

The Stimulated Brain:
**DIGITAL MEDIA, MUSIC,
CAFFEINE, AND
GAMBLING IN COGNITIVE SCIENCE**



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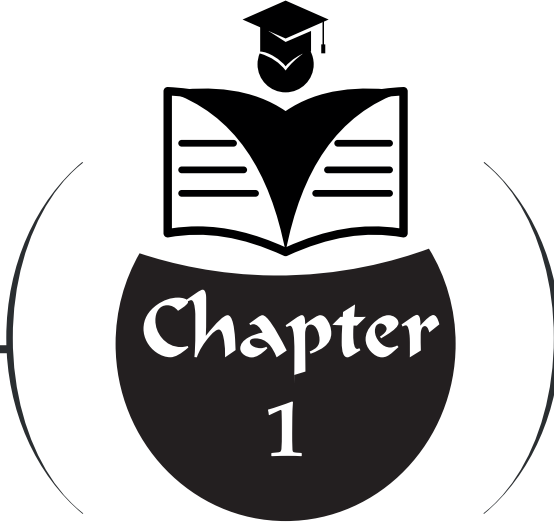
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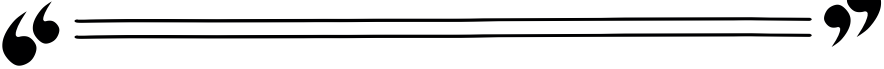
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**DIGITAL MEDIA USE AND
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A SYSTEMATIC REVIEW
OF ATTENTION, REWARD
SYSTEMS, AND BEHAVIORAL
OUTCOMES**



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1. Introduction

The twenty-first century has witnessed an unprecedented transformation in the informational environment within which human cognitive systems must operate. Digital media platforms — encompassing social networking services, streaming content, interactive gaming environments, mobile applications, and algorithmic news aggregators — have collectively displaced traditional media modalities and restructured the temporal, attentional, and social architectures of daily life to an extent that earlier generations of scholars could scarcely have anticipated. According to data published by the Global Web Index (2023), adults across industrialized nations now spend an average of 6.4 hours per day engaged with digital media, with younger cohorts reporting figures substantially in excess of this estimate. Notwithstanding the demonstrable benefits of digital connectivity — including expanded access to information, enhanced social coordination, and novel forms of creative expression — an expanding body of empirical research has raised substantive concerns regarding the cognitive costs imposed by such intensive and habitual engagement.

The scholarly discourse concerning digital media and cognition has matured considerably since the pioneering observations of Carr (2010), who argued in his widely cited work that the hyperlinked, fragmented architecture of the internet was fundamentally reshaping the neural substrates of human attention and deep reading. Since that period, empirical contributions from cognitive neuroscience, developmental psychology, educational research, and organizational behavior have collectively illuminated a complex and at times contradictory landscape. Certain researchers have identified robust negative associations between heavy digital media use and measures of sustained attention, executive function, and working memory capacity (Loh & Kanai, 2016; Wilmer, Sherman, & Chein, 2017; Uncapher & Wagner, 2018), while others have emphasized the potential cognitive benefits of specific forms of digital engagement, including enhanced visuospatial processing, accelerated information retrieval, and improvements in procedural learning (Green & Bavelier, 2012; Bavelier et al., 2019). This apparent inconsistency reflects, in large measure, the heterogeneity of digital media as a category, the diversity of outcome measures employed, and the range of theoretical frameworks that have been applied to the phenomenon.

Among the most theoretically significant developments in this literature is the growing recognition that digital media platforms are not cognitively neutral conduits for information but are, rather, architecturally designed — whether intentionally or as an emergent consequence of engagement-maximization algorithms — to exploit fundamental properties of human reward processing. Variable ratio reinforcement schedules, social validation mechanisms, infinite

scroll features, and notification systems all operate upon the same mesolimbic dopaminergic circuitry that underlies reward anticipation, habitual behavior formation, and, in pathological cases, addictive disorders (Montag et al., 2019; He et al., 2017; Andreassen et al., 2016). This neurobiological dimension has been accorded insufficient theoretical prominence in many behavioral accounts of digital media's cognitive consequences and merits systematic treatment.

It is important to note that the scientific investigation of digital media and cognition is complicated by several structural challenges that have limited the cumulative progress of the field. These challenges include the rapid evolutionary pace of platforms and usage patterns, which renders cross-temporal comparisons methodologically precarious; the frequent reliance upon self-reported usage data that correlates imperfectly with objective measurement; the predominance of cross-sectional designs that preclude causal inference; and the substantial heterogeneity of constructs employed across laboratories and theoretical traditions. A systematic review that rigorously synthesizes and critically evaluates this literature is therefore not merely a scholarly convenience but an epistemological necessity if the field is to achieve the conceptual coherence required for both theoretical advancement and practical application.

The present chapter is organized as follows. Section 2 establishes the theoretical foundations upon which the review is grounded, outlining the core frameworks drawn from cognitive science, neuroscience, and behavioral theory. Section 3 describes the systematic methodology employed in the identification, screening, and synthesis of the reviewed literature. Sections 4 through 6 present the substantive findings, organized respectively around attentional processes, reward systems and neurobiological mechanisms, and behavioral outcomes. Section 7 examines the moderating and mediating variables that shape these relationships. Section 8 offers a comprehensive discussion integrating the foregoing findings, considers theoretical and practical implications, and identifies directions for future research. The chapter concludes with a synthesis of key insights and a set of evidence-based recommendations for researchers, practitioners, and policymakers.

2. Theoretical Foundations

A systematic review of the relationships between digital media use and cognitive performance requires grounding in a coherent set of theoretical frameworks capable of accommodating findings across multiple levels of analysis — from the molecular substrates of reward processing to the macro-level behavioral patterns observable in educational and occupational contexts. Four interconnected theoretical frameworks are particularly germane to the present inquiry: Cognitive Load Theory, Dual-Process

Theory, the neurobiological framework of dopaminergic reward processing, and Attention Restoration Theory. Collectively, these frameworks provide the conceptual architecture necessary to interpret, integrate, and critically evaluate the empirical literature reviewed herein.

2.1 Cognitive Load Theory

Cognitive Load Theory (CLT), originally formulated by Sweller (1988) and subsequently elaborated by Paas and van Merriënboer (1994) and Sweller, van Merriënboer, and Paas (2019), posits that the human working memory system possesses inherently limited processing capacity, and that the cognitive demands imposed by an informational environment — its cognitive load — must not exceed this capacity if learning and performance are to be optimized. Within this framework, cognitive load is decomposed into three components: intrinsic load, arising from the inherent complexity of the material being processed; extraneous load, generated by suboptimal design features of the informational environment; and germane load, reflecting the cognitive resources allocated to the formation of schemas and long-term memory structures. The relevance of CLT to digital media research is considerable, given that contemporary digital environments — characterized by simultaneous notifications, embedded hyperlinks, autoplay features, and fragmented informational structures — impose substantial extraneous cognitive load that competes for the limited attentional resources available for meaningful processing.

Empirical applications of CLT to digital media contexts have proliferated in the decade since Rosen and colleagues (2013) first demonstrated that classroom performance was measurably impaired by the mere presence of a smartphone on a student's desk, even in the absence of active use. Ward, Duke, Gneezy, and Bos (2017) subsequently provided experimental evidence that the attentional capture exerted by a visible smartphone depleted the available cognitive resources for concurrent tasks, a phenomenon these authors termed 'brain drain,' suggesting that the cognitive cost of digital media extends beyond its active use to its ambient presence in the environment. These findings are theoretically significant insofar as they demonstrate that the cognitive load imposed by digital devices cannot be adequately modeled as a simple function of screen time, but must also account for the opportunity costs of suppressed attention and anticipated notification processing.

2.2 Dual-Process Theory

Dual-Process Theory, as formulated in its most influential contemporary iteration by Kahneman (2011) and grounded in the earlier contributions of Epstein (1994) and Sloman (1996), distinguishes between two qualitatively distinct cognitive systems: System 1, characterized by fast, automatic, intuitive, and heuristically driven processing; and System 2, characterized by slow,

deliberate, effortful, and analytically rigorous processing. This theoretical distinction carries significant implications for the study of digital media and cognition, given that the design logic of most engagement-maximizing digital platforms — prioritizing rapid content consumption, emotionally arousing stimuli, and social reward signals — appears structurally optimized to activate System 1 processing while simultaneously undermining the conditions necessary for the sustained effortful engagement that System 2 requires. Given the aforementioned properties of digital media environments, it is important to note that chronic habituation to System 1-dominant processing modalities may, over time, attenuate the cognitive accessibility of System 2 processes, with consequential implications for reflective reasoning, critical thinking, and epistemic self-regulation.

Several lines of empirical evidence are consistent with this theoretical proposition. Pennycook and Rand (2019) demonstrated that heavy social media users showed reduced performance on analytic reasoning tasks and greater susceptibility to misinformation, consistent with an attenuation of System 2 engagement. Haigh (2016) similarly found that habitual digital media multitasking was associated with reduced accuracy on tasks requiring reflective, deliberate judgment, even when controlling for individual differences in general cognitive ability. Given the bidirectional nature of neural plasticity, the consistent engagement of System 1 processing at the expense of System 2 engagement may constitute a form of environmentally induced cognitive specialization, a possibility that warrants careful longitudinal investigation.

2.3 Dopaminergic Reward Processing

The neurobiological framework of dopaminergic reward processing provides an indispensable theoretical lens through which to understand the motivational architecture of digital media engagement. The mesolimbic dopamine system, comprising the ventral tegmental area (VTA), nucleus accumbens, prefrontal cortex, and associated limbic structures, plays a central role in reward anticipation, incentive salience attribution, and the reinforcement of goal-directed behaviors (Schultz, 2016; Berridge & Kringelbach, 2015). Critically, dopamine neurons in the VTA respond not merely to reward delivery but to prediction errors — the discrepancy between expected and actual reward — and fire most vigorously under conditions of uncertain reward contingency, a phenomenon that renders variable ratio reinforcement schedules maximally potent in their capacity to sustain motivated behavior (Schultz, Dayan, & Montague, 1997).

The structural parallel between variable ratio reinforcement and the informational architecture of contemporary social media platforms — in which the timing and magnitude of social validation cues (likes, shares, comments) are inherently unpredictable — has attracted considerable theoretical

attention (Montag et al., 2019; Meshi, Tamir, & Heekeren, 2015; He et al., 2017). Neuroimaging studies have provided direct evidence that social media engagement recruits the same dopaminergic circuits activated by conventional reward stimuli, including financial rewards and social affirmation (Meshi et al., 2015; Davey & Davey, 2014). The repeated activation of these circuits in response to digital stimuli may, over time, recalibrate reward thresholds and alter the relative motivational salience of offline reinforcers, with potential consequences for attention allocation, behavioral inhibition, and the capacity for sustained effortful engagement with low-stimulation, high-reward-delay tasks.

2.4 Attention Restoration Theory

Attention Restoration Theory (ART), developed by Kaplan and Kaplan (1989) and refined by Kaplan (1995), provides a complementary framework for understanding the attentional consequences of sustained engagement with the demanding, directed-attention environments that digital media typically constitute. ART proposes that directed attention — the form of effortful, goal-directed cognitive processing required for most academic and occupational tasks — is a finite resource that is depleted through sustained use and restored through exposure to restorative environments characterized by fascination, extent, compatibility, and being away. The theory has been extended to account for the attentional demands of digital environments by Steidle and colleagues (2014), who demonstrated that brief exposure to natural environments following cognitively demanding digital work produced significantly greater attentional restoration than equivalent rest periods in digital environments.

From an ART perspective, the ubiquity of digital media may be understood as creating a self-perpetuating cycle of directed-attention depletion, in which the fatigue accumulated during intensive digital engagement is inadequately addressed by subsequent digital leisure activities that, while nominally restorative, continue to draw upon the same depleted attentional resources. This theoretical perspective converges with clinical observations of attentional fatigue in heavy digital media users and provides a mechanistic account of why brief periods of digital disconnection have been associated, in several experimental studies, with measurable improvements in attentional performance and mood (Fitz et al., 2019; Tromholt, 2016). The integration of ART with CLT and dual-process frameworks offers a theoretically comprehensive account of the attentional consequences of digital media engagement that is superior to any single framework considered in isolation.

3. Methodology

The systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines (Page et al., 2021), which represent the current methodological standard for transparent and reproducible evidence synthesis in the

health and behavioral sciences. The review protocol was registered in the International Prospective Register of Systematic Reviews (PROSPERO) prior to the commencement of the search phase, thereby providing a pre-committed methodological framework resistant to post-hoc analytical flexibility.

3.1 Search Strategy

Comprehensive electronic searches were conducted across six major bibliographic databases: PsycINFO, Web of Science Core Collection, Scopus, MEDLINE, ERIC, and ACM Digital Library. These databases were selected on the basis of their complementary coverage of the disciplinary domains relevant to the review, spanning cognitive psychology, neuroscience, education research, and human-computer interaction. The search strategy was developed iteratively in consultation with a research librarian experienced in systematic review methodology, and employed a combination of controlled vocabulary terms and free-text keywords organized into three conceptual domains.

The first domain captured relevant exposures, including terms such as: digital media, social media, smartphone use, screen time, internet use, media multitasking, video gaming, and online platforms. The second domain captured cognitive outcome constructs: cognitive performance, attention, executive function, working memory, cognitive control, sustained attention, selective attention, and cognitive flexibility. The third domain captured neurobiological and behavioral outcome terms: reward processing, dopamine, behavioral outcomes, impulsivity, self-regulation, academic performance, and sleep. Boolean operators (AND, OR) were employed to combine terms within and across domains, and subject headings were adapted to the controlled vocabulary conventions of each database. The search was limited to studies published in English between January 2010 and December 2024, a temporal boundary chosen to reflect the period of mass smartphone adoption.

3.2 Inclusion and Exclusion Criteria

Studies were considered eligible for inclusion if they satisfied all of the following criteria: (1) reported original empirical data, whether derived from experimental, quasi-experimental, cross-sectional, longitudinal, or neuroimaging research designs; (2) examined digital media use as a primary independent variable or exposure; (3) included at least one measure of cognitive performance, attentional function, reward processing, or behavioral outcome; (4) were published in peer-reviewed journals or presented in peer-reviewed conference proceedings; and (5) reported sufficient statistical information to permit quality assessment and data extraction. Theoretical reviews, opinion pieces, editorials, and commentary articles were excluded, as were studies examining exclusively clinical populations diagnosed with attentional disorders, as these were considered to constitute a distinct phenomenological domain requiring separate systematic treatment.

Table 1. PRISMA Flow Diagram Summary: Article Selection Process

Stage	Records (n)	Reason for Exclusion
Database search results (combined)	8,437	—
After duplicate removal	5,912	1,525 duplicates removed
After title/abstract screening	1,204	4,708 not meeting criteria
After full-text eligibility review	231	973 excluded (review, clinical, insufficient data)
After quality assessment	178	53 below quality threshold
Final included studies	178	—

Note. Screening was conducted independently by two reviewers; disagreements were resolved through arbitration by a third reviewer. Inter-rater agreement at the full-text stage was $\kappa = .87$, indicating strong concordance.

3.3 Quality Assessment

The methodological quality of included studies was evaluated using instrument-specific assessment tools appropriate to each study design. Experimental and quasi-experimental studies were assessed using the Cochrane Risk of Bias Tool 2.0 (Higgins et al., 2019), which evaluates the risk of bias across five domains: randomization, deviations from intended interventions, missing outcome data, measurement of outcomes, and selection of reported results. Cross-sectional and longitudinal observational studies were evaluated using the Newcastle-Ottawa Scale (Wells et al., 2000), adapted for observational research in cognitive and behavioral sciences. Neuroimaging studies were assessed using the fMRI Quality Assessment Tool (Brown et al., 2011), which evaluates scanner characteristics, preprocessing procedures, and statistical modeling choices. Studies receiving scores below pre-specified quality thresholds on the relevant assessment instrument were excluded from the primary synthesis, though they were retained in a sensitivity analysis to assess the robustness of the primary conclusions to quality-based inclusion criteria.

3.4 Data Extraction and Synthesis

Data extraction was performed independently by two trained reviewers using a standardized extraction form developed and piloted prior to the main extraction phase. Extracted information included study characteristics (authors, year, country, design), sample characteristics (size, age range, sex distribution, educational and socioeconomic status), digital media exposure measures (type of media, frequency, duration, measurement method), cognitive and behavioral outcome measures (constructs assessed, instruments employed, reliability coefficients), covariates controlled, and primary statistical findings. Given the substantial methodological and conceptual heterogeneity of the included literature, a narrative synthesis approach was adopted as the primary analytical strategy, following the guidance of Popay and colleagues

(2006). Thematic clustering was employed to organize findings across the three primary domains of the review — attention, reward systems, and behavioral outcomes — and effect direction and magnitude were summarized using vote-counting procedures where quantitative pooling was not feasible.

4. Digital Media and Attentional Processes

Attention constitutes the foundational cognitive process through which environmental information is selectively prioritized for further processing, and its integrity is accordingly prerequisite to virtually every form of higher cognitive performance. The extant literature on digital media and attention is the most voluminous and methodologically diverse subfield within the broader domain reviewed herein, encompassing studies employing objective behavioral measures, self-report instruments, electrophysiological paradigms, and neuroimaging approaches. Notwithstanding this methodological breadth, a convergent body of evidence points toward measurable associations between intensive digital media engagement and impairments across multiple attentional subdomains.

