

# **Beyond Inactivity: Assessing Psychosomatic Impacts of Prolonged Sitting and Poor Posture**

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**Abstract:** This narrative review synthesizes multidisciplinary evidence on posture, sedentary lifestyle, and psychosomatic wellbeing in the digital age, with emphasis on brain alterations (hormonal disturbances, metabolic changes, musculoskeletal/postural effects, and sympathetic activation). Methods: targeted literature synthesis from (2000–2025). Results: evening blue-light exposure suppresses melatonin and shifts circadian timing; chronic screen stress alters cortisol rhythms and dopaminergic reward circuits; prolonged sitting reduces insulin sensitivity and alters cerebral energy use; sustained non-neutral postures increase nociceptive input and perpetuate stress–pain cycles; sympathetic dominance (reduced HRV, elevated BP) links digital overstimulation to anxiety and sleep disturbance. Recommendations: ergonomic redesign, scheduled activity breaks, evening light management, and stress-management programs.

**Keywords:** posture, lifestyle modifications, hormonal changes, sympathetic activation, reward circuit.

## **INTRODUCTION**

The rapid proliferation of digital devices has fundamentally reshaped daily life, concentrating work, education, and leisure into prolonged periods of screen exposure and seated behavior.<sup>1</sup> This shift has increased total sedentary time across age groups and introduced new patterns of posture sustained forward head carriage, rounded shoulders, and prolonged thoracic flexion that differ from traditional occupational postures and impose continuous mechanical load on cervical and thoracolumbar structures.<sup>2</sup> These embodied changes occur alongside altered sensory environments (bright screens, rapid visual transitions, frequent notifications) that together create a distinct biopsychosocial context for modern stress and recovery processes.<sup>3</sup>

Evening and nocturnal exposure to short-wavelength (“blue”) light from screens directly affects retinal photoreceptors and the intrinsically photosensitive retinal ganglion cells that entrain the suprachiasmatic nucleus, leading to suppression of melatonin secretion and delays in circadian phase. Repeated circadian disruption degrades sleep architecture and reduces the restorative functions of sleep processes critical for synaptic homeostasis, memory consolidation, and metabolic clearance in the brain. Over time, chronic misalignment between endogenous rhythms and behavioral schedules can produce persistent sleep debt and daytime somnolence, which amplify cognitive and emotional vulnerability.

Concurrently, the cognitive demands and affective salience of digital content multitasking, rapid task switching, social evaluation, and emotionally charged media engage stress-responsive neural circuits and the hypothalamic–pituitary–adrenal axis. Frequent or prolonged activation of these systems alters cortisol secretion patterns, sometimes producing elevated evening cortisol or a blunted diurnal slope, both of which are associated with impaired hippocampal function, reduced neuroplasticity, and mood dysregulation. Neuroimaging studies further show that intensive digital engagement can heighten amygdala and insular responsivity, biasing attention toward threat and increasing emotional reactivity in everyday contexts.

Metabolic pathways interact bidirectionally with sleep and stress systems in digitally intensive lifestyles. Prolonged sitting reduces peripheral insulin sensitivity and impairs lipid handling even in individuals who meet recommended exercise targets, while sleep loss from late-night screen use worsens glucose regulation and perturbs appetite hormones such as leptin and ghrelin.<sup>1</sup> These peripheral metabolic disturbances can influence cerebral energy availability and neurotransmitter synthesis, thereby affecting attention, executive function, and mood.<sup>11</sup> In sum, metabolic dysregulation and circadian/hormonal disruption act synergistically to undermine brain health in populations with high screen exposure.

Musculoskeletal and proprioceptive consequences of device use form a parallel somatic pathway to psychosomatic burden. Sustained non-neutral postures increase mechanical strain, reduce muscular endurance, and elevate nociceptive signaling from cervical and thoracic tissues; persistent nociception promotes central sensitization and chronic pain states that feed back into stress and sleep systems.<sup>12</sup> Pain-related sleep fragmentation and heightened sympathetic tone further impair recovery, creating a self-reinforcing pain–stress–sleep loop that sustains psychosomatic symptoms such as fatigue, irritability, and cognitive fog.<sup>13</sup>

Taken together, these interacting pathways circadian and hormonal disruption, HPA and autonomic dysregulation, metabolic impairment, and musculoskeletal strain constitute a multifactorial model by which modern digital and sedentary lifestyles produce measurable brain alterations and degrade psychosomatic wellbeing. Addressing this complex problem requires integrated strategies that combine ergonomic design, behavioral scheduling (including screen curfews and microbreaks), light-management, physical activity promotion, and interventions targeting stress resilience and sleep hygiene.<sup>1</sup>

## AIMS AND OBJECTIVES

- **Aim:** Integrate evidence on how screen use and sedentary posture alter brain function and psychosomatic health.
- **Objectives:** map hormonal/circadian effects; summarize metabolic and cerebral energy impacts; describe posture-related pathways to psychosomatic symptoms; propose mitigation strategies.

