psychiatry international

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Systematic Review

Hyperconnected Minds: A Systematic Review of the Neurobiology of Problematic Internet Use

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Abstract

The rapid expansion of digital technologies has created an unprecedented level of increased connectivity, yet the neurobiological mechanisms underlying these changes remain incompletely understood. This systematic review synthesizes the currently available evidence on neurobiological findings related to problematic internet use, problematic smartphone/social media use, and internet gaming disorder, with a focus on reward processing, attentional control, and emotional regulation. Across the included studies, recurrent findings suggested the involvement of fronto-striatal circuitry, salience- and reward-related regions, and executive-control networks. Alterations in large-scale brain networks, including the default mode, salience, and frontoparietal systems, were also reported, although the evidence base was heterogeneous and consisted largely of cross-sectional studies with modest sample sizes. Overall, the findings support a provisional neurobiological framework linking reward-related processing, inhibitory-control difficulties, and large-scale network alterations, but better-standardized studies are needed to draw conclusions.

Keywords: problematic internet use; neurobiology; review

1. Introduction

Digital technologies have fundamentally transformed modern life, enabling continuous access to virtual environments, social interaction, and entertainment [1–3]. While these advances have generated substantial societal and economic benefits, they have also generated new behavioral patterns, characterized by excessive and dysregulated engagement with digital platforms [1,4,5]. Problematic internet use (PIU), problematic smartphone use (PSU), problematic social media use and internet gaming disorder (IGD) have emerged as clinically and socially relevant phenomena, associated with functional impairment, reduced well-being, and increased psychiatric comorbidities [1,3,5]. Although no consensus regarding terminology, diagnostic criteria, or treatment guidelines has yet been reached apart from internet gaming disorder (IGD), these conditions are increasingly conceptualized as behavioral addictions, sharing phenomenological and neurocognitive features with substance-related and other behavioral addictions, including craving, tolerance, withdrawal-like symptoms, and escalating engagement over time [6–8].

Current research on digital behaviors has consistently linked these phenomena to impulsivity, aggression, and emotional dysregulation, as well as to higher levels of anxiety,

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Academic Editor: Roger C.M. Ho

Received: 15 February 2026
Revised: 24 April 2026
Accepted: 27 April 2026
Published: 13 May 2026

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depression, and stress [4,9,10]. For instance, anxiety symptom severity has been shown to correlate with problematic smartphone use, suggesting that digital engagement may serve both as a coping mechanism and as a factor maintaining anxiety symptoms [4,10,11]. Furthermore, several experimental studies indicate that deprivation of smartphone access can induce measurable stress responses and heightened social threat sensitivity, a phenomenon referred to as "nomophobia" [12–14]. Taken together, these findings suggest that problematic digital behaviors may represent an emerging public health issue and underscore the need to better understand their underlying mechanisms [15–17].

Neuroimaging research has attempted to establish the neural correlates of excessive digital use and its effects on brain function and structure [3,4,11,13]. Structural MRI studies have reported gray matter volume abnormalities in individuals with problematic internet use, particularly in regions implicated in reward processing and inhibitory control [10–12]. Alterations have been observed in the orbitofrontal cortex, striatum, and related corticostriatal circuits, suggesting dysregulation of neural systems involved in reward valuation, habit formation, and impulse control [1,3,10–12]. White matter integrity changes have also been described in young smartphone users, indicating potential microstructural alterations in long-range connectivity pathways [17]. Collectively, these structural findings suggest that chronic digital overuse may be associated with neuroplastic changes in brain systems governing self-regulation and motivational salience [18–20].

Functional neuroimaging studies, including task-based fMRI, have reported altered recruitment of prefrontal control regions and heightened responsivity of limbic and striatal areas during reward processing, decision-making, and inhibitory-control tasks [2,10,11,14,20]. These patterns resemble those described in pathological gambling and substance use disorders, supporting the hypothesis of shared neurobiological substrates across addictive behaviors [15]. Moreover, resting-state fMRI studies have shown disrupted intrinsic connectivity within the default mode network, salience network, and frontoparietal executive control network in problematic internet use, suggesting large-scale network dysregulation [8]. Such alterations may compromise sustained attention, cognitive flexibility, and emotion regulation [17,21].