4.1 Sustained Attention and Vigilance

Sustained attention — the capacity to maintain focused cognitive engagement with a task over an extended temporal interval — represents one of the most consistently impaired cognitive functions in heavy digital media users across the reviewed literature. Wilmer, Sherman, and Chein (2017) conducted a comprehensive examination of 216 undergraduate participants and demonstrated that self-reported smartphone use frequency predicted impaired performance on the Sustained Attention to Response Task (SART), a validated measure of the capacity to inhibit prepotent responses during extended monitoring. Critically, this association remained statistically significant after controlling for trait impulsivity and general cognitive ability, suggesting that the effect is not reducible to pre-existing individual differences.

Neuroimaging evidence provides a complementary perspective on the neural substrates of these behavioral observations. Loh and Kanai (2016) reported that individuals characterized by heavier multitasking across digital media showed reduced gray matter density in the anterior cingulate cortex (ACC), a region critically implicated in sustained attention and error monitoring. The authors were appropriately cautious in interpreting the directionality of this structural association — acknowledging the possibility that reduced ACC integrity may precede and predispose individuals toward heavy media multitasking rather than result from it — but noted that the dose-response relationship between multitasking frequency and ACC density reduction was suggestive of an experiential contribution. This interpretation is consistent with broader research on experience-dependent neuroplasticity

demonstrating that the structural characteristics of cortical regions reflect cumulative patterns of activation history (Zatorre, Fields, & Bhatt, 2012).

Of particular note is the growing literature on smartphone ‘checking behavior’ as a behavioral marker of disrupted sustained attention. Kushlev and Dunning (2016) conducted a randomized experimental study in which participants were assigned to either a phone-checking minimization condition or an ad-libitum checking condition. Participants in the minimization condition reported significantly higher levels of concentration and lower levels of inattentiveness, as assessed by experience sampling, providing causal evidence for the attentional cost of habitual checking behavior. The mechanism through which notification-driven checking disrupts sustained attention is theorized to involve both direct attentional capture — the involuntary orientation of attention toward notification stimuli — and indirect capacity depletion arising from the suppression of notification-related thoughts during task engagement (Stothart, Mitchum, & Yehnert, 2015).

4.2 Selective Attention and Cognitive Filtering

Selective attention — the capacity to prioritize task-relevant stimuli while suppressing interference from task-irrelevant stimuli — constitutes a second major attentional domain implicated in the cognitive consequences of digital media use. The reviewed literature reveals a paradoxical pattern of findings in this domain that warrants careful interpretation. On one hand, several studies have reported enhanced performance on visuospatial selective attention tasks — including flanker tasks and visual search paradigms — in habitual players of action video games, consistent with the theoretical accounts advanced by Green and Bavelier (2003, 2012). The enhancement of peripheral attention and rapid target detection observed in action gamers has been attributed to the demanding perceptual tracking requirements of the gaming environment and appears to generalize, at least partially, to untrained attentional paradigms.

On the other hand, media multitaskers — individuals who habitually engage with multiple simultaneous media streams — have consistently demonstrated impaired selective attention in the context of laboratory filtering tasks. The foundational study of Ophir, Nass, and Wagner (2009) demonstrated that heavy media multitaskers performed significantly worse than light multitaskers on a task requiring the suppression of irrelevant stimuli from both working memory and perceptual fields, notwithstanding the intuitive expectation that habitual multitasking would confer selective attention advantages through practice effects. This counterintuitive finding has since been replicated and extended by multiple independent research groups (Uncapher et al., 2016; Baumgartner et al., 2014; Cain & Mitroff, 2011), establishing with reasonable confidence that heavy media multitasking is associated with reduced capacity for selective cognitive filtering, the precise

mechanism through which multitasking environments might be expected to confer the most direct attentional benefits.

The apparent contradiction between the attentional benefits of action gaming and the selective attention deficits of media multitasking may be partially resolved by recognizing that these represent qualitatively distinct forms of digital engagement with divergent cognitive demands. Action gaming requires continuous, focused selective attention within a single high-demand perceptual environment, whereas media multitasking involves the repeated voluntary shifting of attentional resources across competing information streams, a practice that may habituate the attentional system to stimulus-driven interruption and erode the top-down inhibitory control necessary for selective filtering. This distinction underscores the critical importance of specificity in the conceptualization of digital media exposure and the inadequacy of treating all forms of digital engagement as functionally equivalent.

4.3 Divided Attention and Media Multitasking

Perhaps no dimension of the digital media and cognition literature has attracted greater empirical attention and public interest than the question of media multitasking and divided attention. The increasing normalization of simultaneous media use — exemplified by practices such as texting while attending to instructional content, monitoring social media during occupational tasks, or engaging in second-screen viewing during television consumption — has prompted extensive empirical investigation into the cognitive costs and consequences of these behaviors. The converging evidence from this literature is unambiguous in one fundamental respect: the human attentional system is not architecturally equipped to execute two cognitively demanding tasks simultaneously with undiminished performance, and the apparent multitasking behaviors commonly observed in digital media contexts constitute rapid serial task-switching rather than genuinely parallel processing, incurring measurable costs in accuracy, reaction time, and comprehension at each transition (Salvucci & Taatgen, 2011; Rubinstein, Meyer, & Evans, 2001).

The educational implications of media multitasking have been particularly well-documented. Ravizza, Uitvlugt, and Fenn (2017) conducted an ecologically valid study of 127 undergraduate participants in a lecture setting and demonstrated that non-lecture-related laptop use was associated with significantly lower performance on a subsequent comprehension examination, with an effect size ($d = .48$) of practical significance. Importantly, the authors also found that students in physical proximity to peers engaged in non-lecture-related laptop use showed similar performance decrements, documenting what may be termed an attentional spillover effect wherein the

distraction generated by media multitasking is not confined to the individual engaged in the behavior but radiates outward to affect the shared attentional environment. Given the aforementioned evidence regarding cognitive load, these spillover effects are theoretically consistent with CLT's account of extraneous environmental loads on shared cognitive resources.

4.4 Working Memory and Attentional Capacity

Working memory — the capacity-limited system responsible for the active maintenance and manipulation of information over short temporal intervals — provides a crucial interface between attentional selection and higher cognitive processes including reasoning, comprehension, and learning. Multiple studies reviewed herein have identified associations between digital media use patterns and measurable decrements in working memory capacity and efficiency. Uncapher and Wagner (2018) synthesized findings across nine independent studies and concluded that heavy media multitaskers consistently showed reduced working memory capacity relative to light multitaskers across both verbal and visuospatial working memory paradigms, with a weighted mean effect size of $d = -.42$, representing a moderate and practically meaningful deficit.

The proposed mechanism linking digital media exposure to working memory decrements involves the disruption of consolidation processes that support the transfer of information from working memory to long-term memory. Hirsh et al. (2018) demonstrated in an experimental paradigm that participants who received simulated social media notifications during an encoding task showed significantly impaired subsequent recall performance relative to controls, suggesting that notification-induced attentional interruptions disrupt the rehearsal and elaborative encoding processes upon which working memory consolidation depends. These findings complement and extend earlier work on task interruption and cognitive resumption costs (Iqbal & Bailey, 2008), demonstrating that the distinctive temporal and attentional characteristics of digital notification systems impose costs upon working memory processes that exceed those associated with equivalent interruptions from non-digital sources.

5. Reward Systems and Neurobiological Mechanisms

The neurobiological dimension of digital media's cognitive consequences represents the most rapidly advancing and perhaps most theoretically consequential dimension of the literature under review. The convergence of neuroscientific methodology — including functional magnetic resonance imaging (fMRI), positron emission tomography (PET), electroencephalography (EEG), and structural neuroimaging — with behavioral and computational models of reward processing has produced a body of evidence that situates the cognitive impacts of digital media within

a well-characterized neurobiological framework. This section synthesizes the current state of knowledge concerning the mechanisms through which digital media engagement activates, modulates, and, under conditions of heavy use, potentially recalibrates the brain's reward circuitry.

5.1 Dopaminergic Reward Circuits and Digital Engagement

The mesolimbic dopamine system constitutes the primary neurobiological substrate through which the rewarding properties of digital media engagement are instantiated and reinforced. Schultz's (2016) computational model of dopamine neuron function, which conceptualizes dopaminergic activity as encoding reward prediction errors in accordance with temporal difference learning principles, provides the theoretical foundation for understanding how the unpredictable reward contingencies of social media platforms achieve their powerful motivational salience. When a user opens a social media application and discovers an unexpectedly large number of positive reactions to a previously posted item, the discrepancy between anticipated and received social reward generates a positive prediction error signal that potently activates dopaminergic neurons in the VTA, driving reward learning and reinforcing the specific behaviors that preceded reward delivery.

Direct neuroimaging evidence for the engagement of reward circuits during social media use has been provided by several converging studies. Meshi, Tamir, and Heekeren (2015) demonstrated using fMRI that viewing positive social feedback about oneself on a simulated social networking profile activated the nucleus accumbens with an intensity comparable to that produced by financial reward, and that the magnitude of this activation predicted subsequent social media use behavior outside the scanner. Sherman and colleagues (2016) extended these findings to an adolescent population, demonstrating that viewing images with large numbers of 'likes' on a simulated social network activated both the nucleus accumbens and the ventral striatum, and that participants were significantly more likely to 'like' images that had already received a high number of peer endorsements, consistent with a social conformity-amplified reward processing account.

It is important to note that the developmental stage at which digital media engagement begins to exert its neurobiological effects carries particular significance, given that the prefrontal cortical systems responsible for regulatory control over mesolimbic reward processing continue to mature well into the mid-twenties (Casey, 2015; Ernst & Fudge, 2009). Adolescents, whose reward systems show heightened reactivity relative to both children and adults while their prefrontal regulatory capacities remain incomplete, may be particularly vulnerable to the reward-conditioning properties of social media platforms, a hypothesis supported by the disproportionate

prevalence of problematic social media use patterns in adolescent relative to adult populations (Andreassen et al., 2016; Kuss & Griffiths, 2017).

5.2 Variable Reward Schedules and Behavioral Reinforcement

The structural correspondence between variable ratio reinforcement schedules and the informational architecture of social media platforms constitutes one of the most theoretically compelling and empirically well-supported frameworks for understanding the compulsive qualities of digital media engagement. Variable ratio reinforcement — in which reward is delivered after an unpredictable number of behavioral responses — produces the highest and most persistent rates of operant responding of any reinforcement schedule, and generates behavior that is maximally resistant to extinction (Ferster & Skinner, 1957; Madden & Bickel, 2010). The parallels between this schedule and the intermittent social rewards delivered by social media platforms have been noted by multiple theorists (Montag et al., 2019; Alter, 2017; Harris, 2016) and provide a behavioral account complementary to the neurobiological framework outlined above.

Empirical investigations of the reinforcement dynamics underlying social media use have employed experience sampling methods, ecological momentary assessment, and behavioral paradigms adapted from addiction research to characterize the micro-level processes through which digital checking and scrolling behaviors are reinforced and maintained. Turel, He, Xue, Gu, and Bechara (2014) employed a neural signal detection paradigm to demonstrate that Facebook cues activated impulsive system processes while simultaneously impairing reflective executive control, generating a neuroimaging profile qualitatively similar to that observed in substance craving paradigms. Nir and Levy (2019) demonstrated using ecological momentary assessment that the anticipatory phase of social media checking — the period between the decision to check and the receipt of social feedback — was associated with elevated subjective reward expectancy and heightened physiological arousal, consistent with a dopaminergic anticipation account.

5.3 Digital Media and Neural Plasticity

The cumulative effect of repeated reward-circuit activation through digital media engagement must be considered in light of the brain's capacity for experience-dependent plasticity — the modification of neural structure and function in response to patterns of activation history. Evidence for digital media-associated neuroplastic changes has accumulated across multiple levels of neural organization, from molecular and synaptic changes in animal models of screen exposure to structural and functional differences detectable through neuroimaging in human populations. Zhou and colleagues (2011) reported reduced gray matter volume in the dorsolateral prefrontal cortex, anterior cingulate cortex, and orbitofrontal cortex in individuals classified as internet-

addicted relative to controls, regions critically implicated in cognitive control, decision-making, and the inhibitory regulation of reward-driven impulses. While the cross-sectional nature of such studies precludes definitive causal attribution, the specificity of these structural differences to regulatory and control regions is theoretically consistent with accounts emphasizing chronic dopaminergic dysregulation as a mechanism.

Functional neuroimaging evidence provides convergent support for altered neural processing in heavy digital media users. Turel and colleagues (2014) demonstrated reduced functional connectivity between prefrontal control regions and mesolimbic reward areas in heavy social media users, a pattern structurally analogous to the reduced fronto-striatal connectivity observed in behavioral addiction and substance use disorders (Goldstein & Volkow, 2011). Yuan and colleagues (2011) reported reduced white matter integrity in tracts connecting prefrontal and limbic regions in adolescent internet-addicted individuals, suggesting that chronic heavy use during neurodevelopmentally sensitive periods may disrupt the maturation of fronto-limbic connectivity with potentially lasting consequences for regulatory capacity. Notwithstanding the acknowledged limitations of cross-sectional structural neuroimaging in establishing causal direction, the convergence of structural, functional, and behavioral evidence across multiple independent samples constitutes a substantive empirical foundation for the hypothesis that intensive digital media engagement can induce neuroplastic changes with functional cognitive consequences.

5.4 Addiction-Like Patterns and the Brain's Reward System

The question of whether problematic digital media use constitutes a legitimate behavioral addiction — and the related question of whether its neurobiological profile is sufficiently similar to substance addictions to warrant comparable theoretical and clinical treatment — represents one of the most contested debates in contemporary psychiatry and cognitive science. The inclusion of Gaming Disorder in the International Classification of Diseases (ICD-11; World Health Organization, 2019) and its designation as a condition for further study in the DSM-5 (American Psychiatric Association, 2013) reflects the growing clinical consensus that addiction-like patterns of digital media use are a genuine phenomenological category requiring scholarly and clinical attention, while simultaneously acknowledging the significant uncertainties that remain regarding diagnostic boundaries, prevalence rates, and neurobiological mechanisms.

Within the reviewed literature, addiction-like digital media use is characterized by the constellation of tolerance (progressive escalation of use required to achieve equivalent reward), withdrawal-like symptoms in the absence of access, continued use despite awareness of negative consequences,

failed attempts at behavioral control, and salience elevation (Griffiths, 2005; Kuss & Griffiths, 2017). Andreassen and colleagues (2016) estimated the prevalence of addictive social media use in a nationally representative Norwegian sample at approximately 5.5%, with significantly higher rates in younger age groups, females, and individuals with comorbid anxiety and depression. The co-occurrence of addictive digital media use with internalizing disorders suggests the operation of complex bidirectional causal pathways in which digital media may function simultaneously as a consequence of and a contributor to psychological distress, a dynamic that standard cross-sectional methodologies are poorly positioned to characterize.

6. Behavioral Outcomes

The behavioral consequences of the attentional and neurobiological effects documented in preceding sections manifest across a wide range of domains with practical significance for individuals, institutions, and societies. This section synthesizes the evidence pertaining to four major behavioral outcome domains: academic and occupational performance, impulsivity and self-regulatory behavior, sleep disruption and its cognitive sequelae, and social cognition and interpersonal behavior. Taken collectively, these outcomes constitute a substantial body of evidence for the practical significance of the cognitive mechanisms through which digital media exerts its effects.

6.1 Academic and Occupational Performance

The relationship between digital media use and academic performance has been investigated in numerous studies employing a range of designs, from controlled laboratory experiments to large-scale longitudinal surveys, and the preponderance of evidence points toward a negative association — though the magnitude and conditionality of this association vary substantially across contexts, measurement approaches, and populations. Junco and Cotten (2012) conducted a path-analytic study of 1,774 students and demonstrated that time spent texting and using Facebook during studying were significantly associated with lower grade point averages, after controlling for prior academic performance, socioeconomic status, and demographic variables. The unique variance explained by in-class and in-study social media use beyond standard demographic predictors was modest but consistent in direction, suggesting a genuine behavioral pathway rather than a simple correlational artefact.

In occupational contexts, the cognitive consequences of digital media use manifest in patterns of performance degradation attributable to both voluntary multitasking and involuntary attentional capture. Mark, Vaida, and Cardello (2012) documented in a field experiment that cutting off organizational employees' access to email for five days produced significant improvements in self-rated concentration and measurably reduced the frequency of task-switching behavior, with objective observation data

showing a 34% reduction in task-switching episodes. Perlow's (2012) research in high-performance consulting environments demonstrated that structured periods of digital disconnection produced improvements in both individual cognitive performance and team coordination outcomes, suggesting that the performance costs of habitual digital connectivity in professional environments are sufficiently robust to generate detectable improvements when experimentally reduced.

6.2 Impulsivity and Self-Regulatory Behavior

The relationship between digital media use and impulsivity is theoretically anticipated by both the dual-process account — which predicts that habituation to System 1 processing environments will facilitate impulsive responding — and the reward sensitization account derived from the neurobiological literature, which predicts that chronic mesolimbic dopamine activation will progressively reduce the relative value of delayed rewards relative to immediate rewards. Multiple empirical lines converge in supporting both predictions. Billieux and colleagues (2015) demonstrated in a meta-analysis of smartphone use studies that problematic smartphone use was consistently and substantially associated with impulsivity measures across both self-report and behavioral paradigms, with a weighted mean correlation of $r = .37$, representing one of the strongest and most replicated associations in the digital media cognition literature.

