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The Genetics of Behavioral Addiction: The Neurogenetic Pathways that Link Impulsivity with Behavioral Addiction Risk

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Abstract

People who are more impulsive tend to act without foresight, making them more likely to develop habits such as gambling or excessive gaming. These habits activate the brain's reward system but often lead to lack of control. Currently, there is a lack of research that maps out the system in which genetics influences behavioral addictions. This review paper synthesizes evidence on how impulsivity-related genes may affect brain regions involved with reward and self-control. Dopaminergic and serotonergic systems are neuron networks that use dopamine and serotonin as neurotransmitters, and are crucially involved in reward and motivation. Genetic variants in the aforementioned systems influence neurotransmitter balance, which results in altered signaling across the brain's reward system (which includes the prefrontal cortex, ventral tegmental area and nucleus accumbens). This altered signaling leads to increased impulsivity expressed through insufficient inhibition and high reward sensitivity. In this way, genetic variation in dopamine and serotonin signaling becomes functionally relevant by altering the neural circuits that mediate reward learning and self-control. Because impulsivity may mediate the association between genetic variation and vulnerability to behavioral addictions, understanding this neurogenetic pathway is quite important for human health, as it may allow for early identification of at-risk individuals and guide the development of prevention strategies.

Keywords: impulsivity, behavioral addiction, dopamine, serotonin, neurogenetics, reward system

1. Introduction

Addiction is frequently depicted as a cycle, but what compels a person to take that first step? Perhaps people who often struggle with self-control do so not just because of poor choices, but because of a biological predisposition. Behavioral addictions (BA) are chronic conditions in which individuals compulsively engage in specific activities that do not involve the

use of substances—such as excessive gambling, gaming, shopping, internet use, sex, pornography, work, exercise, etc.—that produce immediate reward. Individuals who suffer from these behavioral addictions continue to compulsively engage in these activities despite them having negative consequences on their lives, including their mental and physical health. Similar to substance-use disorders, they comprise inhibited self-control and are stimulated by interactions between neural circuits, genetic predispositions, and environmental factors (Grant et al., 2010). Although most of these disorders are not clinically recognized since they are not included in the Diagnostic and Statistical Manual of Mental Disorders, 5th ed. (DSM–5), behavioral addictions are very real conditions that affect countless people around the globe (American Psychiatric Association [APA], 2013).

Impulsivity can be understood as a tendency to initiate actions without evaluating potential consequences (Bevilacqua & Goldman, 2013) and could be the factor that pushes someone to engage in addictive behaviors in the first place. Impulsivity is not only psychological, but has a genetic and neurological foundation. There is compelling evidence that impulsivity is heritable, with some studies calculating that around 45% of the variance in impulsivity is due to genetic factors (Congdon & Canli, 2008). Studies have consistently found that multiple brain regions and neurotransmitter systems, particularly the dopaminergic and serotonergic systems, participate in the impulsive behaviors that happen throughout the addiction cycle (Brewer & Potenza, 2008).

Building on genetic evidence, neurotransmitter studies show that dopaminergic systems are involved in several different brain functions, including learning, reward, and control (Ko & Strafella, 2012). Dopamine (DA) neurons in the ventral tegmental area (VTA) regulate both goal-directed and habitual behaviors. Activities that offer instant reward enhance dopamine release and lead to hyperbolic reward signaling, craving, and compulsive addiction-seeking (Poisson et al., 2021a). Serotonin (5-HT) is known to have been associated with several motor, cognitive, and affective functions, including mood disorders. Various studies have found that reduced levels of 5-HT promote impulsive behaviors, such as impulsive action (failing to repress inappropriate actions) and impulsive choice (choosing immediate rewards over delayed rewards) (Miyazaki et al., 2012a).