Electrophysiological studies provide additional evidence that gaming-related and smartphone-related cues elicit exaggerated neural responses in reward- and salience-related regions, including the striatum, amygdala, and insula [6,19]. These findings are consistent with incentive sensitization models, according to which repeated exposure to salient digital stimuli amplifies their motivational value and biases attentional resources toward addiction-relevant cues [4,17]. Problematic internet use has also been associated with greater impulsivity and impaired decision-making, further implicating fronto-limbic circuitry in the maintenance of compulsive digital behaviors [13,14].

Despite these findings, the existing research remains divided and methodologically heterogeneous. Studies vary widely in terminology, diagnostic criteria, psychometric instruments, imaging modalities, and analytic strategies, complicating direct comparison and synthesis of findings [16,18,21]. Moreover, most neurobiological studies have focused on internet gaming disorder, with comparatively fewer investigations addressing problematic smartphone and social media use, despite their increasing prevalence and public health impact [9,22]. As a result, a coherent integrative framework capturing how problematic digital behaviors relate to brain function and structure is still lacking.

The neurobiological literature on problematic digital behaviors remains challenging to integrate because of substantial variation across studies. Included investigations differ not only in behavioral phenotype, but also in age group, diagnostic or screening criteria, imaging modality, analytic pipeline, and comparison strategy. Some studies focus on internet gaming disorder, others on problematic smartphone use, generalized problematic

internet use, or social media-related behaviors; similarly, reported outcomes range from structural alterations and white-matter indices to resting-state connectivity, task-based activation, and cue-reactivity effects. As a result, the field has grown rapidly but unevenly, and its findings are often interpreted in parallel rather than synthesized within a broader conceptual framework.

Despite this heterogeneity, an integrative review remains valuable because recurring patterns may still emerge across modalities and phenotypes. In particular, several strands of research converge on neural systems involved in salience attribution, reward processing, executive control, attentional regulation, and emotion-related processing. Even where findings are not fully consistent, identifying areas of convergence and divergence may help clarify which observations appear robust, which remain phenotype-specific, and where future studies should adopt more harmonized conceptual and methodological approaches. A careful synthesis is therefore needed not only to summarize current findings, but also to refine the interpretive boundaries of the field.

This review aims to synthesize current knowledge on the neurobiological correlates of problematic digital behaviors, with a particular focus on alterations in reward circuitry, executive control networks, and large-scale brain systems involved in attentional and emotional regulation. By integrating findings from neuroimaging, electrophysiology, and related biological research, this review aims to provide a structured overview of the current evidence and to support the development of more refined neurobiological models in this field.

2. Materials and Methods

2.1. Study Design

A systematic review was conducted in accordance with PRISMA 2020 recommendations to identify studies exploring the neurobiology of problematic internet use, problematic smartphone/social media use, and internet gaming disorder in adults [23]. Initially, two reviewers independently screened the titles and abstracts of all identified studies. Any discrepancies between the reviewers were resolved through discussion and consensus. The protocol was registered in PROSPERO (ID: CRD420261292598).

A structured literature search was performed in PubMed, Scopus, and the Cochrane Library. Google Scholar was used as a supplementary source to identify potentially relevant records not captured by the primary databases. Web of Science, Embase, and PsycINFO were not accessible through the institutional subscription at the time of the search. To ensure comprehensive coverage, we included Scopus as a broad multidisciplinary database and conducted supplementary searching via Google Scholar. No additional eligible original neurobiological studies were identified through Google Scholar beyond those already retrieved from the primary databases. The search covered the period from 1 January 2015 to 31 January 2025, aiming to capture the most recent decade of relevant literature. Search terms combined concepts related to problematic digital behaviors and neurobiological outcomes, including problematic internet use, problematic smartphone use, problematic social media use, internet gaming disorder, neuroimaging, EEG, biomarkers, reward processing, attentional control, emotional regulation, and adults. Full database-specific Boolean search strategies are provided in Supplementary Material S2.

2.2. Inclusion Criteria

Eligible studies met the following criteria:

1. Original research involving human adult participants.
2. Studies examining problematic internet use, problematic smartphone use, problematic social media use, or internet gaming disorder as the primary phenomenon of interest.