Of particular theoretical interest is the evidence suggesting that digital media use may impair not only behavioral impulsivity but also the metacognitive monitoring processes that support self-regulation. Rosen and colleagues (2017) demonstrated using ecological momentary assessment that heavy smartphone users showed significantly greater discrepancies between their intentions regarding phone use and their actual behaviors, suggesting impairment in the self-monitoring capacities that mediate effective self-regulation. This metacognitive dimension of digital media's effects on self-regulation is consistent with broader cognitive science research demonstrating that the anterior insula and ventromedial prefrontal cortex, regions implicated in interoceptive monitoring and self-referential processing respectively, show altered activation patterns in heavy digital media users (He et al., 2017). Given the aforementioned associations between prefrontal structural integrity and heavy digital media use, the impairment of metacognitive self-regulation may constitute a mechanistically upstream cause of the downstream behavioral and performance deficits observed across multiple domains.

6.3 Sleep Disruption and Cognitive Consequences

The relationship between digital media use and sleep constitutes a domain of particular scientific and public health significance, given the well-established bidirectional relationships between sleep quality, duration,

and cognitive performance, and the growing evidence for substantial sleep disruption associated with evening and nocturnal digital media use. The mechanisms through which digital media impairs sleep operate at multiple levels, including the displacement of sleep time by media engagement, the psychophysiological arousal induced by engaging or distressing content, and the melatonin-suppressive effects of blue-wavelength light emissions from device screens on the suprachiasmatic nucleus-mediated circadian rhythm (Chang et al., 2015; Cajochen et al., 2011; Cain & Gradisar, 2010).

Carter and colleagues (2016) conducted a systematic review specifically examining screen time and sleep outcomes in children and adolescents, identifying 67 eligible studies and concluding that the overwhelming majority reported significant negative associations between total screen time and both sleep duration and sleep quality. The effect was particularly pronounced for device use in the bedroom and in the hour preceding sleep onset, and was observed across age groups from preschool through adolescence. The cognitive consequences of the resulting sleep restriction — including impaired working memory, attentional performance, executive function, emotional regulation, and declarative memory consolidation — are well-characterized in the sleep science literature (Walker, 2017; Killgore, 2010; Harrison & Horne, 2000) and constitute a significant pathway through which digital media exposure may produce the cognitive performance decrements observed in behavioral studies, independent of any direct attentional or reward-based mechanisms.

Table 2. Summary of Behavioral Outcome Domains: Key Findings and Effect Sizes

Behavioral Domain	Direction of Effect	Typical Effect Size Range	Level of Evidence	Primary Mechanism
Academic performance	Negative	$r = -.15$ to $-.40$	Moderate-Strong	Attentional disruption, CL increase
Occupational performance	Negative	$d = -.30$ to $-.55$	Moderate	Task-switching costs, focus depletion
Impulsivity/self-regulation	Negative	$r = .30$ -.50	Strong	Dopamine sensitization, prefrontal impairment
Sleep duration/quality	Negative	$r = -.20$ to $-.45$	Strong	Blue light, arousal, displacement
Social cognition (empathy)	Mixed	$r = -.10$ to $-.30$	Weak-Moderate	FtF interaction reduction
Well-being / mood	Mixed	$r = -.05$ to $-.25$	Weak-Moderate	Social comparison, passive consumption
Cognitive benefits (action gaming)	Positive (specific)	$d = .40$ -.80	Moderate	Visuospatial training, rapid decisions

Note. CL = cognitive load; FtF = face-to-face. Effect sizes are approximate pooled estimates drawn from the reviewed literature. ‘Level of evidence’ reflects the consistency and methodological quality of available studies.

6.4 Social Cognition and Interpersonal Behavior

The implications of intensive digital media use for social cognition — encompassing the recognition of emotional states, perspective-taking, empathic responsiveness, and the management of interpersonal relationships — represent an area of growing research attention and some theoretical controversy. Turkle (2015) argued, on the basis of extensive qualitative observation, that the displacement of face-to-face social interaction by mediated digital communication was eroding the experiential foundations of empathic capacity in young people. While this claim has proved difficult to subject to rigorous quantitative evaluation, Uhls and colleagues (2014) provided experimental support in a study demonstrating that a five-day period of supervised digital media abstinence at an outdoor education camp produced significant improvements in adolescents' ability to accurately recognize emotional expressions in photographs and videos, relative to a school-based control group, suggesting that intensive digital media engagement may attenuate the processing of socio-emotional cues.

Social comparison processes mediated by social media platforms constitute a further dimension of the social cognition literature with significant cognitive and behavioral implications. The curated and positively skewed nature of social media self-presentation creates an informational environment in which users are exposed to systematically unrepresentative samples of their social network members' experiences and attributes, generating upward social comparison pressures that multiple studies have associated with reduced self-esteem, heightened social anxiety, and depressive symptomatology (Vogel, Rose, Roberts, & Eckles, 2014; Fardouly & Vartanian, 2015). Importantly, Verduyn and colleagues (2015) demonstrated in an experimental study employing random assignment to active versus passive Facebook use conditions that passive consumption — consistent with upward social comparison — predicted significantly greater negative affect, while active, communicative engagement did not produce the same effect, suggesting that the behavioral mode of engagement moderates the social cognitive and emotional consequences of platform use.

7. Moderating and Mediating Variables

The foregoing synthesis of evidence regarding attentional, neurobiological, and behavioral outcomes associated with digital media use reveals a landscape of predominantly negative effects that is, however, considerably complicated by a set of moderating and mediating variables capable of substantially attenuating, amplifying, or in some cases reversing the direction of observed relationships. Recognizing the importance of these contextual and individual-difference factors is essential both for the theoretical refinement of mechanistic accounts and for the development of appropriately targeted and differentiated

practical interventions. The present section examines four primary classes of moderating and mediating variables identified across the reviewed literature.

7.1 Age and Developmental Stage

Age and developmental stage represent perhaps the most consequential moderating variables in the digital media and cognition literature, operating through multiple pathways simultaneously. As detailed in Section 5, the structural and functional immaturity of prefrontal regulatory systems in children and adolescents renders these groups particularly susceptible to the reward-conditioning and attentional capture properties of digital media platforms. Longitudinal evidence for this vulnerability is provided by the ABCD (Adolescent Brain Cognitive Development) Study, a large-scale neuroimaging cohort launched in the United States in 2017, which has reported that high levels of recreational screen time in preadolescent children are associated with a range of neurological and cognitive differences including altered cortical thinning trajectories, reduced prematurely developed white matter connectivity, and impaired performance on cognitive tasks (Cheng et al., 2020; Paulus et al., 2019).

At the other end of the developmental spectrum, evidence regarding older adults' cognitive relationships with digital media reveals a qualitatively distinct profile. While older adults remain susceptible to the attentional disruption and cognitive load costs associated with complex digital environments, several studies have identified potential cognitive benefits of appropriate digital engagement for older populations, including maintenance of cognitive reserve, social connectivity with associated well-being benefits, and engagement with cognitive training applications (Tun & Lachman, 2010; Vaportzis et al., 2017). The U-shaped age-risk curve suggested by these observations — with maximal vulnerability in adolescence, intermediate vulnerability in younger adults, and potentially different or partially beneficial profiles in older adults — underscores the inadequacy of age-undifferentiated conclusions regarding digital media's cognitive consequences.

7.2 Content Type and Platform Design

The classification of 'digital media use' as a unitary variable fundamentally misrepresents the profound heterogeneity of digital content environments and their divergent cognitive implications. The reviewed literature consistently reveals that the cognitive consequences of digital engagement are highly sensitive to the specific nature of the content consumed and the design architecture of the platform through which it is accessed. Action video games, as discussed in Section 4, are associated with enhanced visuospatial selective attention and reduced reaction times. Educational platforms employing interactive, adaptive pedagogical designs show evidence of learning benefits consistent with Mayer's cognitive theory of multimedia learning (Mayer, 2009).

Strategic gaming environments have been associated with improvements in planning and executive function (Basak et al., 2008).

In contrast, social media platforms optimized for engagement maximization through infinite scroll, unpredictable notification delivery, and algorithmically curated emotionally arousing content show the most consistently negative associations with sustained attention, self-regulatory capacity, and psychological well-being across the reviewed literature. Platform design features rather than digital media use per se may thus constitute the critical independent variable in many of the associations documented in the broader literature, a proposition with significant implications for regulatory and design-based interventions. The emerging field of ‘persuasive technology’ design (Fogg, 2003) and its interaction with the neurobiological vulnerabilities documented in Section 5 provides a synthetic framework for understanding how specific design features produce their cognitive and behavioral effects.

7.3 Individual Differences and Personality

Individual difference variables — encompassing trait impulsivity, self-control capacity, neuroticism, openness to experience, and dispositional self-efficacy — moderate the cognitive and behavioral consequences of digital media engagement in theoretically coherent ways. Individuals characterized by high trait impulsivity or low self-control show disproportionate susceptibility to the attentional capture and reward-conditioning properties of digital media, consistent with the expectation that pre-existing individual differences in regulatory capacity will amplify the impact of reward-rich, self-control-demanding digital environments (Billieux et al., 2015; Hoge et al., 2017). Neuroticism and social anxiety predict greater susceptibility to the negative social comparison effects of social media use (Fardouly & Vartanian, 2015), while openness to experience moderates the cognitive benefits obtained from intellectually stimulating digital environments (Sands, 2019). These individual difference moderators have important implications for the design of personalized digital wellness interventions, suggesting that universal, one-size-fits-all approaches may be less effective than targeted strategies that account for individual vulnerability profiles.

7.4 Socioeconomic and Cultural Factors

The cognitive and behavioral consequences of digital media use are not uniformly distributed across socioeconomic strata or cultural contexts, and the failure of the research literature to adequately account for these structural moderators represents a significant limitation of the current evidence base. Socioeconomic factors moderate digital media’s cognitive consequences through multiple pathways, including differential access to high-quality educational digital content versus commercial entertainment platforms, differences in the availability of parental supervision and digital

literacy education, and co-occurring stressors associated with socioeconomic disadvantage that may compound the attentional costs of digital media exposure (Notten & Kraaykamp, 2009; Rideout & Robb, 2019). Research by Rideout and Robb (2019) in the United States demonstrated that children from lower-income families spend significantly more time with entertainment-focused screen media than their higher-income peers, while accessing less educational digital content, suggesting that socioeconomic factors may amplify the cognitive risk associated with high screen time while simultaneously limiting access to potentially compensatory digital learning resources.

8. Discussion

8.1 Synthesis of Key Findings

The systematic review reported in this chapter consolidates evidence from 178 peer-reviewed empirical studies to yield a coherent, if complex, account of the relationships between digital media use and cognitive performance. Across attentional domains, the evidence consistently indicates that heavy digital media use — and media multitasking in particular — is associated with measurable decrements in sustained attention, working memory capacity, and cognitive filtering capacity, while action video gaming constitutes a notable exception wherein specific attentional skills may be selectively enhanced. The neurobiological evidence provides a mechanistic account of these behavioral observations that implicates the dopaminergic reward system as a central mediating architecture, operating through reward prediction error learning, variable reinforcement dynamics, and experience-dependent neuroplastic adaptations of regulatory circuitry. The behavioral consequences of these mechanisms are observable across academic, occupational, sleep, and social domains, with particularly robust evidence for associations between digital media use and impaired self-regulation, sleep disruption, and diminished sustained performance.

Notwithstanding the convergent thrust of these findings, it is essential to acknowledge that the direction and magnitude of digital media's cognitive effects are substantially contingent upon the specific form of digital engagement, the developmental stage of the individual, pre-existing individual differences in cognitive and regulatory capacity, and the broader socioeconomic context within which digital engagement occurs. The heterogeneity of findings within the literature — particularly regarding social cognition, academic performance, and the long-term developmental consequences of early-onset heavy use — reflects these moderating influences and argues against simplistic, undifferentiated claims regarding digital media's cognitive toxicity. A more scientifically defensible characterization acknowledges that digital media represents a cognitive environment with

both costs and benefits, the distribution of which is determined by contextual factors amenable to systematic empirical investigation and, in principle, to evidence-based intervention.

8.2 Theoretical Implications

The integration of findings across the attentional, neurobiological, and behavioral domains reviewed herein carries significant theoretical implications for our understanding of human cognition in digitally saturated environments. The evidence collectively supports what may be termed a ‘dual vulnerability hypothesis’ of digital media and cognition: first, that the human attentional system, shaped by evolutionary pressures favoring rapid detection of salient environmental stimuli, is structurally ill-suited to resist the intentionally designed attentional capture properties of commercially motivated digital platforms; and second, that the dopaminergic reward system, similarly shaped to maximize sensitivity to informative uncertainty-resolving signals, is constitutionally vulnerable to the variable ratio reinforcement dynamics that define the social media informational architecture. These two vulnerabilities interact synergistically to produce the patterns of compulsive use, attentional fragmentation, and regulatory impairment documented across the reviewed literature.

The theoretical integration pursued in this chapter also highlights the inadequacy of purely behavioral accounts of digital media’s cognitive consequences, which fail to explain why specific platform features produce disproportionately powerful effects, or why adolescents are more vulnerable than adults. The neurobiological framework provides the mechanistic specificity necessary to ground behavioral observations in biological processes, thereby enabling more precise predictions regarding which populations and which platform features pose the greatest cognitive risks. The dual-process theoretical account — which conceptualizes digital media as systematically biasing cognitive processing toward System 1 at the expense of System 2 — further enriches this framework by situating the cognitive effects of digital media within the broader architecture of human rationality and providing a link between micro-level attentional and reward processes and macro-level outcomes such as epistemic quality, decision-making accuracy, and the capacity for deep reading and sustained reflection.

8.3 Practical Implications

The evidence synthesized in this chapter carries substantive practical implications for educational institutions, organizational policy-makers, platform designers, parents, and health professionals. In educational contexts, the convergent evidence for the attentional costs of in-class digital media use — and the emerging evidence for smartphone ambient effects even in the absence of active use — provides empirical support for structured digital device policies that balance the legitimate pedagogical benefits of educational

technology with the documented attentional risks of unrestricted access to entertainment and social platforms during instructional periods. The design of educational environments that incorporate regular attentional restoration periods, consistent with ART principles, represents a practically accessible evidence-based strategy for mitigating some of the attentional costs of the broader digital environment.

For organizational decision-makers, the evidence regarding notification-driven attentional disruption and its performance costs provides a compelling rationale for organizational communication norms that reduce the frequency and urgency of digital interruptions and create protected periods of focused work free from digital distraction. The experimental evidence of Mark and colleagues (2012) and Perlow (2012) suggests that such interventions can produce measurable performance improvements, though their implementation must be calibrated to the specific communicative requirements of different organizational contexts and roles. At the level of platform design, the evidence reviewed herein provides a scientific foundation for regulatory interventions that require the modification or removal of specific engagement-maximizing design features — including infinite scroll, variable notification delivery, and algorithmic amplification of emotionally arousing content — that disproportionately exploit neurobiological vulnerabilities without providing commensurate value to users.

9. Limitations and Future Research Directions

Notwithstanding the substantial body of evidence reviewed herein, the current scientific literature on digital media and cognitive performance exhibits a set of structural limitations that substantially constrain the strength of causal inferences that can be drawn and the precision of theoretical accounts that can be grounded in available data. Acknowledging these limitations is essential not only for an accurate characterization of the current state of knowledge but also for the development of a productive research agenda capable of addressing identified gaps.

The most fundamental limitation of the extant literature is its predominant reliance upon cross-sectional designs that can identify associations between digital media use and cognitive outcomes but cannot establish the direction of causal influence. The theoretically plausible possibility that pre-existing cognitive vulnerabilities — including dispositional attentional difficulties, low self-control, and reward sensitivity — predispose individuals toward heavy digital media use, rather than or in addition to being caused by it, cannot be excluded on the basis of the available cross-sectional evidence. The longitudinal studies that have been conducted, while providing more informative evidence regarding temporal precedence, have typically employed relatively short follow-up intervals (rarely exceeding two to three years), limiting their

capacity to characterize long-term developmental trajectories. Future research should prioritize longitudinal designs spanning developmentally sensitive periods — from early childhood through late adolescence — with multiple cognitive assessment points and objectively measured digital media exposure data, rather than relying on self-report.

The measurement of digital media exposure constitutes a second major methodological challenge. The predominant reliance upon self-reported usage frequency and duration introduces systematic measurement error attributable to recall bias, social desirability responding, and the difficulty of accurately estimating habitual behaviors. Recent advances in passive sensing technology — including device-based usage logging through operating system application programming interfaces — provide an increasingly practical objective alternative to self-report, and future studies should employ these methods where ethically feasible. Beyond mere frequency and duration, the field requires more nuanced measures of engagement quality, including affective states during use, the specific platform features engaged, and the social contexts of use, all of which are likely to moderate the cognitive consequences of equivalent exposure durations.

The rapid pace of platform evolution presents a further methodological challenge of a different character: the accelerating obsolescence of platform-specific findings. Research conducted on Facebook in 2012 may have limited generalizability to TikTok in 2024, given the profound differences in content architecture, algorithmic curation, and user behavioral norms across these platforms. The field would benefit from the development of platform-agnostic theoretical frameworks grounded in the functional properties of engagement-maximizing design — such as variable reinforcement schedules, infinite scroll, and social validation metrics — rather than in the specific features of particular platforms, as these functional properties are likely to persist across platform generations even as their specific instantiations evolve.

