

The Effect of Universal Use of Electronic Media on Nerves and Circadian Rhythm: An Overview of the Mechanism of Autonomic Nerves and Neuroendocrine Disorders

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Abstract. The development of digital infrastructure has led to the creation of the heads-down generation. Growing up exposed to digital devices from an early age, these individuals engage in long-term frequent electronic media use, which profoundly reshapes the developmental trajectory of their nervous systems. These findings indicate that prolonged electronic media usage can lead to pathological physiological alterations, which are primarily characterised by dysautonomia, reduced sensitivity of the mesolimbic dopamine system and substantial delay in the circadian rhythm. Its main mechanisms are hyperactivation of the Ventral Tegmental Area (VTA) and Nucleus Accumbens (NAc), suppression of melatonin secretion via the intrinsically photosensitive Retinal Ganglion Cells (ipRGCs) pathway, and destruction of the inhibitory control mechanism of the prefrontal cortex. At the moment, the phenomenon of mental health problems caused by digitalization is becoming more and more common. To maintain human neurophysiological integrity, it is essential to establish a multi-tiered intervention system. Such strategies cover the application of blue light filtering techniques, formulation of healthy digital usage norms, and regular physical activity to improve executive functions.

Keywords: Autonomic Nervous System, Mesolimbic Reward Circuit, Circadian Rhythm, Melanopsin, Sleep Delay, Heart Rate Variability

1. Introduction

The Internet was initially developed in the 1960s with the aim of improving the efficiency of information exchange. Since smartphones became dominant in 2011, human society has officially entered the era of digital saturation. Media usage patterns have shifted: people now rely less on computers and instead use portable electronic devices anytime and anywhere, resulting in a state of constant online connectivity that continuously stimulates humans' innate desire for novel information [1, 2]. Currently, there is a large research gap in the scholarly literature concerning the neurobiological effects of these long-term digital exposures on neurodevelopment.

During human evolution, the ability to detect and respond to new environmental cues has been an innate survival instinct. This instinct is primarily used to avoid predation and acquire energy,

representing an evolutionary principle closely linked to survival. Contemporary digital algorithms are precisely exploiting this evolutionary feature, constantly presenting new content, to achieve sustainable activation of the human primitive biological reward system in artificially created environments [2]. Based on the central idea of the concept of the so-called evolutionary mismatch, this paper examines the inherent mechanisms by which exposure to electronic media causes organic and functional abnormalities of the autonomic nervous system, neuroendocrine system, and cognition, in a systematic way [2].

2. Nerve damage to the brain regulatory system

2.1. Autonomic nerve and heart rate variability disorder

The autonomic nervous system (ANS) maintains bodily physiological homeostasis through dynamic balance between the sympathetic nervous system (fight-or-flight response) and the parasympathetic nervous system (rest-and-digest response). The fact that internet addiction may drastically interfere with this balance has been shown by a research done by Sri Devaraj Urs Academy of Higher Education and Research (SDUAHER) [3].

The current study employs the Standard Deviation of NN Intervals (SDNN) and the Low-Frequency/High-Frequency (LF/HF) ratio as the primary indicators to evaluate autonomic nervous tone. The major results indicate that severe internet addicts exhibit significantly higher SDNN values than mild users, with a statistically significant difference. In spite of the fact that high SDNN is typically connected with good cardiovascular elasticity, in the situation of severe addiction, it denotes a disorder of autonomic nerve signal transmission and the inability of the body to maintain its stable regulation system [3].

Furthermore, the LF/HF ratio, a core marker reflecting sympathetic nervous system predominance, was remarkably higher among severe internet addicts than mild users, showing statistically significant differences. In contrast, high-frequency power (HF), which represents vagal and parasympathetic nerve activity, was markedly lower in the severe addiction group relative to the mild user group, also reaching statistical significance. This is a sign of reduced physiological resistance of the body to environmental stress and self-repair [3]. The subsequent sympathetic-parasympathetic dysfunction puts the body into a state of continuous excessive allostatic load, rendering people susceptible to both the risk of psychological discomfort and raised vulnerability to cardiovascular disorders.

2.2. Mesolimbic dopamine reward circuit: Ventral Tegmental Area—Nucleus Accumbens pathway

The core neurochemical mechanism underlying smartphone addiction lies in the mesolimbic reward pathway, an evolutionarily formed neural circuit that mainly reinforces survival-related behaviors such as feeding and reproduction. All types of notifications of electronic devices, social media likes and information updates constitute distinct rewards that can trigger the Ventral Tegmental Area (VTA) of the midbrain, which will subsequently cause the VTA to release dopamine into the Nucleus Accumbens (NAc) and create a short-term feeling of happiness [2, 4], and eventually develop a loop of behavior reinforcement between compulsive checking and an immediate reward.

