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Problematic Smartphone Use And Its Psychosocial Correlates: A G-Deeg–Based Paradigm Proposal

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Abstract

Background: The deep embedding of smartphones into everyday life has intensified debates about their links with psychological well-being and social functioning. Much of the literature reports small, context-sensitive associations between technology use and mental health indices; the magnitude and direction vary by use characteristics (e.g., passive social comparison, notification intensity), timing (especially late-night use), and individual susceptibilities (e.g., FoMO, impulsivity) [1, 2, 3, 4, 5, 6, 7, 8].

Objective: We conceptualize *Problematic Smartphone Use (PSU)* not as a standalone diagnosis but as a risk syndrome at the intersection of loss of control, tolerance/withdrawal-like patterns, and salient functional impairment. We integrate evidence within a multilayered framework (developmental, digital-design, emotional/psychological, social/environmental, biological) [9, 10, 11].

Methods: We narratively synthesize longitudinal/panel/EMA studies, systematic reviews and meta-analyses, and selected neuroimaging evidence published between 2013 and 2024 [12, 1, 13, 14, 15, 16, 17].

Findings (synthesis): (i) Average associations are small; however, context-sensitive large effects can emerge via sleep disruption, high-intensity notification architectures, and FoMO/emotion-regulation difficulties [12, 18, 19]. (ii) Neuroimaging studies note striatal–prefrontal differences in at-risk subgroups, with limited causal inference [20, 14]. (iii) Digital therapeutics yield small-to-moderate benefits for depression/anxiety; human support and measurement transparency appear to enhance effects [17, 21, 22].

Conclusions: Moving beyond "addicted everywhere for everyone," PSU should be approached through functional impairment and context-based clinical decisions. At school/work, phone-free windows and notification diets are advisable; in clinics, stepped-care and preferably human-supported digital interventions are recommended. Future work should prioritize causal mapping through self-report–log–EMA triads, preregistered RCTs, and longitudinal neuroimaging [1, 13, 4].

Keywords: Adolescents; Digital therapeutics; Emotion regulation; Ecological momentary assessment (EMA); Fear of missing out (FoMO); Notification architecture; Problematic smartphone use; Smartphone addiction; Smartphone; Sleep disturbance.

1. INTRODUCTION

Smartphones consolidate communication, productivity, health, and learning applications within a single ecosystem, enabling cognitive off-loading and robust social connectedness. At the same time, an increasing number of individuals exhibit patterns of *problematic smartphone use (PSU)*—loss of everyday control, aimless time loss, and functional impairment across school/work/relationships [23, 10]. PSU is not a distinct diagnosis in DSM-5/ICD-11; nevertheless, it remains debated due to phenomenological overlaps with behavioral addiction clusters (loss of control, tolerance, withdrawal, and persistence despite harms) [23, 10]. Rather than a universal "addiction" narrative, we conceptualize PSU as a risk syndrome that varies along axes of functional impairment and context.

1.1. Scale of the Issue and "Small-but-Context-Sensitive" Effects

Analyses using large, multi-country samples show negative but **small** associations between digital engagement and adolescent well-being; explained variance is limited on average [1, 4]. The "Goldilocks hypothesis" suggests nonlinearity—relative advantages for moderate use compared with very low or very high engagement [2]. EMA and longitudinal evidence generally reports weak/unstable daily links between use and psychopathology [13]. Thus, while mean effects are small, risks can concentrate in "pockets" defined by context (content, timing) and person-level differences [6, 24].

1.2. Why *Who Uses What, When, and How* Matters

Total duration is a poor stand-alone marker. The **quality** of use (e.g., passive comparison), **timing** (especially at night), and **interface architecture** (dense notification cascades, infinite scroll, variable reinforcement) are key conduits of risk [12, 25]. When paired with FoMO and emotion-regulation difficulties, small average effects can scale to clinically meaningful outcomes [18, 19]. Risk is therefore more sensitive to the contextual profile of engagement than to "screen time" per se.

1.3. Mechanisms: Sleep, Attentional Fragmentation, FoMO, and Habit Loops

Sleep. Smartphone use at bedtime/bedroom is consistently linked with shorter/poorer sleep, delayed sleep onset, and daytime sleepiness [12]. Light exposure, arousal, and emotionally salient content may disrupt circadian processes, undermining next-day cognition and mood.

Attention/impulsivity. Notification density and infinite feeds leverage variable reinforcement, strengthening habit loops and *loss of control* perceptions [11]. Task-switching costs rise, producing attentional fragmentation; effects amplify among individuals with fragile self-control.

FoMO and emotion regulation. FoMO mediates PSU–anxiety/depression links; structural equation models support this pathway [18, 26]. FoMO-driven social comparison and instant-response expectations widen use windows.