At the neural systems level, these genetic changes converge in the mesolimbic reward circuitry. The mesolimbic system, or reward system, is made up of different brain areas that are in control of processing reward. Reward is defined as the "natural process during which the brain associates diverse stimuli (substances, situations, events, or activities) with a positive or desirable outcome" (Lewis et al., 2021). This process results in changes in a person's behavior, eventually causing them to seek that particular positive stimulus (Lewis et al., 2021). Three specific brain areas are part of the mesolimbic system, which are directly involved with the release of DA and 5-HT: the VTA, the nucleus accumbens (NAc), and the prefrontal cortex (PFC). The VTA is located in the ventral midbrain, and projections from the VTA release DA throughout the neural circuit to the NAc and PFC when reacting to a motivationally significant stimulus (Kalivas & Volkow, 2005; Pistillo et al., 2015). The NAc is situated in the ventral striatum and plays an integrative role in processing motivation, reinforcement, and reward by coordinating inputs from the PFC and the VTA (Bayassi-Jakowicka et al., 2021). The PFC plays an essential role in evaluating motivationally relevant events and predicting rewards. This area is activated by cues associated with reward and determines if a behavioral response will happen (Kalivas & Volkow, 2005). These different factors are linked in a particular pathway in which DA neurons in the VTA project to the NAc and the PFC (Poisson et al., 2021). Functionally, alterations in these regions translate into measurable differences in impulsive behavior. Understanding this gene-brain-behavior pathway may be critical to gain insight into how impulsivity is linked to behavioral addictions, and therefore how to prevent individuals from beginning that addictive cycle due to impulsivity.

Recent literature on behavioral addictions often focuses solely on genes, brain regions, or behaviors instead of the pathway as a whole. Research that does have a more comprehensive outlook is focused on substance use disorders as opposed to behavioral addictions (also considered impulse-control disorders or ICDs). Although both BAs and ICDs involve impairments in inhibitory control, they do not manifest in the same way, as BAs are driven by reward seeking, whereas ICDs reflect impulsive actions intended to relieve internal tension. Currently, there is a lack of investigation into the genetics, neuroimaging, and behavioral factors of behavioral addictions, which generates a limited understanding of how genetics shape the vulnerability to these disorders.

This review aims to address how impulsivity-related genes (such as DRD2, DRD4, DAT1, MAOA, SLC6A4, HTR1B, HTR2C, and HTR2A) affect brain regions involved with reward and self-control (such as the VTA, NAc, and PFC) and how this contributes to the elaboration of behavioral addictions. This paper argues that understanding how these impulsivity-related genes affect these specific brain regions could reveal key mechanisms that are fundamental to behavioral addictions and may aid in early identification and prevention of these disorders. Taken together, these findings suggest a unified pathway in which genetic variation affects DA and 5-HT signaling, which in turn alters activity in the VTA, NAc, and PFC, increasing impulsive actions and ultimately heightening vulnerability to BAs.

2. The Scope of Behavioral Addictions

In the past, behavioral addictions (BAs) have been equated with impulse-control disorders (ICDs), due to the similarities in certain characteristics, specifically compulsivity and inhibited impulse regulation (Brewer & Potenza, 2008; Geller, 2008). Nevertheless, as asserted by Grant et. al (2010), this fusion is inaccurate. This is because not all BAs are ICDs and not all ICDs are BAs (illustrated in Figure 1). This difference became apparent when gambling disorder, formerly included in the ICD section of the DSM-4, was reclassified under the Substance-Related and Addictive Disorders section in the DSM-5 (Pinna et al., 2015). This change highlights how BAs are different from ICDs and that they warrant their own classification.

Currently, the DSM-5 does not officially recognize BAs as mental disorders "because at this time there is insufficient peer-reviewed evidence to establish the diagnostic criteria and course descriptions needed to identify these behaviors as mental disorders" (American Psychiatric Association, 2013). However, this lack of official recognition may itself be contributing to the lack of research available on BAs, and this might be creating a futile cycle in which the lack of official recognition deters research and the lack of research impedes clinical recognition. This is why it is imperative to develop further investigations into this matter, as individuals worldwide would benefit from a deep understanding of the disorders at hand.