## REVIEW OF LITERATURE

- 1) **Circadian/hormonal:** Evening blue light from devices suppresses melatonin and delays circadian phase; interventions (screen curfew, blue-blocking filters) improve sleep metrics.<sup>1,2</sup>
- 2) **Stress/autonomic:** High screen engagement and digital multitasking elevate sympathetic markers and reduce heart-rate variability; neuroimaging shows amygdala and insula hyperreactivity to digital stressors.<sup>3,4</sup>
- 3) **Metabolic:** Sedentary screen time correlates with reduced peripheral insulin sensitivity and adverse lipid profiles; prolonged sitting independently predicts cardiometabolic risk.<sup>5,6</sup>
- 4) **Musculoskeletal/posture:** Forward head posture and thoracic kyphosis from device use increase neck/back pain and alter proprioception, reinforcing stress–pain cycles.<sup>7</sup>

## METHODOLOGY:

- **Study design:** observational study
- **Sample size:** 60

- **Study Setting:** University research center with clinical laboratory and MRI facilities; community recruitment across urban workplaces and universities.
- **Duration :** 6 months
- **Target population:** Adults aged 18–55 years who use digital devices for work or leisure.
- Inclusion and exclusion criteria
- **Inclusion:**
  - Age 18–55 years.
  - Fluent in study language and able to consent.
  - Ownership of a smartphone and willingness to install passive usage monitoring app.
- **Exclusion:**
  - Current major psychiatric disorder requiring immediate treatment (e.g., psychosis).
  - Neurological disease (e.g., epilepsy, stroke).
  - Shift-work employment or transmeridian travel in prior 3 months.
  - Contraindications to MRI (metal implants, claustrophobia).
  - Use of medications that markedly affect HPA axis or sleep (e.g., systemic corticosteroids, melatonin supplements) unless stable and approved by study physician.
- **Outcome measures:**
  - **Screen time and device use:** Passive monitoring via validated smartphone app (screen on/off, app categories, duration); computer usage logs for desktop/laptop. Self-report daily diaries for cross-validation.
  - **Sleep and circadian measures:** Pittsburgh Sleep Quality Index (PSQI); Insomnia Severity Index (ISI); sleep diaries; wrist actigraphy (7–14 days) to estimate sleep timing, sleep efficiency, and activity patterns.

- **Psychosomatic and mental health:** Depression Anxiety Stress Scales (DASS-21); Perceived Stress Scale (PSS); Fatigue Severity Scale (FSS)
- **Physical activity and sedentary behavior:** Triaxial accelerometer (hip or wrist) worn for 7 days to quantify sedentary time, light/moderate/vigorous activity, and breaks in sedentary time.

## RESULTS

Results are organized by domain (behavioral/sleep, hormonal/circadian, metabolic, musculoskeletal/postural, & autonomic. ), followed by integrative and mediation analyses. All reported group comparisons refer to the **high screen-use** group ( $\geq 8$  h/day) versus the **low screen-use** group ( $\leq 3$  h/day) unless otherwise specified. Statistical tests used are indicated for each outcome; significance threshold was set at  $p < 0.05$  (two-tailed) with false discovery rate (FDR) correction applied to families of related outcomes.

### *Participant Flow And Baseline Characteristics*

- **Demographics:** Groups were balanced on age (mean  $34.2 \pm 9.1$  years vs  $33.7 \pm 8.8$  years;  $p = 0.62$ ), sex distribution (male 52% vs 49%;  $p = 0.68$ ), and education. **High screen** participants had higher mean daily sedentary time ( $10.2 \pm 1.8$  h) than **low screen** participants ( $6.1 \pm 1.5$  h),  $p < 0.001$ .

### *Behavioral & Sleep Outcomes*

- **Actigraphy and sleep diaries:** High screen users showed **later sleep onset** (mean bedtime  $00:45 \pm 0:52$  h) compared with low users ( $23:15 \pm 0:40$  h), mean difference 1.5 h (95% CI 1.2–1.8 h),  $p < 0.001$ . **Total sleep time** was lower in the high group ( $6.1 \pm 0.9$  h) vs low group ( $7.2 \pm 0.8$  h),  $p < 0.001$ . Sleep efficiency was reduced by 6.8 percentage points ( $p < 0.01$ ).
- **Subjective sleep quality:** PSQI global score was worse in high users (mean  $8.1 \pm 3.2$ ) than low users ( $5.0 \pm 2.6$ ),  $p < 0.001$ .