3. Studies using neurobiological methods, including structural MRI, functional MRI, diffusion imaging, EEG, PET, or biological markers.
4. Articles published in English.
5. Articles published between 1 January 2015 and 31 January 2025.

Exclusion criteria:

1. Review articles, systematic reviews, meta-analyses, narrative reviews, editorials, commentaries, letters to the editor, conference abstracts, and opinion papers.
2. Studies conducted exclusively in pediatric or adolescent populations.
3. Animal studies or in vitro research.
4. Studies not involving problematic internet use, problematic smartphone use, problematic social media use, or internet gaming disorder as the primary phenomenon of interest.
5. Studies lacking neurobiological outcomes (e.g., purely behavioral, psychometric, or epidemiological studies without neuroimaging, electrophysiological, or biological measures).
6. Methodological or computational studies using neurobiological data solely for classification or prediction purposes (e.g., machine learning or artificial intelligence approaches) without mechanistic neurobiological interpretation.
7. Case reports or case series with insufficient methodological rigor or sample size.
8. Studies published outside the predefined time window (before 1 January 2015 or after 31 January 2025).
9. Studies not published in English.
10. Studies for which full-text articles were not accessible or did not provide sufficient methodological detail to assess eligibility.

2.3. Study Selection

Following the database searches, records were screened for duplicates by comparing titles, authors, publication year, and journal details across sources. Two reviewers independently screened titles and abstracts for relevance. Full texts of potentially eligible studies were then assessed independently using the predefined inclusion and exclusion criteria, and disagreements were resolved through discussion and consensus. Extracted data included study design, sample characteristics, problematic digital behavior phenotype, neurobiological modality, brain regions or systems implicated, and the main findings related to reward processing, inhibitory control, emotional regulation, and large-scale brain networks.

2.4. Quality Assessment and Risk of Bias

Because the included literature comprised mainly observational neuroimaging and electrophysiological studies, methodological quality was assessed using a structured narrative appraisal based on study design. Cross-sectional studies were evaluated with attention to sampling strategy, clarity of phenotype definition, ascertainment of neurobiological outcomes, management of confounding, and transparency of statistical reporting. Randomized studies were considered in relation to allocation procedures, outcome assessment, and interpretability of intervention effects. Overall, the evidence base was judged to be limited by predominantly cross-sectional designs, modest sample sizes, heterogeneous phenotype definitions, and variability in imaging paradigms and analytic strategies. These factors were taken into account when interpreting the strength and generalizability of the findings. Given the methodological heterogeneity of the included neuroimaging studies and the absence of a universally adopted risk-of-bias tool specifically designed for this type of literature, study quality was assessed using a structured, review-specific framework

adapted to the aims of the present synthesis. The appraisal considered the following domains: (1) clarity of participant characterization and phenotype definition; (2) adequacy of control/comparison group selection, where applicable; (3) sample size and potential limitations related to statistical power; (4) transparency of neuroimaging acquisition and analytic procedures; (5) control of relevant confounding variables, such as age, sex, psychiatric comorbidity, and substance use; and (6) completeness of reporting and interpretability of the findings. These domains were used to guide a narrative evaluation of methodological strengths and limitations across studies rather than to derive a single summary score.

3. Results

The systematic literature search was conducted in PubMed, Scopus, and the Cochrane Library. Google Scholar was additionally used as a supplementary source to identify potentially relevant records that may not have been captured by the primary databases.

The Cochrane Library search yielded four records. After title and abstract screening, two were excluded: one because it was published outside the predefined time window (in 2026) and one because it focused exclusively on adolescent populations. The remaining two records were retained for full-text assessment, both addressing neurobiological interventions in internet gaming disorder.

The PubMed search identified 288 records. After title and abstract screening, 271 were excluded because they were not relevant to the research question, did not report neurobiological outcomes, involved non-problematic digital use populations, or did not meet other predefined eligibility criteria. Seventeen records were retained for full-text assessment.

The Scopus search yielded 159 records. Following title and abstract screening, 152 were excluded based on the predefined eligibility criteria, mainly because of limited relevance to problematic digital behaviors, lack of neurobiological outcomes, or focus on unrelated psychiatric or technological topics. Seven records were retained for full-text assessment.

