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Publication Date

2023

Peer reviewed|Thesis/dissertation

UNIVERSITY OF CALIFORNIA

Santa Barbara

Neurobiological Predispositions and Developmental Trajectories of Gaming Disorder in Adolescents: A Longitudinal ABCD Study Analysis

A Thesis submitted in partial satisfaction of the

requirements for the degree Master of Arts

in Communication

by

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December 2023

The thesis of Kylie Sarah Cole Woodman is approved.

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December 2023

ACKNOWLEDGEMENTS

In the culmination of this academic journey, I find myself surrounded by a network of incredible individuals whose contributions and support have shaped this thesis. First and foremost, a profound thank you is due to my committee members—Rene Weber, Michael Miller, and Kristy Hamilton. Your time, expertise, and prompt responses played a pivotal role in the completion of my Master's thesis. Your guidance and commitment to my academic success are deeply appreciated.

To my esteemed cohort, your collective achievement in completing your Master's theses not only blazed a trail but also illuminated a clear path forward. Your genuine care, support, and encouragement were beacons guiding me through challenges and uncertainties. My gratitude extends to my lab mates—Paula, Sungbin, Musa, Brittney, and Yibei—whose support and encouragement were integral to the completion of this work. Their camaraderie made the research journey more meaningful, and without them, the finish line might have seemed insurmountable. Additionally, my heartfelt thanks go to Bedlam, Casey, and Cookie, whose unconditional care, expressed through their words, actions, and boundless dog love, provided solace and joy during the twists and turns of this endeavor.

A special acknowledgment is reserved for my partner, Caleb. Amidst the long days and late nights dedicated to crafting this thesis, your unwavering support and insistence on self-care were my anchors, grounding me in moments of exhaustion. Lastly, I am indebted to my parents for their enduring encouragement and unwavering support, regardless of the academic paths I chose to tread. Your belief in me fueled my determination to reach this milestone.

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ABSTRACT

Neurobiological Predispositions and Developmental Trajectories of Gaming Disorder in Adolescents: A Longitudinal ABCD Study Analysis

by

Kylie Sarah Cole Woodman

Adolescents are at three times higher risk for developing gaming disorder compared to children or adults, and this concern is only growing as video games establish themselves as a ubiquitous aspect of development. Utilizing data from the Adolescent Behavioral Cognitive Development (ABCD) Study examining 1,367 adolescents across two years, this study investigates the intricate relationships between predisposing factors and the evolution of gaming disorder in a longitudinal context. Cross-sectional regression analyses revealed significant associations between gaming disorder and male sex, low household income, high impulsivity (T1 & T2), low self-esteem, high peer victimization (T1), high depression symptoms, high family conflict, low inhibition, and delayed puberty (T2) with notable exclusions of ADHD, anxiety, social competency. Moreover, the longitudinal analysis unravels the directional nature of these associations, demonstrating the enduring impact of risk factors over time, such as high depression symptoms, high ADHD symptoms, high peer aggression, and low self-esteem. The presented research extends the understanding of gaming disorder dynamics, offering insights into the development of more tailored models

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for gaming disorder in adolescents— providing insights into future interventions for this complex issue.

Neurobiological Predispositions and Developmental Trajectories of Gaming Disorder in Adolescents: A Longitudinal ABCD Study Analysis

Video gaming is a core part of the developmental experience in the United States, with 91 - 99% of children and adolescents playing video games for at least one hour per day (Granic et al., 2014; Statista, 2021). Video games are particularly appealing during adolescence, characterized by a heightened sensitivity to rewards due to a fully developed limbic system and poor behavioral inhibition due to an underdeveloped prefrontal cortex (Casey et al., 2008). This developmental imbalance results in adolescents being drawn to high-reward and low-effort activities, such as video games. Compared to other forms of media, video games are optimized to provide immediate rewards and punishments to help guide players through the game environment; however, these same mechanisms condition compulsive and addictive gaming behaviors. In 2015, the World Health Organization (WHO) officially recognized gaming disorder in the International Classification of Diseases as a diagnosable mental health condition (ICD-11; WHO, 2023). A recent meta-analysis estimated that 3% of video gamers worldwide met the criteria for a gaming disorder – with 8.5% of adolescents meeting the criteria (Gentile, 2009; Kim et al., 2022; Stevens et al., 2021). Some models exist outlining predictors and risk factors of gaming disorder, but none account for neurocognitive changes that occur during adolescence across time. The lack of longitudinal models also highlights a significant limitation of the current literature, which focuses on cross-sectional findings that do not account for developmental factors or the directionality of relationships. Traditional cross-sectional techniques ignore developmental factors that are vital to understanding the accurate relationship between potential predisposing factors and gaming disorder. Thus, the present study aims to longitudinally

examine the development of gaming disorder through self-reported and neurological activation within the Adolescent Brain Cognitive Development (ABCD) study– the largest study on adolescent brain development in the world. The findings from this research will test the scope and validity of previous research longitudinally with a large adolescent population in the United States.

To begin, we explicate the terms *video games* and *gaming disorder*, as these concepts have many different definitions and are foundational for this research study.

It is common in the video game literature to assume the term 'video games' holds a similar meaning and heuristic of a video game; for scientific research, the conceptualization of video games needs further explication. Video games vary greatly across platforms (e.g., mobile phones, computers, tablets, handheld gaming devices, or a game console), and can include varied aspects of different platforms, including social, physical, and/or violent components genres (i.e., role-playing, flighting, first-person shooter, etc.). In a broad sense, video games are electronic or digital games that can be played across various platforms (Halbrook et al., 2019; Jones et al., 2014). Games are voluntary activities with a goal, set rules, and a feedback system (McGonigal, 2011). However, this broad conceptualization of video games does not capture the nuances that have made it a multi-billion-dollar industry (Technavio, 2022). Video games, as compared to non-digital games are uniquely composed of immediate feedback due to pre-programmed digital content, this includes as rewards and punishments such as winning coins or losing health, which help to guide the player toward achieving the end goal (Madigan, 2020). Based on these definitions of games and video games, the current study conceptualizes video games as digital or virtual games programmed with rewards and punishments to engage and direct players toward a goal through immediate feedback. Taken together, the immediate and rewarding features of video games make them an appealing pass time for individuals; however, these features can also exploit the reward systems of the brain – especially in developing minds.

Gaming disorder, also referred to as gaming addiction, is characterized by impaired control over gaming behaviors, prioritizing gaming over other interests, and continued escalation of gaming despite adverse consequences. WHO officially recognized gaming disorder as a diagnosable behavioral addiction in its International Classification of Diseases-11 (ICD-11) in 2015. The American Psychological Association (APA) acknowledged Internet gaming disorder as a 'diagnosis under consideration' in the Diagnostic and Statistical Manual-V (DSM-V) in 2013. Both gaming disorder (GD) and Internet gaming disorder (IGD) involve an inability to control video game-related behaviors and an increased prioritization of gaming over other aspects of life, resulting in adverse consequences. This classifies gaming disorder as a behavioral addiction, a category of disorder characterized by excessively performed behaviors that lead to suffering. Behavioral addictions exhibit parallels with substance addictions, manifesting comparable addiction patterns and involving similar underlying brain mechanisms. Distinct from substance addiction, where external substances alter brain chemistry, behavioral addictions arise through the exploitation of the brain's inherent learning systems, activated by engagement in specific behaviors.

The present study adopts the term 'gaming disorder' to address the diagnosable criteria outlined in the ICD-11; however, the DSM-V criteria are considered complementary. Thus, gaming disorder is defined as a behavioral addiction reinforced through content features in video games, resulting in (1) a pattern of impaired control over gaming behaviors,

(2) an increase in salience of video games, and (3) negative consequences to an individual's life.

Adolescents

Adolescence represents the transitional phase between childhood and adulthood in which humans undergo significant developmental changes that amplify the allure of video games. The transition from childhood to adulthood is marked by key developmental changes that enhance the appeal of video games (Cohen et al., 2010; Galvan, 2013). However, research on gaming disorders in adolescents often lacks a comprehensive understanding of the psychological and physical transformations occurring during this critical period, leading to spurious correlations and unwarranted speculations (King & Delfabbro, 2014; Shapira et al., 2000; Torres-Rodríguez et al., 2018). Researching the relationship between gaming disorders in adolescents requires a complete understanding of this unique developmental period. Although the pubertal flood of hormones signals the onset of physical changes, such as the development of primary and secondary sex characteristics, it also triggers fundamental changes neurologically, psychologically, and socially.

Neurological Changes

Pubertal hormonal release spurs the rapid development of specific brain regions, such as the limbic system, known for reward processing. However, not all brain areas develop at the same rate. The prefrontal cortex, an area used for higher-level executive functioning, develops relatively slowly—creating a developmental imbalance known as the maturation imbalance hypothesis (Casey et al., 2008). According to the maturation imbalance hypothesis, the mature limbic regions, relative to the prefrontal regions, enhance the motivational salience of rewarding stimuli, decrease inhibition, and increase sensation

seeking. This neurological imbalance is especially pronounced during fMRI rewardprocessing tasks. Galvan and colleagues (2006) conducted a study on youth, teens, and adults to examine reward processing using the Monetary Incentive Delay (MID) task. During the reward anticipation phases of the task, they found higher activation in the nucleus accumbens (NAcc, part of the reward system) and lower activation in the prefrontal cortex in adolescents compared to children and adults. This study corroborated the maturation imbalance hypothesis and demonstrated that this imbalance can be neurologically analyzed through reward anticipation tasks, such as the MID. This imbalance manifests in enhanced responsiveness to rewarding stimuli and poses challenges in behavioral regulation, laying the groundwork for understanding how adolescents interact with video games and are at an increased risk for developing a gaming disorder.

Social Changes

Adolescence is characterized by substantial social transformations driven by an emerging sense of autonomy and the desire to challenge the 'status quo' (Lerner & Steinberg, 2009; McElhaney et al., 2009; Steinberg, 2008). Increased autonomy and control motivate teens to challenge parental rules and boundaries—shifting their focus and resources from parents to peers (Mastrotheodoros et al., 2020; Dishion & Tipsord, 2011). Research on the neuroscience of adolescents found that peer faces increase activity in the reward centers of the adolescent brain and teens are also more sensitive to the nuances in emotional peer faces (Pfeifer & Allen, 2021; Sandre et al., 2022). The accelerated development of adolescents' social brains allows them to strengthen connections with peers but also makes them more sensitive to negative peer evaluations.