Despite BAs not being widely recognized as clinical disorders beyond pathological gambling, research by Bevilacqua & Goldman (2013) and Congdon & Canli (2008) found genetic underpinnings to these types of impulsive behavior, involving impulsivity-related genes in the serotonergic and dopaminergic pathways that echo previous findings in substance use disorders (SUDs) (Poisson et al., 2021). Comprehensive research (such as Brewer & Potenza (2008), Leeman & Potenza (2012), and Lobo & Kennedy (2006)) further demonstrates that BAs have a neurobiological and genetic foundation, in which genes and brain regions alike influence these types of behaviors. Grant et al. (2010) and Pinna et al. (2015) have argued that these disorders should not be ignored, despite their current lack of clinical recognition. The public health burden of BAs is substantial. A global meta-analysis estimates that between 0.2 and 2.1% of individuals develop BAs like pathological gambling,

yet fewer than 15% receive treatment due to limited access and insurance coverage (Petry et al., 2017; Slutske et al., 2010; Stucki & Rihs-Middel, 2007). Similarly, internet gaming addiction affects an estimated 1.7-10% of the US population, but inconsistent diagnostic criteria have impeded clinical interventions (Video Game Addiction, n.d.). For the most part, research on BAs demonstrates the validity of BAs, yet this lack of approved and accessible treatments highlights the need for further research and recognition that may enable early identification and prevention.

For this review, behavioral addictions (BA) will be defined as chronic conditions in which individuals compulsively engage in activities that do not involve the use of substances. These activities exhibit similar features to addictions, such as inhibited self-control, compulsive cravings, and persistence in engaging in such activities despite them having a negative effect on their lives. This includes pathological gambling, internet gaming, shopping, internet use, compulsive sexual behavior, pornography, work, and exercise, as neurological and clinical similarities with substance-use disorders have surfaced (Geller, 2008; Grant et al., 2010; Pinna et al., 2015). Whereas, behaviors such as oppositional defiant disorder, intermittent explosive disorder, conduct disorder, antisocial personality disorder, pyromania, kleptomania and trichotillomania, which are all ICDs (American Psychiatric Association, 2013), will all be excluded from the BA category (as shown in Figure 1) because they lack the similar neuroadaptive mechanisms that are distinctive of addictions (Grant et al., 2010).

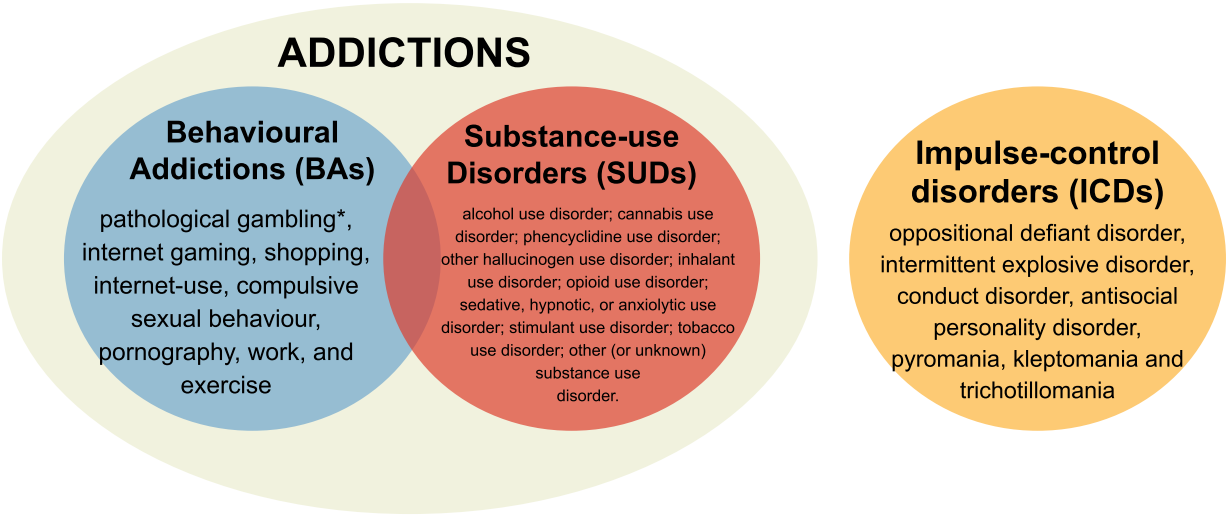

Figure 1: The Spectrum of Addictive and Impulsive Disorders.