The Google Scholar search returned approximately 4580 records. The first 200 results, sorted by relevance, were screened. All potentially relevant records identified through Google Scholar were review articles or opinion papers and therefore did not meet the inclusion criteria for original neurobiological studies. As a result, no additional eligible studies were identified from this source.

In total, 26 records were retained for full-text assessment: 2 from the Cochrane Library, 17 from PubMed, and 7 from Scopus. After full-text evaluation, 12 records were excluded, including 9 review articles, 2 studies with insufficiently interpretable neurobiological findings, and 1 duplicate. This left 14 studies eligible for inclusion in the qualitative synthesis.

A PRISMA flow diagram summarizing the identification, screening, eligibility, and inclusion process is presented in Figure 1.

The 14 included studies were methodologically heterogeneous and comprised predominantly cross-sectional designs, with only two randomized intervention trials. The evidence base was weighted toward internet gaming disorder and problematic internet use phenotypes, whereas problematic smartphone and social media use were represented by comparatively fewer studies. Across the included literature, neurobiological assessment methods included structural MRI, task-based fMRI, resting-state fMRI, diffusion MRI, EEG-based paradigms, and biomodulatory intervention designs. All included studies involved adult samples, although the age composition of the samples was not reported uniformly across studies. A structured summary of study characteristics and main findings is presented in Table 1 and Supplementary Material S1.