Such dopaminergic signaling is not confined to the midbrain; instead, it manifests as complex interactions between glutamatergic and monoaminergic neurotransmitters within the amygdala and prefrontal cortex (PFC). The amygdala is used to process the emotional value of online social

feedback whereas the prefrontal cortex is engaged in the processes of executive decision-making and impulse control. In the case of pathological media consumption, chronic activation of this loop results in desensitization of dopamine receptors and decrease in their number - a phenomenon very closely related to substance addiction. According to studies conducted by Ianni et al. and Westbrook et al., the ability of striatal dopamine to synthesize has been directly linked to the amount of online social activity on smartphones, which indicates a clear relationship between digital behavioral phenotypes and the biochemical potential of the brains reward system [5, 6].

2.3. Changes in neuroplasticity and prefrontal cortex atrophy

With the shift between habitual media consumption and addiction, there occurs an adaptive shift in the structural neuroplasticity of the brain, the most common of which is thinning of the prefrontal cortex. Prefrontal cortex is the central hub of the so-called a top-down inhibitory control, which regulates the bottom-up impulsive wishes created by the reward circuit [2]. Longitudinal brain developmental data, including findings from the Adolescent Brain Cognitive Development (ABCD) Study, reveal that excessive social media browsing is associated with atypical development of the prefrontal cortex and its associated neural circuits [7].

Prefrontal cortical thinning impairs complex cognitive decision-making and impulse control abilities, thereby shifting individuals' self-regulation pattern from active self-restraint to passive loss of control. The breakdown of this prefrontal inhibitory system directly exacerbates behavioral disturbances associated with sleep deprivation. Deficient executive function fails to restrain the dopamine-driven urge to use digital devices before sleep [8]. The weakening of synaptic bonds reinforces the dependence cycle even more and with the gradual transformation of the organic structure of the brain, people become more inclined to place higher value on the instant gratification of the digital environment over the need to satisfy the biological requirements of the body through a long-term state of physiological homeostasis.

3. Sleep structure and circadian rhythm disorder

Excessive exposure to electronic media is a major cause of disrupted sleep architecture, as chronic sleep loss and fragmented restorative sleep cycles prevent people from sustaining adequate sleep duration and satisfactory sleep quality. Its most immediate effect is the suppression of the melatonin secretion, which is a fundamental hormone involved in the regulation of neuroendocrine sleep onset stimuli, causing a general delay of 2 to 3 hours in circadian rhythm [9], which seriously disrupts the sleep-wake cycle of the individual with regard to external light and social timing demands [2].

Sleep serves as a critical period when the brain's glymphatic system eliminates metabolic wastes such as beta-amyloid and other protein fragments, and this system reaches its peak activity during slow-wave sleep (SWS). Slow-Wave Sleep is very vulnerable to homeostatic sleep pressure, and the uninterrupted cognitive arousal caused by electronic media makes the person less aware of homeostatic sleep pressure, which causes a delay in sleep onset. The decrease of Slow-Wave Sleep duration does not only harm the ability of the brain to clear metabolic wastes but also causes excessive activation of amygdala emotional reactions on the next day, which results in emotional instability and cognitive dysfunction [2].

4. Intrinsic pathogenic mechanisms of physiology and behavior

4.1. Screen light: photosensitive Ganglion Cells—melanopsin—blue light pathway

Spectral emission characteristics are the core factor leading to the interference of electronic devices with human physiological responses. High-intensity blue light emitted by modern electronic screens has a peak wavelength of around 480 nm, which is right in the sensitive peak region of intrinsically photosensitive Retinal Ganglion Cells (ipRGCs) [9], which are located in the human eye. These are the cells that have melanopsin and are not involved in conventional visual imaging processes but are the primary receptors used by the human body in calibrating circadian rhythms.

By stimulating ipRGCs via the retinohypothalamic tract (RHT), 480-nm blue light transmits signals to the suprachiasmatic nucleus (SCN) in the hypothalamus, the central pacemaker governing the human circadian clock. The SCN then inhibits the pineal gland with an inhibitory signal, biochemically inhibiting the synthesis and release of melatonin. Night-time screen use delivers false daylight cues to the body, which does not allow it to enter a state of lower arousal during sleep, and also contributes to even further circadian rhythm delay and loss of deep sleep [2].

4.2. High neural arousal: Fear of Missing Out and amygdala activation

Besides the biological consequences of light, digital media content may keep the brain at a high level of neural excitement. A key trigger of psychological and behavioral responses is Fear of Missing Out (FoMO), which overexcites the amygdala—a central brain region involved in the "fight-or-flight" response [2] —by inducing excessive sensitivity and emotional arousal in individuals exposed to digital social stimuli.