1.4. Neurobiological Sensitivities and the Causality Problem

Neuroimaging indicates structural/functional differences in striatal–prefrontal circuits among heavy-use groups [15, 14]. However, most studies are cross-sectional with small samples; causality and temporal precedence remain unclear [14]. Converging with clinical/epidemiological findings, the picture suggests person- and context-dependent risk.

1.5. Diagnostic Debate: "Addiction" or "Problematic Pattern"?

One view frames smartphone use as a behavioral addiction; a critical stance emphasizes heterogeneous evidence and the limited explanatory power of a single disease label [23, 10]. We adopt a function-first approach: diagnosis should appraise **Control** (loss of control/failed restriction), **Consequences** (sleep, school/work, relationships), and **Context** (night-time use, hazardous situations, notification design) in tandem.

1.6. Measurement and Method: The Self-Report–Digital Log–EMA Triad

Self-reported durations often diverge from device logs, introducing measurement error and biasing effect sizes [27]. A comprehensive meta-analysis recommends caution toward studies ignoring the self-report vs. log distinction [28]. To approach causality, combine EMA with passive logs and contextual content, alongside preregistered intervention/quasi-experimental designs [13, 1].

1.7. Social Context and Inequalities

Cross-national evidence shows that the quality of family/friend relationships predicts life satisfaction more strongly than time-spent metrics, arguing against one-size-fits-all restriction policies and in favor of targeted, context-sensitive approaches [5].

1.8. The Turkish Context: Summary of Evidence and Localization

Among Turkish university samples, PSU correlates positively with poor sleep and depression/anxiety [29]. While non-causal, this justifies stepped-care interventions focusing on sleep hygiene, notification architecture, and FoMO.

1.9. Positioning of the Present Study

We synthesize evidence across five interacting layers—**Developmental/Generational; Digital-Design; Emotional/Psychological; Social/Environmental; Genetic/Biological**—and link PSU to a **Control–Consequences–Context** clinical decision tree, generating actionable recommendations from school/work policies to human-supported digital therapeutics [17, 16, 22, 11].

2. THEORETICAL FRAMEWORK: THE G-DEEG PARADIGM

2.1. Rationale and Architecture

The G-DEEG paradigm conceptualizes problematic smartphone use (PSU) as the emergent product of five interacting layers: **G**enerational/Developmental, **D**igital/Design, **E**motional/Psychological, **E**nvironmental/Social, and **G**enetic/Biological. Rather than treating "screen time" as a sufficient cause, the framework emphasizes **contextualized exposure**, **individual susceptibility**, and **design affordances** that canalize behavior toward or away from impairment. This multilevel perspective accommodates the empirical reality that average associations between digital engagement and mental health tend to be **small** yet **context-sensitive**, with effects magnifying under specific conditions (e.g., late-night use, high notification density, passive social comparison) [1, 4, 7, 12, 36].

2.2. Layer 1 — Generational/Developmental (G)

During early–mid adolescence, elevated reward sensitivity relative to still-maturing executive control systems increases vulnerability to immediate, variable reinforcement schedules common to app ecosystems. Developmental risk concentrates in windows where autonomy expands while regulatory capacities lag, thereby potentiating loss-of-control experiences and sleep-related dysfunctions that feed next-day mood and cognition [1, 4, 7]. Differential susceptibility theory further predicts that the same adolescents who are most sensitive to adverse contexts may benefit disproportionately from supportive arrangements (e.g., notification hygiene, phone-free routines) [39].

Operational cues: age-normed cut-points on the Smartphone Addiction Scale–Short Version (SAS-SV), daytime sleepiness scores, and diary/EMA markers of self-control lapses [29].

2.3. Layer 2 — Digital/Design (D)

Interface architecture—**infinite scroll**, **autoplay**, **pull-to-refresh**, **streaks**, and **dense notification cascades**—implements variable-ratio/interval reinforcement that entrenches cue-response loops. These affordances interact with night-time availability to degrade sleep onset and continuity, with spillovers to affect and executive function [7, 12]. Discrepancies between self-reported and passively logged use are non-trivial, underscoring the need to treat "exposure" as a **measured, multi-facet construct** (content, timing, intensity, contingency) rather than a single duration metric [36, 37].
Operational cues: device logs for notification frequency and session timing; content classification (active vs. passive use); bedtime proximity metrics.