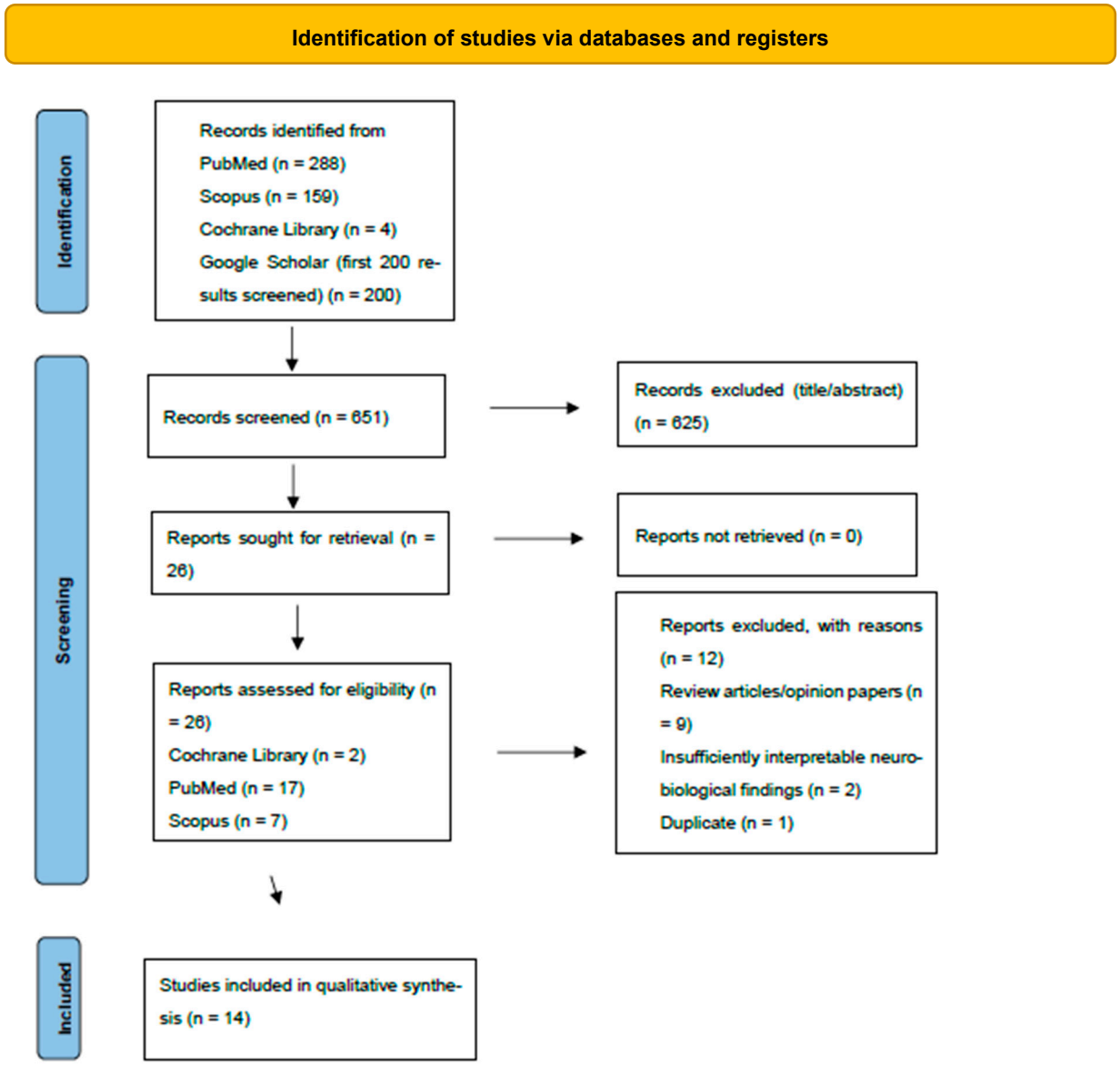


Figure 1. PRISMA 2020 flow diagram of study selection.

Table 1. Main findings of the selected studies.

No.	Study	Design	Sample (*n*)	Population	Modality	Brain Regions	Main Findings
1	[24]	Randomized, single-blind, placebo-controlled trial	55	IGD	fMRI (cue reactivity)	Paracentral lobule, precuneus, insula	IGD showed bias toward gaming cues and increased neural synchronization in paracentral lobule, precuneus, and insula during cue exposure
2	[25]	Randomized, double-blind trial	46	IGD	EEG (P300) + tDCS	Parieto-occipital cortex (Pz)	P300 (Pz) discriminated IGD vs. HC and correlated with craving; cathodal tDCS during cue exposure reduced craving, gaming time, and P300 alpha, with improved decision-making
3	[26]	Cross-sectional	51	IGD	EEG–fNIRS (Stroop)	Left frontal cortex	IGD showed reduced hemodynamic activity and ERP amplitudes with prolonged latency; nonlinear EEG features (left frontal) differentiated IGD despite no behavioral differences

Table 1. *Cont.*

No.	Study	Design	Sample (*n*)	Population	Modality	Brain Regions	Main Findings
4	[27]	Cross-sectional	82	PIU	Task fMRI (PVT; fatigue)	Left precuneus; left medial, middle, and superior frontal gyri	PIU severity predicted subjective mental fatigue; task-induced BOLD changes in precuneus and frontal gyri correlated negatively with PIU and fatigue change
5	[28]	Cross-sectional	59	Problematic smart-phone & social media use	Resting-state fMRI	Language, visual, auditory, sensorimotor, executive, frontoparietal networks	Severity of problematic smartphone and social media use was negatively associated with within-network functional connectivity across multiple networks, with lower connectivity in executive and frontoparietal networks at higher symptom severity.
6	[29]	Cross-sectional	65	PIU & excessive smart-phone use	Task fMRI (EFE)	Amygdala connectivity with PCG, ACG, SMG, MTG, frontal pole	PIUQ obsession associated with increased amygdala FC to cognitive control and social cognition regions during emotional face processing
7	[30]	Cross-sectional	42	Problematic smart-phone & social media use	Resting-state fMRI	Attention (frontoparietal/ cingulo-opercular), visual, language networks	Increased static FC in attention networks and reduced temporal variability in dynamic networks; decreased FC in visual/language and executive/frontoparietal networks
8	[31]	Cross-sectional	65	IGD	fMRI (cue-craving)	Precuneus; hippocampal and middle frontal gyrus connectivity	IGD severity correlated with precuneus activation and volume, increased hippocampal → precuneus FC, and reduced middle frontal → precuneus FC
9	[32]	Cross-sectional	144	IA (women)	Structural MRI (VBM)	Right pars opercularis (IFG)	Increased gray matter mass and volume in the right pars opercularis were associated with IA severity, implicating regions involved in inhibitory control and social cognition.
10	[33]	Cross-sectional	60	PIU	Task fMRI (Stroop)	DMN (precuneus, PCC); ICN (left IFG, frontal opercular, insula)	DMN deactivations negatively correlated with PIUQ; ICN activity positively correlated with PIUQ during incongruent trials
11	[34]	Cross-sectional (exploratory)	51	Internet addiction subgroups	Task fMRI (Emotional Stroop)	Left MTG, left STG	Gaming addicts showed reduced MTG/STG activation during social-word interference, suggesting altered social semantic processing
12	[35]	Cross-sectional	82 (females)	Subclinical PIU	Structural MRI (volumetry)	Putamen, nucleus accumbens, orbitofrontal cortex	Putamen volume correlated with control disorder; nucleus accumbens volume correlated with obsession; reward-system structural correlates of PIU tendencies
13	[36]	Cross-sectional	19	IGD	Task fMRI (gaming stimuli)	SPL, insula, precuneus, cingulate gyrus, STG, brainstem	IGD showed increased activation in attention, salience, and visuospatial regions and left brainstem; frontal hypofunction and right-hemisphere laterality
14	[37]	Cross-sectional	42	IGD	Diffusion MRI (FA)	ATR, ALIC, CST, IFOF, corpus callosum, ILF	IGD associated with increased FA in multiple tracts involved in reward integration and sensory–motor coordination