Note: This diagram demonstrates the classification of addictive and impulsive disorders for the purpose of this review. The outermost circle represents all addictions, divided into two main categories, Behavioral Addictions (BAs) and Substance-use Disorders (SUDs). All SUDs and Impulse-control Disorders (ICDs) shown are based on the DSM-5 (American Psychiatric Association, 2013). Pathological gambling (marked with an asterisk) is included in the DSM-5 as well. Other BAs are included for this review, but are not currently recognized as clinical disorders by the DSM-5.

3. Key Genes and Their Role in Impulsivity

The heritability of impulsivity is a factor that has been demonstrated for both impulsive traits that are included in the DSM

categorization of mental disorders and those outside of these categories. Evidence from twin studies has calculated that around 45% of self-reported impulsivity is due to genetic factors (Congdon & Canli, 2008). Genes can affect impulsivity by regulating and changing reward sensitivity and inhibitory control. Previous studies have associated neurotransmitters with impulsivity, specifically dopaminergic and serotonergic pathways, which predominate in brain regions related to impulse control (Bevilacqua & Goldman, 2013). Because the DA and 5-HT genes discussed here are also implicated in both ICDs and SUDs, current evidence suggests substantial biological overlap across the three diagnostic categories. This convergence may challenge the idea that BAs are neurologically separate conditions and instead supports a continuum model of addiction vulnerability driven by shared neurogenetic pathways.

Genetic variations in DA receptors (e.g., DRD2 and DRD4) and transporters (e.g., DAT1) have been implicated in impulsive traits (Bevilacqua & Goldman, 2013; Congdon & Canli, 2008), which increase susceptibility to both SUDs and BAs (Albekairi et al., 2025; Brewer & Potenza, 2008). D2 dopamine receptors (DRD2) manage DA neuron activity by creating negative feedback. A large availability of DRD2 reduces compulsive seeking behavior, while low DRD2 availability is linked to high impulsivity and compulsive behaviors (Poisson et al., 2021; Wise & Jordan, 2021). A lack of DRD2 has been associated with contributing to both compulsive internet gaming and pathological gambling (Brewer & Potenza, 2008; Leeman & Potenza, 2013). D4 dopamine receptors (DRD4) are expressed in brain regions involved in behavioral inhibition and self-control, specifically in the PFC. Knockout studies of DRD4 revealed that mice lacking the receptor show reduced impulsive decision-making (Congdon & Canli, 2008). Some studies also found an association between DRD4 and PG (Brewer & Potenza, 2008). DA transporter 1 (DAT1) regulates DA signaling by removing DA from the extracellular space (Congdon & Canli, 2008). Immoderate levels of DAT1 can excessively reduce DA levels and potentially weaken behavioral inhibition (Brewer & Potenza, 2008). Together, these variants converge on altered reward sensitivity and inhibition, forming the first step of the gene-brain-behavior pathway.

Genetic variations in 5-HT receptors (e.g., HTR1B and HTR2B) and transporters (e.g., SLC6A4) have been implicated in impulsive traits (Bevilacqua & Goldman, 2013; Congdon & Canli, 2008) and increase vulnerability to both SUDs and BAs (Brewer & Potenza, 2008). 5-HT enhances motivation via its effects on the NAc, but it inhibits activity in the VTA (Bayassi-Jakowicka et al., 2021). The 5-hydroxytryptamine receptor 1B (HTR1B) inhibits the release of 5-HT and has been implicated in impulsive behavior by different pharmacological studies (Bevilacqua & Goldman, 2013). It has an involvement in mood regulation and impulsivity, factors that influence addiction vulnerability (Albekairi et al., 2025). The 5-hydroxytryptamine receptor 2B (HTR2B) regulates 5-HT reuptake and is associated with impulsive violence. HTR3B knockout mice showed increased compulsive seeking and reward reactivity (Bevilacqua & Goldman, 2013). Another key receptor implicated is the 5-HT2A receptor (HTR2A). Individuals with the prevalent HTR2A -1438A/A genotype displayed significantly higher maladaptive impulsivity scores (Tomson et al., 2016). Because this receptor regulates cortical serotonergic tone, variation in it may influence reward sensitivity and impulse control (Tomson et al., 2016). The 5-hydroxytryptamine transporter gene (SLC6A4) removes 5-HT from the extracellular space, managing signal duration and magnitude (Charnay & Léger, 2010). Variation of SLC6A4 is linked to impulsivity, SUDs, and ICDs (Brewer & Potenza, 2008). Finally, monoamine oxidase A (MAOA) is an enzyme that breaks down 5-HT and DA (Charnay & Léger, 2010). Higher levels of DA and 5-HT and increased aggressive behavior were found in MAOA knockout mice (Bevilacqua & Goldman, 2013). Low levels of MAOA predispose the risk for impulsive aggression because of dysregulated serotonin signaling (da Cunha-Bang & Knudsen, 2021).