2.4. Layer 3 — Emotional/Psychological (E)

FoMO, negative affectivity, and emotion-regulation difficulties are robust correlates of PSU. FoMO in particular mediates associations between heavy, late, or passive engagement and anxiety/depressive symptoms, while impaired regulation transforms minor trigger exposures into prolonged sessions [12, 14, 18]. Meta-analyses of human-supported digital therapeutics show small-to-moderate improvements in common mental disorders, implying that **human scaffolding** is a key effectiveness moderator for app-based interventions [16, 42].
Operational cues: FoMO scales, momentary affect (EMA), rumination indices, and just-in-time adaptive intervention (JITAI) responsiveness.

2.5. Layer 4 — Environmental/Social (E)

Family climate, peer norms, school/work expectations, and socioeconomic context shape both the **meaning** and **impact** of smartphone use. Cross-national data indicate that **relationship quality** and **offline supports** explain more variance in adolescent well-being than time-spent alone, arguing for **context-sensitive** rather than universal restriction policies [5]. In university settings, PSU clusters with poor sleep and elevated depression/anxiety, pointing to campus-level levers (sleep hygiene education, notification diets, time-boxed study blocks) [30].
Operational cues: family cohesion/monitoring scales, classroom/office phone policies, peer-norm audits.

2.6. Layer 5 — Genetic/Biological (G)

Candidate neural correlates include striatal and prefrontal systems implicated in reward learning and control; structural/functional differences have been reported in high-risk subgroups, though designs are predominantly cross-sectional and cannot secure causal precedence [10, 9]. Biological sensitivity to context likely intersects with developmental and psychosocial moderators, aligning with differential susceptibility predictions [39].
Operational cues: sleep-actigraphy and circadian markers; where feasible, longitudinal neuroimaging embedded within preregistered designs.

2.7. Cross-Layer Dynamics and the 3C Decision Tree

We operationalize clinical decision-making through **Control–Consequences–Context (3C)**:

- **Control:** failed restriction, loss-of-control episodes, time-loss with regret.
- **Consequences:** sleep disturbance, academic/occupational underperformance, relational conflict, mood/anxiety escalation [30].
- **Context:** late-night exposure, hazardous situations (e.g., driving), high notification density, predominantly passive consumption [7, 12, 36].

Severity tiers (watchful waiting → brief intervention → stepped care) are assigned by intersecting **control** failure with **functional harm** in risky **contexts**; human-supported digital therapeutics are preferred over app-only approaches when impairment is present [16, 42].

2.8. Measurement Strategy: The "Moderator–Mechanism–Metric" (M³) Matrix

To align evaluation with theory, we recommend an **M³ Matrix** that links:

- **Moderators** (age, FoMO, regulation capacity, notification density),
- **Mechanisms** (sleep disruption, attentional fragmentation, social comparison stress),
- **Metrics** (SAS-SV; PHQ-4 or equivalent mood screens; EMA micro-surveys; device-log indicators; bedtime proximity; weekly notification counts).

This triad enables **triangulation** across self-report, logs, and EMA, reducing single-method bias and clarifying when small average effects conceal large, person-specific risks [29, 36, 37].

2.9. Testable Propositions

P1. After adjusting for relationship quality and sleep, total duration will show attenuated associations with distress relative to **timing** and **notification intensity** [1, 4, 36].
P2. Adolescents with high FoMO and low regulation will display **disproportionate** impairment under dense notification architectures, consistent with differential susceptibility [14, 18, 39].
P3. Bedtime-proximal passive use will predict next-

day mood/cognition decrements more strongly than daytime active use [7, 12].
P4. Human-supported digital interventions will outperform app-only modules at equal dose, particularly in high-FoMO/high-notification subgroups [16, 42].
P5. Self-report–only exposure models will **underestimate** true effect heterogeneity compared with log-augmented, EMA-linked designs [36, 37].
P6. Neurobiological markers will show **state-tracking** rather than stable trait-level differences when examined in longitudinal, preregistered cohorts [9, 10].

3. METHODS

3.1. Design and Scope

We conducted a critical **narrative synthesis** of the empirical literature on Problematic Smartphone Use (PSU) and psychosocial/clinical outcomes, integrating longitudinal/panel and ecological momentary assessment (EMA) studies, systematic reviews/meta-analyses, and selected neuroimaging reports published between 2013 and 2024. The review prioritizes **contextualized exposure** (content, timing, notification density), **person-level susceptibilities** (e.g., FoMO, emotion regulation), and **design affordances**, consistent with the G-DEEG paradigm articulated in Section 2 [1, 4, 7, 12, 36].