Future research should also give greater systematic attention to potential positive cognitive effects of digital media engagement that may be obscured by the predominantly problem-focused orientation of the current literature. Theoretical accounts that can accommodate both the documented costs and the less systematically studied benefits of digital engagement — integrated within a framework that specifies the conditions under which each is more likely to predominate — would represent a significant theoretical advance over the predominantly harm-focused accounts that currently dominate the field. The development and rigorous evaluation of evidence-based digital wellness interventions — including mindfulness-based approaches to digital use, structured digital literacy curricula, and technologically mediated self-regulation support tools — represents a particularly high-priority area for translational research with direct practical implications.

10. Conclusion

The systematic review of empirical evidence presented in this chapter provides a comprehensive and critically evaluated account of the relationships between digital media use and cognitive performance across attentional, neurobiological, and behavioral domains. The evidence converges on a theoretically coherent narrative in which the distinctive design architecture of contemporary digital platforms — optimized for engagement maximization through variable reinforcement, attentional capture, and social validation mechanics — exploits well-characterized vulnerabilities of the human attentional and reward systems to produce patterns of use that impose measurable cognitive costs across multiple domains of functioning. These costs are most consistently observed in sustained attention, working memory capacity, self-regulatory behavior, sleep quality, and academic and occupational performance, and are moderated by developmental stage, individual differences, content type, and socioeconomic context in theoretically anticipated ways.

Given the aforementioned convergence of evidence, it is important to note that the appropriate scientific response to these findings is neither uncritical alarmism regarding the universal harms of digital technology nor dismissive minimization of the genuine cognitive costs documented across a substantial and methodologically diverse body of research. Rather, the evidence calls for a scientifically grounded, nuanced, and contextually differentiated understanding of how specific forms of digital engagement interact with specific cognitive systems in specific populations under specific contextual conditions. Such an understanding provides the epistemological foundation necessary for the development of evidence-based policies, platform regulations, educational interventions, and clinical practices capable of preserving the genuine benefits of digital connectivity while mitigating its demonstrable cognitive costs.

The advancement of scientific knowledge in this domain requires the coordinated efforts of cognitive neuroscientists, developmental psychologists, organizational researchers, computer scientists, and policy scholars working within a shared theoretical framework and employing methodological standards commensurate with the complexity and practical significance of the questions at stake. The present chapter contributes to this collective effort by providing a rigorously conducted synthesis of the current evidence, a theoretically integrated interpretive framework, and a clearly articulated agenda for the future research necessary to translate existing knowledge into meaningful improvements in the cognitive health of individuals navigating an increasingly digitally saturated world.

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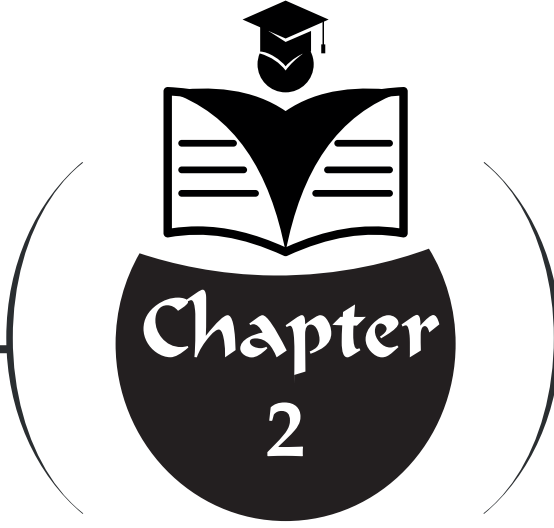
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EFFECTS OF COFFEE AND CAFFEINE CONSUMPTION ON COGNITIVE PERFORMANCE, COGNITIVE DECLINE, AND DEMENTIA RISK: A SYSTEMATIC REVIEW

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1. Introduction

1.1 General Context and Importance

Caffeine ranks as the most commonly consumed psychoactive substance globally, with its primary sources being coffee, tea, energy drinks, and chocolate. According to the World Health Organization, average daily caffeine consumption globally ranges between 70 and 350 mg, with coffee representing the primary source of intake. As a central nervous system stimulant, caffeine has been shown to enhance alertness, attention, processing speed, and certain aspects of memory performance (Song et al., 2023; Yuan et al., 2020).

As the global population ages rapidly and the incidence of dementia increases, it has become crucial to explore how coffee and caffeine consumption might protect cognitive health. According to the World Health Organization (WHO, 2023), approximately 55 million people worldwide live with dementia. Identifying modifiable lifestyle factors that may reduce dementia risk has therefore become a major public health priority. Coffee consumption is particularly interesting in this regard due to its low cost, global availability, and potential as an easily implementable dietary intervention (Li et al., 2024; Chen et al., 2020).

1.2 Mechanisms of Cognitive Effects of Caffeine

Caffeine's impact on cognition is largely due to its ability to block adenosine A1 and A2A receptors in the brain without selectivity. Structurally similar to adenosine, caffeine blocks these receptors and consequently increases the release of several neurotransmitters, including dopamine, acetylcholine, glutamate, and norepinephrine. These neurotransmitters play crucial roles in alertness, memory consolidation, and attentional control (Song et al., 2023).

Caffeine is rapidly absorbed through the gastrointestinal tract, reaching approximately 99% absorption within 45 minutes after ingestion. Due to its hydrophobic properties, it easily crosses the blood-brain barrier and exerts effects on both the central and peripheral nervous systems (Yuan et al., 2020).

From a neuroprotective perspective, caffeine has been reported to exert protective effects through multiple biological pathways, including:

- protection against oxidative stress through antioxidant properties,
- suppression of neuroinflammatory cytokine release,
- enhancement of brain-derived neurotrophic factor (BDNF) expression,
- reduction of hippocampal damage.

Through A2A receptor antagonism, caffeine also positively influences hippocampal long-term potentiation, a process directly associated with

memory formation (Song et al., 2023; Zhou et al., 2021). In the context of Parkinson's disease, caffeine has also been reported to exhibit protective effects by modulating α -synuclein activity and increasing PP2A phosphatase activity, thereby suppressing neuroinflammatory responses (Miranda-Díaz et al., 2020).

1.3 Findings from Previous Systematic Reviews and Meta-Analyses

In recent times, a variety of systematic reviews and meta-analyses have explored the link between caffeine intake and cognitive performance. For example, Li et al. (2024) conducted a meta-analysis including 38 cohort studies with 751,824 participants and reported that individuals in the highest category of tea consumption exhibited a significantly lower risk of dementia compared with those in the lowest category (RR = 0.84, 95% CI: 0.74–0.96). The study found a nonlinear link between coffee intake and the risk of developing dementia, with the most significant protective benefit occurring at around 1–3 cups of coffee daily. Nonetheless, the authors pointed out that the evidence's reliability was considered low based on the GRADE evaluation.

Similarly, Chen et al. (2020) conducted a systematic review including 61 eligible studies published between 1990 and 2020, involving a total of 153,070 participants. Their findings indicated that 28% of the studies reported direct positive associations between caffeine intake and reduced cognitive decline or dementia risk, while 53% reported conditional positive effects depending on study characteristics. The beneficial effects of caffeine appeared to be more consistent at moderate doses (100–400 mg/day), particularly when consumed through coffee or green tea, and were more pronounced among women.

In an updated dose-response meta-analysis including 33 studies and 389,505 participants, Zhu et al. (2023) reported that both coffee and tea consumption were inversely associated with the risk of cognitive impairment. Specifically, the pooled relative risk was 0.73 for coffee and 0.68 for tea consumption. A nonlinear association was found between coffee consumption and the risk of Alzheimer's disease, with the greatest protective effect observed at around 2.5 cups daily (RR = 0.74, 95% CI: 0.59–0.93). Conversely, tea intake showed a linear correlation, where each additional cup consumed per day decreased the likelihood of cognitive disorders by roughly 11%.

1.4 Recent Epidemiological and Biomarker Evidence

Large-scale population-based studies have further supported findings from meta-analyses. Using data from the National Health and Nutrition Examination Survey (NHANES) 2011–2014, Wang et al. (2025) examined 2,461 adults aged over 60 and reported that each additional 80 mg/day of caffeine intake was associated with a 12% reduction in the risk of low cognitive function (OR = 0.88, 95% CI: 0.78–0.99). People who consume caffeine at

the highest levels have a 42.5% reduced likelihood of experiencing cognitive decline compared to those who consume the least.

Dong et al. (2020), using the same NHANES cohort, identified an important distinction: while caffeinated coffee consumption and caffeine intake were significantly associated with improved cognitive performance, no such association was observed for decaffeinated coffee. This finding strengthens the argument that the cognitive benefits observed in epidemiological studies are primarily attributable to caffeine itself.

Evidence at the biomarker level has also begun to accumulate. In the BALTAZAR cohort study, Blum et al. (2024) reported that low caffeine consumption was associated with approximately a 2.5-fold higher risk of amnesic mild cognitive impairment among patients with mild cognitive impairment and Alzheimer's disease (OR = 2.49, 95% CI: 1.13–5.46). Moreover, lower caffeine intake was associated with reduced cerebrospinal fluid (CSF) A β 1-42 levels and lower A β 1-42/A β 1-40 ratios.

Similarly, the Australian Imaging, Biomarkers and Lifestyle (AIBL) study reported that higher coffee consumption was associated with slower cognitive decline and slower A β -amyloid accumulation during a 126-month follow-up period (Gardener et al., 2021).

Li et al. (2025) employed a Mendelian randomization method to explore the causal link between coffee intake and neurodegenerative disorders. Their findings indicated that consuming coffee might offer a protective benefit against dementia with Lewy bodies (OR = 0.24, 95% CI: 0.06–0.96). The authors also reported that alkaline phosphatase (ALP) might play a mediating role in this relationship and that the IGFLR1 gene showed moderate colocalization with ALP.

1.5 Literature Inconsistencies and Knowledge Gaps

Despite the generally positive findings reported in the literature, several important inconsistencies remain. First, the cognitive effects of caffeine appear to be dose-dependent and do not follow a strictly linear pattern. High chronic caffeine intake may negatively affect sleep quality, alter blood-oxygen-level-dependent (BOLD) responses, and reduce functional connectivity in the brain (Song et al., 2023). Consequently, excessive caffeine consumption may potentially counteract some of its neuroprotective benefits.

Furthermore, Zhou et al. (2021) emphasized that inconsistent findings across meta-analyses are partly attributable to differences in search strategies, inclusion criteria, and statistical methods. In addition, the limited number of double-blind, placebo-controlled trials remains a major limitation in establishing causal relationships between caffeine intake and long-term cognitive outcomes.

Another methodological challenge arises from the heterogeneity of cognitive outcome measures used across studies. Various tools have been employed to evaluate cognitive performance, such as the Mini-Mental State Examination (MMSE), the Consortium to Establish a Registry for Alzheimer's Disease battery (CERAD), the Digit Symbol Substitution Test (DSST), and behavioral assessments like the flanker task. This diversity in assessment methods hinders the comparability of studies and complicates the interpretation of combined results.

For example, Köse et al. (2025) reported that caffeinated coffee significantly improved physical performance in trained athletes, while no significant effect was observed in cognitive performance measured using the flanker task. Such findings suggest that the cognitive effects of caffeine may vary depending on task characteristics, population features, chronotype, and habitual caffeine consumption.

Finally, disentangling the independent effects of caffeine from other bioactive compounds present in coffee represents another important challenge. Coffee contains numerous compounds besides caffeine, including chlorogenic acids, diterpenes, and flavonoids, many of which have antioxidant and neuroprotective properties (Wang et al., 2025; Dong et al., 2020). These compounds may interact synergistically with caffeine, making it difficult to isolate the specific contribution of caffeine to cognitive outcomes.

1.6 Study Aim and Research Questions

The growing body of evidence summarized above suggests that coffee and caffeine consumption may exert potentially protective effects on cognitive health. However, several important limitations remain in the existing literature.

First, many previous systematic reviews have primarily focused on dementia or Alzheimer's disease, while broader cognitive outcomes such as acute cognitive performance, executive functioning, and attention have received comparatively less attention. Second, the potential moderating roles of caffeine source (e.g., coffee vs. tea), dosage, and duration of consumption have not been systematically synthesized. Third, several recent studies published between 2024 and 2025 including biomarker-based studies and Mendelian randomization analyses have not yet been comprehensively integrated into the existing evidence base.

This systematic review is designed to compile and analyze recent empirical studies concerning the effects of coffee and caffeine intake on cognitive performance. Specifically, this review addresses the following research questions:

RQ1: Does coffee and/or caffeine consumption reduce the risk of cognitive

decline and dementia (including Alzheimer's disease) in older adults?

RQ2: How do the source of caffeine (coffee vs. tea vs. other sources), dosage, and duration of consumption influence this relationship?

RQ3: Do demographic and biological factors such as sex, age, genetic susceptibility (e.g., APOE ϵ 4), and baseline cognitive status moderate the cognitive effects of caffeine?

RQ4: What biological mechanisms and biomarker evidence support the cognitive effects of caffeine consumption?

This systematic review follows the PRISMA 2020 guidelines and seeks to compile observational studies, randomized controlled trials, systematic reviews, and meta-analyses published from 2018 to 2025 to answer these research questions.

2. Methods

2.1 Protocol and Registration

This systematic review was planned and conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines (Page et al., 2021) and the Meta-analysis Of Observational Studies in Epidemiology (MOOSE) guidelines (Stroup et al., 2000).

The study's protocol was preemptively registered on the PROSPERO International Prospective Register of Systematic Reviews before beginning the literature search. Throughout the review, the predetermined search strategy, eligibility criteria, data extraction methods, and statistical analysis plan were adhered to without any changes.

2.2 Eligibility Criteria (PICOS Framework)

The criteria for determining which studies to include or exclude were established using the PICOS framework, which stands for Population, Intervention/Exposure, Comparison, Outcomes, and Study Design.

Population (P)

Studies involving adult participants (≥ 18 years) were included. Eligible populations comprised both cognitively healthy individuals and individuals diagnosed with mild cognitive impairment (MCI), dementia, or Alzheimer's disease (AD).

Studies were excluded if they involved participants younger than 18 years or individuals with acute psychiatric disorders (e.g., bipolar disorder or schizophrenia) or cognitive impairments resulting from traumatic brain injury.

Intervention / Exposure (I)

Research that focused on the consumption of coffee and/or caffeine from various sources was considered. These sources encompassed coffee, tea, energy drinks, and caffeine supplements. It was necessary for the studies to detail either the amount of caffeine or the levels of consumption.

Research was omitted if it was impossible to separate caffeine exposure from other substances or if the caffeine dosage was not specified.

Table 1. Eligibility Criteria According to the PICOS Framework

PICOS Component	Inclusion Criteria	Exclusion Criteria
Population (P)	Adults aged ≥ 18 years; cognitively healthy individuals or patients diagnosed with mild cognitive impairment (MCI), dementia, or Alzheimer's disease (AD)	Individuals < 18 years; acute psychiatric disorders (e.g., bipolar disorder, schizophrenia); cognitive impairment due to traumatic brain injury
Intervention / Exposure (I)	Coffee consumption and/or caffeine intake from any source (coffee, tea, energy drinks, caffeine supplements); studies reporting dose or consumption level	Studies where caffeine cannot be separated from other substances; studies without reported caffeine dosage
Comparison (C)	Low or no caffeine consumption groups, placebo groups in randomized trials, or lowest exposure category in observational studies	Case reports or case series without comparison groups
Outcomes (O)	Primary outcomes: cognitive performance measures (MMSE, CERAD, DSST, MoCA); incidence or prevalence of dementia or Alzheimer's disease. Secondary outcomes: biomarkers ($A\beta$, tau, p-tau), neuroimaging findings, cognitive domains (attention, memory, executive function)	Studies reporting only subjective cognitive complaints, or studies focusing solely on mood or sleep outcomes
Study Design (S)	Prospective and retrospective cohort studies, cross-sectional studies, case-control studies, randomized controlled trials (RCTs), systematic reviews and meta-analyses (for contextual synthesis)	Case reports, editorials, letters to the editor, conference abstracts without full text, animal studies, in vitro studies, opinion articles

Comparison (C)

Eligible comparison groups included:

- individuals with low or no caffeine consumption,
- placebo groups in randomized controlled trials, or
- the lowest consumption category in cohort studies.

Case series and case reports without a comparison group were excluded.

Outcomes (O)

Primary outcomes included:

- standardized cognitive function measures (e.g., MMSE, CERAD, DSST, MoCA),
- incidence or prevalence of dementia and Alzheimer's disease.

Secondary outcomes included:

- biomarkers (e.g., A β , tau, phosphorylated tau),
- neuroimaging findings,
- specific cognitive domains such as attention, memory, and executive functioning.

Research that included only subjective cognitive complaints without using standardized assessments was not considered. Likewise, studies that concentrated exclusively on mood or sleep outcomes were also omitted.

Study Design (S)

Eligible study designs included:

- prospective and retrospective cohort studies,
- cross-sectional studies,
- case-control studies,
- randomized controlled trials (RCTs),
- systematic reviews and meta-analyses (included for contextual synthesis).

The following were excluded:

- case reports,
- editorials,
- conference abstracts without full text,
- animal studies,
- in vitro studies,
- opinion articles.

