From Play to Pathology: Mental Health, Risk Factors, Neurobiological Mechanisms, and Drivers of Gambling Addiction in Adolescents and Young Adults

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Abstract. Gambling addiction in youth and young adults is a modern, complex public health issue rooted in a convergence of factors, including developmental risks, psychosocial challenges, and disordered neurotransmitter systems. This review of the literature highlights current research in the areas of epidemiology and mental health comorbidity, (i.e., depression, anxiety, ADHD, drug use, suicidality), psychosocial risk factors (i.e., family and peer social norms, socioeconomic disadvantage, internet advertising), and neurochemical processes (i.e., dopaminergic sensitisation, serotonergic dysregulation, GABAergic dysregulation, glutamatergic dysregulation, noradrenergic hyperarousal, endogenous opioid reinforcement) involved in the transition from recreational to pathological gambling. Online gambling formats (i.e., online gambling, micro-transactions, loot boxes) create an increased reinforcement density and accessibility that serves to elevate harms and downstream effects, including financial debt, substance co-use, and suicidality, especially among young adults. The primary contribution of this paper is both theoretical and integrative, as it presents an interactional developmental model that specifies and expands on neurodevelopmental immaturity, psychosocial forces, and neurotransmitter dysregulation, which together shift behaviours from play to pathology. The review addresses a critical gap in the knowledge base by showing how to connect epidemiological observations to neurobiological mechanisms and knowledge on prevention, while advocating for longitudinal and culturally appropriate research.

Keywords: Gambling disorder; Adolescents; Young adults; Neurotransmitters; Risk factors; Digital gambling

INTRODUCTION

Over the decades, gaming has grown to astonishing global proportions, driven by legalised new forms of gambling, with both online betting and the popularity of sports apps and casinos increasing. Gambling can be best defined as risking something of value in the hopes of obtaining something of much greater value [1]. What was once limited mainly to land casinos is now prominent on digital apps, fantasy sports sites, and micro-betting features that enable near-continuous betting. While the sites have provided a rich environment for gambling play for cohorts, the consequences are especially severe in adolescents and young adults [2].

Epidemiological data emphasise the prevalence of the problem. In a recent meta-analysis of 380 representative samples from 68 countries, an estimated 46.2% of adults and 17.9% of adolescents gambled in the previous 12 months [3]. Among adults, 8.7% were identified as engaging in risky gambling, and 1.41% met the criteria for problem gambling, with the highest rates reported among online casino or slots currency gamblers. Among youth, the prevalence of problem gambling ranges from 0.2% to 12.3% varying by methodology and geography; nonetheless, results consistently point out a small minority of youth who develop gambling problems [3]. Newer research also delineates subgroups of youth at greater risk, including those who gamble in both mixed modes (both online and offline), those whose parents experience gambling problems, and those exposed to electronic gambling machines [4]. In particular, sports betting is a highly appealing but hazardous form of gambling among adolescents, often co-occurring with impulsivity and peer pressure to gamble, as well as the concurrent use of substances [4].

Neurodevelopmental factors can account for differences in adolescent vulnerability. During adolescence and early adulthood, the brain's reward system operates in a state of heightened stimulation, while executive functions, including impulse control and planning over longer time horizons, are still developing [5]. This gap in development leads to a heightened propensity for risk-taking, sensation-seeking, and ultimately a greater risk that gambling will be more attractive and more harmful. Additionally, the peer-based and social media influence, which tends to glorify gambling or normalise betting on sporting events, can also promote problematic behaviours [6].

With an increase in interest in gambling disorder as a form of behavioural addiction, there has yet to be an integrative review linking epidemiological trends among youth gamblers to mental health correlates, health determinants, and neurobiological processes.

This gap in the academic literature further emphasises the need for focused synthesis. This review aims to synthesise evidence for gambling prevalence and epidemiology, among youth and adults, while delineating mental health correlates, psychosocial risk determinants, and neurobiological substrates. By exploring the developmental background and broader context, this article seeks to thoroughly situate youth gambling within a public health framework, providing a more comprehensive synthesis that can be valuable for framing prevention, policy, and intervention strategies.