Abbreviations: IGD = Internet gaming disorder; PIU = Problematic internet use; IA = Internet addiction; PIUQ = Problematic Internet Use Questionnaire; rs-fMRI = resting-state functional MRI; DMN = Default mode network; ICN = Inhibitory control network; IFG = inferior frontal gyrus; ACG = anterior cingulate gyrus; PCG = posterior cingulate gyrus; SMG = supramarginal gyrus; MTG = middle temporal gyrus; STG = superior temporal gyrus; SPL = superior parietal lobule; PCC = posterior cingulate cortex; FA = fractional anisotropy; ATR = anterior thalamic radiation; ALIC = anterior limb of internal capsule; CST = corticospinal tract; IFOF = inferior fronto-occipital fasciculus; ILF = inferior longitudinal fasciculus; EFE = emotional face expression; PVT = psychomotor vigilance task.

4. Discussion

This systematic review synthesized neurobiological evidence from fourteen original studies examining problematic internet use, problematic smartphone use, and internet gaming disorder. Across multimodal neuroimaging and electrophysiological approaches, recurrent findings involved fronto-striatal circuits, large-scale intrinsic brain networks, and salience- and reward-related systems. However, the evidence base was methodologically heterogeneous, relied predominantly on cross-sectional designs, and provided the strongest support for internet gaming disorder, with comparatively fewer studies addressing problematic smartphone and social media use.

An important interpretive limitation concerns the heterogeneity of the behavioral phenotypes included under the broader umbrella of problematic digital use. Although synthesizing findings across problematic internet use, internet gaming disorder, problematic smartphone use, and problematic social media use may help identify convergent neurobiological patterns, such pooling may also obscure potentially distinct underlying mechanisms. For example, cue-reactivity and reward-related processes may be more prominent in gaming-related phenotypes, whereas social salience, reassurance-seeking, and compulsive checking may be more relevant in social media- or smartphone-related behaviors. Accordingly, the present synthesis should be interpreted as highlighting shared tendencies across related phenotypes, while acknowledging that these categories may not be neurobiologically equivalent.

4.1. Fronto-Striatal Dysregulation and Impaired Inhibitory Control

A consistent pattern across included neuroimaging studies was the involvement of prefrontal and striatal regions implicated in inhibitory control and reward valuation. Structural MRI findings suggested gray matter alterations in components of the reward system, including the putamen, nucleus accumbens, and orbitofrontal cortex, which were associated with control disorder and obsession dimensions of problematic internet use [25,32]. Structural and diffusion-based findings should also be interpreted with caution. Alterations in gray matter volume, cortical thickness, or white-matter microstructure cannot be assumed to reflect straightforward pathological change. Depending on study design, developmental context, and analytic approach, such findings may reflect vulnerability markers, experience-dependent neuroadaptation, compensatory processes, or nonspecific correlates with uncertain functional significance. This interpretive ambiguity is particularly important in a literature dominated by cross-sectional designs, where the temporal and mechanistic meaning of observed brain differences remains difficult to establish.

Functional MRI studies further revealed altered activation and connectivity within prefrontal–striatal loops during inhibitory control and cue-craving paradigms, with particular involvement of the precuneus, inferior frontal gyrus, and middle frontal gyrus [31,33]. Taken together, these findings are consistent with impaired top-down control and altered reward-related processing, although they do not establish causality.