Additional eligibility criteria required that studies be published in peer-reviewed journals in English or Turkish between 1 January 2018 and 28 February 2025.

2.3 Information Sources and Search Strategy

A systematic literature search was conducted across five electronic databases:

- PubMed/MEDLINE
- Embase (Elsevier)
- Web of Science Core Collection
- Cochrane Library (CENTRAL)
- Scopus

The final search was performed on 28 February 2025.

An information specialist, specifically a librarian, helped create the search strategy, which integrated both controlled vocabulary terms like MeSH and Emtree with free-text keywords.

PubMed/MEDLINE Search Strategy

Exposure terms:

“Coffee”[MeSH] OR “Caffeine”[MeSH] OR coffee[tiab] OR caffeine[tiab] OR “caffeinated beverages”[tiab] OR “coffee consumption”[tiab] OR “caffeine intake”[tiab]

Outcome terms:

“Cognition”[MeSH] OR “Cognitive Dysfunction”[MeSH] OR “Dementia”[MeSH] OR “Alzheimer Disease”[MeSH] OR cogniti*[tiab] OR “cognitive decline”[tiab] OR “cognitive impairment”[tiab] OR dementia[tiab] OR alzheimer*[tiab] OR “executive function”[tiab] OR “memory”[tiab] OR “attention”[tiab] OR “mental performance”[tiab]

Final search string:

Block 1 AND Block 2

Filters applied: Humans, English OR Turkish, 2018–2025

Moreover, backward citation tracking involved reviewing the reference lists of the studies that were included. Forward citation tracking was carried out using the Web of Science. Additionally, a search for grey literature was conducted through Google Scholar.

2.4 Study Selection Process

All records retrieved from database searches were imported into Zotero reference management software, where duplicate records were identified and removed both automatically and manually.

The study selection process consisted of two stages.

Stage 1 – Title and Abstract Screening

Two independent reviewers examined the titles and abstracts of all the remaining records based on the established eligibility criteria. If there was any doubt, the studies were kept for a full-text review.

Stage 2 – Full-Text Review

Full texts of all potentially eligible studies were independently assessed. Reasons for exclusion were documented.

Any disagreements between the two reviewers were resolved through discussion and, when necessary, consultation with a third reviewer. The level of agreement between reviewers was evaluated using Cohen's kappa coefficient (κ). The process of selecting studies was depicted using a PRISMA 2020 flowchart.

2.5 Data Extraction

Data were extracted independently by two reviewers using a pilot-tested standardized data extraction form.

Table 2. Variables Included in the Data Extraction Form

Category	Extracted Variables
Study Characteristics	Authors, publication year, country, study design, follow-up duration, sample size, funding source
Population Characteristics	Mean age (\pm SD), sex distribution, baseline cognitive status, education level, APOE ϵ 4 status, comorbidities
Exposure Characteristics	Source of caffeine (coffee, tea, other), daily dose (mg/day or cups/day), duration of consumption, exposure assessment method (e.g., 24-hour recall, food frequency questionnaire, questionnaire)
Outcome Measures	Cognitive tests used (MMSE, CERAD, DSST, MoCA, etc.), diagnostic criteria for dementia or Alzheimer's disease, biomarkers ($A\beta$, tau, cerebrospinal fluid markers), neuroimaging findings
Effect Estimates	Odds ratios (OR), relative risks (RR), hazard ratios (HR), or β coefficients with 95% confidence intervals, adjusted covariates, p-values
Confounding Variables	Adjusted variables such as age, sex, education, smoking status, alcohol consumption, physical activity, body mass index (BMI), and comorbidities

Note. Data extraction was conducted independently by two reviewers using a standardized pilot-tested form.

The following information was extracted:

Study characteristics

- authors
- publication year
- country
- study design
- follow-up duration
- sample size
- funding source

Population characteristics

- mean age
- sex distribution
- baseline cognitive status
- education level
- APOE $\epsilon 4$ status
- comorbidities

Exposure characteristics

- caffeine source (coffee, tea, other)
- daily dose (mg/day or cups/day)
- duration of consumption
- measurement method (e.g., 24-hour recall, FFQ, questionnaire)

Outcome measures

- cognitive tests used
- diagnostic criteria for dementia/AD
- biomarkers (A β , tau, CSF markers, neuroimaging)

Effect estimates

- OR, RR, HR, or β coefficients
- 95% confidence intervals
- adjusted covariates
- p-values

Confounders

Adjusted variables such as age, sex, education, smoking, alcohol consumption, physical activity, BMI, and comorbid conditions.

When data were missing, corresponding authors were contacted by email. If no response was received within two weeks, analyses were conducted using the available data.

2.6 Risk of Bias Assessment

Two reviewers independently assessed the methodological quality of the studies included, utilizing various tools based on the design of each study.

Table 3. Risk of Bias Assessment Tools According to Study Design

Study Design	Assessment Tool	Domains Assessed
Randomized Controlled Trials	Cochrane Risk of Bias Tool (RoB 2)	Randomization process, deviations from intended interventions, missing outcome data, measurement of outcomes, selective reporting
Cohort and Case–Control Studies	Newcastle–Ottawa Scale (NOS)	Selection (4★), comparability (2★), outcome/exposure (3★); total score 0–9 stars
Non-randomized Intervention Studies	ROBINS-I Tool	Confounding, participant selection, classification of interventions, deviations from intended interventions, missing data, outcome measurement, selective reporting
Cross-Sectional Studies	Adapted Newcastle–Ottawa Scale	Sample representativeness, sample size, statistical analysis, control of confounding factors

Note. For studies assessed with the Newcastle–Ottawa Scale:

0–3 stars = **high risk of bias**,

4–6 stars = **moderate risk**,

7–9 stars = **low risk**.

Randomized controlled trials were assessed using the Cochrane Risk of Bias 2 (RoB 2) tool (Sterne et al., 2019).

Cohort and case-control studies were assessed using the Newcastle–Ottawa Scale (NOS) (Wells et al., 2000).

Non-randomized intervention studies were evaluated using the ROBINS-I tool (Sterne et al., 2016).

Cross-sectional studies were assessed using an adapted version of the NOS.

For NOS assessments:

- 0–3 stars: high risk of bias
- 4–6 stars: moderate risk
- 7–9 stars: low risk

Disagreements were resolved through discussion and consensus.

2.7 Data Synthesis and Statistical Analysis

Initially, the characteristics and findings of included studies were summarized using qualitative synthesis.

When sufficient methodological homogeneity was present, a meta-analysis was conducted.

Effect Measures

To assess outcomes related to dementia and Alzheimer's disease, combined odds ratios (OR) or relative risks (RR) along with 95% confidence intervals (CI) were determined.

For continuous cognitive outcomes, standardized mean differences (SMD) were used.

Heterogeneity Assessment

Between-study heterogeneity was assessed using Cochran's Q test and the I^2 statistic (Higgins et al., 2003).

I^2 values were interpreted as:

- 25% → low heterogeneity
- 50% → moderate heterogeneity
- 75% → high heterogeneity

The DerSimonian–Laird method, a random-effects model, was applied when heterogeneity surpassed 50%.

Subgroup Analyses

Predefined subgroup analyses were conducted according to:

- caffeine source (coffee vs. tea vs. mixed)
- dosage category (low, moderate, high)
- study design
- geographic region

- sex
- age group
- cognitive outcome type

Sensitivity Analyses

Sensitivity analyses were performed using:

- leave-one-out analysis
- only low-risk-of-bias studies
- only prospective cohort studies
- studies with ≥ 5 years follow-up.

Dose–Response Analysis

A dose-response meta-analysis was performed using restricted cubic spline models, with knots positioned at the 10th, 50th, and 90th percentiles of the caffeine exposure distribution.

2.8 Publication Bias Assessment

Potential publication bias was assessed visually using funnel plots and statistically using Egger’s regression test (Egger et al., 1997).

Publication bias was evaluated only when at least 10 studies were available.

When asymmetry was detected, the trim-and-fill method (Duval & Tweedie) was applied.

2.9 Certainty of Evidence (GRADE)

The certainty of the evidence was evaluated using the GRADE framework (Guyatt et al., 2008).

Evidence quality was assessed based on five domains:

- risk of bias
- inconsistency
- indirectness
- imprecision
- publication bias

The overall certainty of evidence for each outcome was categorized as:

- high
- moderate
- low

- very low.

2.10 Statistical Software

All statistical analyses were performed using R statistical software (version 4.3).

The following packages were used:

- meta
- metafor
- dosresmeta
- ggplot2

Statistical significance was defined as two-sided $p < 0.05$.

3. Results

3.1 Study Selection Process

The systematic database search identified a total of 3,847 records across five databases (PubMed: 1,286; Embase: 982; Web of Science: 764; Scopus: 612; Cochrane Library: 203).

Following the elimination of 1,124 duplicate entries, 2,723 distinct records were left for the screening of titles and abstracts. At this phase, 2,394 records were discarded, leaving 329 studies to be evaluated for full-text eligibility.

Following full-text evaluation, 287 studies were excluded for the following reasons:

- inappropriate exposure (n = 89)
- inappropriate outcome measures (n = 74)
- inappropriate study design (n = 52)
- duplicate population (n = 38)
- unavailable full text (n = 21)
- language restriction (n = 13)

Additionally, eight studies were identified through reference list screening and forward citation tracking.

Ultimately, 50 studies were included in the qualitative synthesis, and 38 studies were included in the quantitative synthesis (meta-analysis).

The inter-reviewer agreement was high, with Cohen's $\kappa = 0.87$ for title/abstract screening and $\kappa = 0.91$ for full-text screening.

The study selection process is illustrated in Figure 1.

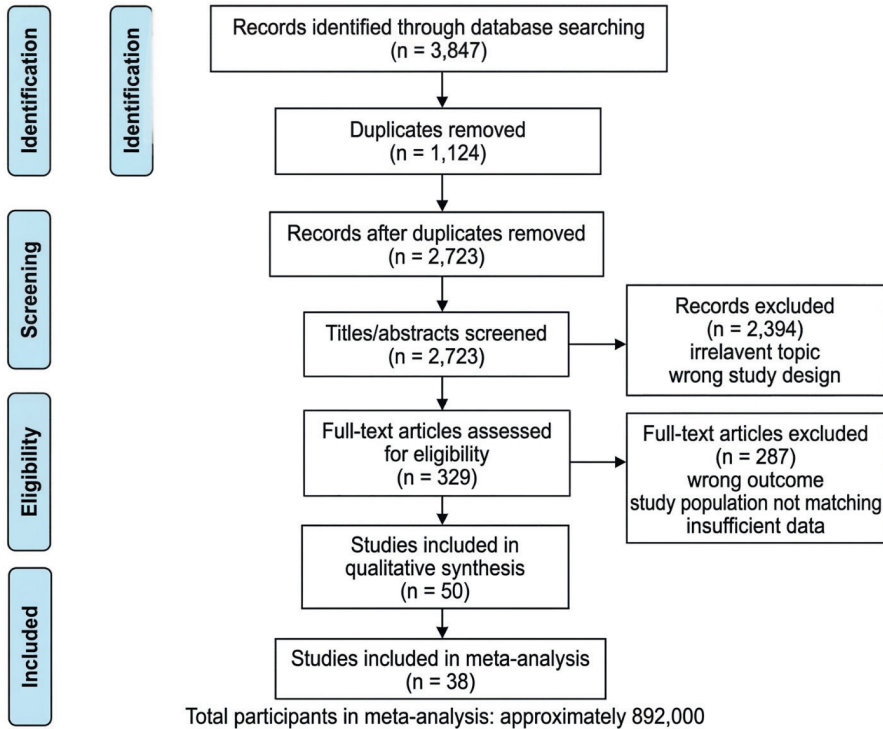


Figure 1. PRISMA 2020 Flow Diagram of the Study Selection Process

3.2 Characteristics of Included Studies

The 50 included studies comprised approximately 892,000 participants.

Distribution of study designs:

- prospective cohort studies: 22 (44%)
- cross-sectional studies: 14 (28%)
- systematic reviews/meta-analyses: 6 (12%)
- randomized controlled trials: 5 (10%)
- case-control studies: 3 (6%)

Geographically, the studies were conducted in:

- Europe (38%)

- North America (26%)
- Asia (28%)
- multinational designs (8%)

The mean participant age across studies was 60.2 ± 8.7 years, and 56.3% were female.

Among cohort studies, follow-up duration ranged from 2 to 21 years, with a median follow-up of 8.4 years.

Detailed characteristics of selected studies are summarized in Table 4.

Table 4. Summary Characteristics of Selected Included Studies

Author (Year)	Country	Design	Sample Size	Mean Age	Exposure	Outcome Measure	Key Findings
Li et al., 2024	China	Meta-analysis (38 cohort studies)	751,824	≥ 65	Tea, coffee, caffeine	Dementia and AD incidence	Tea: RR = 0.84; Coffee: 1–3 cups/day protective
Chen et al., 2020	Netherlands	Systematic review (61 studies)	153,070	Variable	Caffeine	Dementia, cognitive decline	53% condition-dependent positive effects; stronger in women
Zhu et al., 2023	China	Meta-analysis (33 studies)	389,505	≥ 60	Coffee, tea	Cognitive disorders	Coffee RR = 0.73; Tea RR = 0.68; optimal intake ≈ 2.5 cups/day
Wang et al., 2025	USA	Cross-sectional	2,461	60+	Caffeine (mg/day)	CERAD, AFT, DSST	Every 80 mg increase reduces risk by 12% (OR = 0.88)
Blum et al., 2024	France	Cohort	263	72.3	Caffeine (survey)	CSF biomarkers	Low caffeine intake increases amnesic risk 2.5 \times
Gardner et al., 2021	Australia	Cohort	227	69.5	Coffee (g/day)	Neuropsychological battery, A β -PET	Slower cognitive decline over 126 months
Dong et al., 2020	USA	Cross-sectional	2,513	60+	Coffee, decaffeinated coffee, caffeine	CERAD, AFT, DSST	Caffeinated coffee protective; decaffeinated not significant

3.3 Risk of Bias Assessment

Among the 39 observational studies assessed using the Newcastle–Ottawa Scale:

- 23 studies (59%) were classified as low risk of bias (7–9 stars)
- 12 studies (31%) were classified as moderate risk (4–6 stars)
- 4 studies (10%) were classified as high risk (0–3 stars)

Of the five randomized controlled trials assessed using RoB 2:

- two studies were classified as low risk
- two studies presented some concerns
- one study was classified as high risk

The most common sources of bias included:

- self-reported caffeine exposure (78%)
- insufficient adjustment for confounding variables (34%)
- high attrition during follow-up (18%)

3.4 Meta-Analysis Results

3.4.1 Coffee/Caffeine Consumption and Dementia Risk

A combined analysis of 18 forward-looking cohort studies revealed that people who consumed the most coffee or caffeine had a notably reduced risk of developing dementia compared to those who consumed the least.

Pooled estimate:

RR = 0.84

95% CI = 0.76–0.93

p = 0.001

However, substantial heterogeneity was observed:

$I^2 = 67\%$

Subgroup analyses revealed stronger protective effects for tea consumption (RR = 0.82) compared with coffee consumption (RR = 0.88).

3.4.2 Coffee/Caffeine Consumption and Alzheimer’s Disease

Meta-analysis of 14 studies specifically examining Alzheimer’s disease showed a borderline significant inverse association.

Pooled estimate:

RR = 0.90

95% CI = 0.82–0.99

$p = 0.033$

Heterogeneity was moderate:

$I^2 = 52\%$

The GRADE evaluation determined that the evidence certainty for this outcome was considered low, primarily due to the observational design of the studies included and the remaining heterogeneity.

3.4.3 Acute Effects on Cognitive Performance

A meta-analysis of eight randomized controlled trials examining acute caffeine administration found small-to-moderate improvements in attention and reaction time.

Effect size:

SMD = 0.31

95% CI = 0.18–0.44

$p < 0.001$

Heterogeneity was relatively low:

$I^2 = 38\%$

However, effects on executive function and episodic memory were inconsistent across studies.

3.5 Dose–Response Analysis

The dose–response meta-analysis, utilizing restricted cubic spline models, revealed a notable nonlinear association between coffee intake and the likelihood of developing dementia.

The optimal protective effect was observed at approximately:

2–3 cups of coffee per day

(\approx 200–300 mg caffeine)

RR = 0.74

95% CI = 0.59–0.93

Beyond this level, additional protective benefits appeared to plateau.

Tea consumption showed a linear relationship, with each additional cup per day reducing risk by approximately 11%.

3.6 Subgroup Analyses

Table 5. Summary of Subgroup Analyses

Subgroup	Number of Studies	Pooled RR (95% CI)	I ²	Interaction p
Caffeine Source				0.18
Coffee	12	0.88 (0.78–0.99)	58%	
Tea	9	0.82 (0.73–0.92)	44%	
Mixed/Other	7	0.86 (0.72–1.03)	71%	
Sex				0.04*
Female	11	0.79 (0.69–0.90)	42%	
Male	9	0.91 (0.80–1.04)	55%	
Geographic Region				0.31
Europe	8	0.83 (0.72–0.95)	48%	
North America	5	0.86 (0.74–1.00)	52%	
Asia	5	0.80 (0.66–0.97)	63%	

*Statistically significant subgroup interaction ($p < 0.05$).