Adopting digital media and video games as a mechanism for social connection is a natural outcome, considering the increased importance of peers during adolescence. A survey on adolescent's motivations for playing video games found that the number one motivation for playing video games was social, closely followed by stress reduction (Ferguson & Olson, 2013). Playing online multiplayer video games or engaging in offline conversations about gaming experiences helps to facilitate healthy adolescent social development, such as feeling connected (78% of teen gamers) or relaxed and happy (81% of teen gamers; Lenhart et al., 2015). In addition, the increased stressors experienced by adolescents, such as increased family conflict and negative peer judgment, often result in many teens turning to video games to cope with stressors and escape unpleasant feelings. In a longitudinal study on gaming motivation, Wang and colleagues (2022) found that teens used video games primarily as a form of escapism for stressors such as family conflict. Teens turning to video games as a form of escapism were more likely to have higher depression, anxiety, and gaming disorder symptoms (Wang et al., 2022).

Psychological Changes

Another aspect of adolescence that informs how individuals interact with video games is the emergence and onset of mental health disorders. Many mental health disorders begin to emerge around ages 11-14, coinciding with the onset of puberty (Kessler et al., 2005). Research studies examining adolescent mental health and media effects often do not account for the pubertal onset of mental health in cross-sectional analyses of media use and consequently lead to spurious correlations found between mental health and media use (Desai et al., 2010; Drummond et al., 2020; von der Heiden et al., 2019). A hallmark study by Coyne and colleagues (2022) shows that positive correlations between digital media use, depression, and anxiety are common but insignificant when studied in longitudinal designs and adjusted by particular developmental stages. Some evidence shows that relationships between mental health and media use might be reciprocal. For instance, internalizing disorders such as depression and anxiety can lead to the onset of gaming disorder and result in intensified depression and anxiety symptoms –creating a positive feedback loop of symptoms (Kuss et al., 2018; Volkow et al., 2016; Yip et al., 2017). To better parse these potentially reciprocal or bidirectional relationships between mental health and gaming disorders in adolescents, it is imperative to examine mental health symptoms and the development of gaming disorders from a longitudinal perspective.

Theoretical Frameworks of Gaming Disorder

Over the past two decades, gaming disorder research has burgeoned, unveiling a plethora of correlates and factors related to problematic gaming behaviors. However, many of these factors are based primarily on correlational data. Therefore, claims cannot be made about causality and directionality, correlational data is also prone to spurious relationship, especially in the context of confounds introduced by adolescent development. One of the most popular models to research gaming disorder is I-PACE, the Interaction of Person-Affect-Cognition-Execution Model (Brand et al., 2016; 2019). I-PACE is used to explain the development of general internet-related addictions (e.g., gaming disorder, online gambling disorder, online shopping disorder, problematic online pornography viewing). However, it does not outline all of the specific predisposing factors of gaming disorder but rather provides general constructs of internet-related behavioral addictions. For our study, we account for the general constructs proposed in I-PACE; however, we rely on Richard et al. 's (2022) model for the development of Gaming Disorder Across the Lifespan (GDAL) to

provide specific variables related to gaming disorder during adolescence. Similarly, for the neurological features of gaming disorder, we lean on I-PACE to provide a general framework but rely on current fMRI research on gaming disorder for the operationalizations. Through the guiding framework of the I-PACE model and GDAL, we are able to narrow down the variables of interest from the over 8,000 variables available in the ABCD study to sixteen variables of interest.

The I-PACE model is a comprehensive theoretical framework that explains the development and progression of addictive behaviors related to internet use and is the most common predictive model used in the gaming disorder literature (Brand et al., 2016; 2019). The I-PACE consists of four major components: the *person component* outlining predisposing factors, the *affect component* emphasizing emotional responses to gaming triggers, the *cognition component* examining the cognitive processes, and the *executive component* representing the behavioral aspects of gaming disorders. The I-PACE model outlines predisposing factors but also active decision-making processes. Since we are using the I-PACE model as a guide for predicting the development of gaming disorder, we focus on the person, affect, and cognitive components.

Person and Affect Component

The person component, also known as the predisposing component of I-PACE, encompasses individual characteristics, including biopsychological constitutions, psychopathology, using motives, social cognitions, and personality traits, which can increase vulnerability to gaming disorder. Affect involves emotional responses to external and internal stressors or triggers, such as subjectively perceived stressful situations. To operationalize the person and affect components, we mapped them to Richard and colleagues' (2022) twentytwo risk factors and outcomes for gaming disorder in adolescents. These variables were compiled through a scoping review of 34 longitudinal research studies on gaming disorder across adolescents, identifying twenty-two risk factors and outcomes. Of the 34 longitudinal research studies, only two of them were conducted in the United States. Currently, no study exists examining all of these factors longitudinally in a United States sample of adolescents. Sixteen variables of interest were selected based on a cross-check of the variables from the GDAL model, I-PACE, and the available ABCD variables (see Table 1 for specifics).

Table 1

I-PACE	GDAL	Risk Factor and/or Outcome (Based on GDAL)	ABCD Time 1 (2YR)	ABCD Time 2 (4YR)
Biopsychological constitution	Adverse life events*	Risk factor	Negative life events	Negative life events
Biopsychological constitution	Male	Risk factor	Demographics	Demographics
Biopsychological constitution	Respiratory sinus arrhythmia		Х	Х
Biopsychological constitution	Galvanic Skin Response		Х	Х
Psychopathology	Anxiety	Outcome	CBCL - anxiety subscale	CBCL - anxiety subscale
Psychopathology	Depression	Outcome & Risk Factor **	CBCL - depression subscale	CBCL - depression subscale
Psychopathology	Conduct problems	Outcome	CBCL - rule- breaking subscale	CBCL - rule- breaking subscale

Cross-Comparison of Variables Between Variables From Models with ABCD Variables

Psychopathology	Physical aggression	Outcome & Risk factor	CBCL - aggression subscale	CBCL - aggression subscale
Psychopathology	ADHD	Risk factor	CBCL- ADHD subscale	CBCL- ADHD subscale
Using motives	Reward seeking*	Outcome & Risk factor	Behavioral activation system	Behavioral activation system
Using motives	Identification with avatar		Х	Х
Social cognition	Loneliness		X***	X***
Social cognition	Social vulnerability		Х	Х
Social cognition	Social competence	Outcome & Risk factor	CBCL - social subscale	CBCL - social subscale
Personality	Emotion dysregulation		Х	Х
Personality	Self-esteem	Outcome & Risk factor	Brief problem monitor - self- esteem question	Brief problem monitor - self- esteem question
Personality	Impulsivity	Outcome & Risk factor	UPPS-P	UPPS-P
Personality	Reduced behavioral control	Risk factor	Behavioral inhibition system	Behavioral inhibition system
Subjectively perceived situations	Peer victimization	Risk factor	Peer experiences questionnaire - victimization	Peer experiences questionnaire - victimization
Subjectively perceived situations	Relational aggression	Outcome	Peer experiences questionnaire - relational aggression	Peer experiences questionnaire - relational aggression
Subjectively perceived situations	Life satisfaction		Х	Х

Subjectively perceived situations	Academic difficulties		Х	Х
Subjectively perceived situations	Parental relationships	Outcome & Risk factor	Family environment scale	Family environment scale
Subjectively perceived situations	Parental communication		Х	Х

Note. Time 1 in this chart corresponds with the ABCD two-year follow-up visit, and Time 2 corresponds with the four-year follow-up visit. The variables chosen were from the core ABCD study; the ABCD substudy variables were not considered. Grayed-out rows indicate variables that were excluded. CBCL = Child Behavior Checklist, GD = gaming disorder, UPPS =

* These are variables proposed by Richard et al., 2022 that needed additional longitudinal research.

** GDAL reports in their model that depression is exclusively an outcome of gaming disorder; however, more recent longitudinal research also indicates it as a risk factor (for example, Liu et al., 2021)

*** The only question on loneliness is already captured within the CBCL - depression subscale. We chose not to look at this question specifically to uphold the validity and reliability of the CBCL.

Biopsychological Constitution

Biopsychological predictors of gaming disorder encompass the earliest predisposing genetic and early life factors. Of the biopsychological factors, the most prominent correlate of gaming disorder is *sex*, specifically being male. Currently, males not only play more video games but also have a four times higher diagnosis rate of gaming disorders compared to females (Bagot et al., 2022; Lopez-Fernandez et al., 2019). Since the COVID-19 pandemic, more females have begun to play video games, yet this has not resulted in an increase in gaming addiction scores (Han et al., 2022). A study by Dong and colleagues (2018) identified that gaming elicited more craving-related activations in the brain (e.g., thalamus)

for males than females. These results provide a neurological basis for why males may be more vulnerable than females in developing Internet gaming disorder.

Negative early life events are another reported biopsychological stressor outlined in the I-PACE model and suggested by GDAL. Early life stressors such as early trauma and emotional or physical abuse are all related to later life maladaptation, resulting in the development of gaming disorder. A recent longitudinal study by Jhang (2023) examined 848 high school students and found that negative life events were strongly associated with the development of gaming disorder symptoms six months later. Negative early life experiences, such as negative early life events and family conflict, are additional biopsychological factors that may heighten susceptibility to mental disorders and gaming disorder (Bussone et al., 2022). These experiences can lead to insecure attachment styles and lower oxytocin levels, both correlated with Internet-use disorders (Jhang, 2023).

Psychopathology

Comorbidities, also known as the coexistence of multiple health disorders, are deeply intertwined with gaming disorder to the extent that the DSM-V and ICD-11 provide specific instructions to clinicians to examine comorbidities before determining a final diagnosis and treatment plan. Common comorbid mental disorders with IGD include 92% anxiety, 89% depression, 85% attention deficit hyperactivity disorder (ADHD), and 75% social phobia (González-Bueso et al., 2018). In addition to these four psychopathologies, GDAL emphasizes aggression (Li et al., 2023) and conduct problems (Brunborg et al., 2014; Lemmens et al., 2010) as additional psychological issues related to the development of gaming disorder.

In the current literature, it is still unclear if there is a causal relationship between gaming disorder and psychopathology. For example, depression symptoms might lead individuals to develop a gaming disorder, or excessively playing video games might lead to heightened depression symptoms. Research using cross-lagged panel analyses (Gentile et al., 2011; Teng et al., 2021; Yao et al., 2015; Yip et al., 2017), provides insights into causal directionality; however, the findings are still unclear. A longitudinal study by Gentile and colleagues (2011) found that anxiety and depression increased due to pathological gaming – indicating depression as an outcome of gaming disorder. In contrast, other research studies found no significant relationship between gaming disorder and the development of anxiety one year later (Teng et al., 2021; Yao et al., 2015; Yip et al., 2017). Conversely, Krossbakken et al. (2018) found that depression symptoms predict gaming disorder (similar to findings by Liu et al., 2021), and gaming disorder predicts the development of anxiety. Internalizing psychopathology and the development of gaming disorders likely define a complex, mutually reinforcing relationship where cause and effect over time are difficult to separate. Nevertheless, it is important to try to decipher this relationship, especially in the context of emergent mental health concerns in adolescence.