4.2. Cue Reactivity, Salience Attribution, and Reward Sensitivity

Several of the included studies highlighted exaggerated neural responses to gaming-related or emotionally salient cues, particularly within regions associated with salience detection and reward processing. Increased synchronization and activation in the insula, paracentral lobule, and precuneus during gaming cue exposure were observed in individuals with internet gaming disorder [24,37]. These findings align with incentive sensitization models of addiction, according to which repeated exposure to highly salient stimuli enhances their motivational value and biases attentional resources toward addiction-relevant

cues [38,39]. Electrophysiological evidence pointed in a similar direction, with cue-related P300 alterations being associated with craving and symptom severity [24,34,37].

4.3. Large-Scale Network Alterations and Attentional Control

Resting-state fMRI studies suggested evidence towards dysregulation of intrinsic brain networks in problematic smartphone and social media use. Decreased within-network functional connectivity was observed in executive control, frontoparietal, visual, language, and sensorimotor networks, while increased static connectivity and reduced temporal variability were noted in attention-related networks [34,35]. Such large-scale network alterations suggest a reconfiguration of neural systems supporting sustained attention, cognitive flexibility, and internal mentation. Altered default mode and frontoparietal dynamics observed during cognitive control and fatigue-induction paradigms further support this interpretation [29,31].

4.4. White Matter Integrity

Evidence from a diffusion MRI study indicated altered white-matter integrity across multiple tracts involved in reward integration and sensory–motor coordination, including the anterior thalamic radiation, corpus callosum, and inferior longitudinal fasciculus [33].

Taken together, the findings synthesized in this review suggest a provisional integrative framework involving three recurrent domains: reward-related cue reactivity, inhibitory-control and executive-regulation difficulties, and large-scale network alterations affecting attention and emotional processing. However, this synthesis should be regarded as hypothesis-generating rather than as a formally validated or universally applicable neurobiological model, particularly given the predominance of internet gaming disorder studies and the heterogeneity of the available evidence.

At a broader conceptual level, the current literature also illustrates the difficulty of interpreting problematic digital behaviors as if they reflected a single neurobiological entity. Although overlap across phenotypes is plausible, the behavioral architecture of internet gaming disorder, problematic smartphone use, generalized problematic internet use, and problematic social media use is unlikely to be identical. These patterns may share certain transdiagnostic features, such as impaired inhibitory control, attentional capture by salient cues, reward-related learning, or emotion-regulation difficulties, while still differing in the relative contribution of habit formation, compulsive checking, social reinforcement, escape-based coping, or immersive reward seeking. This distinction is important because it suggests that apparent convergence at the neural level may coexist with meaningful phenotype-specific variation at the behavioral and mechanistic levels. Accordingly, future models should aim not only to identify shared neurobiological tendencies across problematic digital behaviors but also to clarify where these phenotypes diverge in psychologically and clinically relevant ways [27,36,37].

Future work would benefit from more harmonized definitions and more phenotype-specific models of problematic digital behavior. Rather than assuming that internet gaming disorder, problematic smartphone use, generalized problematic internet use, and problematic social media use reflect a single neurobiological entity, future studies should test both shared and distinct mechanisms across these categories. Greater use of longitudinal designs, multimodal imaging approaches, preregistered analyses, and better control of psychiatric and behavioral confounders may help clarify whether currently reported findings reflect vulnerability markers, consequences of repeated digital over-engagement, compensatory adaptations, or mixed processes. Such advances would substantially strengthen the interpretability and translational relevance of the field.

5. Limitations

Several limitations should be considered when interpreting the findings of this systematic review.

First, most of the included studies employed cross-sectional designs, which preclude causal inference regarding whether the reported neurobiological alterations represent pre-existing vulnerability factors or neuroplastic consequences of prolonged digital engagement.

Second, sample sizes across studies were generally modest, reducing statistical power and limiting the robustness and generalizability of the reported neural effects.

Third, substantial heterogeneity was present in the operationalization and assessment of problematic digital behavior. Studies employed different diagnostic criteria, psychometric instruments, and cut-off thresholds to define problematic internet use, problematic smartphone use, problematic social media use and internet gaming disorder. This lack of standardization complicates direct comparison across studies and limits the feasibility of quantitative meta-analysis.