Collectively, these genes influence the gene-brain-behavior pathway by changing the neurotransmitter pathways in the PFC, VTA, and NAc. These genetic influences might help understand differences in individual susceptibility to BAs, setting an outline for future research in prevention and treatment strategies.

4. The Gene-Brain-Behavior Pathway

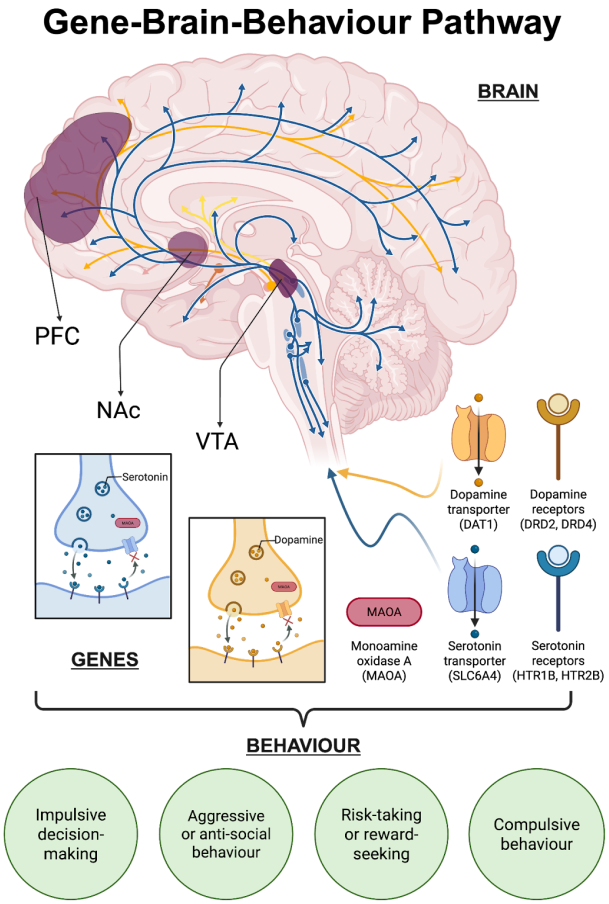

Figure 2: The Gene-Brain-Behavior Pathway

Note: Schematic representation of the influence of serotonergic and dopaminergic genes on the PFC, the NAc, and the VTA, and the associated behavioral outcomes. Created with BioRender.

Given what is known about impulsivity-related genes and behavioral addictions (BA), a potential model that explains the way genes ultimately influence the susceptibility to BAs is a gene-brain-behavior pathway (illustrated in Figure 2). In this model, the genetic variations previously discussed influence the dopaminergic and serotonergic systems, and these systems in turn alter the function of specific brain areas and form impulsive traits. These changes in the brain modify reward processing and decision-making, which manifest as behaviors (such as impulsive traits). Since the environment can alter gene expression, it could also provide an explanation for why environmental factors can also influence addiction susceptibility (Albekairi et al., 2025; Grant et al., 2010; Koob & Volkow, 2010; Lobo & Kennedy, 2006).

The aforementioned serotonergic genes (HTR1B, HTR2B, SLC6A4, and MAOA) manage 5-HT signaling, which influences mood regulation, control inhibition, and reward processing (Charnay & Léger, 2010; da Cunha-Bang & Knudsen, 2021; Miyazaki et al., 2012). Early childhood adversity increases impulsivity risk partly through epigenetic modification of MAOA expression (Charnay & Léger, 2010; da Cunha-Bang & Knudsen, 2021; Miyazaki et al., 2012). The dopaminergic pathway genes (DRD2, DRD4, and DAT1) regulate reward processing and sensitivity through the mesolimbic system. One proposed mechanism is the signaling between the VTA and the NAc (shown in Figure 2) (Ko & Strafella, 2012; Poisson et al., 2021; Wise & Jordan, 2021). These genes can specifically modify PFC activity, which regulates control over impulses (Congdon & Canli, 2008).