Social Cognitions

Social cognition, defined as an individual's perception of their social standing and support, is seen as a motivator for excessive online video game playing, in addition to being a risk factor for gaming disorder. Adolescent reports of social cognitions such as loneliness and social competence are seen as longitudinal outcomes and risk factors of gaming disorder (Richard et al., 2022). Social competencies may be more of a risk factor than an outcome of gaming disorder. Based on Lee and colleagues' (2017) description of problematic gaming

profiles, socially conditioned gamers are individuals who develop a gaming disorder as a way to meet their social needs and reduce loneliness in 'real life.' Within the literature, poor social cognitions, such as loneliness and social competencies, are predictive of gaming disorder, but additional research is needed to examine the potential bidirectional effects of gaming disorder on social cognitions.

Personality

Personality and temperament are identified in I-PACE as a significant predisposing variable, exhibiting consistent associations with gaming disorders. Specific personality traits linked to gaming disorder features include high impulsivity, low inhibition, and high sensation seeking. In particular, impulsivity stands out as a consistent predictor of gaming disorder (Cyders & Smith, 2008), reflecting the inability to consider alternative solutions, acting on immediate urges without assessing consequences, and lacking self-control in the face of rewards and punishments (as defined in Şalvarlı & Griffiths, 2022). In a systematic review by Şalvarlı and Griffiths (2022), impulsivity was identified as a recurring and strong relationship to gaming disorder and was related to deficits in executive control in the brain. Deficits in executive control also underlie the relationship between gaming disorder and other personality traits, such as reduced inhibition and high sensation seeking (Gervasi et al., 2017).

Subjectively Perceived Situations

Lastly, stressful experiences or environmental triggers contribute to the development of mental disorders and addictive behaviors. Subjectively perceived situations include stressful family, school, and peer environments. As previously mentioned, familial environment, especially family conflict, plays a role in shaping children's and adolescents'

gaming use. Increased family conflict can result in video games being used as a coping mechanism and catalyzes the emergence of an Internet-use disorder (Bussone et al., 2022). Emotionally vulnerable gamers are a subset of individuals who developed problematic gaming behaviors as a method for coping with subjectively perceived stressful situations (Lee et al., 2017). Video gaming as a coping tool for family, academic, and peer stressors is related to the development of gaming disorder (Bányai et al., 2021); however, this relationship is also bidirectional, indicating that gaming disorder can also exacerbate these problems (Richard et al., 2022). Further, individuals experiencing stress from peer victimization (i.e., being bullied) and relational aggression (i.e., bullying others) are positively linked to gaming disorder, but few longitudinal studies exist examining the directionality of the relationship (Li et al., 2022).

Cognitive and Execution Components

The last components of I-PACE are the cognitive and executive components, which create a reinforcing feedback loop of gaming behavior and cognitive functioning. The cognitive component refers to the individual's expectations of positive and rewarding experiences while engaging in gaming behaviors. Execution represents the behavioral manifestation of gaming disorders and what rewards are received from engaging in gaming disorders. This cycle of positive cognitive expectations and positive reinforcement of video games can train the brain to develop an imbalance in the decision-making process, resulting in more impulsive gaming behaviors. This imbalance is best explained through the Competing Neurobehavioral Decision System (CNDS) model of addiction. The CNDS model examines the decision-making process as two competing systems: the impulsive decision system (System 1) and the executive decision system (System 2; Bickel et al., 2012;

Bickel et al., 2014). System 1 is an impulsive decision system located in the limbic (e.g., midbrain, amygdala, habenular commissure, & striatum) and paralimbic (i.e., insula & NAcc) brain regions. The limbic and paralimbic regions incite desires for immediate rewards (Bickel et al., 2014). On the other hand, System 2 is an executive decision system located in the prefrontal cortex of the brain. The prefrontal cortex drives high-level thinking, including planning and suppressing impulses in favor of long-term outcomes (Bickel et al., 2014). The dual-system model of CNDS states that these two systems are in constant competition for control during decision-making processes. When these two systems are balanced, individuals can modulate their behavior and have a dynamic range of appropriate decisions (Sussman, 2020; Bickel et al., 2018). Conversely, when the systems become imbalanced, one will default take control of behaviors - limiting an individual's behavioral range (Sussman, 2020). For example, a teenager with a gaming addiction would have a prominent System 1, and when presented with a game, System 1 will automatically decide to engage in the behavior before System 2 can have input, limiting the teen's decision options. The dualsystem framework of CNDS ameliorates the recurring findings of physiological measures on gaming disorder with reward processing and executive functioning.

Video game content provides immediate, novel, and high rewards to players for minimal effort compared to other tasks (i.e., socializing, studying, and sex; Bickel & Athamneh, 2019). The low effort-to-high reward ratio of video games leads individuals to place excessive value on brief video game rewards (as explained in Acuff et al., 2022). Secondly, the excessive preference for immediate rewards can develop from individual characteristics (e.g., impulsivity, social support, or socioeconomic status) or conditioned reinforcement (Bickel et al., 2014). High-value rewards like those presented in video games

increase dopamine (reinforcing neurotransmitter) release to the limbic and paralimbic areas of the brain, reinforcing System 1 and the development of a neurological imbalance.

The CNDS model for gaming disorder closely mirrors that of the maturation imbalance phenomenon observed in adolescents. The period of adolescence, specifically with a maturation imbalance, exhibits the same pattern seen in individuals with a higher risk for gaming disorder –making the period of adolescence a developmental risk factor for developing gaming disorder.

The Present Study

The existing body of literature on gaming disorders has predominantly examined the constructs outlined in I-PACE, GDAL, and neurological mechanisms in isolation—rather than completed model. Within this literature, numerous studies have delved into a range of factors to comprehend the emergence of gaming disorder, particularly during the pivotal period of adolescence. However, a notable gap exists in the absence of longitudinal investigations into the prominent correlates of gaming disorder concerning developmental factors within the context of American adolescents, which deprives the field of a complete understanding of the directionality and overlapping interactions which may occur. Our study addresses this gap by drawing upon I-PACE, GDAL, and CNDS to delineate sixteen variables of interest and identify two neurological brain systems (System 1 and System 2) for a comprehensive and simultaneous examination of their association with gaming disorder over time. Through this analysis we progress the research on gaming disorder to account for developmental factors and provide specific variables for gaming disorder within the I-PACE model.

Many variables linked to gaming disorder have been predominantly explored in cross-sectional studies, posing challenges, especially given the developmental intricacies of adolescence marked by hormonal changes, maturation imbalances in the brain, the onset of psychopathology, and significant familial and social transformations that may lead to spurious correlations with gaming disorder. This study aims to fill this void by investigating the intertwined influences of individual characteristics, affective components, cognitive and executive functions, and developmental factors associated with gaming disorders. Leveraging data from a large, representative sample of U.S. adolescents provided by the Adolescent Brain Cognitive Development (ABCD) study, we employ advanced structural equation modeling and cross-lagged panel analysis to elucidate temporal relationships and directionalities among these multifaceted factors. In light of emerging research insights, we hypothesize that gaming disorder scores in early adolescents will exhibit cross-sectional correlations with the identified predisposing factors, as detailed in Table 2 (H1). Although it is predicted that all variables of interest will be significantly correlated, it is unclear the amount of shared variance between these variables that could be underlying the relationship to gaming disorder; for example, pubertal status and sex are highly related to psychopathology in addition to gaming disorder. Thus, we also investigate the relationship between all the variables of interest and gaming disorder to decipher which variables explain the most variance in gaming disorder (RQ1). Additionally, as a research question, we explore the temporal sequence of the variables of interest using a Cross-Lagged Panel Model (H2). While existing studies provide some guidance based on European and Asian samples, at this time, certain variables (i.e., anxiety, depression, conduct disorder, aggression, ADHD, impulsivity, inhibition, and sensation seeking) lack clear directional evidence and are

untested in American samples. Table 2 outlines specific expectations for the variables of

interest within Hypotheses 1 and 2, grounded in the theoretical frameworks of I-PACE,

GDAL, and CNDS.

Table 2

Directional Cross-Sectional and Longitudinal Hypotheses Based on GDAL, I-PACE, and

CNDS Models as T	heoretical Frameworks
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	H1: Cross-sectional Relationship to gaming disorder (GD)	H2: Longitudinal Relationship to gaming disorder (GD) based on GDAL
Adverse Life Events	More negative life events are related to higher GD scores	Higher negative life events will predict higher GD scores two years later. Whereas GD scores will not predict negative life events two years later.
Sex	Male sex is related to higher GD scores	-time-constrained factor-
Anxiety	Higher anxiety scores are related to higher GD scores	Higher gaming disorders scores will predict higher anxiety scores two years later. Whereas anxiety scores will not predict GD scores two years later.
Depression	Higher depression scores are related to higher GD scores	Higher gaming disorders scores will predict higher depression scores two years later, and depression scores will predict GD scores two years later.
Conduct Problems	More conduct problems are related to higher GD scores	Higher gaming disorders scores will predict more conduct problems two years later. Whereas conduct problems will not predict GD scores two years later.
Aggression	Higher aggression scores are related to higher GD scores	Higher aggression scores will be related to higher GD scores two years later, and higher gaming disorder scores will predict aggression scores two years later.
ADHD	Higher ADHD scores are related to higher GD scores	Higher ADHD scores will be related to higher GD scores two years later, but gaming

		disorder scores will not predict ADHD scores two years later.
Social Competence	Lower social competence is related to higher GD scores	Lower social competence scores will be related to higher GD scores two years later, and higher gaming disorder scores will predict lower social competence scores two years later.
Peer Victimization	Higher peer victimization scores are related to higher GD scores	Higher peer victimization scores will be related to higher GD scores two years later, but gaming disorder scores will not predict peer victimization scores two years later.
Relational Aggression	Higher relational aggression scores are related to higher GD scores	Higher gaming disorder scores will predict higher relational aggression scores two years later. Whereas relational aggression scores will not predict GD scores two years later.
Family Conflict	Higher family conflict is related to higher GD scores	Higher family conflict will predict higher GD scores two years later, and gaming disorder scores will predict family conflict two years later.
Self-Esteem	Lower self-esteem scores are related to higher GD scores	Lower self-esteem scores will predict higher GD scores two years later, and higher gaming disorder scores will predict lower self-esteem scores two years later.
Impulsivity	Higher impulsivity scores are related to higher GD scores	Higher impulsivity scores in Time 1 will predict higher GD scores in Time 2, whereas gaming disorder scores in Time 1 will not predict impulsivity in Time 2.
Inhibition	Lower inhibition scores are related to higher GD scores	Higher gaming disorder scores will predict lower inhibition scores two years later. Whereas inhibition scores will not predict GD scores two years later.
Sensation Seeking (Behavioral Approach)	Higher reward-seeking scores are related to higher GD scores	Higher reward-seeking scores will predict higher GD scores two years later, and higher gaming disorder scores will predict higher reward-seeking scores two years later.
Prefrontal System Activation	Higher prefrontal system activation during loss anticipation related to	Higher prefrontal system activation during loss anticipation will predict higher GD scores at Time 2, whereas gaming disorder

	higher GD scores	scores in Time 1 will not predict prefrontal activation at Time 2.
Limbic System Activation	Hyperactivity in limbic systems during reward anticipation is related to higher GD scores	Hyperactivity in limbic systems during reward anticipation will predict higher GD scores at Time 2, whereas gaming disorder scores in Time 1 will not predict limbic activation at Time 2.