3.2. Guiding Questions

Primary questions were\
(i) What is the magnitude and stability of associations between smartphone engagement and mental health/functional outcomes when exposure is measured beyond total duration (e.g., timing, notification architecture)? [1, 4, 36, 37]
(ii) Which person-level moderators and mechanisms (sleep disruption, FoMO, attentional fragmentation) most consistently account for heterogeneity? [7, 12, 18, 29]
(iii) What is the comparative effectiveness of **human-supported** versus app-only digital interventions? [16, 42]

3.3. Eligibility Criteria

Inclusion. Peer-reviewed human studies in adolescents or adults; designs including RCTs, quasi-experiments, prospective/longitudinal cohorts, daily diary/EMA, cross-sectional studies with validated measures; exposures encompassing usage **timing**, **content/active vs. passive patterns**, and **notification density**; outcomes spanning sleep, mood/anxiety, cognition/executive function, academic/occupational performance, and functional impairment; meta-analyses/systematic reviews of digital therapeutics or exposure–outcome linkages [1, 7, 12, 16, 36].
Exclusion. Case reports; studies lacking clear exposure or outcome definitions; non-validated scales; samples restricted to clinical conditions unrelated to PSU without separate analyses.

3.4. Information Sources and Search Strategy

Databases (e.g., PubMed, PsycINFO, Web of Science) were queried for 2013–2024 using terms for smartphone/screen use, timing/notifications, sleep, FoMO/emotion regulation, EMA/logs, and digital therapeutics. Reference lists of key syntheses were hand-searched to capture additional studies [1, 4, 16, 36]. Because this is a targeted synthesis aligned with an a priori theoretical model, we did not aim for exhaustive retrieval.

3.5. Study Selection and Data Extraction

Titles/abstracts were screened against eligibility; full texts were then appraised for design quality, exposure/measure validity, and analytic adequacy. We extracted: population/age, design, exposure operationalization (duration vs. timing vs. notifications; active vs. passive), measurement method (self-report vs. device logs), outcomes, effect estimates (with covariates), moderators/mediators (e.g., FoMO), and risk-of-bias indicators [29, 36, 37].

3.6. Measures and Operational Definitions

Exposure.

- **Timing:** bedtime/bedroom proximity and night-time usage windows [7, 12].
- **Notification architecture:** notification counts/frequency, batch vs. continuous delivery [36].
- **Content/Mode:** passive vs. active engagement; social comparison features.
- **Total duration:** retained but treated as a **coarse proxy** [1, 4].

Outcomes.

- **Sleep:** duration, latency, quality/sleepiness indices [7, 12].
- **Affect and symptoms:** depression/anxiety/stress (e.g., PHQ-4 or analogous brief scales).
- **Function:** academic/occupational performance, relationship conflict.
- **PSU severity:** validated scales such as **SAS-SV** for adolescents/young adults [29].

Mechanism/Moderator constructs.

- **FoMO** and **emotion regulation** (self-report scales) [18].
- **Self-control/impulsivity** (brief trait/state indices).
- **Measurement mode: device logs** vs. self-reports flagged explicitly given known discrepancies [36, 37].
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3.7. Quality Appraisal and Risk of Bias

We used design-appropriate heuristics: RoB-2 for RCTs; ROBINS-I or Newcastle–Ottawa criteria for non-randomized designs; and targeted checks for **exposure misclassification** (self-report vs. logs), **temporal precedence** (lagged models), **confounding** (relationship quality, sleep), and **analytic flexibility**. Neuroimaging reports were appraised for sample size, correction for multiple comparisons, and preregistration status where available [10, 15].

3.8. Synthesis Approach

Given heterogeneity in exposure operationalization and outcomes, we conducted a **best-evidence narrative synthesis** structured by the **Moderator–Mechanism–Metric (M^3) Matrix** (Section 2.8), privileging studies that: (i) measure timing/notifications or passive vs. active content; (ii) triangulate **self-report–log–EMA**; (iii) test moderators/mediators (sleep, FoMO) [29, 36, 37]. Where multiple high-quality syntheses existed (e.g., digital therapeutics, log vs. self-report), we summarized **pooled estimates** and **consensus directionality** rather than recomputing meta-analytic models [16, 36, 42].

3.9. Sensitivity and Robustness Checks

Findings were examined for robustness to: (i) measurement mode (self-report only vs. log-augmented) [36, 37]; (ii) age band (early/middle adolescence vs. young adulthood) [1, 4]; (iii) outcome domain (sleep vs. affect vs. function) [7, 12]; and (iv) presence of human support in digital therapeutics [16, 42].

3.10. Ethics, Registration, and Reporting

This review synthesizes published data and did not require institutional review board approval. Reporting follows good-practice guidance for narrative reviews and **SWiM** principles for synthesis without meta-analysis. No preregistered protocol is available; key questions and inclusion logic were specified prior to screening.

3.11. Planned Outputs

We pre-specified (i) a **conceptual decision tree** (Control–Consequences–Context) to guide stepped care; (ii) an **M^3 Matrix** mapping moderators → mechanisms → measurable metrics; and (iii) a **translation layer** for policy/clinic (school/work phone-free windows, notification diets; human-supported vs. app-only interventions) [7, 12, 16, 36, 42].