The changes in the DA and 5-HT pathways affect the whole mesolimbic system. The PFC, which regulates self-control and determines if there will be a behavioral response to reward cues (Kalivas & Volkow, 2005), is particularly affected by DA transmissions (Congdon & Canli, 2008). Because projections from the VTA release DA to the NAc and the PFC (shown in Figure 2), any change in this brain region can influence the neural reaction to rewarding stimuli (Kalivas & Volkow, 2005; Pistillo et al., 2015). In addition, since the NAc processes motivation, reinforcement, and reward by coordinating inputs from the PFC and the VTA (Bayassi-Jakowicka et al., 2021), changes in this region may affect the function of both regions. This paper proposes that the gene-brain-behavior pathway provides a unified model uniquely suited to explaining BAs. Understanding this pathway may explain how genetic risk factors translate into behavioral traits.

4.1. Ventral Tegmental Area (VTA) in the BA genetic pathway

The VTA, located in the midbrain, is the beginning of the mesolimbic system, which is linked by neuroimaging studies to reward learning and emotion (Ko & Strafella, 2012). The VTA houses most of the midbrain DA neurons, which mainly project to the NAc and the PFC in the mesostriatal pathway (Poisson et al., 2021). The DA signaling from the VTA to the NAc supports motivation, craving, and reward anticipation (Kalivas & Volkow, 2005). Since DA projections from the VTA influence reward learning and motivation, balanced signaling of this pathway is crucial for adapting reward-seeking and decision-making. Therefore, extreme activity may cause compulsive motivation and impaired learning or motor control (Poisson et al., 2021). Because these DA neurons are essential to generate a behavioral response to addictive activities, gene variants (DRD2, DRD4, and DAT1) that affect DA firing alter how the brain assigns motivational value to rewarding stimuli (Bayassi-Jakowicka et al., 2021). Together, these findings suggest genetic variation affecting dopaminergic signaling in the VTA can dysregulate reward prediction and motivation, contributing to the heightened impulsive drive observed in BAs. However, there is currently a lack of investigation into the effects of 5-HT in the VTA and how that may influence BA predisposition.

Some neuroimaging studies involve the monetary incentive delay task (MIDT), which is based on previous evidence that dopaminergic firing in the VTA shifts from experiencing rewards to reward-predicting cues, and is used to measure VTA responses during reward anticipation (Knutson et al., 2000; Schultz et al., 1997). In pathological gambling, studies show reduced VTA activation to generic cues but normal or even heightened activation to gambling-related cues, suggesting that VTA-driven reward-predicting cues become selectively tuned to addiction-specific stimuli (consistent with genetic and neurochemical alterations in the VTA that increase vulnerability to BAs) (Fujiwara et al., 2022).

4.2. Nucleus accumbens (NAc) in the BA genetic pathway

The NAc, located in the ventral striatum, is made up of a shell and a core. The NAc shell regulates motivational relevance

through its connection with the VTA, because it receives DA from the VTA and regulates mesolimbic reward functions (Bayassi-Jakowicka et al., 2021). The NAc core handles learned behavioral responses to specific cues by predicting motivationally relevant events (Brewer & Potenza, 2008). The NAc is central to reward processing, evaluation, and incentive-based learning, and plays a central role in the acute reinforcing effects of addictive activities because it receives motivational information from the PFC (Kalivas & Volkow, 2005; Koob & Volkow, 2010). Through its connections across the brain, the NAc manages DA release to mesocortical areas, combining reward processing with motor and cognitive functions (Bayassi-Jakowicka et al., 2021).