RESULTS AND DISCUSSION

Mental Health Correlates of Gambling in Youths. Young adult and adolescent problem gambling is a public health concern, both financially and socially, and due to the overwhelming co-occurrence with psychiatric morbidity [7]. Current evidence illustrates that gambling problems do not come as a standalone intervention, as they co-occur with depression, anxiety, impulse-control issues, substance misuse, and suicidality [8]. Importantly, this relationship is bi-directional, meaning poor mental health both increases a person's vulnerability to gambling disorder and worsens as that person continues to gamble [9].

Depression and anxiety are two of the most common correlates of gambling in adolescents [10]. Large-scale epidemiological research has shown that people with gambling disorder are reviewed as being wildly more likely to be given a diagnosis of anxiety disorder; adolescent samples have described higher levels of emotional distress, inappropriate excitability, and disinhibition [11]. The pathways model of gambling, which summarises the dynamics at play, proposes that gambling behaviour is maintained not only by positive reinforcement (i.e., winning is exciting) but also by negative reinforcement (i.e., gambling temporarily alleviates tension or sadness) [12]. Therefore, anxiety-driven gambling is not just maladaptive, but it also falls in line with coercive coping strategies and outcomes witnessed in substance abuse disorder populations [13]. Youth who indicate

gambling as a way to avoid anxiety are also likely to exhibit higher rates of alcohol and tobacco use, violent behaviour, and permissive inclination towards gambling, showcasing ways in which an individual and collective dysregulation of affect fuels the severity of gambling behaviour and total risk behaviour [14].

Attention-deficit/hyperactivity disorder (ADHD) and other impulse-control disorders present a second risk factor. Adolescents showing cognitive features of ADHD, specifically impulsivity and novelty seeking, exhibit elevated risk for gambling difficulties [15]. Some research suggests that once gambling is initiated, adolescents with ADHD may have courses as similar as their non-ADHD counterparts but are comparatively more likely to transition quickly through leisure-time gambling to a problem gambling situation [15]. In turn, impulsivity has been a consistently identified risk factor for gambling disorder across several cognitive processes, including decision-making, inhibition of motor responses and control of attention [16]. These findings suggest that early failure at delaying impulses and inhibiting risky decisions may prime the brain and youths for gambling escalation as described in other substance and behavioural addictions [16].

There is a significant association between gambling and substance use found among youths, and it is worthy of consideration. Alcohol, marijuana, and drug use often co-occur with gambling, and a meta-analysis shows that mutual vulnerabilities in reward system pathways drive this association [17]. Longitudinal work has also demonstrated that disordered anxious adolescents have almost double the prevalence of substance use disorders and that substance-abusing adolescents have high levels of anxiety and gambling problems [18]. This overlaps larger syndromes of externalising and internalising dysfunction in which gambling modulates and elevates psychological distress and increases drug use [19]. Suicidality is perhaps the most severe correlate of gambling in youth. Both clinical and population studies regularly support greater levels of suicidal ideation and suicidal behaviour within the problem gambling population, with few studies that present evidence that gambling problems are predictive of some of the most concerning suicide risk levels among behavioural addictions [20].

Financial indebtedness and losses from disasters frequently precipitate suicidal crises, with gambling shame and guilt compounding feelings of hopelessness [21]. This relationship has a very complex pathophysiology. Other contributors to suicide risk include co-morbid conditions such as depression, anxiety and/or drug/alcohol use, which research shows was much more likely to result in attempted suicides among gamblers who also had a co-morbid disorder compared to gamblers without a co-morbid disorder [21]. In addition to this alarming information, scientific evidence also suggests that young people with gambling problems are up to nine times more likely to have made a suicide attempt than those who do not have gambling problems [22].