4. RESULTS / EVIDENCE SYNTHESIS

4.1. Overall magnitude and stability of associations

Across longitudinal, panel, and EMA designs, average links between general smartphone engagement and mental health indices are **near-zero to small**. Effect directions vary by *context of use* (timing, content, notification density) and *person-level susceptibilities* (FoMO, emotion regulation, self-control) [1, 4, 13, 36, 37]. When exposure is operationalized as **total duration alone**, associations tend to attenuate further after adjustment for sleep and relationship quality, indicating confounding by proximal mechanisms and social context [1, 4].

4.2. Timing and sleep-proximal exposure

Bedtime/bedroom use exhibits the **most consistent** adverse pattern: shorter sleep duration, longer sleep latency, poorer sleep quality, and next-day sleepiness—effects that generalize to attentional control and mood the following day [7, 12]. Studies that isolate **bedtime-proximal windows** (e.g., last 60–90 minutes) detect larger and more stable associations than day-time analyses, supporting the hypothesis that **circadian disruption** is a key pathway from use to functional impairment [7, 12].

4.3. Notification architecture and attentional fragmentation

High **notification density** and continuous delivery (vs. batched modes) are linked to frequent task switching, subjective loss of control, and perceived time-loss. Measurement work comparing **device logs** with self-reports shows that ignoring notifications underestimates exposure–outcome heterogeneity and biases effect sizes toward the null [36, 37]. Variable reinforcement features (infinite scroll, autoplay, streaks) interact with notification cascades to strengthen cue–response loops and prolong sessions [7, 12, 36].

4.4. Content mode: passive vs. active engagement

Passive consumption (scrolling, social comparison) relates more robustly to negative affect and lower well-being than active, goal-directed use (messaging, creation). Mediation analyses implicate **FoMO** and **rumination** as proximal mechanisms connecting passive use with distress [12, 18]. By contrast, active, socially supportive use shows weaker and sometimes beneficial associations when exposure is disentangled from night-time timing [1, 4, 12].

4.5. Person-level moderators: FoMO, emotion regulation, developmental windows

FoMO and difficulties in emotion regulation **amplify** the impact of high-density notifications and night-time use on mood/sleep outcomes [12, 18]. Developmentally, early–mid adolescence—characterized by heightened reward sensitivity and still-maturing control systems—presents a **risk window** in which the same exposure yields larger impairments than in older groups [1, 4, 7]. Consistent with **differential susceptibility**, some adolescents appear both more vulnerable to adverse contexts and more responsive to supportive arrangements (notification hygiene, phone-free routines) [39].

4.6. Measurement mode: self-report versus device logs

Across studies that include both metrics, **self-reported duration** diverges substantially from **logged** use, with systematic error correlated with symptoms in some samples [36, 37]. Evidence syntheses argue that exposure needs to be **triangulated**—duration + timing + notification architecture—ideally with log-augmented EMA, to reduce misclassification and clarify causal direction [36, 37, 13].

4.7. Functional outcomes: academic/occupational performance and relationships

Associations with school/work performance are generally small when modeled on total duration, but become **larger and more consistent** when models include sleep disruption, night-time use, and notification density, suggesting **indirect** pathways via fatigue and attentional fragmentation [7, 12, 36]. Relationship conflict correlates with perceived loss of control and problematic patterns (e.g., checking while conversing), again contingent on timing and notification profiles [12].

4.8. Clinical outcomes and digital therapeutics

Human-supported digital interventions (CBT-informed or well-being apps with coaching/feedback) show **small-to-moderate** benefits for depression/anxiety; app-only modules perform less consistently [16, 42]. Heterogeneity is partly explained by design quality, adherence, and whether interventions **target mechanisms** (sleep, FoMO, regulation) rather than duration alone [16, 42]. Studies embedding **notification hygiene** and sleep protocols achieve larger functional gains in high-risk subgroups [7, 12, 16].

4.9. Neuroimaging and biological correlates

Structural/functional differences have been reported in striatal–prefrontal circuits among heavy-use or high-PSU groups; however, samples are often small and cross-sectional, limiting causal inference [10, 9]. Longitudinal work remains sparse; a priori expectation is **state-tracking** (dynamic coupling with sleep/affect) rather than stable trait-like abnormalities [9, 10].

4.10. Country-specific evidence (Turkey)

University cohorts in Türkiye replicate positive correlations between PSU severity and **poor sleep**, **depressive** and **anxiety** symptoms, in line with international evidence [30, 29]. While not causal, these findings motivate campus-level strategies addressing notification architecture, bedtime routines, and FoMO-targeted psychoeducation [29, 30].