DA neuron activity from the VTA in the NAc creates a motivational "pull" toward cues and predicted rewards, which is essential for effective reward-seeking and decision-making (Poisson et al., 2021). Dopamine released from the NAc is regulated by DRD2 and DRD4, and is essential to forming motivation, reward learning, and adaptive behaviors because of synaptic plasticity (Bayassi-Jakowicka et al., 2021). 5-HT receptors, such as HTR1B and HTR2B, modulate the inhibition of premature actions while waiting for rewards, an action that takes place in the core of the NAc (Miyazaki et al., 2012). Reduced MAOA expression might elevate neural DA levels in the NAc, which influences reward sensitivity (Kalivas & Volkow, 2005). Neurons in the NAc show anticipatory activity in reward delay periods, further implicating the NAc in impulsive traits (Miyazaki et al., 2012).

Over time, chronic addictions induce structural and synaptic changes in neural circuits that regulate reward. In the later stages of addiction, self-control of behaviors can begin to shift from the NAc, where positive reinforcement happens, to the dorsal striatum for habitual use (Albekairi et al., 2025; Brewer & Potenza, 2008). It has also been found that BA cue exposure, especially in internet gaming, creates heightened activation in the NAc (Leeman & Potenza, 2013). A neuroimaging study conducted by Liu et al. (2016) indicates that individuals with pathological gambling have heightened cravings to gambling cues that correlate with increased activation in the NAc, suggesting hypersensitized reward prediction cues consistent with BA-related genetic vulnerability. Overall, gene-driven variations in DA and 5-HT signaling within the NAc appear to amplify reward sensitivity and cue reactivity, reinforcing compulsive reward-seeking behaviors characteristic of BAs.

4.3. Prefrontal Cortex (PFC) in the BA genetic pathway

The PFC, located in the anterior part of the frontal lobe, mainly modulates DA levels by two routes, the corticonigral and corticostriatal pathways (Ko & Strafella, 2012). Through these pathways, the PFC regulates DA release in the NAc and plays a key role in seeking behaviors (Albekairi et al., 2025). During decision-making tasks, the OFC has increased DA release levels for immediate choices, and the lateral PFC and OFC are linked to delayed rewards, with all parts of the PFC being involved in the decision-making process (Bevilacqua & Goldman, 2013). The OFC and mPFC encode reward expectation, which influences the ability to wait for delayed rewards, implicating the PFC in impulsive conditions such as addictions (Miyazaki et al., 2012). Projections from the VTA to the PFC drive risky decision-making, and in risky contexts, these projections decrease with punishment risk (Poisson et al., 2021).

The PFC is crucial in decision-making, reward evaluation, cognitive function, and inhibiting intrusive compulsive-seeking thoughts (Brewer & Potenza, 2008). The mPFC and OFC, along with the NAc, modulate patience in the wait to obtain reward through 5-HT signaling (Miyazaki et al., 2012b). The PFC responds to motivationally relevant and reward-predicting events (Kalivas & Volkow, 2005), and dysfunction of the PFC leads to an increase in impulsive choices, as shown in impaired performances in the Iowa Gambling Task (Brewer & Potenza, 2008). Low PFC activity is implicated in impulsive

decision-making and greater cue reactivity, similar to abnormalities seen in SUDs and pathological gambling (Brewer & Potenza, 2008; Grant et al., 2010). Cue-induction neuroimaging studies on internet gaming and pathological gambling reveal impaired activity in the PFC and the NAc, finding that stronger craving is associated with reduced NAc-PFC connectivity (Limbrick-Oldfield et al., 2017).

While pathological gambling shows reduced PFC activity during risk, reward, and control tasks, research also found poor white matter integrity in the PFC when suffering from this condition. Taken together, evidence that genetic variation impacts neurotransmission in the PFC supports a model in which impaired inhibitory control and risky decision-making increase vulnerability to BAs.

5. Conclusion

The discussion above regarding the role of the dopaminergic and serotonergic pathway genes in impulsivity has shown that variations in impulsivity-related genes (DRD2, DRD4, DAT1, HTR1B, HTR2B, SLC6A4 and MAOA) alter the neural regions involved in the brain's reward system (the VTA, the NAc and the PFC), which increases impulsivity in individuals and therefore their predisposition to BAs. As behaviors become habitual, addictions begin in the VTA-NAc-PFC circuits, because of their role in reward processing and decision-making. This circuit reinforces habitual behavior through learned stimuli, DA receptor variations, and compulsive seeking caused in the NAc (Brewer & Potenza, 2008). Both SUDs and BAs activate the same key regions in the brain (PFC, VTA, and NAc) during motivationally relevant event exposure, showing the influence of dopaminergic and serotonergic pathways in these regions due to their overlap (Grant et al., 2010).