Methods

ABCD Study

The current study utilizes data from the Adolescent Behavioral Cognitive Development (ABCD) Study dataset release 5.0 (released in July 2023, <u>https://abcdstudy.org/scientists/data-sharing/</u>), which we downloaded using NDA Tools (https://github.com/NDAR/nda-tools). The ABCD study is an adolescent cohort study of 11,572 youth examining teen development through questionnaires, behavioral tasks, and neurological markers. Consent was obtained from parents and assent from children by ABCD investigators at each time point. A centralized review board approved all procedures at the University of California San Diego (Garavan et al., 2018).

The 5.0 data release includes four complete annual time points following youth from ages 9 to 14. During the annual visits, a parent and their child completed a series of over 38 validated questionnaires; the youth also completed neurocognitive tasks and biological samples. During even time points, baseline, two-year follow-up, and four-year follow-up, there were additional MRI and fMRI components of the study, which examined neurological changes over a two-year time span. For the present study, we focus on two time points as they include the fMRI components and a measure of our dependent variable– gaming disorder. Going forward, we will refer to the two-year follow-up as 'Time 1' and the fouryear follow-up as 'Time 2'. Time 1 contains youth in emerging adolescents ages 11-12; these participants return two years later at Time 2 when the teen participants are ages 13-14.

Participants

The ABCD study uses multistage probability sampling to collect a closely representative sample of the United States population. Participants were recruited primarily through schools at 21 sites across the United States. The BSL cohort consisted of youth ages 9-10 years of age who did not have any severe impairments preventing them from completing the study requirements (e.g., a current diagnosis of schizophrenia, autism spectrum disorder - moderate to severe, intellectual disability, claustrophobia). Only a subset of the original 11,875 participants were analyzed based on the following criteria. Participants who completed the Time 1 and Time 2 fMRI tasks are included (N = 4,785). Moreover, participants' anatomical images were assessed for quality. Low-quality images will be excluded from the analyses (low-quality images are defined by ABCD as having movement artifacts or severe incidental findings; Rapuano et al., 2022). Participants with missing data for any variables of interest or covariates will be further excluded; this includes participants who reported playing no video games - and would not receive the video game addiction questionnaire. The final sample size at Time 1 is 1,367, and at Time 2 it is 1,406; for additional demographic information, please reference Table 3. The subsample demographics were compared to the larger ABCD data and were found to be comparable; however, our final sample had an additional 5% of male participants, which aligns with current video game playing statistics (Entertainment Software Association, 2023).

Measures

Demographic Information

Demographic information is self-reported by a single caregiver at each annual time point. Caregivers report on themselves, their child, and household members' sex, annual household income, and their highest level of education. Questions regarding race and ethnicity for their child are only recorded at Baseline. See Table 3 for complete demographic information.

Pubertal Development

Pubertal status was assessed using the Pubertal Development Scale (PDS), measuring the perceived development of secondary sex characteristics (Petersen, 1988). The questionnaire consists of five items completed by youth, each rated on a 4-point scale, with higher scores indicating more advanced pubertal development. This includes questions about growth spurts, body hair growth, skin changes, breast development, menarche in females, voice changes, and growth of testes in males. The PDS has demonstrated reliability and validity, showing high correlations with hormone levels and Tanner stages (Koopman-Verhoeff, 2020).

Table 3

Demographic Information and Descriptive Statistics by Time Point

Characteristic	Time 1 , $N = 1,367^{1}$	Time 2 , $N = 1,406^{1}$
Age (months)	143.4 (8.0)	168.9 (8.4)

Household Income (Ordinal Variable)	7.6 (2.2) 7 = \$50,000 - \$74,999 8 = \$75,000 - \$99,999	7.8 (2.1) 7 = \$50,000 - \$74,999 8 = \$75,000 - \$99,999
Missing	84	123
Parental Education (Ordinal Variable)	18.1 (3.2) 18 = Bachelor's degree	18.0 (3.2) 18 = Bachelor's degree
Missing	3	17
Sex		
1 - Male	738/ 1,367 (54%)	745 / 1,406 (53%)
2 - Female	628 / 1,367 (46%)	430 / 1,406 (47%)
Missing	1	1
Race and Ethnicity		
1 - White	792/ 1,367 (58%)	815 / 1,406 (58%)
2 - Black	137/ 1,367 (10%)	140 / 1,406 (10%)
3 - Hispanic	376/ 1,367 (20%)	282/ 1,406 (20%)
4 - Asian	27/ 1,367 (2%)	28 / 1,406 (2%)
5 - Other	137/ 1,367 (10%)	141 / 1,406 (10%)

Missing	0	0
Puberty		
1 - Pre-Puberty	179 / 1,307 (14%)	20 / 1,359 (1.5%)
2	349 / 1,307 (27%)	81 / 1,359 (6.0%)
3	532 / 1,307 (41%)	437 / 1,359 (32%)
4	243 / 1,307 (19%)	726 / 1,359 (53%)
5 - Post-Puberty	4 / 1,307 (0.3%)	95 / 1,359 (7.0%)
Missing	60	47
Gaming Disorder Score	12.1 (5.9)	13.0 (6.3)
Missing	411	295
Minutes Gaming per day	115.9 (151.7)	176.1 (208.6)
Social Anxiety	1.3 (2.1)	1.0 (1.8)
Missing	6	16
Depression	1.5 (2.2)	1.8 (2.7)

Missing	6	16
ADHD	2.3 (2.7)	2.0 (2.6)
Missing	6	16
Aggression	2.8 (3.6)	2.4 (3.4)
Missing	6	16
Conduct Disorder	1.1 (1.7)	1.0 (1.6)
	25	22
Behavioral Inhibition	5.0 (2.8)	5.4 (3.0)
Missing	2	4
Behavioral Motivation	16.3 (6.4)	16.6 (6.4)
Missing	2	3
Family Conflict	1.8 (1.8)	2.3 (2.2)
Missing	2	2
Negative Life Events	5.0 (5.4)	4.3 (4.7)

Self-Esteem	.29 (.12)	.19 (.33)
Missing	39	22
Impulsivity	39.3 (7.8)	41.5 (7.8)
Missing	2	4
Relational Aggression	10.4 (1.8)	10.2 (1.8)
Missing	17	7
Peer Victimization	12.5 (4.1)	11.8 (3.5)
Missing	17	7

¹ Mean (SD); n / N (%)

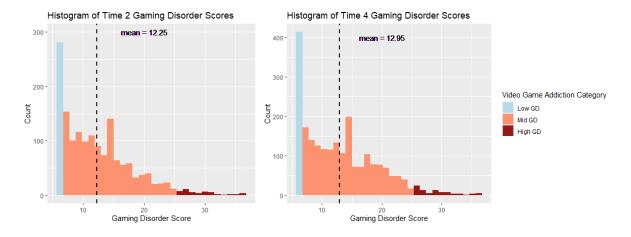
Gaming Disorder

The Video Game Addiction Questionnaire (VGAQ) is a self-report youth questionnaire on youths' thoughts about their video game habits. The VGAQ was adapted from the Bergen Facebook Addiction scale and added to the ABCD study during 2YR and 3YR (Andreassen et al., 2012; Bagot et al., 2022). Only a subset of youth who previously reported playing video games in the STQ, completed the video game addiction questionnaires. Based on the DSM-V criteria (2013) for Internet gaming disorder, the six questions assessed preoccupation with media, experienced withdrawal, developed tolerance,

loss of control, escapism, and risk opportunities. Example statements include, "I feel the need to play video games more and more" and "I play video games so much that it has had a bad effect on my schoolwork or job" (Bagot et al., 2022). Youth can respond on a scale of one to six, with one being "Never," four being "Sometimes," and six being "Very often." Each scale is summed together to create a continuous score for video game addiction; higher scores indicate higher symptoms of addiction. Gaming disorder scores are considered 'abnormal' variables, indicating a strong right-skew (see Figure 1 below) of scores, with the first quartile indicating a score less than seven. To best understand the descriptives of the ABCD data, gaming disorder scores were divided into three categories: Low, Medium, and High gaming disorder scores, as previous studies have suggested (Macur & Pontes, 2021). The low scores are classified based on the 1st quartile of responses, with a score less than seven. The high gaming disorder scores are based on the DSM-V diagnostic criteria of Internet Gaming Disorder and the fifth quartile (5% of high scores > 24). The DSM-V criteria for experiencing at least five or more of the symptoms establishes eligibility to be diagnosed with Internet Gaming Disorder. For the VGAQ, participants reporting a five (e.g., "Often") or higher on five or more questions would meet the criteria for being diagnosed with IGD. During Time 1, 49 participants (3.6%) reached the diagnostic criteria for IGD, increasing to 83 participants (5.7%) of adolescents in Time 2. The medium scorers fall within the larger range, between seven and twenty-four (see Figure 1 below for details). Additional analyses at a later point may benefit from using a Generalized Linear Modeling approach in which the skew will best be accounted for by an appropriate transfer function (e.g., a Poisson or a negative binomial distribution).