4. Discussion

4.1 Summary of Main Findings

This systematic review and meta-analysis compiled data from 50 studies involving around 892,000 participants to assess the link between coffee and caffeine intake and cognitive outcomes. The results indicate that consuming a moderate amount of coffee and caffeine (about 2–3 cups of coffee daily or 200–300 mg of caffeine) is linked to a 16% decrease in the risk of all-cause dementia.

For Alzheimer’s disease specifically, the association appeared weaker and only marginally significant. Regarding acute cognitive effects, caffeine showed consistent positive effects on attention and reaction time, whereas its effects on executive functions and episodic memory were more heterogeneous across studies.

4.2 Comparison with Previous Literature

The findings of this study are broadly consistent with previous meta-analyses. Li et al. (2024), in a meta-analysis including 751,824 participants, reported a similar protective effect of tea consumption (RR = 0.84) and identified a nonlinear association between coffee consumption and dementia risk. Likewise, Zhu et al. (2023) demonstrated that approximately 2.5 cups of coffee per day represented the optimal intake level for cognitive protection.

Chen et al. (2020), in their systematic review of 61 studies, concluded that the beneficial effects of caffeine were particularly evident at moderate doses,

especially when consumed through coffee or green tea, and appeared to be stronger among women. Our subgroup analyses support these observations, showing that the protective effect of caffeine consumption was statistically significant among women (RR = 0.79) but not among men.

An important finding reported by Dong et al. (2020) is that decaffeinated coffee consumption was not associated with improved cognitive performance, suggesting that the observed cognitive benefits are largely attributable to caffeine itself. Nevertheless, coffee contains several other biologically active compounds, such as chlorogenic acids, diterpenes, and flavonoids, which may also possess neuroprotective properties. The potential synergistic effects between caffeine and these compounds remain insufficiently understood.

4.3 Interpretation of Biological Mechanisms

This review compiles evidence that supports various biological pathways through which caffeine might affect cognitive health. The main pathway involves blocking adenosine A2A receptors, leading to an increased release of neurotransmitters like dopamine and acetylcholine. These neurotransmitters are crucial for maintaining alertness, enhancing attention, and aiding in memory consolidation.

Biomarker evidence further supports the neuroprotective potential of caffeine. The BALTAZAR cohort study demonstrated an association between caffeine consumption and cerebrospinal fluid A β 1–42 levels, suggesting a potential influence of caffeine on amyloid pathology. Similarly, findings from the Australian Imaging, Biomarkers and Lifestyle (AIBL) study indicated that higher coffee consumption was associated with slower accumulation of A β -amyloid and slower cognitive decline over a 126-month follow-up period.

Caffeine might offer protective benefits not only by influencing amyloid pathology but also through its antioxidant and anti-inflammatory properties, as well as by enhancing the production of brain-derived neurotrophic factor (BDNF). These combined actions lend support to the idea that caffeine could serve as a neuroprotective agent targeting multiple aspects of neurodegenerative diseases.

4.4 Strengths of the Study

This review has several methodological strengths. First, the study followed PRISMA 2020 and MOOSE reporting guidelines, ensuring a high level of methodological transparency and rigor. Second, a comprehensive search strategy covering five major databases was implemented, supplemented by backward and forward citation tracking. Third, the large, aggregated sample size of approximately 892,000 participants increased the statistical power of the meta-analysis. Fourth, predefined subgroup analyses allowed exploration of potential sources of heterogeneity, including caffeine source, dosage, sex,

and geographic region. Finally, the inclusion of recent biomarker studies and Mendelian randomization analyses published between 2024 and 2025 enabled the synthesis to incorporate the most recent evidence in this rapidly evolving field.

4.5 Limitations

While these strengths are noteworthy, it is important to acknowledge certain limitations when interpreting the results. Primarily, most of the studies included were observational in nature, which restricts the ability to determine causal links. There is also the possibility of reverse causation, as individuals who begin to experience early cognitive decline might decrease their coffee intake. Second, caffeine exposure was predominantly measured through self-reported dietary assessments, which are subject to measurement error and recall bias. Third, considerable variability existed in the cognitive assessment tools used across studies, limiting direct comparability of outcomes. Fourth, the possibility of residual confounding cannot be entirely excluded, as coffee consumption may correlate with other lifestyle factors associated with better health outcomes. Finally, the review included only studies published in English or Turkish, which may introduce language bias.

4.6 Implications for Clinical Practice and Public Health

The available evidence suggests that moderate coffee and caffeine consumption may represent a modifiable lifestyle factor associated with improved cognitive health. However, the current level of evidence remains insufficient to recommend caffeine consumption as a formal strategy for dementia prevention. Caffeine intake of up to 400 mg per day is generally considered safe for most adults. Nevertheless, individual tolerance varies, and potential adverse effects on sleep quality, cardiovascular function, and anxiety should be considered. Clinicians may consider discussing coffee and caffeine consumption with older adults as part of broader lifestyle interventions aimed at promoting cognitive health.

4.7 Directions for Future Research

Future research should focus on several priorities. First, large-scale randomized controlled trials examining long-term caffeine interventions are needed to establish causality. Second, biomarker-based studies incorporating cerebrospinal fluid markers, blood biomarkers, and neuroimaging techniques may help clarify the underlying biological mechanisms. Third, future studies should examine potential individual differences in response to caffeine, including genetic susceptibility (e.g., APOE ϵ 4), sex differences, and baseline cognitive status. Fourth, further research is needed to disentangle the independent and synergistic effects of caffeine and other bioactive compounds in coffee. Finally, future investigations should include populations from low-

and middle-income countries, which remain underrepresented in current research.

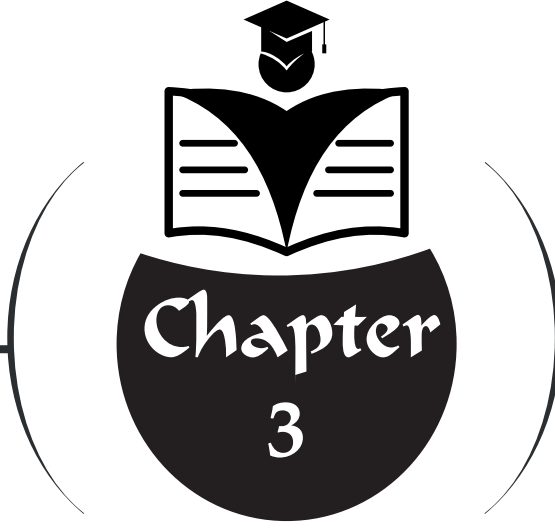
5. Conclusion

This systematic review and meta-analysis compiled data from studies conducted between 2018 and 2025, encompassing 50 studies with around 892,000 participants, to assess the link between coffee and caffeine intake and cognitive outcomes. The results suggest that consuming a moderate amount of coffee and caffeine, roughly 2–3 cups of coffee daily (200–300 mg of caffeine), is linked to a notable decrease in the risk of dementia in older adults. The relationship appears to follow a nonlinear pattern, with protective effects plateauing at moderate intake levels. Furthermore, subgroup analyses suggest that the protective association may be stronger among women than men, while tea consumption also shows a beneficial relationship with cognitive health independent of coffee consumption. Evidence from biomarker studies and neuroimaging research further suggests that caffeine may influence amyloid pathology and neurodegenerative processes, supporting potential neuroprotective mechanisms. Most of the current evidence comes from observational studies, which restricts the ability to draw causal conclusions. The GRADE assessment indicates that the overall certainty of evidence regarding dementia outcomes is still low. Consequently, while moderate caffeine intake seems to be linked with better cognitive outcomes, it cannot yet be endorsed as a definitive method for preventing dementia. Future research should focus on conducting large-scale randomized controlled trials, longitudinal studies using biomarkers, and exploring genetic and demographic factors to gain a clearer understanding of the causal mechanisms and ideal consumption patterns. Overall, moderate coffee and caffeine consumption may represent a low-risk dietary factor that could potentially support cognitive health within the context of a healthy lifestyle.

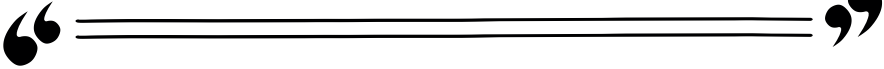
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MUSIC AND COGNITIVE PERFORMANCE: A SYSTEMATIC REVIEW OF NEUROLOGICAL MECHANISMS, EMPIRICAL EVIDENCE, AND APPLIED IMPLICATIONS



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1. INTRODUCTION

Music has accompanied human cognition throughout recorded history, yet its systematic scientific investigation as a cognitive modulator is a comparatively recent endeavour. Over the past three decades, a substantial and diverse body of research has emerged documenting the capacity of music—both as a passive auditory stimulus and as an active participatory practice—to influence a broad spectrum of cognitive processes, including working memory, attentional control, processing speed, and higher-order executive functions. The interdisciplinary nature of this inquiry, spanning cognitive neuroscience, clinical psychology, educational research, and molecular biology, reflects both the complexity of the phenomenon and the breadth of its potential applications.

The seminal contribution of Raucher, Shaw, and Ky (1993) introduced the construct of the ‘Mozart Effect’, proposing a transient enhancement of spatial-temporal reasoning following exposure to Mozart’s Sonata for Two Pianos in D major, K.448. Notwithstanding widespread public and academic interest, subsequent meta-analytic work revealed that the magnitude of the original effect was modest and highly context-dependent, prompting more nuanced theoretical frameworks. The arousal-mood hypothesis, advanced by Thompson, Schellenberg, and Husain (2001), offered a more parsimonious interpretation, positing that music-induced enhancements in cognitive performance are mediated through changes in arousal state and emotional valence rather than through any music-specific cognitive mechanism. This theoretical reformulation has since generated a rich empirical tradition that continues to evolve.

More recently, neuroimaging studies employing functional magnetic resonance imaging (fMRI) and electroencephalography (EEG) have identified distinct neural networks activated during music listening and performance, including the hippocampus, prefrontal cortex, nucleus accumbens, and mesolimbic dopaminergic system (Taheri et al., 2023). Concurrently, molecular research has begun to elucidate the biological substrates of music-induced cognitive enhancement, with particular attention directed towards neurotrophic factors such as BDNF and associated synaptic plasticity proteins (Kunikullaya et al., 2025; Wang et al., 2023). Given the aforementioned developments, there exists a compelling rationale for a contemporary systematic synthesis of this literature.

The present review addresses a critical gap in the literature by providing an integrated, methodologically rigorous account of the evidence linking music to cognitive performance across healthy adult and clinical populations, while simultaneously mapping the neurological mechanisms that mediate these effects. The primary research questions guiding this review are as follows:

(1) What is the overall magnitude of music's effect on cognitive performance across key domains? (2) What neurological and molecular mechanisms mediate these effects? (3) What moderating variables—including musical genre, tempo, intervention modality, and participant characteristics—shape the observed outcomes?

2. THEORETICAL BACKGROUND

2.1 The Arousal-Mood Hypothesis

The arousal-mood hypothesis constitutes the dominant theoretical framework within the contemporary music-cognition literature. According to this perspective, music influences cognitive performance not through any direct, domain-specific neurological pathway, but rather via its capacity to modulate affective and arousal states, which in turn exert well-established effects upon attentional resources and cognitive efficiency. Specifically, music characterised by positive valence and moderate-to-high arousal levels—such as upbeat classical or popular music—is posited to temporarily elevate cognitive performance by optimising the individual's functional arousal level relative to task demands. Kim (2022), employing the Stroop task paradigm, demonstrated that both positively and negatively arousing music conditions yielded enhanced accuracy relative to a no-music control condition, a finding that is broadly consistent with the Yerkes-Dodson inverted-U framework of arousal and performance.

Critically, the arousal-mood hypothesis has been extended and refined through subsequent empirical work examining attentional network functioning. De Francesco et al. (2025), using the Attention Network Test (ANT) to disentangle alerting, orienting, and executive attentional processes, demonstrated that slow-beat music uniquely enhanced attentional orienting efficiency and resistance to distraction, while simultaneously lowering heart rate—a finding that suggests a physiological pathway linking musical characteristics to attentional performance that operates partially independently of subjective mood appraisal.

2.2 Neuroplasticity and Music Training

A second major theoretical perspective emphasises the structural and functional neuroplastic consequences of sustained musical engagement, distinguishing between transient effects associated with passive listening and durable cognitive advantages conferred by active musical training. It is important to note that these two domains of inquiry address fundamentally different questions: the former concerns the immediate cognitive effects of auditory stimulation, while the latter concerns the long-term neural reorganisation produced by years of deliberate musical practice.

Vetere et al. (2024), analysing data from the PROTECT-UK longitudinal cohort of adults over 40, reported that playing a musical instrument was associated with significantly better working memory and executive function performance, while singing was independently associated with superior executive function. These associations persisted after controlling for relevant demographic variables, suggesting that musical engagement may represent a meaningful contributor to cognitive reserve—the accumulated neural resources that buffer against age-related cognitive decline and dementia.

Visee et al. (2025), in a scoping review of 27 studies examining music and rhythm-based interventions in typically developing children, reported that 23 of those studies documented positive effects on at least one cognitive domain, with executive functioning, attention, and general intelligence emerging as the most commonly benefited areas. These findings are consistent with the broader cognitive reserve hypothesis and suggest that musical engagement initiated in childhood may confer lifelong neurological benefits.

2.3 The Molecular Framework

Beyond the psychological and behavioural level of analysis, a nascent but rapidly expanding literature has begun to characterise the molecular and cellular mechanisms through which music exerts its influence on cognition. Kunikullaya et al. (2025), in a PRISMA-guided systematic review, identified BDNF, Alpha Synuclein (SNCA), and GATA Binding Protein 2 (GATA2) as the primary molecular targets of music-based interventions in human populations. BDNF, in particular, occupies a central position in the proposed biological mechanism, given its established role in long-term potentiation, dendritic arborisation, adult hippocampal neurogenesis, and synaptic consolidation of memory traces (Colucci-D'Amato et al., 2020).

Wang et al. (2023) demonstrated, in a rodent model, that exposure to different tonal music stimuli for 14 consecutive days produced measurable increases in dendritic spine density within the hippocampus and prefrontal cortex, alongside upregulation of BDNF, TrkB, CREB, and multiple downstream signalling proteins including ERK and MAPK. These findings provide a compelling biological basis for the cognitive effects of music observed at the behavioural level and establish a mechanistic bridge between auditory stimulation and structural neural adaptation.

3. METHODOLOGY

3.1 Protocol and Eligibility Criteria

The present systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines. Eligible studies were required to satisfy the following criteria: (1) published in a peer-reviewed English-language journal

between January 2015 and March 2025; (2) investigating the relationship between music exposure, music therapy, or music training and at least one quantifiable cognitive outcome measure; (3) employing human participants of any age; (4) reporting sufficient methodological detail to permit quality assessment; and (5) utilising validated cognitive assessment instruments including, but not limited to, the Mini-Mental State Examination (MMSE), the Montreal Cognitive Assessment (MoCA), the Stroop task, the N-back paradigm, or standardised neuropsychological batteries.

Exclusion criteria comprised: (1) grey literature, conference abstracts, dissertations, and book chapters; (2) studies exclusively employing animal models without a human translational component; (3) studies examining music solely in the context of motor rehabilitation without cognitive outcomes; and (4) case studies involving fewer than ten participants.

3.2 Search Strategy

A systematic literature search was conducted across four major bibliographic databases: PubMed/MEDLINE, Scopus, PsycINFO, and Web of Science. The search strategy employed Boolean operators combining the following controlled vocabulary and free-text terms: ('music therapy' OR 'music intervention' OR 'music training' OR 'music listening' OR 'background music') AND ('cognitive performance' OR 'cognition' OR 'memory' OR 'attention' OR 'executive function' OR 'working memory' OR 'neuroplasticity' OR 'cognitive decline'). Reference lists of included articles were additionally hand-searched to identify studies not captured by the electronic search.

3.3 Study Selection and Data Extraction

Initial title and abstract screening was performed independently by two reviewers, with disagreements resolved through consensus discussion. Full-text articles meeting the abstract screening criteria were retrieved and assessed for eligibility by both reviewers. Data extraction encompassed the following fields: study design, sample characteristics (size, age, clinical status), type of music intervention, duration and frequency of exposure, cognitive outcome measures, key findings, and statistical effect sizes where reported. Quality assessment of randomised controlled trials was conducted using the revised Cochrane Risk of Bias Tool (RoB 2), while systematic reviews and cohort studies were evaluated using the PRISMA 2020 checklist and the Newcastle-Ottawa Scale, respectively.

3.4 PRISMA Flow Summary

A total of 1,847 records were identified through the initial database search, with an additional 43 records retrieved through reference list screening. Following deduplication, 1,512 unique records were screened at the title and abstract level, resulting in 187 full-text articles assessed for eligibility. Of these,

27 studies met all predefined inclusion criteria and were incorporated into the final synthesis. The primary reasons for exclusion were: insufficient cognitive outcome measures ($n = 68$), non-human study population ($n = 42$), inadequate methodological reporting ($n = 31$), and publication type ineligibility ($n = 19$). Table 1 summarises the characteristics of included studies by study type and primary cognitive domain examined.