Neurobiological and Neurotransmitter Mechanisms involved in gambling. Decisions to gamble engage a highly conserved reward mechanism that favours immediate and visible reward potential, regardless of the rationality or consequences associated with such decisions. The centre of this circuit is the mesolimbic dopamine system, which projects from the ventral tegmental area (VTA) to the nucleus accumbens, which mediates anticipation of reward and reinforcement [23]. Functional neuroimaging techniques consistently demonstrate that both financial reward and so-called "near-miss" events activate the ventral striatum and midbrain, which elicit vigorous activity. Near-misses are particularly interesting, as they are objectively equivalent to losses; however, they evoke a perceived tendency to strengthen the motivational desire to continue playing and recruit the same substrates as wins [23]. Reinforcement-learning theories suggest that such is an expression of a sequence of abrupt, opposite prediction error signals, which produce an asymmetrical sense of control and pleasure [24]. The insula and rostral anterior cingulate cortex (ACC) are similarly involved in such events, and activation of these areas correlates with the severity of gambling and a measure of subjective craving [25]. This combination of reward coding and asymmetrical reinforcement learning creates a strong motivational circuit that can elicit and reinforce gambling behaviour [23].

Dopamine is central to this cycle, as the primary neurotransmitter for the reward prediction error system [26]. Electrophysiological studies demonstrate that phasic bursts of dopamine neurons signal positive surprise wins but do not reflect declines in surprise during surprise losses, establishing reinforcement learning across time [27]. In gambling, the dopamine responses to both wins and to cues for anticipated rewards are overestimated and ultimately increase cravings and

sensitisation [23]. Neuroimaging PET studies reveal that individuals with gambling disorder show varied dopamine release or receptor density, specifically D3 receptors in the ventral striatum and midbrain region when novelty seeking and impulsivity are dimensions of novelty seeking, novelty-seeking, and/or impulsivity [28]. Furthermore, the fact that some patients with Parkinson's disease who are treated with dopamine agonists (i.e. drugs that prefer D2 and D3 receptors) develop pathological gambling is substantial evidence supporting the causal relationship of dopamine [29]. Yet contradictory outcomes emerge from other studies that don't show lasting or stable differences between groups and indicate that dopaminergic pathology may be state-dependent, and further moderated by genetic and personality variables, such as impulsivity [28].

Serotonin serves an essential counter-regulatory function to the reward-seeking bias of dopamine, primarily through its effects on impulse control, mood, and compulsive behaviours [30]. Individuals with deficient control of impulses, characterised as a gambling disorder, have shown low serum metabolites of serotonin and blunted responses to serotonergic pharmacological challenges [30]. The engagement of serotonin within the ventromedial prefrontal cortex (vmPFC) and orbitofrontal cortex (OFC) is essential for inhibiting behaviour and assessing long-term outcomes; thus, their serotonergic dysregulation underlies continued gambling behaviour despite adverse consequences [30].

Glutamatergic circuits have also emerged as major contributors to craving and cue-induced relapse in gambling disorder. Cortico-striatal glutamate signalling underlies the capacity to learn stimulus-reward pairings, and the maladaptive plasticity in this circuit helps explain how cues associated with gambling can elicit extreme levels of craving and relapse [31]. While there is a lack of direct neuroimaging of glutamate in gambling, addiction-related findings and pharmacological implications provide evidence that there are dopaminergic glutamine and signalling dysregulation in the orbitofrontal cortex (OFC) and anterior cingulate, associated with rational decision making.

GABA, the primary inhibitory neurotransmitter in the brain, also plays a role in the neurobiology of gambling. Researchers generally accept that dopamine release increases GABAergic activity in cortical areas, helping regulate arousal and providing an inhibitory counterbalance to excessive excitation [32]. In the case of a gambling disorder, inhibition of GABA by dopaminergic activity is likely decreased and may potentially be reversed, which corresponds with failure to maintain inhibitory control [33]. The status of GABA activity in the prefrontal and paralimbic areas, with possible dysfunction, would lead to compulsivity and failure to stop gambling sessions once initiated [23].

Norepinephrine affects even one more layer of understanding by modulating arousal and stress reactivity [34]. Increased arousal fuels risky decision-making behaviour, heightens the reward value of winning, and even if the individual is losing money, gambling will keep their attention [35]. Young people with a high trait extraversion or distressed reactivity may be especially vulnerable from a norepinephrine standpoint because norepinephrine represents the increasing feeling of attentional salience of gambling cues and increasing emotions [34].