Figure 1

Histograms of Gaming Disorder Scores



Note. "Low GD" = Low gaming disorder scores, "Mid GD" = Medium gaming disorder scores, and "High GD" = High gaming disorder scores. The dotted horizontal line indicates the mean score each year. Time 1 contains a sample size of 1,367 and Time 2 contains a sample of 1,406.

Adverse Life Events

At each time point, Time 1 and Time 2, youth participants complete an Adverse Life Events Scale, from the PhenX toolbox collection, about events that the youth has experienced (Barch et al., 2018). The Life Events Scale is one of the most frequently used measures of stress exposure and provide strong reliability and validity, although there are some errors reported with the reliance on retrospective recall (Wethington, 2016). Participants read through various examples of life events and respond "Yes" if they have occurred or "No" if they have not. If the event occurred, the participant is then asked if the event was a good or bad experience and to rate how much the event affected them on a scale of zero to three, zero being "Not at all" and three being "A lot." We calculate a negative life events score by taking the average of the affected negative life score divided by the total number of negative life events.

Child Behavior Checklist

The Child Behavior Checklist (CBCL) employs 112 questions to track changes in internalizing and externalizing behaviors at each time point (Achenbach & Ruffle, 2000; Clark et al., 2021). Caregivers reflect on their child's behavior over the past six months and respond on a scale of 0 "Not True," 1 "Somewhat or Sometimes True," and 2 "Very True or Often True." The CBCL has multiple subscales that incorporate symptoms associated with depression, social problems, anxiety, ADHD, rule-breaking, and aggression (Clark et al., 2021). Parental reports of the subscale items from the CBCL are strongly related to clinical diagnoses of their respective DSM-V disorders. Examples of the depression question include "Complains of loneliness" and "Feels worthless or inferior." Examples of social problem questions include, "Doesn't get along with peers." The anxiety subscale includes questions such as "Nervous or tense" or "Worries." ADHD examples include "impulsive" or "Can't Concentrate." Lastly, examples of aggression questions are "Argues," "Bullies," or "Disobedient at school." 71 questions were not included in the analysis as they were not identified as relevant predictors of gaming disorder. The questions administered in the CBCL remained consistent for parents across all time points.

Peer Experiences

The peer experiences questionnaire is an assessment of whether the youth has either experienced overt, relational, or reputational victimization from peers or perpetrated overt, relational, or reputational aggression towards peers. Youth participants rated the occurrence of events in the past year using a 5-point scale, 1 = Never' and 5 = A Few Times a Week'

(Landoll et al., 2013). The first subscale that is assessed is the peer victimization subscale, which is a summation of questions on relational, overt, and reputational victimization. The second subscale, peer aggression, had the same questions but asked the participants if they had done the actions to another person. An example of a question would be, "A kid tried to damage my social reputation by spreading rumors about me." Similarly, perpetrating relational aggression is the sum score of relational, overt, and reputational questions, such as "I gossiped about another kid so others would not like him/her."

Family Conflict

The Family Environment Scale (FES) measures family dynamics, cohesion, expressiveness, and conflict. The ABCD modified the FES and family conflict subscales from the PhenX toolkit. Youth completed the family conflict subscale, which included nine binarized True/False questions: "We fight a lot in our family" or "Family members sometimes hit each other." Higher scores are indicative of more severe family conflict within the household (Moos & Moos, 1984). These questions are consistent throughout each followup visit.

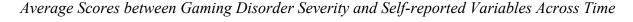
Self-Esteem

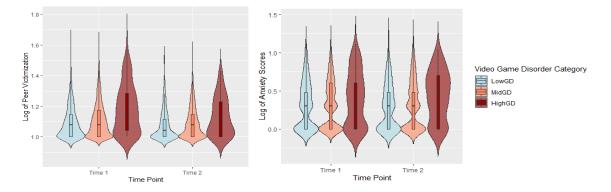
The self-esteem question was selected from the Brief Problem Monitor for youth, which is an abridged questionnaire derived from the child behavior checklist that is suited for youth participants. Although this questionnaire does not have a specific subscale for selfesteem, it does contain one self-esteem question, "I feel worthless or inferior/less good [compared to other kids my age]." Participants can respond with 0 = 'Not True', 1 ='Somewhat True', and 2 = "Very True." Thus, a higher score on self-esteem indicates lower self-esteem for the youth.

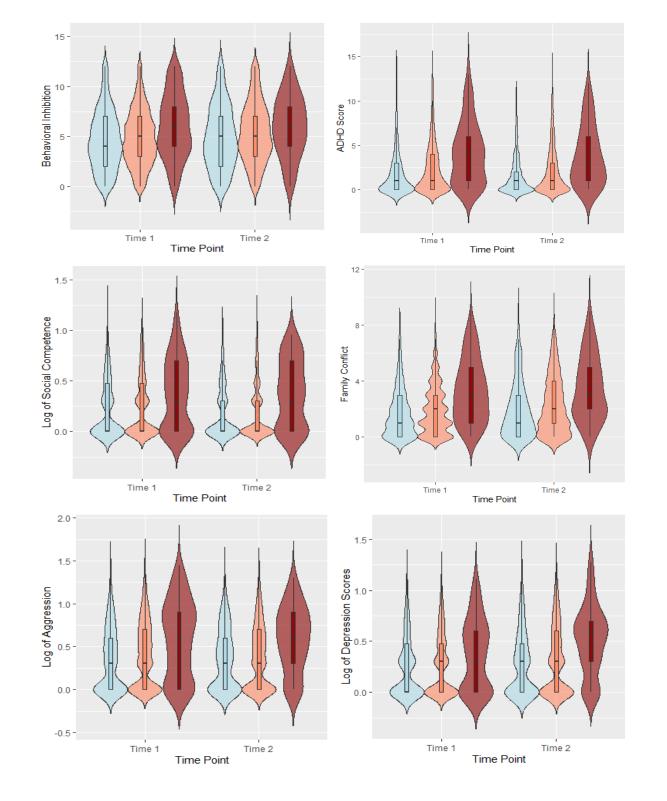
Impulsivity

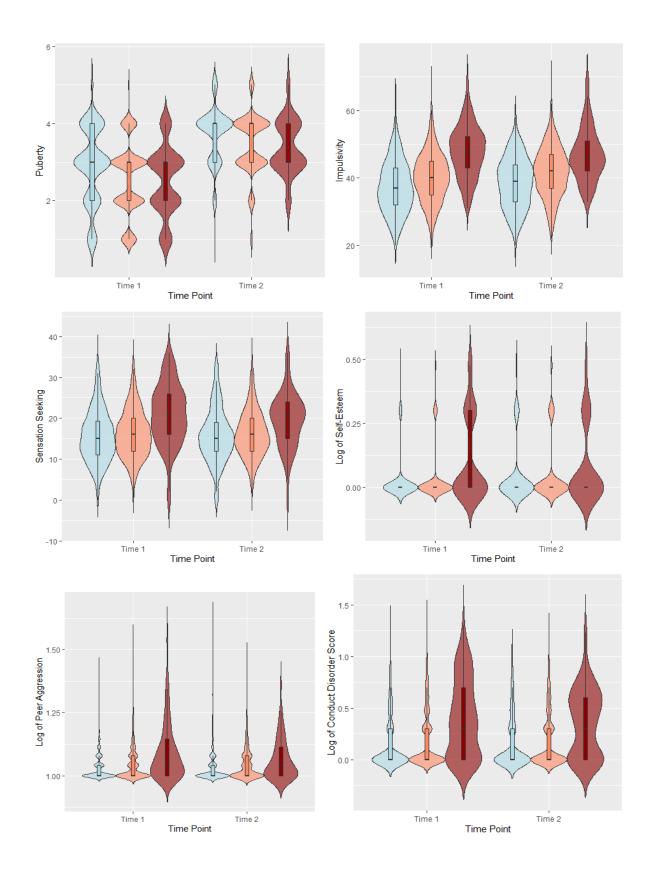
The present study utilizes the Urgency-Premeditation-Perseverance-Sensation Seeking-Positive Urgency (UPPS-P) scale, which is used to measure five dimensions of impulsivity. A shortened version of the UPPS-P was used in the ABCD study to be more youth-appropriate. A pilot study of the shortened measure demonstrates robust internal consistency (Cronbach's $\alpha = 0.99$) to measures of impulsivity (Barch et al., 2018). The present study examines impulsivity using all 5-dimensions of the UPPS-P (i.e., negative and positive urgency, sensation seeking, and lack of preservation and premeditation). Examples of these questions include: "Sometimes when I feel bad, I keep doing something even though it is making me feel worse" or "When I feel rejected, I often say things that I later regret." Participants can respond on a four-point Likert-type scale, with one indicating "Not at all like me" and four "Very much like me." Positively valanced statements like "I finish what I start" and "I like to stop and think about things before I do it" are reverse coded, meaning that higher total scores on impulsivity reflect elevated levels of impulsivity.

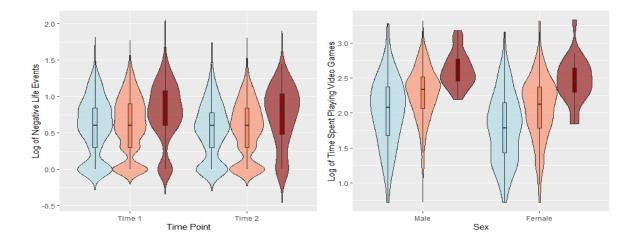
Figure 2











Note. The last chart indicates differences in time spent playing video games by sex rather than by time point. Some variables have been transformed using log₁₀ to create a more normal distribution.

Behavioral Inhibition/Behavioral Approach System Scales (BIS/BAS)

The Behavioral Inhibition/Behavioral Approach System Scales (BIS/BAS) is a youth self-administered 20-item questionnaire that measures individual differences in motivation, which was modified from PhenX Toolkit protocol (Pagliaccio et al., 2016). The BIS measures response inhibition to aversive stimuli, examining the participants' inhibition of behaviors that may lead to detrimental consequences. In contrast, the BAS can be explained as sensation seeking or the motivational responses to positive reinforcement, driving individuals toward actions aimed at obtaining rewards (Elliot & Thrash, 2002). Examples of the BIS are, "I worry about making mistakes" or "I feel pretty upset when I think that someone is angry with me." Examples of the BAS include questions such as, "I feel excited and full of energy when I get something that I want" or "I crave excitement and new sensations." Participants can respond on a zero to three-point scale, with zero indicating "Not true" and three indicating "Very true." The BIS questions are summated to create a measure

of behavioral inhibition, and the BAS is summated to get a measure of motivated behaviors (See Figure 2 for distributions and descriptives of each variable).

