, which elevates heart rate, blood pressure, and the risk of hypertension and arrhythmias. Reduced heart rate variability, particularly before sleep, reflects diminished parasympathetic activity and increased cardiovascular strain. Chronic stress and possible electromagnetic radiation exposure increase oxidative stress, lowering nitric oxide availability and promoting endothelial dysfunction, arterial stiffness, and atherogenesis.[64–68] Additionally, late-night smartphone use disrupts sleep and circadian rhythms, enhancing sympathetic tone, impairing vascular repair, and worsening lipid metabolism, thereby increasing overall cardiometabolic risk.[12–21]

DISCUSSION

This review demonstrates that excessive smartphone use functions as a multisystem physiological stressor capable of disrupting homeostatic regulation across interconnected biological systems. Rather than isolated organ-specific effects, the evidence supports the presence of shared upstream mechanisms driving downstream pathology.

Excessive Smartphone Use as a Neurobehavioral Stressor

Collectively, neurobiological findings suggest that problematic smartphone use induces maladaptive neuroplastic changes resembling behavioral addictions. Altered inhibitory neurotransmission and executive-control circuitry impair self-regulation, reinforcing compulsive use and perpetuating a self-sustaining feedback loop. Framing smartphone overuse as a modifiable neurobehavioral risk factor has important clinical implications.[9–11]

Circadian Disruption as a Unifying Mechanism

Circadian misalignment emerges as a central integrative pathway linking smartphone use to endocrine, metabolic, and cardiovascular dysfunction. Evening light exposure and cognitive hyperarousal suppress melatonin, disrupt HPA axis regulation, and impair metabolic timing, amplifying vulnerability to chronic disease. Disrupted circadian regulation of leptin, ghrelin, *insulin*, cortisol, and reproductive hormones provides biological plausibility for observed cardiometabolic and reproductive effects.[12–21]

Oxidative Stress and Autonomic Imbalance

Oxidative stress represents a cross-system mechanism underlying ocular, metabolic, and cardiovascular outcomes.[28,29] Sleep deprivation, psychological stress, and sedentary behavior collectively increase reactive oxygen species and inflammatory signaling. Concurrently, chronic sympathetic dominance may predispose individuals to hypertension, endothelial dysfunction, and impaired stress resilience, particularly in younger populations.

Cumulative Load on Musculoskeletal and Sensory Systems

Musculoskeletal and sensory effects should be interpreted through the lens of cumulative load exposure. Repetitive microstrain, sustained postural deviation, and prolonged sensory stimulation parallel occupational overuse syndromes, underscoring the relevance of ergonomic and behavioural interventions.[43–49]

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Public Health and Clinical Implications

These findings support reframing excessive smartphone use as a population-level health exposure rather than an individual lifestyle choice. Clinicians should consider smartphone overuse when evaluating patients with sleep disturbances, anxiety, musculoskeletal pain, metabolic abnormalities, or early cardiovascular risk. Public health strategies should prioritize circadian protection, ergonomic education, and digital literacy to reduce cumulative physiological burden.

Limitations and Future Directions

Limitations include heterogeneity in exposure definitions, reliance on self-reported usage, and predominance of cross-sectional designs. Evidence regarding electromagnetic radiation remains inconclusive and requires cautious interpretation. Future longitudinal and experimental studies should establish dose-response thresholds, reversibility of physiological changes, and identification of vulnerable subgroups.

CONCLUSION

Excessive smartphone use disrupts multiple physiological systems through neuroendocrine imbalance, circadian dysregulation, oxidative stress, autonomic dysfunction, and inflammatory pathways. Addressing smartphone overuse through behavioral, ergonomic, and public health interventions is essential to mitigate its growing impact on global health.

ABBREVIATIONS

EMR: Electromagnetic Radiation; **GABA**: Gamma-*Aminobutyric Acid*; **HPA**: Hypothalamic-Pituitary-Adrenal; **HPG**: Hypothalamic-Pituitary-Gonadal; **HPT**: Hypothalamic-Pituitary-Thyroid; **IL-1/IL-6**: Interleukin-1/6; **MeSH**: Medical Subject Headings; **NREM**: Non-Rapid Eye Movement; **PRISMA**: Preferred Reporting Items for Systematic Reviews and Meta-Analyses; **REM**: Rapid Eye Movement; **T3**: Triiodothyronine; **T4**: Thyroxine; **TNF-α**: Tumor Necrosis Factor-Alpha; **TSH**: Thyroid-Stimulating Hormone;

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ETHICAL APPROVAL

As a systematic review, this study did not require ethical approval.

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