4.11. Sensitivity and robustness

Findings are robust to (i) adjustment for relationship quality and sleep (attenuating duration-only associations) [1, 4]; (ii) stratification by age (larger effects in early–mid adolescence) [1, 4, 7]; and (iii) exposure measurement (log-augmented models reveal stronger timing/notification effects than self-report-only analyses) [36, 37]. Publication bias is minimized in meta-analyses focused on **logged** exposure, though heterogeneity persists owing to operational differences in content/timing [36].

4.12. Interim synthesis

Taken together, the evidence favors a **context-first** interpretation: small mean effects conceal **large, actionable pockets** of risk, primarily via **sleep**, **notification-driven attentional fragmentation**, and **FoMO-mediated affective reactivity**. Measurement precision (logs + EMA), mechanism-targeted interventions (sleep hygiene, notification diets), and **human support** consistently improve outcomes relative to duration-focused or app-only strategies [7, 12, 16, 36, 42].

5. DISCUSSION

5.1. Principal findings

This review advances a **context-first** account of problematic smartphone use (PSU). Average associations between generic "use" and mental health are **small** and often unstable across designs, yet **large, actionable pockets of risk** emerge under specific **timing** (bedtime/bedroom), **interface** (high notification density; infinite scroll), and **person-level** susceptibilities (FoMO, emotion-regulation difficulties) [1, 4, 7, 12, 18, 36, 37]. Evidence favors **mechanism-targeted** interventions—sleep hygiene, notification diets, and human-supported digital therapeutics—over duration-centric prescriptions [7, 12, 16, 42]. Methodologically, failure to measure **timing and notification architecture** and reliance on **self-report only** obscure effect heterogeneity and bias estimates toward the null [36, 37].

5.2. Reconciling small averages with large contextual effects

The apparent paradox—small mean effects but clear clinical cases—resolves when exposure is **decomposed** into *when*, *what*, and *how* people engage. Late-night, passive, and notification-saturated use propagates impairment through **sleep disruption** and **attentional fragmentation**, which intensify affective reactivity the following day [7, 12]. Further, the **differential susceptibility** lens explains why the same exposure harms some individuals much more than others: youth with high FoMO or weaker regulation experience stronger, non-linear responses; the converse holds under supportive arrangements (notification hygiene, phone-free routines) [39].

5.3. Measurement implications

Treating exposure as **total duration** is insufficient and potentially misleading. Studies triangulating **device logs** with **EMA** consistently show (i) substantive **self-report–log** discrepancies and (ii) stronger, more precise links for **timing** and **notifications** than for hours-of-use alone [36, 37, 13]. Pragmatically, researchers and clinicians should adopt a **measurement triad**—duration **plus** timing **plus** notification density—ideally with log-augmented EMA, and should report exposure with standardized fields (e.g., last-90-min bedtime proximity; daily notification counts; passive/active proportion) [36, 37].

5.4. Mechanisms: sleep, attentional fragmentation, and FoMO-mediated reactivity

Across designs, bedtime/bedroom use predicts **shorter sleep**, delayed onset, and **next-day sleepiness**; these translate into poorer executive functioning and lower mood [7, 12]. Notification cascades and variable reinforcement (infinite scroll, streaks) impose **switching costs**, perceived loss of control, and time-loss, especially in high-FoMO or low-regulation profiles [12, 18]. A parsimonious account is that **sleep disruption** and **notification-driven attentional fragmentation** serve as **proximal conduits** from context-specific phone use to functional and affective outcomes [7, 12, 36].

5.5. Clinical implications: a Control–Consequences–Context (3C) decision tree

Findings support a **function-first** approach operationalized via **3C**:

- **Control:** repeated failed restriction, loss-of-control episodes, regret-laden time-loss.
- **Consequences:** sleep disturbance, daytime sleepiness, academic/occupational underperformance, relationship conflict, mood/anxiety escalation [30].
- **Context:** bedtime proximity, hazardous settings (e.g., driving), dense notification architecture, predominantly passive consumption [7, 12, 36].

Screening. Use **SAS-SV** in adolescents/young adults, brief mood screens (e.g., PHQ-4), and a two-item sleep/adherence check; add a one-minute "notification census" (batching on/off; counts/day) [29, 30, 36].

Stepped care.

- *Tier 1:* psychoeducation; **notification diets** (batch delivery; quiet hours), **phone-free windows** aligned with sleep, and passive→active content rebalancing.
- *Tier 2:* mechanism-targeted modules (sleep hygiene; FoMO/regulation skills) with **log-EMA feedback**.
- *Tier 3:* **human-supported** digital therapeutics or brief psychotherapy, given superior and more reliable effects vs. app-only formats [16, 42].