Imaging Procedure

Imaging procedures were harmonized between 3 MRI scanners: Siemens Prisma, General Electric T3/GE T3, and Phillips MRI machines across all 21 sites through standardized imaging protocols and adjustments for each machine. The neuroimaging paradigm included structural scans, resting state, and three fMRI tasks, one of which is the monetary incentive delay (MID) task - used to evoke reward processing (Casey et al., 2018). T1 images were collected using 1 mm isotropic voxels, and fMRI acquisitions were collected with multiband EPI with slice acceleration factor six, 2.4 mm isotropic voxel size, and a TR of 800 milliseconds (Casey et al., 2018). Before the beginning of the MRI scan, youth completed metal screening questionnaires and practice rounds of the fMRI tasks, including practice rounds of the MID task, to ensure participants understood the game before entering into the scanner.

Monetary Incentive Delay Task

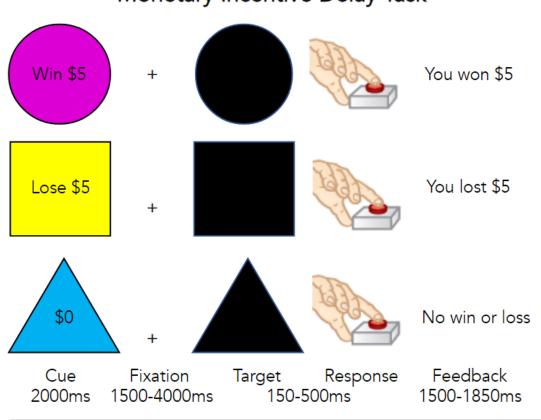
In the MID task, participants have the chance to win real money by providing a quick responses to reward and punishment queues (Casey et al., 2018). During the task, participants are presented with the stimuli of winning money, losing money, or earning nothing. The MID is a well-known standard task that evokes neural correlates of reward processing. During each time series of the MID task, participants are presented with a shape indicating a monetary outcome, the participants then waits till they see a black shape appear, quickly press a button to receive the monetary outcome, and then receive feedback on their response (see Figure 3 for a visualization of the MID task). During the practice rounds outside of the MRI scanner, participants learned three shapes with different rewards and punishments: Win \$5, Win \$0.20, Lose \$5, Lose \$0.20, and neutral (no money at stake). Researchers obtain the participant's base reaction time from the practice rounds of the MID, which are then used to personalize an adaptive algorithm that adjusts the task's difficulty to each participant's skill level. Once the participant starts the MID in the scanner, they see the shape indicating a win, lose, or neutral condition. They then wait for a black shape to appear before quickly pushing a button. If the participant responds too fast (before the black shape appears) or too slow (after the black shape disappears), they do not receive the reward or punishment. After the participant responds, a large message appears to provide feedback on their response, displaying: "You won \$5.00", "You did not win \$5.00", "You did not lose \$5.00", "You lose \$5.00", or "No money at stake." Participants completed two rounds of each task in the MRI using a two-button button box. At the end of both rounds, the amount of money won by participants is reported on the screen. **Calculating System 1 and System 2 Variables**

Preprocessing

ABCD data processing was completed using the standardized ABCD-BIDS data processing pipelines; for additional details and procedures, visit the DCAN-labs GitHub page (https://github.com/DCAN-Labs/abcd-hcp-pipeline). Briefly, the preprocessing steps include (1) head motion correction, (2) B0 distortion correction, (3) gradient warping correction, (4) within-scan motion correction (estimating movement based on respiration; Fair et al., 2018), and (5) registration to T1w structural images.

Figure 3

Visualization of the Monetary Incentive Delay Task



Monetary Incentive Delay Task

Note. Each row represents a different time series of the MID stimuli. The image is modified from Casey et al. (2018).

Beta Estimates

The ABCD dataset provides estimated beta activation for fMRI tasks, employing modeled contrasts for each run and integrating them into a general linear model. In our study, we utilized standardized Beta weights and standard error scores from the average time courses calculated for cortical surface-based parcellations, including the gyral- and sulcalspecific Desikan-Killiany parcellation (34 cortical parcels, Desikan et al., 2006), and the thirty subcortical parcellations from Freesurfer segmentation (Fischl et al., 2002). Despite acknowledging the limitations of the Beta aggregate approach, similar methods have been successfully employed in previous research, such as Nakua et al. (2023) and Shen et al. (2021), providing a more reproducible analysis through established parcellations and contrasts with ABCD consortium data.

Two a-priori contrasts were selected based on Yao and colleagues' (2020) findings regarding brain activation in the prefrontal and limbic areas using the MID task. The first contrast examines reward anticipation moments before receiving the reward confirmation message, compared to the neutral condition. Adolescents with gaming disorder exhibited heightened reward sensitivity (hyperactivity in System 1) during reward anticipation compared to healthy controls. The second contrast investigates loss anticipation compared to neutral, revealing less activation in the prefrontal cortex (System 2) in adolescents with gaming disorder than in healthy controls. The first contrast combines all reward anticipation phases with the neutral phase, where no money is won or lost, while the second contrast focuses on the combined anticipation loss activations just before receiving loss feedback.

A-priori regions of interest (ROI) in the brain were identified based on the I-PACE, CNDS, and gaming disorder literature. System 1 comprises the ventral striatum (VS), medial orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), dorsolateral prefrontal cortex (DLPFC), posterior cingulate cortex (PCC), nucleus accumbens (NAC), hippocampus (HPP), amygdala (AM), and globus pallidus (GP) and represents the reward processing network and dopamine pathways related to addiction. System 2, or the executive system, includes the

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anterior inferior frontal gyrus (IFG), posterior insula (INS), and precentral and postcentral gyri (PCG).

Beta weights were calculated for each ROI individually per contrast during the firstlevel GLM analysis conducted by the ABCD consortium. To create system-level variables, a significance threshold was set, and the standardized betas were averaged to ensure homogeneity before combining. Combining highly correlated areas created a standardized beta weight or an artificial region of interest. Each brain region of interest within a system was highly correlated with others. Consistent with previous studies (Balodis & Potenza, 2015; Yao et al., 2020), we expected individuals with gaming disorder to show stronger activation in System 1 and less activation in System 2 during the reward anticipation versus neutral contrast. Conversely, during the loss anticipation versus neutral contrast, we anticipated lower activation in System 1 and higher activation in System 2.

Results

Data was downloaded using the NDA download manager

(https://github.com/NDAR/nda-tools), and variables of interest were merged into a singular data frame using pandas in Python. The data analyses and visualizations were conducted in R (version 4.3.1). Each variable was examined for normality and heterogeneity (see Figure 2). Seven variables were identified as having significant skew and kurtosis; a skewness greater than '1' or less than '-1' was considered extremely skewed, and a kurtosis score greater than three indicates that the distribution of numbers spans the tail of the curve rather than around the mean. Seven variables were identified as being abnormal (e.g., peer victimization, peer aggression, self-esteem, aggression, social competency, anxiety, depression, negative life events). They were transformed using log base ten plus one $[log_{10} (x + 1)]$. Our dependent

variable, gaming disorder scores, was highly skewed (skew = 1.1), but the kurtosis was within an acceptable range (kurtosis = .98); thus, we only slightly corrected for the skew through square rooting $(x^{1/2})$ gaming disorder scores.

Descriptive Statistics

Means, standard deviations, and correlations among the variables were calculated to identify preliminary data patterns (see Table 2). Our final sample of gaming adolescents included a sample of 54% Males with an average household income of \$50,000 - \$99,999, with the average parental education being a bachelor's degree, and 58% identifying as White non-Hispanic. At Time 1, participants were, on average, 12 years of age and reported being early/ mid-way through pubertal development and playing an average of 115.9 minutes (1.9 hours) of video games per day. At Time 2, participants were, on average, 14 years of age and reported being in the mid/late stages of pubertal development and playing an average of 176.1 minutes (2.9 hours) of video games per day.

Cross-sectional Analysis

H1: Correlational Statistics

Hypothesis 1 aims to examine the relationship between each variable of interest and gaming disorder. The cross-sectional correlations indicated significant relationships for most variables during to gaming disorder during Time 1 and Time 2. At Time 1, only three of the nineteen variables showed no significant correlation with gaming disorder, specifically System 1 and System 2 for the Loss versus neutral contrast, as well as System 2 for the reward versus neutral contrast. During Time 2, there was a different pattern; only anxiety indicated an insignificant relationship with gaming disorder (see Table 4 for details).

RQ1: Linear Regression

Research Question 1 aims to examine the relationship between all variables of interest and gaming disorder. We run two linear regressions, one for each time point, to further understand the relationship between the variables of interest and gaming disorder crosssectionally. For each regression, we control for race, income, research site, family relationships (i.e., siblings and twins), and pubertal status. The Time 1 regression [*F*(24, 1396) = 27.7, Adjusted $R^2 = .31, p < .001$] indicated that the following variables as significantly related to gaming disorder scores: male sex [t(1396) = .14.4, p < .001], high impulsivity [t(1396) = 8.7, p < .001], high ADHD [t(1396) = 2.99, p = .003], low self-esteem [t(1396) = 3.74, p = .002], high family conflict [t(1396) = 2.62, p = .009], high impulsivity [t(1396) = 2.99, p = .003], and high bullying victimization [t(1396) = 2.4, p = .02] (see Table 4). Accounting for the control variables through a hierarchical regression, the regression model explained 27% of the variance in gaming disorder [$\Delta R^2 = .27, p < .001$], with sex explaining 16% of the remaining variance.

The Time 2 regression echoed similar results, showing significance with the following variables: male sex [t(1626) = -13.3, p < .001], high impulsivity [t(1626) = 8.62, p < .001], high ADHD symptoms [t(1626) = 2.3, p = .02], high depression symptoms [t(1626) = 3.9, p < .001], low aggression [t(1626) = -2.3, p = .02], high family conflict [t(1626) = 3.5, p < .001], low inhibition [t(1626) = 5.3, p < .001], high peer victimization [t(1626) = 1.98, p = .05], and high peer aggression [t(1626) = 2.1, p = .03]. Overall, this model explained 19.6% of the variance in gaming disorder during Time 2 [F(24, 1626) = 19.72, Adjusted $R^2 = .216, \Delta R^2 = .196, p < .001$], with sex explaining 7% of the model's variance.

Surprisingly, within both the Time 1 and Time 2 regression models, social

competence, anxiety, conduct disorder, sensation seeking (BAS), and the fMRI Systems were insignificant.