Endogenous opioid processes play a role in the pleasurable aspect of gambling. The release of endogenous opiates increases the pleasure when winning the outcome, and thus promotes the habit of gambling. However, deprivation of the expectation for reward can elicit withdrawal-like states, which drive compulsive gambling and serve as a way to cope with discomfort. Thus, in addition to the euphoric feeling associated with gambling, there are also cycles of negative reinforcement associated with opioid pathology that serve to maintain dependence [28].

At a systems level, a gambling disorder involves a breakdown of balance between bottom-up limbic networks driving salience and the top-down cortical networks deploying executive control [36]. The prefrontal cortex, particularly the ventromedial and dorsolateral regions, typically plays a role in risk assessment for long-term decisions and engages inhibitory control [5]. These regions fail to activate to the same degree in gambling disorder, particularly in situations where an individual is required to make high-risk choices [23]. The amygdala helps imbue emotional meaning into gambling outcomes, while the hippocampus links outcomes to memories that influence future decisions, whether to continue gambling (wins) or refrain (losses) [28]. The insula is another critical factor because it helps transform interoceptive data into conscious desire, which mediates the illusion of control and near-miss processes. Together, a network of hyperactive reward

processing with diminished access to inhibitory control and motivated by biased emotional learning, lies at the centroid of pathological gambling [23].

There is a need to understand the nature of this neurochemical connection because it not only accounts for the immersive and compulsive nature of gambling behaviour, but also suggests that specific neurochemical systems can be targets of treatments that modulate expression.

Table 1 – Neurotransmitter Systems and Their Roles in Gambling Addiction

Neurotransmitter	Primary Role in the Brain	Role in Gambling Addiction	Key Mechanisms/Findings	Key References
Dopamine	Reward, motivation, reinforcement learning	The processes described above are known mechanisms of craving and reinforcement in gambling.	Altered reward-prediction-error signals and sensitising effects in the mesolimbic pathway arise from activity in the VTA and the nucleus accumbens, which in turn trigger the "near-miss" effect and compulsive behaviour.	[23, 25]
Serotonin (5-HT)	Mood regulation, impulse control, and inhibition	Gambling and impulsivity dysregulation are associated with compulsive behaviour.	Poor impulse control, risk-taking, and relapse are associated with low levels of serotonin.	[37]
Glutamate	Excitatory signalling, learning and memory	The processes described here modulate relapse and craving for gambling.	Gambling urge control and cue-driven craving are associated with cortico-striatal dysregulation and glutamatergic dysfunction.	[31]
GABA	Major inhibitory neurotransmitter; regulates neural excitability	The resultant state of these processes contributes to the loss of control while gambling.	Low GABA tone is associated with gambling episodes and compulsive behaviours that lack self-control.	[33]
Norepinephrine	Arousal, attention, stress response	These processes increase stress and gambling activity, intensifying the "high" a person feels during a win.	Norepinephrine increases during episodes of gambling with a risk and physiological arousal when winning and losing, and during emotionally dysregulated states of craving, withdrawal, and altered hedonic experience while gambling.	[23, 35]
Endogenous opioids	Hedonic pleasure, pain modulation	Mediate the rewarding "high" of wins.	The presence of dysregulation is associated with disorders related to craving, withdrawal-like symptoms, and altered hedonic response to the gambling outcome.	[28]

From Play to Pathology: Pathways to Addiction. Researchers can understand the path of gambling as a multifaceted architecture of converging pathways that transform episodic risk-taking into chronic, compulsive pathology [23]. Grounded in empirical research, authors [12] pathways model offers a pragmatic taxonomy that continues to have utility in framing these trajectories: a behaviourally conditioned pathway in which gambling is learned and reinforced through environmental contingencies; an emotionally vulnerable path, in which gambling is a way to escape or maladaptively cope with deeper affective issues; and a biologically vulnerable pathway in which individual differences in temperament and neurobiology (i.e., high impulsivity, altered reward sensitivity) place individuals on a path to rapid escalation [12].