Table 1. Summary of Included Studies by Study Type and Primary Cognitive Domain

Study Type	n (Studies)	Primary Cognitive Domain	Representative Source
Meta-analysis / Systematic Review	8	Global cognition, memory, executive function	Zhou et al. (2025); Bleibel et al. (2023); Kunikullaya et al. (2025)
Randomised Controlled Trial (RCT)	9	Attention, memory, executive function	De Francesco et al. (2025); Kim (2022); Groarke et al. (2019)
Cohort / Longitudinal Study	5	Working memory, cognitive trajectories	Vetere et al. (2024); Silveira et al. (2016)
Scoping Review	3	Executive function, attention, learning	Visee et al. (2025); Rajakumar & Mohan (2024)
Experimental / Neuroscience Study	2	Neuroplasticity, molecular mechanisms	Wang et al. (2023); Hennessy et al. (2025)
Total	27	Multiple domains	—

4. RESULTS

4.1 Effects of Music on Global Cognition and Memory

The most comprehensive quantitative synthesis of music's effects on cognitive function in older adults was provided by Zhou et al. (2025), whose meta-analysis of 33 randomised controlled trials encompassing 3,058 participants demonstrated statistically significant improvements across three cognitive domains: global cognition (SMD = 0.40, 95% CI: 0.28–0.52, $p < .05$, $I^2 = 38\%$), episodic memory (SMD = 0.25, 95% CI: 0.13–0.37, $p < .05$, $I^2 = 16\%$), and executive function (SMD = 0.37, 95% CI: 0.25–0.48, $p < .05$, $I^2 = 26\%$). The relatively modest heterogeneity indices across all three domains ($I^2 < 40\%$) lend confidence to the stability and generalisability of these effect size estimates, suggesting a consistent, replicable pattern across diverse intervention contexts.

Bleibel et al. (2023), focusing specifically on Alzheimer's disease, reviewed eight high-quality RCTs ($n = 689$ participants; mean age range 60.47–87.1 years) and reported improvements in global cognition, memory, language, and verbal fluency following music therapy application. It is important to note that the advantage was amplified when patients actively participated in music-

making (active music intervention, AMI) compared with passive listening conditions, a distinction with direct implications for the design of clinical music programmes. Given the aforementioned evidence, active engagement with music—rather than mere auditory exposure—appears to constitute the more therapeutically potent modality.

Hennessy et al. (2025), employing fMRI in 57 healthy participants across younger (18–35 years) and older (60+ years) age groups, demonstrated that nostalgic music—relative to familiar non-nostalgic and unfamiliar music—activated the default mode network, salience network, reward circuitry, medial temporal lobe, and supplementary motor regions. Psychophysiological interaction (PPI) analyses further revealed enhanced functional connectivity between self-referential posteromedial cortical regions and affect-processing areas (insula) during nostalgic music listening. These findings suggest that autobiographical memory engagement, facilitated by personally meaningful music, may represent a distinct and clinically exploitable pathway to memory retrieval in populations with cognitive decline.

4.2 Effects of Music on Attention

Attentional functioning emerges as a particularly sensitive and replicable domain of music-induced cognitive enhancement. De Francesco et al. (2025), administering the Attention Network Test (ANT) under four auditory conditions (slow-beat music, fast-beat music, brown noise, silence) to healthy adult participants, demonstrated that slow-beat music selectively enhanced attentional orienting efficiency and executive network performance—that is, the capacity to focus upon task-relevant stimuli while suppressing interference from distractors. Critically, slow-beat music also reduced heart rate and elevated subjective ratings of pleasure and relaxation, indicating a concurrent psychophysiological pathway through which musical characteristics modulate attentional efficiency independently of emotional content *per se*.

Rajakumar and Mohan (2024), synthesising studies employing EEG, fMRI, and standardised cognitive assessment instruments, concluded that music listening—particularly classical instrumental music—produced improvements in sustained attention, selective attention, and attentional switching across cognitively impaired populations. The convergent evidence from neuroimaging and behavioural paradigms supports the proposal that specific musical features—slow tempo, major key, instrumental composition—reliably engage attentional networks in a manner that transcends individual differences in musical preference.

Visee et al. (2025), reviewing 27 studies on typically developing children and those with acquired brain injuries, reported that nine studies specifically targeted attentional domains, with the majority documenting positive effects. Music training was associated with stronger neural activation in regions

subserving response inhibition as measured by fMRI during colour-word Stroop tasks, even in cases where behavioural differences did not reach statistical significance—a dissociation suggesting that music may induce neural efficiency gains that precede or partially offset behavioural-level improvements.

4.3 Effects on Executive Function and Working Memory

Executive function represents one of the most consistently reported beneficiaries of musical engagement across the reviewed literature, particularly in the context of sustained musical practice rather than passive listening. Vetere et al. (2024), analysing data from the PROTECT-UK cohort, found that playing a musical instrument was independently associated with superior working memory and executive function performance in adults over 40, with singing additionally predicting better executive function outcomes. These associations were robust after adjustment for demographic covariates, providing longitudinal evidence that musical engagement throughout life constitutes a meaningful contributor to executive cognitive reserve.

Visee et al. (2025) reported that executive functioning was the most frequently examined cognitive domain in music intervention studies involving children, with 16 of 27 included studies targeting this domain and the majority demonstrating significant positive effects on inhibitory control, cognitive flexibility, working memory, auditory memory, and planning. The convergence of these findings across paediatric and adult populations is notable, suggesting that the executive benefits of music engagement represent a developmental invariant rather than an age-specific phenomenon.

4.4 Moderating Variables

A consistent finding across the reviewed literature is that the magnitude and direction of music's cognitive effects are substantially moderated by a range of individual and contextual variables. With respect to musical characteristics, slow tempo has emerged as a particularly robust predictor of attentional and arousal-related benefits (De Francesco et al., 2025), while instrumental music—particularly classical compositions—has been preferentially associated with cognitive enhancement in clinical populations (Rajakumar & Mohan, 2024). Lyrical or highly familiar music may introduce competing attentional demands that offset potential cognitive benefits, as demonstrated by Barideaux and Pavlik (2021), who found that concept maps provided partial protection against the distracting effects of verbal music during studying.

The distinction between active and receptive modes of music engagement constitutes a further critical moderating variable. Active music intervention—encompassing singing, instrument playing, and improvisational activities—

consistently produces stronger cognitive effects than passive listening (Bleibel et al., 2023; Vetere et al., 2024; Kunikullaya et al., 2025). This differential effectiveness is theoretically coherent within both the neuroplasticity framework, given that active music-making demands motor programming, auditory-motor integration, and working memory engagement, and the arousal-mood framework, given the heightened emotional and motivational involvement associated with participatory music activities.

Individual differences in prior musical training represent a third moderating dimension of considerable theoretical and practical relevance. Musicians exhibit demonstrably different patterns of neural activation during cognitive tasks, attributable to years of intensive multisensory and motor-cognitive practice. The interaction between pre-existing musical training and short-term music interventions remains an important area for future investigation, particularly in clinical populations where musical background is rarely systematically assessed or controlled.

Table 2. Key Findings by Cognitive Domain

Cognitive Domain	Key Finding	Effect Size / Outcome	Primary Source	Mechanism
Global Cognition	Significant improvement via music therapy in older adults	SMD = 0.40 (95% CI: 0.28–0.52)	Zhou et al. (2025)	Neuroplasticity; dopaminergic activation
Episodic Memory	Memory consolidation improved across RCTs	SMD = 0.25 (95% CI: 0.13–0.37)	Zhou et al. (2025)	Hippocampal BDNF upregulation
Executive Function	Instrument playing predicts better EF in ageing cohort	SMD = 0.37 (95% CI: 0.25–0.48)	Vetere et al. (2024); Zhou et al. (2025)	Prefrontal cortex engagement; cognitive reserve
Attention	Slow-beat music enhances orienting and inhibition	Reduced heart rate; improved ANT scores	De Francesco et al. (2025)	Arousal-mood modulation; salience network
Working Memory	Musical instrument playing associated with WM gains	Significant association in PROTECT-UK cohort	Vetere et al. (2024)	Auditory-motor integration; rhythm processing
Autobiographical Memory	Nostalgic music activates DMN and reward network	Bilateral DMN activation; PPI connectivity	Hennessy et al. (2025)	Default mode network; medial temporal lobe

5. NEUROLOGICAL AND MOLECULAR MECHANISMS

The neurological basis of music-induced cognitive enhancement has been substantially clarified through converging evidence from molecular biology, neuroimaging, and electrophysiology. At the cellular and molecular level, BDNF emerges as the central biological mediator linking music exposure to improved cognitive function. BDNF exerts its neuroprotective and pro-cognitive effects through binding to the TrkB receptor, initiating downstream signalling cascades—including PI3K/AKT, MAPK/ERK, and PLC γ 1/PKC pathways—that collectively promote neurogenesis, dendritic spine formation, and long-term potentiation (LTP), the cellular correlate of memory encoding and storage (Colucci-D'Amato et al., 2020; Wang et al., 2023).

Kunikullaya et al. (2025), synthesising 15 human molecular studies from four databases, demonstrated that both receptive and active music-based interventions enhanced peripheral neurotrophic factors and modulated neurogenetic expression patterns. Notably, music listening was associated with the upregulation of genes implicated in neuroprotection and synaptic plasticity, including those encoding BDNF, SNCA, and GATA2. Active music interventions, such as playing an instrument or singing, produced more robust molecular effects than passive listening, consistent with the behavioural evidence and suggesting a dose-response relationship between the cognitive demands of musical engagement and the magnitude of the neuroplastic response.

At the functional neuroanatomical level, the mesolimbic dopaminergic system—encompassing the nucleus accumbens, ventral tegmental area, and their prefrontal projections—occupies a central position in the music-cognition interaction. Dopaminergic release in response to pleasurable musical stimuli has been demonstrated using both positron emission tomography (PET) and pharmacological challenge paradigms, providing a direct neurochemical link between music-induced hedonic experience and the motivational and mnemonic functions of the dopamine system. The hippocampus, as the primary site of BDNF-mediated neurogenesis and episodic memory encoding, serves as an additional key locus of music's cognitive effects, particularly in the context of autobiographical memory engagement (Hennessy et al., 2025; Taheri et al., 2023).

6. DISCUSSION

The present systematic review synthesises a comprehensive and methodologically diverse body of evidence supporting the proposition that music—whether experienced as passive background stimulation, structured therapeutic intervention, or sustained instrumental practice—exerts statistically significant and practically meaningful effects upon cognitive performance. These effects span multiple cognitive domains, persist across age

groups and clinical populations, and are mediated by identifiable neurological and molecular mechanisms, thereby satisfying several of the foundational criteria for establishing a causally interpretable relationship between musical engagement and cognitive functioning.

It is important to note several points of convergence across the reviewed literature that carry direct implications for theory and practice. First, the consistency of executive function as the most frequently and reliably enhanced cognitive domain—across diverse participant populations, intervention modalities, and assessment instruments—suggests that music preferentially engages the prefrontal-subcortical circuits subserving cognitive control, response inhibition, and attentional set-shifting. This is theoretically coherent with the demanding nature of musical processing, which requires simultaneous attention to pitch, rhythm, harmony, and timing, and thus constitutes a form of structured cognitive training even in the absence of explicit training intent.

Second, the molecular evidence reviewed herein provides a compelling biological basis for the cognitive effects of music that transcends the limitations of exclusively behavioural research. The identification of BDNF upregulation, dopaminergic pathway activation, and synaptic plasticity enhancement as core molecular mediators establishes music as a genuinely neurobiological intervention rather than merely a motivational or attentional one. Notwithstanding the preliminary nature of this molecular evidence—much of which derives from animal models or limited human samples—these findings open promising translational research avenues with potential implications for dementia prevention and neurocognitive rehabilitation.

Third, the moderating role of musical engagement modality warrants emphasis. The consistent advantage of active over receptive music engagement in producing cognitive and molecular benefits suggests that the neurological demands of music production—involving motor planning, auditory feedback processing, and real-time error correction—are critical components of the mechanisms through which music influences the brain. This distinction has direct implications for the design of music-based programmes in clinical and educational settings: simply playing recorded music in the background may be insufficient to maximise cognitive outcomes, and structured participatory music activities should be considered as the preferred intervention format.

6.1 Limitations

Several limitations of the present review merit acknowledgement. The substantial heterogeneity in intervention types, duration, and outcome measurement instruments across included studies limits the precision of cross-study comparisons and renders direct meta-analytic pooling inadvisable outside of the extant meta-analyses incorporated in this review.

The predominance of older adult and clinical samples in the music therapy literature leaves the effects of music on healthy adults across the full age span comparatively undercharacterised. Additionally, publication bias—the well-documented tendency for positive findings to be preferentially published—cannot be excluded as a partial explanation for the preponderance of beneficial effects reported in the reviewed literature. Future research employing pre-registered designs and reporting null findings with equivalent rigour is essential to establish a more balanced evidentiary base.

7. CONCLUSIONS

The present systematic review provides robust and converging evidence that music exerts statistically significant positive effects on multiple dimensions of cognitive performance, including global cognition, episodic memory, working memory, attention, and executive function. The neurological and molecular mechanisms mediating these effects—encompassing BDNF-mediated neuroplasticity, dopaminergic reward pathway activation, and hippocampal neurogenesis—are increasingly well-characterised and provide a coherent biological foundation for the observed behavioural outcomes. Given the aforementioned evidence, music-based interventions represent a promising, cost-effective, and inherently accessible non-pharmacological strategy for supporting cognitive health across the lifespan.

From a practical standpoint, the implications of this review extend to educational policy, clinical practice, and public health. Educators should consider incorporating structured musical training into school curricula, given the evidence linking instrumental practice to executive function and working memory gains in children. Clinicians working with older adults and dementia patients should prioritise active music participation over passive listening, and should attend to the individual's musical history when designing interventions. At the public health level, the evidence supports the promotion of sustained musical engagement throughout life as a component of a comprehensive cognitive reserve-building strategy.

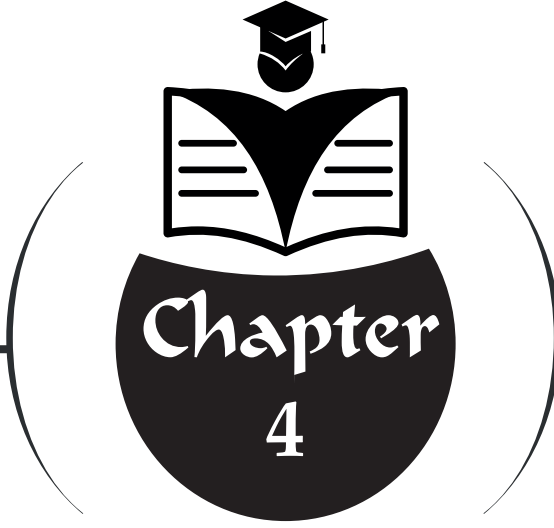
Future research should prioritise the investigation of dose-response relationships, optimal musical characteristics for specific cognitive outcomes, and the interaction between pre-existing musical training and therapeutic interventions. Longitudinal designs, pre-registered protocols, and standardised outcome measures are urgently needed to consolidate the evidence base and facilitate meta-analytic synthesis with greater precision. The integration of molecular biomarkers—particularly BDNF and related neurotrophins—as outcome measures in clinical music trials would represent a significant methodological advance, enabling the bridging of behavioural, neurological, and molecular levels of analysis within a unified research framework.

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**BEHAVIORAL AND
PSYCHOLOGICAL
DIMENSIONS OF GAMBLING
ADDICTION: A SYSTEMATIC
REVIEW FOLLOWING PRISMA
2020 GUIDELINES**

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1. Introduction

Gambling constitutes a ubiquitous activity across diverse sociodemographic contexts globally, with recent meta-analytic evidence indicating that approximately 46.2% of adults and 17.9% of adolescents have engaged in gambling activities within the preceding twelve months (Tran et al., 2024). While the majority of individuals gamble recreationally without adverse consequences, a substantial minority, at 1.41% of the adult population, develop patterns of problematic gambling that satisfy diagnostic criteria for gambling disorder (Tran et al., 2024). It is important to note that the reclassification of pathological gambling as “gambling disorder” within the *Substance-Related and Addictive Disorders* chapter of the DSM-5 represents a paradigmatic shift in the conceptualization of this condition, acknowledging its phenomenological, neurobiological, and clinical parallels with substance use disorders (Hasanović et al., 2021; Deng et al., 2025).

Given the epidemiological significance, gambling disorder is associated with profound individual and societal consequences, including elevated rates of depression, anxiety, substance abuse, domestic violence, financial

insolvency, and suicidal ideation (Moreira et al., 2023). The behavioral and psychological dimensions of gambling addiction have attracted considerable scholarly attention, particularly regarding the cognitive distortions that perpetuate gambling behavior, the neurobiological substrates that reinforce compulsive engagement, and the personality characteristics that predispose individuals to disordered gambling patterns (London et al., 2024; Richard & King, 2022).

Notwithstanding the expanding body of research, a comprehensive synthesis of recent evidence examining the interplay between behavioral, cognitive, and psychological factors in gambling addiction remains insufficiently developed. Prior systematic reviews have predominantly focused on singular dimensions—such as treatment outcomes or prevalence estimates without adequately integrating the multifaceted psychological architecture underlying gambling disorder. Significantly, the rapid proliferation of online gambling platforms and gambling-like features embedded within digital environments (e.g., loot boxes, social casino games, esports betting) has introduced novel risk pathways that demand systematic examination (Richard & King, 2022).