Table 4

Linear Regression Table with Time 1 and Time 2 Using Standardized Betas and Zero-Order Correlations

	Tim	ne 1	Time 2		
	Zero-Order Correlation (r)	Standardized Beta (β)	Zero-Order Correlation (r)	Standardized Beta (β)	
(Intercept)		1.14 ***		1.15 ***	
Site Number	-0.01	-0.00 -0.05*		-0.04	
Income	-0.15***	-0.12 *** -0.05*		-0.09 ***	
Education	-0.03	0.01	0.00	0.01	
Race	0.05	0.03	0.06*	0.05 *	
Sex	-0.39***	-0.39 ***	-0.27***	-0.37 ***	
Puberty	-0.12***	0.04	-0.11***	0.04	
Impulsivity	0.36***	0.23 ***	0.27***	0.22***	
ADHD	0.24***	0.09 **	0.15***	0.07 *	
Depression	0.10***	-0.02	0.13***	0.12 ***	
Aggression	0.14***	0.00	0.06**	-0.08 *	
Negative Life Events	0.09***	-0.04`	0.05*	-0.02	
Esteem	0.17***	0.09***	0.09***	0.03	

Social Competence	0.11***		-0.01	0.06*		-0.03
Anxiety	0.05*		0.05	0.04		-0.02
Conduct Disorder	0.15***		-0.01	0.07**		-0.06`
Family Conflict	0.19***		0.06**	0.17***	0	.09 ***
BAS	0.16***		0.02	0.07**		0.01
BIS	0.07**		0.08**	0.08***	0	.14 ***
Peer Victimization	0.20***		0.07 *	0.12***		0.05*
Peer Aggression	0.21***		0.03	0.15***		0.05*
System 1 Loss v. Neutral	-0.04		-0.05	0.06*		-0.11
System 2 Loss v. Neutral	-0.02		0.13	0.05*		0.08
System 1 Reward v.	-0.06*		-0.02	0.07**		0.15`
Neutral System 2 Reward v. Neutral	-0.04		-0.09	0.05*		-0.06
Ν		1392			1632	
Adjusted R2			0.31			0.22

The output of the regression is reported as standardized Betas. *** p < 0.001; ** p < 0.01; * p < 0.02; ` p < 0.10

RQ2: Cross-Lagged Panel Models

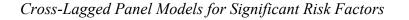
The goal of research question 2 is to examine the longitudinal relationships between the predisposing factors in the I-PACE model and GDAL. In RQ2 two, we employ a crosslagged panel model (CLPM) using the lavaan package in R. The CLPM uses two waves of measurements to estimate the influence of a predictor at Time 1 on an outcome at Time 2 while controlling for the Time 1 variables (Maxwell & Delaney, 2004). Nineteen CLPMs were conducted for each variable of interest except for sex since it is included as a time-invariant predictor along with site, family, and race.

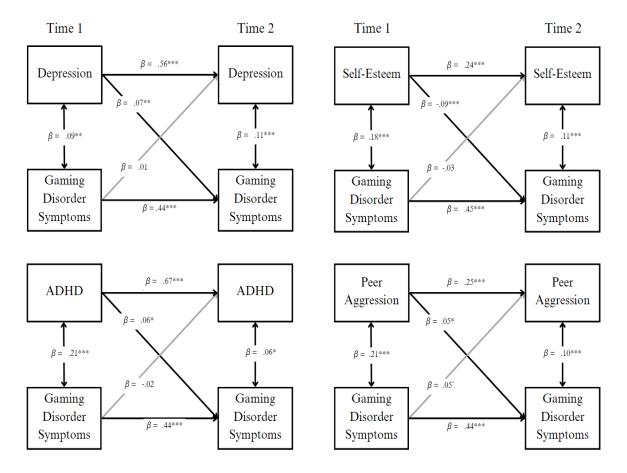
Risk Factors for Gaming Disorder

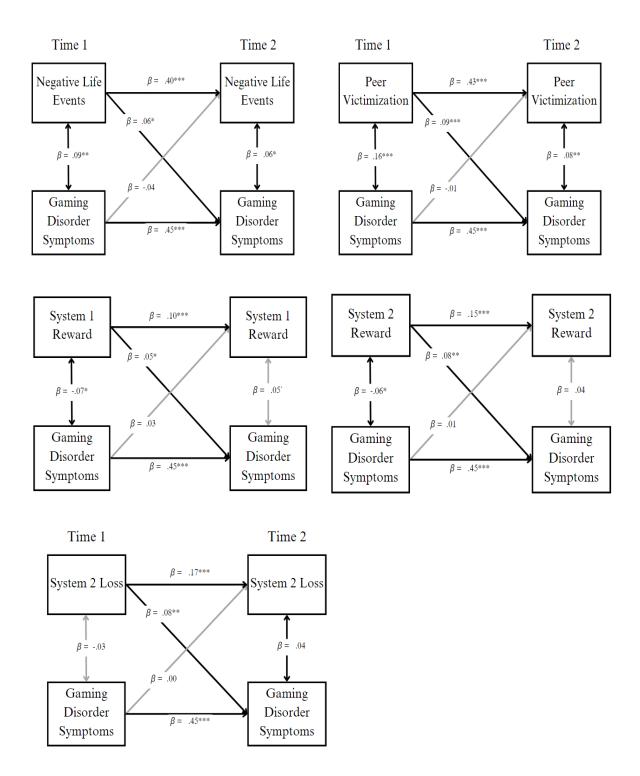
Results from the CLPM indicated nine significant risk factors for developing gaming disorder. Items are considered risk factors if the relationship between the Time 1 independent variable and the Time 2 dependent variable (gaming disorder) is significant, but the inverse relationship is insignificant. The nine significant risk factors are higher depression symptoms $[\beta = .07, p = .004]$, higher ADHD symptoms $[\beta = .06, p = .001]$, lower self-esteem $[\beta = .09, p = .004]$ p < .001 higher relational peer aggression [$\beta = .05$, p = .001], more negative life events [$\beta =$.07, p = .002], higher peer victimization [$\beta = .07$, p < .001], hyperactivation in System 1 [$\beta =$.05, p = .002] and System 2 [$\beta = .08$, p = .002] during reward anticipation, and hyperactivation in System 2 during loss anticipation [$\beta = .08, p = .002$]. Further, there was trending significance for increased family conflict [$\beta = .09, p = .07$], higher impulsivity [$\beta =$.05, p = .051], and hyperactivation in System 1 during loss anticipation [$\beta = .05$, p = .053]. For example, in the CLPMs for depression (Figure 4), the cross-sectional relationships echo the findings in the regression, indicating that at Time 1 and Time 2, depression symptoms are significantly related to gaming disorder [(T1; $\beta = .09$, p = .002), (T2; $\beta = .11$, p < .001)]. Further, the horizontal lines indicate the stability of the trait over time, indicating that Time 1 depression scores are significantly related to Time 2 depression scores [$\beta = .56, p < .001$], and a similar relationship is seen with Time 1 and Time 2 [$\beta = .44$, p < .001] gaming disorder. Lastly, from each CLPM model, we can examine the cross-sectional relationships,

which indicate the directionality of the relationship between depression and gaming disorder. Depression is found to be a risk factor because the relationship between depression (T1) and gaming disorder (T2) [$\beta = .07$, p = .004] must have a stronger relationship than gaming disorder (T1) and depression (T2) [$\beta = .01$, p = .61]. Similar patterns are seen for the other eight variables- indicative of a temporal order.

Figure 4





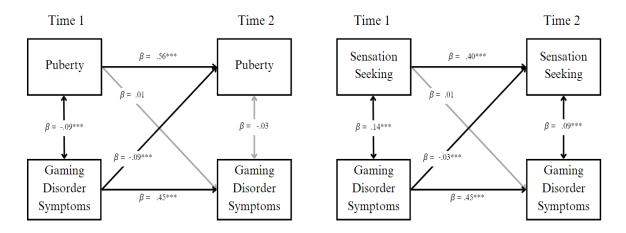


Note. All values are mean-centered. Positive numbers indicate increases, and negative numbers indicate a decrease from the average. *** p < 0.001; ** p < 0.01; * p < 0.05, `p < 0.09

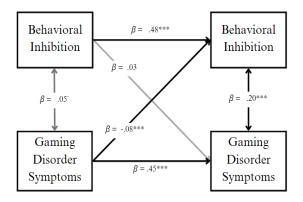
Outcomes of Gaming Disorder

Further, higher gaming disorder symptoms at Time 1 significantly predicted lower inhibition [β = -.08, p < .001], lower sensation seeking [β = -.03, p = .001], and less pubertal development [β = -.09, p < .001] (see Figure 5). These results indicate that from Time 1 to Time 2, behavioral inhibition increases [β = .48, p < .001]; however, for those who experience high gaming disorder symptoms, their inhibition decreases over time, and having a low inhibition at Time 1 does not predict developing gaming disorder at Time 2. Sensation seeking has a similar pattern, although sensation seeking increases over time; for individuals with gaming disorder, sensation seeking decreases over time compared to those without gaming disorder. Lastly, although we included pubertal development as a controlling factor, it revealed unexpected findings that higher gaming disorder symptoms relate to slower pubertal development two years later.

Figure 5



Cross-Lagged Panel Models for Significant Outcome Factors

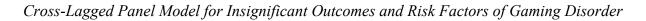


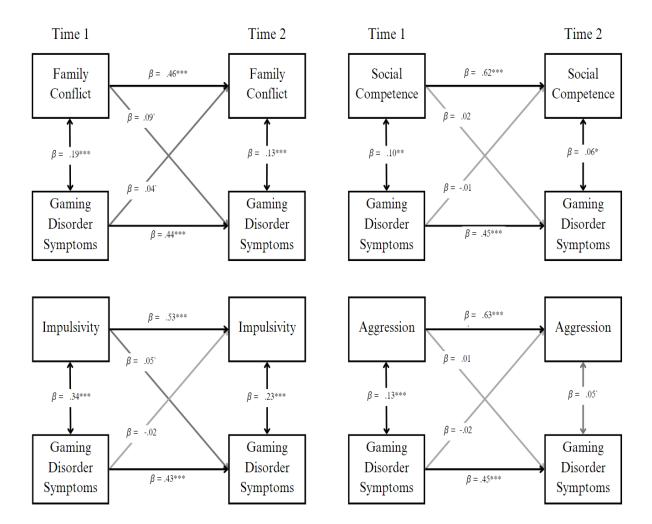
Note. All values are mean-centered. Positive numbers indicate increases, and negative numbers indicate a decrease from the average. *** p < 0.001; ** p < 0.01; * p < 0.05, ` p < 0.09.