Documentation. Record **timing**, **notification density**, and **passive/active ratio** at each visit—these variables are *treatable levers*.

5.6. Schools and workplaces: design the defaults, not just the rules

Policies centered on blanket bans or generic hour caps underperform. Instead, institutions should **engineer defaults**: protected **phone-free windows** (instructional blocks; meetings), **batched notifications** by default on campus/work Wi-Fi, and **night-mode** prompts with pre-set quiet hours. These contextual nudges reduce the *opportunity* for bedtime-proximal and interruption-prone use without stigmatizing routine, beneficial engagement [7, 12, 36]. Communication campaigns should emphasize **sleep-first** and **notification hygiene** rather than moralizing duration.

5.7. Equity and cultural context

Cross-national evidence shows that **relationship quality** and **offline supports** explain more variance in adolescent well-being than time-spent metrics, underscoring the primacy of **social context** [5]. In Türkiye, university-based cohorts align with international patterns—PSU co-occurs with poor sleep and elevated depression/ anxiety—pointing to pragmatic campus levers (sleep education, Wi-Fi–level batching, FoMO-targeted psychoeducation) [30, 29]. Equity-sensitive implementation matters: lower-resource students may benefit disproportionately from **default** protections (quiet hours pre-configured on institutional networks).

5.8. Neurobiological interpretation and the causality problem

Neuroimaging reports implicate **striatal–prefrontal** systems in high-PSU groups, but samples are small and predominantly cross-sectional, limiting causal inference [10, 9]. A theoretically coherent expectation is **state-tracking**—dynamic neural coupling with sleep, affect, and control—rather than stable trait abnormalities. Longitudinal, preregistered imaging embedded within **log-EMA** designs is needed to adjudicate whether neural differences are antecedents, consequences, or **bidirectional** correlates [9, 10].

5.9. Methodological limitations of the evidence base

Four issues recur. **First**, overreliance on **self-reported duration** inflates measurement error and obscures contextual mechanisms [36, 37]. **Second**, insufficient modeling of **timing** and **notifications** leaves mechanistic variance unexplained [36]. **Third**, cross-sectional and convenience samples remain common, hindering temporal inference [1, 13]. **Fourth**, heterogeneity in PSU scales and variable cut-points complicates synthesis; adoption of **age-normed** thresholds would improve comparability [29]. Where meta-analyses pool studies ignoring logs, effects are plausibly **underestimated** relative to log-augmented designs [36].

5.10. Future research agenda

We outline testable priorities aligned with the **M³ Matrix** (moderators–mechanisms–metrics).

1. **Measurement standards.** Mandate reporting of **bedtime proximity**, **notification counts** (and batching), and **passive/active proportions** alongside total duration [36, 37].
2. **Triangulated designs.** Pair **device logs** with **EMA** micro-surveys and mechanistic outcomes (sleep-actigraphy, cognitive tasks) to reduce misclassification and clarify directionality [13, 36].
3. **Mechanism-focused trials.** Preregistered RCTs targeting **sleep** and **notification architecture** should test mediation by FoMO/regulation, with **human support** as a planned moderator [7, 12, 16, 42].
4. **Developmental sensitivity.** Stratify by early–mid adolescence to evaluate **differential susceptibility** and to identify *for-whom* effects are largest [1, 4, 39].
5. **Open science.** Share de-identified log features and EMA codebooks; harmonize PSU thresholds (e.g., SAS-SV) by age bands to facilitate cross-study meta-analyses [29].

5.11. Implications for theory and practice

The present synthesis supports a **paradigm shift** away from duration-centric models toward **contextualized exposure** and **susceptibility**. Conceptually, PSU is best framed as a **risk syndrome** defined by **Control**, **Consequences**, and **Context** rather than a monolithic "addiction." Practically, the **treatable levers** are clear: **sleep windows**, **notification architecture**, **passive→active rebalancing**, and **human support** in digital therapeutics [7, 12, 16, 42]. By aligning measures and interventions with mechanisms, small mean effects translate into **meaningful clinical gains** for high-risk subgroups.

6. CONCLUSIONS & PRACTICAL RECOMMENDATIONS

6.1. Key conclusions

Problematic smartphone use (PSU) is best understood as a **context-contingent risk syndrome**, not a monolithic disorder. Average associations between general use and mental health are **small**, yet **clinically meaningful harms** arise under specific exposure profiles—**bedtime/bedroom timing**, **dense notification architecture**, and **passive, comparison-oriented content**—particularly in adolescents with **high FoMO** and **weaker regulation** [1, 4, 7, 12, 18, 36, 37]. Evidence favors **mechanism-targeted** interventions (sleep hygiene, notification diets) and **human-supported** digital therapeutics over duration-only prescriptions [7, 12, 16, 42].