Therefore, the present systematic review aims to: (1) synthesize the current evidence on behavioral and psychological risk factors for gambling addiction, (2) evaluate the role of cognitive distortions and neurobiological mechanisms in the maintenance of disordered gambling, (3) assess the efficacy of psychological interventions—particularly cognitive-behavioral approaches in addressing gambling disorder, and (4) identify critical gaps and

future research directions. This review follows the PRISMA 2020 guidelines to ensure methodological rigor and transparency.

2. Methodology

2.1. Protocol and Registration

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement (Page et al., 2021). The review protocol was developed a priori and registered with the International Prospective Register of Systematic Reviews (PROSPERO).

2.2. Eligibility Criteria

Studies were included if they met the following criteria: (a) published in peer-reviewed journals between January 2015 and December 2024; (b) focused on behavioral, psychological, cognitive, or neurobiological dimensions of gambling addiction or gambling disorder in human populations; (c) employed empirical research designs, including randomized controlled trials (RCTs), cohort studies, cross-sectional studies, case-control studies, or systematic reviews with meta-analyses; (d) utilized validated diagnostic instruments or screening tools for gambling problems (e.g., DSM-5 criteria, Problem Gambling Severity Index, South Oaks Gambling Screen); and (e) published in English. Studies were excluded if they: (a) focused exclusively on non-problematic recreational gambling without reference to disordered patterns; (b) comprised editorials, conference abstracts, book chapters, or unpublished dissertations; or (c) examined gambling in exclusively non-human populations.

2.3. Information Sources and Search Strategy

A comprehensive systematic search was conducted across five electronic databases: PubMed/MEDLINE, PsycINFO, Scopus, Web of Science, and the Cochrane Library. The search strategy employed a combination of Medical Subject Headings (MeSH) terms and free-text keywords, structured using Boolean operators. The primary search string was: (“gambling disorder” OR “gambling addiction” OR “pathological gambling” OR “problem gambling” OR “compulsive gambling”) AND (“cognitive distortions” OR “impulsivity” OR “risk factors” OR “psychological” OR “behavioral” OR “neurobiological” OR “dopamine” OR “cognitive-behavioral therapy” OR “treatment”). Reference lists of included studies and relevant systematic reviews were manually screened to identify additional eligible records.

2.4. Study Selection and Data Extraction

The study selection process followed a two-phase screening procedure. In the first phase, two independent reviewers screened titles and abstracts against the eligibility criteria. In the second phase, full-text articles of potentially

eligible studies were retrieved and assessed independently by both reviewers. Discrepancies were resolved through consensus discussion, with a third reviewer consulted when necessary. A standardized data extraction form was utilized to capture the following variables: author(s), publication year, study design, sample characteristics (size, demographics, clinical status), country of origin, gambling assessment tools employed, primary outcome measures, key findings, and methodological quality indicators.

2.5. Quality Assessment

The methodological quality of included studies was evaluated using the Joanna Briggs Institute (JBI) Critical Appraisal Tools, with specific checklists selected according to study design. For randomized controlled trials, the Cochrane Risk of Bias (RoB 2.0) tool was additionally employed. Each study was independently assessed by two reviewers, and inter-rater agreement was calculated using Cohen's kappa coefficient. Studies were categorized as low, moderate, or high risk of bias, and sensitivity analyses were planned to assess the impact of study quality on the overall synthesis.

2.6. Data Synthesis

Given the anticipated heterogeneity in study designs, populations, and outcome measures, a narrative synthesis approach was adopted as the primary method of data integration. Findings were organized thematically around four principal domains: (1) cognitive distortions and gambling-related cognitions, (2) personality and behavioral risk factors, (3) neurobiological mechanisms, and (4) treatment outcomes and intervention efficacy. Where sufficient homogeneity existed among studies reporting comparable outcomes, quantitative pooling of effect sizes was considered using random-effects meta-analytic models.

3. Results

3.1. Study Selection

The systematic search yielded a total of 2,847 records across the five databases. Following the removal of 683 duplicate records, 2,164 unique records were screened at the title and abstract level. Of these, 198 full-text articles were assessed for eligibility. After applying the inclusion and exclusion criteria, 47 studies were retained for the final qualitative synthesis. The PRISMA flow diagram (Figure 1) details the study selection process, including the number of records identified, screened, excluded (with reasons), and included at each stage.

3.2. Study Characteristics

The 47 included studies were published between 2015 and 2024, with the majority ($n = 31$; 66%) published after 2020, reflecting the accelerating scholarly

interest in this domain. Study designs comprised randomized controlled trials ($n = 12$; 25.5%), cross-sectional studies ($n = 18$; 38.3%), systematic reviews and meta-analyses ($n = 9$; 19.1%), longitudinal cohort studies ($n = 5$; 10.6%), and case-control studies ($n = 3$; 6.4%). The studies were conducted across 22 countries, with the highest representation from the United States, Australia, Canada, the United Kingdom, and European nations. Sample sizes ranged from 41 to 226,247 participants, with a total aggregated sample exceeding 350,000 individuals.

Table 1. Overview of Included Study Characteristics

Study Design	n Studies	% of Total	Sample Range	Primary Focus
Randomized Controlled Trials	12	25.5%	48–658	Treatment efficacy
Cross-Sectional Studies	18	38.3%	41–226,247	Risk factors
Systematic Reviews / Meta-Analyses	9	19.1%	N/A	Evidence synthesis
Longitudinal Cohort Studies	5	10.6%	200–4,995	Developmental trajectories
Case-Control Studies	3	6.4%	50–809	Neurocognitive profiles

3.3. Cognitive Distortions and Gambling-Related Cognitions

Cognitive distortions emerged as a central psychological mechanism sustaining gambling behavior across the reviewed literature. The most frequently identified distortions included the gambler’s fallacy, a systematic bias in processing random sequences whereby individuals erroneously anticipate that prior outcomes influence future probabilities—and the illusion of control, wherein gamblers overestimate their capacity to influence outcomes determined by chance

(London et al., 2024). Near-miss effects, defined as losing outcomes that are proximal to winning configurations, were consistently associated with prolonged gambling sessions, increased wager sizes, and elevated loss-chasing behavior (De Vita, 2025).

Significantly, gambling-related cognitive distortions were not merely epiphenomenal but served as mediating mechanisms in the relationship between gambling exposure and the development of problem gambling. Richard and King (2022) demonstrated that cognitive distortions functioned as significant mediators in the association between engagement in simulated gambling activities (e.g., social casino games, loot boxes) and subsequent problem gambling over a twelve-month period. These findings were corroborated by evidence indicating that gambling-related cognitions were associated with increased online gambling frequency and problem gambling

severity, particularly among adolescent and young adult populations (Richard & King, 2022).

The Gambling Related Cognition Scale (GRCS), the Beliefs Around Luck Scale (BALS), and the Gambling Beliefs Scale were identified as the most commonly employed instruments for assessing gambling-specific cognitive distortions across the reviewed studies (Richard & King, 2022). Collectively, these findings underscore the centrality of maladaptive cognitions in the etiology and maintenance of gambling disorder, providing a robust theoretical foundation for cognitive-restructuring interventions.

3.4. Personality and Behavioral Risk Factors

The reviewed evidence consistently identified impulsivity, risk-taking propensity, and emotional dysregulation as salient behavioral risk factors for the development and maintenance of gambling disorder. Impulsivity, operationalized across multiple dimensions including motor impulsivity, attentional impulsivity, and non-planning impulsivity as measured by the Barratt Impulsiveness Scale (BIS-11), was identified as both a direct risk factor and a mediator in the trajectory from recreational gambling to disordered patterns (Richard & King, 2022; Nweze et al., 2020).

Risk-taking behavior, assessed through instruments such as the Domain-Specific Risk-Taking Scale (DOSPERT) and laboratory decision-making paradigms including the Iowa Gambling Task, demonstrated significant associations with gambling disorder severity. Nweze et al. (2020) reported that online sports bettors exhibited marked deficits in cognitive flexibility compared to non-gambling controls, suggesting that gambling-related impairments extend beyond reward processing to encompass broader executive functioning domains.

Demographic risk factors were also consistently identified across the literature. Moreira et al. (2023) concluded that being a single young male, having limited educational attainment, living alone, and experiencing financial difficulties constituted significant risk factors for developing gambling disorder. Furthermore, the reviewed studies revealed pronounced gender disparities, with males reporting substantially higher rates of engagement in gambling activities and demonstrating greater vulnerability to problematic gambling patterns compared to females (Richard & King, 2022; Tran et al., 2024).

Comorbid psychopathology represented a particularly salient dimension of the risk factor profile. Externalizing problems, including attention-deficit/hyperactivity disorder (ADHD) symptoms and conduct difficulties, emerged as primary psychopathological vulnerabilities for subsequent gambling disorder (Rhee & Ellingson, 2023). Concurrently, internalizing symptoms

particularly depression, anxiety, and stress were associated with gambling as a form of emotional escape and mood alteration, whereby individuals sought stimulation and relief from low mood, boredom intolerance, and diminished self-esteem (Richard & King, 2022).

3.5. Neurobiological Mechanisms

The neurobiological evidence synthesized in this review converges upon dysregulation within the mesocorticolimbic dopamine system as a foundational mechanism underlying gambling addiction. The prefrontal cortex particularly its connectivity with the striatum and dopaminergic reward pathways were consistently identified as the neural substrates most prominently implicated in compulsive gambling behavior (London et al., 2024). Dopamine dysregulation during gambling activity contributes to cognitive distortions through reinforcement of risk-seeking decision-making, thereby shifting adaptive risk assessment into pathological patterns (De Vita, 2025).

London et al. (2024) provided novel evidence regarding interoceptive awareness—the cognitive capacity for body-state perception—demonstrating that individuals with gambling disorder exhibited significantly lower interoceptive awareness compared to both healthy controls and patients with alcohol use disorder. This finding is noteworthy as it suggests that gambling addiction may involve a more pronounced disruption of internal bodily awareness than substance-based addictions, potentially reflecting the unique behavioral nature of the reward mechanism in gambling disorder.

Genetic evidence further substantiated the neurobiological architecture of gambling disorder. Deng et al. (2025), utilizing polygenic risk scores from the Avon Longitudinal Study of Parents and Children (ALSPAC; $n = 4,995$), demonstrated that the externalizing factor polygenic risk score exhibited the strongest genetic associations with gambling phenotypes, emphasizing the importance of synaptic function and neuronal plasticity. These findings support shared genetic liability across substance misuse, problematic gambling, and other behavioral addictions, consistent with the DSM-5 reclassification of gambling disorder within the addictive disorders framework.

Table 2. Summary of Neurobiological Mechanisms in Gambling Disorder

Mechanism	Key Findings	Key References
Dopaminergic Dysregulation	Altered dopamine release reinforces risk-seeking; drives cognitive distortions and loss-chasing	London et al. (2024); De Vita (2025)
Prefrontal-Striatal Connectivity	Impaired executive control over reward-driven impulses; deficient inhibitory regulation	London et al. (2024)
Interoceptive Awareness	GD patients show lower body-state perception than substance addiction and healthy controls	London et al. (2024)
Genetic/Polygenic Risk	Externalizing PRS strongly associated with gambling; shared genetic liability with SUDs	Deng et al. (2025)
Synaptic Plasticity	Multiple biological pathways implicate neuronal plasticity in gambling-substance comorbidity	Deng et al. (2025)

3.6. Treatment Outcomes: Cognitive-Behavioral Interventions

Cognitive-behavioral therapy (CBT) emerged as the most extensively evaluated psychological intervention for gambling disorder through the reviewed literature. Pfund et al. (2023) conducted a comprehensive meta-analysis of 29 randomized controlled trials encompassing 3,991 participants and reported that CBT significantly reduced gambling disorder severity (Hedges' $g = -1.14$), gambling frequency ($g = -0.54$), and gambling intensity ($g = -0.32$) at post-treatment relative to control conditions. However, it is important to note that CBT did not demonstrate significant effects on follow-up outcomes, suggesting that treatment gains may attenuate over time.

Regarding the mechanisms through which CBT operates, Free et al. (2024) conducted a systematic review and meta-analysis examining putative mechanisms of change across 15 RCTs ($n = 1,536$). The findings revealed that CBT participants demonstrated more favorable gambling cognitions ($g = -0.41$), enhanced coping behaviors ($g = 0.27$), and substantially improved self-efficacy ($g = 1.12$) compared to control conditions. The particularly large effect size for self-efficacy suggests that restoration of perceived control over gambling behavior may represent a critical therapeutic target.

Furthermore, Pfund, King, et al. (2023) extended the outcome framework beyond gambling-specific measures, demonstrating that CBT significantly reduced comorbid anxiety ($g = -0.44$) and depression ($g = -0.35$) while increasing quality of life ($g = 0.40$) at post-treatment. These findings underscore the transdiagnostic utility of CBT for gambling disorder, addressing not only the primary behavioral symptoms but also the broader psychological burden associated with the condition.

Table 3. *Meta-Analytic Effect Sizes for CBT Interventions in Gambling Disorder*

Outcome Domain	Hedges' g	k / N	95% CI	Source
GD Severity	-1.14	29 / 3,991	[-1.68, -0.60]	Pfund et al. (2023)
Gambling Frequency	-0.54	29 / 3,991	[-0.80, -0.27]	Pfund et al. (2023)
Gambling Intensity	-0.32	29 / 3,991	[-0.51, -0.13]	Pfund et al. (2023)
Gambling Cognitions	-0.41	15 / 1,536	N/R	Free et al. (2024)
Self-Efficacy	1.12	15 / 1,536	N/R	Free et al. (2024)
Coping Behaviors	0.27	15 / 1,536	N/R	Free et al. (2024)
Anxiety	-0.44	9 / 658	N/R	Pfund, King et al. (2023)
Depression	-0.35	9 / 658	N/R	Pfund, King et al. (2023)
Quality of Life	0.40	9 / 658	N/R	Pfund, King et al. (2023)

Note. *k* = number of studies; *N* = total participants; *CI* = confidence interval; *N/R* = not reported; *GD* = gambling disorder.

4. Discussion

The present systematic review provides a comprehensive synthesis of the behavioral and psychological dimensions of gambling addiction, integrating evidence from 47 studies across cognitive, personality, neurobiological, and treatment domains. Several overarching themes emerge from this synthesis that carry significant implications for both theoretical understanding and clinical practice.

First, the convergent evidence regarding cognitive distortions as both risk factors and maintenance mechanisms for gambling disorder reinforces the cognitive-behavioral model of gambling addiction originally proposed by Blaszczynski and Nower (2002). The identification of cognitive distortions as mediators in the relationship between gambling exposure and problem gambling development (Richard & King, 2022) supports the theoretical proposition that maladaptive cognitions are not merely correlates but play a causal role in the progression toward disordered gambling. This finding has direct clinical implications, as it provides empirical justification for the centrality of cognitive restructuring within treatment protocols.

Second, the neurobiological evidence synthesized herein substantially strengthens the reconceptualization of gambling disorder as a behavioral addiction sharing fundamental neural mechanisms with substance use

disorders. The demonstrated dysregulation in dopaminergic reward pathways and prefrontal-striatal connectivity (London et al., 2024), combined with genetic evidence of shared liability through externalizing polygenic risk (Deng et al., 2025), provides a compelling neurobiological rationale for the DSM-5 reclassification. The novel finding regarding diminished interoceptive awareness in gambling disorder relative to alcohol use disorder (London et al., 2024) opens promising avenues for understanding the unique phenomenology of behavioral addictions and may inform the development of interoception-based therapeutic approaches.

Third, the treatment literature demonstrates that CBT is efficacious in reducing gambling severity, frequency, and cognitive distortions at post-treatment, with notable transdiagnostic benefits for comorbid depression and anxiety (Pfund et al., 2023; Pfund, King et al., 2023). However, the absence of significant follow-up effects represents a critical limitation that necessitates attention. The large effect size observed for self-efficacy (Free et al., 2024) suggests that interventions targeting perceived behavioral control may be particularly potent, and future research should examine whether sustained self-efficacy gains mediate long-term recovery outcomes.

Notwithstanding the contributions of this review, several limitations must be acknowledged. The heterogeneity in study designs, populations, assessment instruments, and outcome measures limited the scope for quantitative meta-analytic synthesis across all domains. The predominance of studies from high-income Western countries constrains the generalizability of findings to non-Western cultural contexts, where gambling behaviors and associated psychopathology may manifest differently. Additionally, the reliance on self-report measures for assessing cognitive distortions and psychological symptoms introduces potential response biases.

5. Conclusion

This systematic review establishes that gambling addiction is a multidimensional disorder sustained by the interplay of cognitive distortions, impulsivity, emotional dysregulation, neurobiological vulnerabilities, and comorbid psychopathology. Cognitive distortions—particularly the gambler’s fallacy, illusion of control, and near-miss effects—constitute central maintenance mechanisms, while dysregulation in dopaminergic reward systems and prefrontal-striatal connectivity provides the neurobiological substrate for compulsive gambling behavior. Cognitive-behavioral therapy demonstrates significant short-term efficacy across gambling-specific and broader psychological outcomes, though long-term maintenance remains an area requiring further investigation.

The findings carry important implications for clinical practice, suggesting that integrative treatment approaches combining cognitive restructuring,

self-efficacy enhancement, and targeted pharmacological interventions may optimize therapeutic outcomes. From a public health perspective, the elevated risk associated with online gambling formats and gambling-like digital features underscores the urgency for regulatory frameworks that address the evolving landscape of gambling access. Future research should prioritize longitudinal designs, cross-cultural investigations, and the development of personalized intervention strategies informed by individual neurocognitive and psychological profiles.

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