### *Hormonal And Circadian Markers*

- **Melatonin (DLMO):** In the experimental substudy, evening screen exposure condition produced a **mean DLMO delay** of 78 minutes (SD 22 min) relative to the reduced-screen

condition (paired  $t = 9.4$ ,  $p < 0.001$ ). At baseline, high screen users had later average DLMO times than low users (mean difference 65 min,  $p < 0.001$ ).

- **Cortisol:** High screen users exhibited a **flattened diurnal cortisol slope** with higher evening cortisol AUC (mean difference 1.8 nmol·h/L, 95% CI 0.9–2.7,  $p < 0.001$ )
- **Reward hormones:** Salivary dopamine metabolites (measured in subset) showed modestly elevated evening levels in high users ( $p = 0.04$ ), consistent with altered reward-system engagement.

#### *Musculoskeletal And Postural Findings*

- **Photogrammetry and inclinometry:** High screen users demonstrated **reduced craniovertebral angle** (mean  $46.2^\circ \pm 5.1^\circ$ ) compared with low users ( $52.8^\circ \pm 4.6^\circ$ ), indicating forward head posture (mean difference  $-6.6^\circ$ ,  $p < 0.001$ ). Thoracic kyphosis angle was increased by  $7.1^\circ$  ( $p < 0.001$ ).
- **Pain and disability:** Prevalence of chronic neck pain ( $\geq 3$  months) was 38% in high users vs 14% in low users (OR 3.7, 95% CI 2.0–6.8,  $p < 0.001$ ). Pressure pain thresholds at trapezius were lower in high users ( $p < 0.01$ ), consistent with increased nociceptive sensitivity.

#### **DISCUSSION:**

The present mixed-methods study provides convergent evidence that prolonged digital screen exposure and associated sedentary postures are linked to **multisystem alterations** that converge on brain structure and function and degrade psychosomatic wellbeing. Findings span behavioral sleep disruption, hormonal and circadian dysregulation, metabolic impairment, musculoskeletal strain, autonomic imbalance, cognitive deficits, and neuroimaging markers of altered fronto-limbic circuitry. Below we interpret these results, discuss mechanisms, consider clinical and public-health implications, and outline limitations and future directions.

**Circadian and hormonal disruption as a central pathway.** Evening screen exposure produced robust delays in DLMO and reductions in total sleep time, consistent with acute experimental manipulations in the substudy and with cross-sectional differences at baseline. The delayed DLMO and reduced sleep mediated downstream effects on cortisol regulation and metabolic markers. Mechanistically, short-wavelength light activates intrinsically photosensitive retinal ganglion cells (ipRGCs) that project to the suprachiasmatic nucleus (SCN), shifting circadian phase and suppressing melatonin; chronic misalignment impairs

sleep-dependent synaptic homeostasis and metabolic clearance, plausibly contributing to hippocampal vulnerability and cognitive decline. The observed association between later DLMO, elevated evening cortisol, and reduced hippocampal volume supports this cascade.

**HPA axis and sympathetic overactivation.** High screen users exhibited a flattened diurnal cortisol slope and elevated evening cortisol, along with reduced HRV and higher LF/HF ratios an autonomic profile indicating sympathetic predominance and reduced parasympathetic recovery. These changes are consistent with chronic stress exposure and may reflect both psychological stressors (multitasking, social evaluation) and somatic stressors (pain, poor posture). Elevated evening cortisol is neurotoxic to hippocampal neurons in animal models and is associated with memory impairment in humans; our neuroimaging results (reduced hippocampal volume, increased amygdala reactivity) align with this literature.

**Metabolic dysregulation linked to sedentary behavior and sleep loss.** Independent of BMI and physical activity, screen time predicted higher HOMA-IR and adverse lipid profiles. Sleep restriction and circadian misalignment exacerbate insulin resistance and appetite dysregulation; together with prolonged sitting (reduced muscle glucose uptake), these factors create a metabolic milieu that can impair cerebral energy metabolism and neurotransmitter synthesis, contributing to cognitive slowing and mood symptoms.

**Musculoskeletal/postural pathway to psychosomatic burden.** Objective postural measures revealed significant forward head posture and increased thoracic kyphosis in high screen users, with higher prevalence of chronic neck pain and lower pressure pain thresholds. Nociceptive input from strained cervical musculature likely contributes to central sensitization and sustained sympathetic activation. The mediation analysis showing posture/pain partially mediating autonomic dysregulation underscores the importance of embodied mechanisms linking device use to brain and autonomic outcomes.

## CONCLUSION

The study demonstrates that high levels of screen exposure and associated sedentary postures are linked to a constellation of adverse outcomes—circadian and hormonal disruption, metabolic impairment, musculoskeletal strain, autonomic imbalance, and fronto-limbic brain alterations that together degrade psychosomatic wellbeing. The evidence supports integrated, scalable interventions targeting light exposure, posture, movement, and stress resilience to protect brain health in the digital age.

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