Such heightened neural arousal manifests as restlessness and anxiety, which are entirely contrary to the tranquil mental state required to fall asleep. This mental disorder interferes with the ability of the brain to switch to the resting mode of the parasympathetic system [9]. The joint action of increased neural arousal, as well as the suppression of melatonin by blue light creates a kind of double whammy to seriously disrupt the natural process of sleep onset.

4.3. Two-step sleep deprivation model

The two-stage sleep deprivation mechanism proposed by Exelmans and van den Bulck clearly illustrates how electronic media invades people's sleep time [8]. The first stage is termed as the bedtime delay: individuals are compelled to push back their intended time of going to bed because of a dopamine-induced next page phenomenon that encourages them to surf through digital material without end [2]; the second stage is called sleep onset delay: once they have already lain down, the smartphone is still available at hand, which causes further exposure to media (and in many cases accompanied with rumination or compulsive social checking) [7].

The given two steps directly decrease total sleep time as well as the time accessible to the brain to conduct memory consolidation and the glymphatic metabolic clearance. TAs mentioned earlier, reduced prefrontal cortical volume has been confirmed as the organic pathological cause underlying impaired self-regulatory behaviors in the second stage.

4.4. Potential impact of electromagnetic radiation on sleep

Growing clinical attention has been paid to the potential impacts of radio frequency (RF) and electromagnetic radiation (EMR) on adolescents' developing nervous systems. Nervous systems of

digital native groups are at a fast-developing phase, and therefore they are highly susceptible to long-term cumulative exposure to RF-emitting gadgets. There have been longitudinal studies indicating that greater frequency of mobile phone use is related to changes in cognitive response time, which implies that the non-thermal effects of electromagnetic radiation have possible and significant effects on neurodevelopmental health [10, 11].

5. Scientific intervention and prevention strategies

To mitigate the adverse physiological impacts of electronic media exposure, researchers can establish a multidimensional intervention system integrating technical means, behavioral guidance and clinical intervention strategies. At the technical level, the main focus is on minimizing the light-induced impact of the Suprachiasmatic Nucleus. Reflective electronic paper technology, which uses reflected ambient lighting instead of active backlighting, has the potential to substantially decrease the likelihood of inhibiting melatonin [9]; blue light filtering features in software and hardware filters on phone screens are also effective in reducing the disruptive effects of 480 nm blue light on the ipRGCs-melanopsin pathway.

Behavioral interventions, also known as digital hygiene, serve as core measures to restore self-regulatory ability, which involves creating the mobile phone-free zone and applying the fifteen-minute duration of use with the inclusion of a posture reset [2]. The reset may relieve musculoskeletal problems related to head lowering and wrist extension and clinical evidence indicates that it may prevent symptoms in over 70 percent of regular users.

Exercise is one of the most effective biological intervention strategies. A synthesis of studies published in *Advances in Psychological Science* indicates that the ancient focus regulation mechanism developed through human brain evolution can only be triggered by physical exercise, including hunting or fleeing threats, a stimulus which is lacking in the current sedentary way of life [9]. High-intensity physical training outperforms ordinary walking in alleviating anxiety and boosting cognitive focus.

Mindfulness intervention may also assist in supporting the aforementioned measures by training the brain to regulate its attention, recognize impulsive urges without acting on them, strengthen the functional connection between the prefrontal cortex and the amygdala, and improve the ability to switch between explicit regulation and implicit self-regulation. Digital addiction screening needs to become a regular practice at university mental health centers, and its promotion as a part of the above multidimensional approach to prevent and control addiction needs to be systematic [2].

6. Conclusion

This paper reviews the effects of widespread electronic media use on the nervous system and circadian rhythms based on relevant literature. Mechanistic evaluation of multi-dimensional physiological signals such as heart rate variability, dopaminergic neural circuits, prefrontal cortex structure and melatonin secretion indicate that the use of electronic media is significantly harmful to the neurophysiological system of humans. Imbalance of the autonomic nervous system indicates that the body is subjected to chronic excessive allostatic load; desensitization of the mesolimbic reward circuit and atrophy of the prefrontal cortex lead to the loss of inhibitory control function; inhibition of the ipRGCs-melanopsin-blue light pathway induces circadian rhythm disorders and sleep deprivation, which in turn interfere with cerebral glymphatic metabolism and memory consolidation.

The focus on future research should be more on longitudinal brain imaging investigations as they can help explain the causality between particular digital behavioral phenotype and organic brain

injury. Given the high degree of integration of digital technology into people's daily lives, implementing proactive prevention and control measures to safeguard the neurophysiological integrity of current and future generations has become a major healthcare concern.

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