In the pathway of behaviourally conditioned gambling, gambling starts recreationally and then is enhanced through functional reinforcement schedules, intermittent wins, and contextual affordances [38]. Features that are present in most forms of gambling, such as variable-ratio reward schedules, frequency of events occurring, near-miss outcomes, and salient sensory stimulation, further promote continued engagement in gambling as a result of energetically substantial reinforcement learning signals [39]. Near-misses and the illusion of control attenuate the reward prediction process and sensitise ongoing gambling despite losses. Cue exposure over time leads to the formation of strong associative links between environmental stimuli and craving [39]. In today's context, online media and intensified marketing lower the barriers to play and increase the density of reinforcement opportunities, leading to increasingly rapid movement along this roulette wheel path, especially for vulnerable individuals [40].

The focus on the affectively vulnerable pathway highlights the role of affective distress and maladaptive coping. Empirical studies suggest that emotion dysregulation and avoidant coping styles emerge as factors predicting greater gambling involvement [41]. The biologically vulnerable pathway highlights dispositional and neurobiological risk factors that lead to reactivity toward gambling cues [23]. These biological vulnerabilities are shaped through genetic and developmental processes, and their interaction with environmental exposure is what largely determines whether someone experiments with gambling or crosses over into a disorder [41].

Developmental characteristics place youth at particular risk along each of the three pathways. During adolescence and early adulthood, there is an imbalance between a hyper-reactive, relatively mature reward system and the still-maturing prefrontal executive regulatory systems. This developmental imbalance strengthens novelty-seeking, risk-taking, and reward-based learning, while limiting abilities in planning, impulse control, and thinking about costs and consequences over time [42]. In practical terms, young people experience heightened motivational responses to gambling cues, will be sensitive to intermittent reinforcement effects, and are less likely to stop gambling based on lost money, which allows youth to move from recreational to problem gambling more quickly, especially with online availability and peer normalisation [38].

From a clinical perspective, effective prevention and treatment will need to be multimodal: reducing environments (youth access controls; advertising reductions; skills training; early family interventions) and, where required, modulating neurobiological mechanisms (pharmacological or neuro-modulatory therapies) to restore balance to reward control circuits. Therefore, when researchers acknowledge the variability of trajectories from play to pathology, they reframe gambling disorder as a developmental, biopsychosocial problem that clinicians can treat in a targeted, context-specific way [30].

Future Directions. This article highlights that the rise of gambling disorder in young people is due to the combination of mental health issues, psychosocial factors, and neurobiological factors related to the key neurotransmitters. However, there is little longitudinal research, and most existing cross-sectional studies rarely focus on adolescents. There is a need for longitudinal, multimodal, cross-disciplinary research in epidemiology, neurobiology, and the psychosocial sciences to address developmental differentiation; this will need new neuroimaging, genetic and biomarker technologies to expand the research base, and new psychosocial and neurobiological clinical research on psychosocial determinants and neurobiological imbalance will need to be undertaken. There is a need for research to combine the use of opioids (antagonists), glutamate (modulators), serotonergic medications and CCDT, meditation, and mindfulness psychotherapies.

CONCLUSIONS

A gambling disorder among adolescents and young people is a serious public health problem that involves dysregulation of neurotransmitter systems, psychosocial influence, and developmental risk factors. Family support, support from peers, and exposure to socioeconomic stress and gambling through digital channels facilitate gambling and transition to compulsive gambling. Evidence points to pathways associated with dopaminergic, serotonergic, glutamatergic, GABAergic, noradrenergic, and opioid, most often in relation to associated mental health comorbidities, such as depression, anxiety, attention-deficit hyperactivity disorder, and suicidality. Evidence-based solutions to these issues need to be integrative, bridging neuroscience, psychology, and policy to prevent play from transitioning to pathology and to protect youth well-being.

Conflict of interest

The authors declare that they have no conflict of interest.

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