The mesolimbic system is central to reward processing in both SUDs and BAs, as found in different brain imaging studies, with reduced activity in these key neural regions leading to impulsive decision-making and heightened cue reactivity (Grant et al., 2010). Variations in the dopaminergic and serotonergic pathways might lead to impaired functioning of the PFC, and are therefore implicated in impulsive decision-making and reward sensitivity in both SUDs and BAs, with neuroimaging studies finding an overlap between the inhibitory control and reward processing circuits across the spectrum of addictive disorders (Bevilacqua & Goldman, 2013; Congdon & Canli, 2008; Grant et al., 2010). Interactions between the key brain regions of the mesolimbic system regulate the patience an individual has when it comes to waiting for a reward, linking 5-HT and DA pathways to the control of impulsivity. Simultaneously, variations in the VTA, NAc, and PFC due to levels of DA and 5-HT genes create a pathway that increases impulsive behavior, which in turn heightens susceptibility to BAs. Understanding the genetic regulation of DA and 5-HT pathways across the VTA, NAc, and PFC offers a potential basis for biomarker development, enabling earlier identification of at-risk individuals. Such mechanistic insight also highlights these neurotransmitter systems as therapeutic targets, suggesting that future interventions may involve correcting pathway-specific dysregulation rather than treating behavioral symptoms alone.

5.1. Limitations

Although this review aimed to provide a comprehensive report on the potential mapping of BAs, summarizing a range of neurobiological reports linking genes related to impulsivity to the mesolimbic system brain regions, several limitations constrain the strength of its conclusions. The review relies heavily on previous reviews done on the matter, which can preserve the biases or methodological gaps of other authors. It also creates a methodological inconsistency across studies, as results may be varied due to these differences in neuroimaging studies, behavioral tasks, and others. Most of the included

articles are correlational and not longitudinal, and the observed genetic variations in these disorders may be due to the disorders themselves, and not their cause.

When suggesting further research directions, this review considers the studies of Lang et. al (2016), and Lind et. al (2013), which aimed to make a genome-wide association study (GWAS) of pathological gambling. Notably, the results of these studies were not included in the review because they found no statistically significant genetic associations with the BA. However, this does not mean that there is no genetic foundation to the disorder, but it could be due to the studies' reliance on the candidate gene approach. This review recommends that future GWAS of BAs should avoid this approach by experimenting without a narrow hypothesis and scanning in large and diverse samples. Conducting GWAS in BAs without this potential limitation might lead to more concrete evidence of the influence of dopaminergic and serotonergic pathway genes or might reveal novel genetic contributions to the literature. The review also recommends further neuroimaging studies using functional magnetic resonance imaging (fMRI) to map the functional changes in reward processing and impulsive decision-making in the proposed gene-brain-behavior pathway across different types of BAs. Pairing neuroimaging studies with behavioral task studies might also be able to create a link between genetic variation, brain activity, and behavioral traits. If further studies are able to provide more concrete evidence that links this proposed pathway, clinical studies could be conducted to develop prevention and treatment strategies for BAs, targeting the specific genes that influence these brain pathways.

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Mentor Contribution Statement

Dr. Jorge Avila served as an academic mentor for this project. He provided high-level guidance on the scientific framing and coherence of the review. He offered feedback on how to structure the review, suggested ways to clarify the research question, and helped the author refine the theoretical framework presented in it. Dr. Avila also reviewed different drafts, providing suggestions for improvement. Dr. Avila did not write any portion of the manuscript, but his comments and suggestions were taken into account for improvements made by the author.

Bre Calhoun served as an academic mentor for this project, primarily supporting the development of the manuscript's structure and clarity. She provided feedback on early drafts, suggesting ways to improve flow and align each section more clearly with the proposed pathway. She also offered advice on how to present complex concepts in a way that would be accessible to non-expert readers. Bre Calhoun did not write any part of the manuscript, but guided the author through her feedback to improve the review.