Fourth, methodological heterogeneity across neurobiological paradigms and analytic approaches further constrains interpretability. The included studies used diverse modalities, including structural MRI, diffusion MRI, task-based fMRI, resting-state fMRI, and electrophysiological methods. In addition, task paradigms, pre-processing pipelines, connectivity metrics, and statistical thresholds varied substantially across studies. Such variability increases the risk of inconsistent or modality-specific findings and reduces overall reproducibility.

Fifth, the evidence base was unevenly distributed across phenotypes. Most included studies focused on internet gaming disorder or closely related problematic internet use constructs, whereas problematic smartphone use and problematic social media use were represented by comparatively fewer studies. This imbalance limits the extent to which integrative conclusions can be generalized across the broader spectrum of problematic digital behaviors.

Finally, publication bias and database bias cannot be excluded. Although multiple databases and supplementary Google Scholar searches were employed, relevant studies indexed exclusively in inaccessible databases may have been missed.

6. Conclusions

This systematic review suggests that problematic digital behaviors may be linked to alterations in neural systems involved in reward processing, salience, and executive control. Across structural MRI, functional neuroimaging, electrophysiology, and interventional studies, the most recurrent findings pointed to fronto-striatal circuitry and large-scale brain networks, particularly the default mode, salience, and frontoparietal systems. At the same time, the evidence base remains limited by modest sample sizes, methodological heterogeneity, and the predominance of cross-sectional designs. It is also important to note that the strongest evidence comes from studies on internet gaming disorder, while findings related to problematic internet use, problematic smartphone use, and problematic social media use are still relatively sparse and more heterogeneous.

Taken together, the current literature is more suggestive of a provisional, hypothesis-generating neurobiological framework than of a clearly established unified mechanism across problematic digital behaviors. Although some findings appear broadly consistent with addiction-related models, this interpretation should be made with caution until it is supported by larger, longitudinal, and better-standardized studies.

For this reason, the proposed framework should be regarded as provisional and hypothesis-generating rather than as evidence of a single unified neurobiological model across all problematic digital behavior phenotypes.

Future research should prioritize longitudinal and multimodal designs to help clarify whether the reported neurobiological differences reflect pre-existing vulnerabilities, markers of symptom severity, or consequences of prolonged digital engagement. Clearer diagnostic definitions, more harmonized neuroimaging protocols, and greater attention to problematic smartphone and social media use will be important for refining neurobiological models in this field.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/psychiatryint7030113/s1, Material S1: PRISMA Checklist, Material S2: Database-specific search strategies.

Author Contributions: Conceptualization: R.-I.M. and A.M.; methodology, O.-R.B.; software, O.-R.B.; validation R.-I.M. and A.M.; formal analysis, R.-I.M. and A.M.; investigation, R.-I.M., A.M.; resources, R.-I.M. and A.M.; data curation, R.-I.M. and A.M.; writing—original draft preparation, R.-I.M., A.M. and O.-R.B.; writing—review and editing R.-I.M., A.M. and O.-R.B.; visualization, R.-I.M., A.M. and O.-R.B.; supervision A.M. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: No new datasets were generated or analyzed beyond the data extracted from the published studies included in this systematic review. The data supporting the findings of this study are available within the article and its Supplementary Materials.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript: IGD = Internet gaming disorder; PIU = Problematic internet use; IA = Internet addiction; PIUQ = Problematic Internet Use Questionnaire; rs-fMRI = resting- state functional MRI; DMN = Default mode network; ICN = Inhibitory control network; IFG = inferior frontal gyrus; ACG = anterior cingulate gyrus; PCG = posterior cingulate gyrus; SMG = supramarginal gyrus; MTG = middle temporal gyrus; STG = superior temporal gyrus; SPL = superior parietal lobule; PCC = posterior cingulate cortex; FA = fractional anisotropy; ATR = anterior thalamic radiation; ALIC = anterior limb of internal capsule; CST = corticospinal tract; IFOF = inferior fronto-occipital fasciculus; ILF = inferior longitudinal fasciculus; EFE = emotional face expression; PVT = psychomotor vigilance task.

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