6.2. Clinical practice recommendations (3C-based)

Screen & stage using the 3C decision tree—Control, Consequences, Context.

- **Control:** Identify failed restriction, loss-of-control episodes, regret-laden time-loss.
- **Consequences:** Document sleep disturbance, daytime sleepiness, academic/occupational underperformance, relationship conflict, mood/anxiety escalation [30].
- **Context:** Record bedtime proximity (last 60–90 minutes), hazardous settings (e.g., driving), notification density (counts/day; batching on/off), and passive/active ratio [7, 12, 36].

Minimum measurement set for visits.

- **SAS-SV** (adolescents/young adults) for PSU severity; **brief mood** screener (e.g., PHQ-4 equivalent); **two-item sleep** check; **notification census** (batched vs. continuous, quiet hours) [29, 30, 36].
- Where feasible, **device logs** for timing and notifications; consider **EMA** micro-surveys for momentary affect/self-control [36, 37, 13].

Stepped-care pathway.

- **Tier 1 (brief):** Psychoeducation; **notification diets** (batch delivery; quiet hours), **phone-free windows** aligned with sleep; shift from **passive** to **active** engagement.
- **Tier 2 (targeted):** Modules focused on **sleep hygiene** and **FoMO/regulation skills**, with **log-EMA feedback** to surface trigger contexts [7, 12].
- **Tier 3 (enhanced): Human-supported** digital therapeutics or brief psychotherapy; these outperform app-only approaches on average [16, 42].

Documentation & follow-up.

- Track **timing**, **notification density**, and **passive/active ratio** longitudinally; treat these as **modifiable levers**.
- Reassess at **4–8 weeks**; escalate if Control or Consequences remain impaired despite Tier 1 adherence [7, 12].

6.3. Schools and workplaces

- Replace blanket bans with **engineered defaults**: protected **phone-free instructional/meeting blocks**, **Wi-Fi–level batched notifications**, and **institutional quiet hours** prompting night-mode by default [7, 12, 36].
- Embed **sleep-first** messaging and **notification hygiene** in student/employee onboarding.
- For high-risk subgroups (e.g., early–mid adolescents; high FoMO), add **skills micro-modules** targeting comparison stress and response inhibition [1, 4, 18].

6.4. Public health and policy

- Prioritize **sleep protection** and **notification governance** (vendor-level batching, quiet-hour nudges) over generic screen-time caps.
- Encourage **interoperable logging APIs** for research/clinical use with privacy safeguards to enable **log–EMA** triangulation at scale [36, 37].
- Address equity: default protections disproportionately benefit students and shift workers with limited control over schedules.

6.5. Reporting standards for researchers

- Report exposure with **three fields**: **bedtime proximity**, **notification counts/batching**, **passive/active proportion**, alongside total duration [36, 37].
- Distinguish **self-reported** vs. **logged** measures; where both exist, analyze discrepancies explicitly [36, 37, 13].
- Pre-register **mechanism tests** (sleep, FoMO, attentional fragmentation) and moderator analyses (age, regulation capacity) [7, 12, 18].
- Favor **log-augmented EMA** and **quasi-experimental/RCT** designs to clarify directionality and reduce misclassification [13, 36].

6.6. Future directions

- **Causal mapping:** Preregistered trials that jointly manipulate **notification architecture** and **sleep windows**, measuring mediation by **FoMO/regulation** and outcomes in mood/executive function [7, 12, 16].
- **Developmental sensitivity:** Stratify early–mid adolescence; test **differential susceptibility** predictions with mechanism-targeted supports [1, 4, 39].
- **Biology in context:** Longitudinal neuroimaging embedded within **log-EMA**, prioritizing **state-tracking** over trait assumptions [9, 10].
- **Implementation science:** Evaluate campus/workplace **default engineering** (batched notifications; quiet hours) for real-world adherence and spillover benefits.

6.7. Take-home messages

1. **Context beats duration.** Timing (especially bedtime), notifications, and content mode explain more risk than hours alone [7, 12, 36].
2. **Measure to manage.** Use a triad—**logs + EMA + brief scales**—to reveal hidden heterogeneity [36, 37, 13].
3. **Target mechanisms.** Protect sleep, batch notifications, and reduce passive comparison; prefer **human-supported** over app-only interventions [7, 12, 16, 42].
4. **Adolescence matters.** Early–mid adolescence is a **risk window** and an opportunity for disproportionate benefit under supportive defaults [1, 4, 7, 39].
5. **Function-first.** Let **Control, Consequences, Context** define severity and guide stepped care.

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