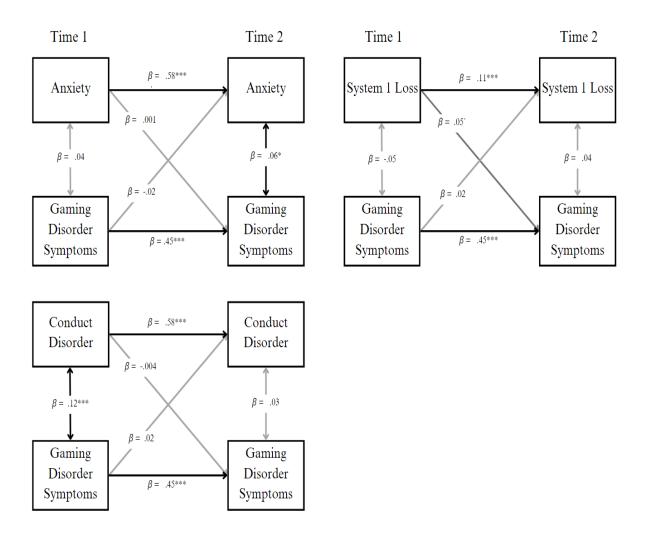
Insignificant Longitudinal Relationships to Gaming Disorder

Lastly, the longitudinal analysis revealed that many of the variables of interest were insignificant despite the extensive previous research. For the loss anticipation compared to neutral contrast in the MID task, no significant predictive relationship was found for gaming disorder scores and System 1 [β = .05, p = .06] nor as an outcome [β = .02, p = .43]. Further, anxiety, conduct disorder, family conflict, social competence, impulsivity, and aggression do not indicate significance for being either outcomes or risk factors for gaming disorder but do indicate significance cross-sectionally (See Figure 6).

Figure 6







Note. All values are mean-centered. Positive numbers indicate increases and negative numbers indicate a decrease from the average. *** p < 0.001; ** p < 0.01; * p < 0.05, ` p < 0.09

Discussion

This study investigated the associations between adolescent development, predisposing factors, and neurobiological functioning with gaming disorder. The intricate nature of gaming disorder prompted a longitudinal exploration into its progression, associated factors, and neurological aspects. Our findings revealed seven key risk factors for gaming disorder development from early to mid-adolescence, including depression, ADHD, peer aggression, peer victimization, low self-esteem, and negative early life events.

Additionally, two main outcomes of gaming disorder in early adolescence were identified as decreased behavioral inhibition and diminished sensation-seeking. These findings underscore the complexity of gaming disorder, revealing numerous cross-sectional relationships without longitudinal implications and vice versa.

Cross-Sectional Results

In examining cross-sectional relationships between predisposing factors and gaming disorder, our findings revealed significant associations with all variables of interest, supporting hypothesis 1. However, not all variables remained significant when applying regression analysis with control variables such as demographic factors, site location, family relationships, and pubertal status. Specifically, at both Time 1 and Time 2, higher gaming disorder scores were associated with male sex, low household income, and high impulsivity. Time 1 showed associations with low self-esteem and higher peer victimization, while Time 2 exhibited connections with higher inhibition, family conflict, depression symptoms, and pubertal development. Intriguingly, contrary to existing literature, social competencies (Hygen et al., 2019), anxiety (Wang et al., 2017), sensation seeking (Xiang et al., 2021), and conduct issues (Gustirani & Amin, 2022) did not a significant cross-sectional relationship with gaming disorder scores.

Effects of Sex

The observed lack of significance in certain variables within our comprehensive regression analysis raises the possibility of significant overlap among these variables. For instance, the strong relationship between male sex and gaming disorder (β = -0.30, p < .001), alongside associations with ADHD (r = -.16, p < .001), impulsivity (r = -.13, p < .001),

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anxiety (r = .19, p < .001), and self-esteem (r = .26, p < .001) underscores the intricate interplay of these variables with sex. Thus, when variance from sex is removed, factors such as impulsivity and ADHD are no longer significant. In a recent meta-analysis by Koncz and colleagues (2023), examining the relationship between factors such as ADHD and gaming disorder concluded that sex has a robust impact on the effects between hyperactivity/impulsivity (ADHD) and gaming disorder, such that larger male samples will result in inflated significance results.

Effects of Puberty

Our study considered the role of pubertal development, a factor often neglected in cross-sectional research. Factors such as relational conflict, cognitive imbalances, and psychopathology are spurred on during puberty. Controlling for pubertal development (β = 0.02, p < .05), which is significantly related to peer victimization (r = .10, p < .001), behavioral inhibition (r = .14, p < .001), and self-esteem (r = .10, p < .001), reduced their relationship to gaming disorder. Controlling for pubertal development significantly reduced its relationship with gaming disorder, shedding light on a potential third-variable effect not fully addressed in the current adolescent literature.

System 1 & System 2

Significant cross-sectional correlations emerged from the neuroimaging results, validating that the a-priori Systems are significantly related to gaming disorder. However, the results did not hold within the linear regression, resulting in insignificant cross-sectional results for both contrasts. Possible limitations of our approach, involving within-person parcellated beta weights and a diverse sample, warrant consideration. The non-significant findings could result from having a larger and more diverse sample compared to any other fMRI research study on adolescent gaming disorder, and the previous research findings do not sufficiently generalize (Marek et al., 2019). However, it is also worth noting that fMRI media addiction research was conducted with ABCD, and significant results were found, but previous research has examined exclusively resting-state fMRI network connectivity that does not encompass the a-priori System 1 and System 2 of interest (Miller et al., 2023; Song et al., 2023). The inconclusiveness of our null results prompts reflection on potential flaws in the study design or the generalizability of previous research methodologies to the MID task within the ABCD population.

Longitudinal Results

Hypothesis 2 delved into the intricate longitudinal relationships between predisposing factors outlined in the I-PACE and GDAL models on gaming disorder development.

Risk Factors of Gaming Disorder

Our CLPM analysis unveiled key insights into the risk factors contributing to gaming disorder through the cross-lagged panel models. Notably, low self-esteem ($\beta = 0.09$) and high peer victimization ($\beta = 0.09$) emerged as the strongest predictors, followed by depression ($\beta = 0.07$), ADHD ($\beta = 0.06$), negative life events ($\beta = 0.06$), peer aggression ($\beta = 0.05$), and the neurological indicators: System 1- Reward ($\beta = 0.5$), System 2- Reward ($\beta = 0.8$), and System 2-Loss ($\beta = 0.8$). ADHD corroborated the prediction outlined in GDAL as it was exclusively a risk factor. Adolescents with ADHD and impulsivity, often linked to reward processing difficulties, showed susceptibility to the high rewards and novelty in video games, contributing to problematic gaming behaviors (Hygen et al., 2020). While GDAL predicted impulsivity and self-esteem as both outcomes and risk factors, our findings identified them

solely as risk factors. Self-esteem is a recurring risk factor for gaming disorder and is explained by self-determination theory, indicating that individuals will engage in activities to fulfill physiological needs such as competency and expertise introduced in games to boost self-esteem (Bender & Gentile, 2020; Kavanagh et al., 2023). Depression and peer aggression, initially predicted as outcomes, emerged as significant risk factors, emphasizing the dynamic nature of gaming disorder development. Depression is one of the most common comorbidities with gaming disorder; however, even in recent reviews of depression and gaming disorder (specifically Mihara & Higuchi, 2017 and Ostinelli et al., 2021), it is still unclear if depression is a risk factor or outcome. The unexpected link between depression in early adolescence and the risk of developing gaming disorder supports ongoing debates suggesting problematic gaming as a coping mechanism for depression (van Rooij et al., 2018). Additionally, we speculate that adolescents with higher peer aggression tendencies may turn to gaming as a form of escapism or an outlet for toxic communication. Finally, peer victimization was anticipated as a risk factor; however, no association was observed in a positive cross-sectional relationship during Time 1. These findings underscore the complex and dynamic nature of gaming disorder, suggesting that these variables interact over time to contribute to the evolution of gaming disorder.

Outcomes of Gaming Disorder

Our findings revealed that gaming disorder symptoms correlated with future issues in behavioral inhibition, lower sensation and novelty seeking, and marginally slower pubertal development. GDAL outlined that behavioral inhibition would be a risk factor and an outcome; however, we only found support for it as an outcome. Per the I-PACE model, the expected positive feedback loop between impulsivity/inhibition and gaming behaviors

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revealed complexities and potential limitations in our operationalization of inhibition and impulsivity measures. Inhibition and impulsivity were measured as self-reported trait measures; while validated and reliable measures, the inhibition and impulsivity conceptualized by I-PACE may require an objective measure of behavioral inhibition through a task such as a Stroop task or Stop-Signal task. Higher sensation seeking was a risk factor proposed in Lee's taxonomy of gamers (2017) and outlined in I-PACE, but minimal longitudinal research supported that claim. Our study showed decreased sensation seeking, suggesting that the gaming environment might fulfill adolescents' novelty and sensation needs, reducing their inclination to seek these experiences outside gaming. Surprisingly, gaming disorder at time one predicted lower pubertal development scores at time two. This was an unexpected finding, as the pubertal CLPM was meant as a control for hormonal changes. We postulate a potential mediation effect between gaming disorder and pubertal development by environmental stress (Avci et al., 2022) or poor nutrition (Yen et al., 2019), both of which could impact puberty. The existence of these connections underscores the multifaceted nature of this phenomenon and highlights the significance of devising strategies to prevent and manage gaming disorder once symptoms arise in adolescents.

Implications for I-PACE and GDAL

While the I-PACE model offers a comprehensive framework for understanding the complexities of addictive behaviors related to internet use, it is important to acknowledge its limitations – many of which are highlighted in our findings. One notable limitation is it does not account for age-related differences in how the brain processes rewards, as proposed by the maturation imbalance hypothesis. This omission is significant given that age-related changes in brain development can impact susceptibility to addictive behaviors. Based on our

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findings, many variables, such as anxiety, social competencies, and behavioral inhibition, are highly correlated with puberty, which may moderate their relationship to gaming disorder. We suggest that I-PACE incorporate an age-related factor for children and adolescents still undergoing developmental changes. Within the most recent update of I-PACE, Brand and colleagues (2019) discuss the inconsistencies in fMRI research, with some studies finding that individuals with gaming disorder experience hyperactivity in System 1, while other research studies find hypoactivity in System 1 during reward-related cues compared to individuals without gaming disorder. They ameliorate these inconsistencies by stating that it may be related to the type of reward (either a gaming or monetary cue) that explains these differences. However, based on our findings, we offer an alternative explanation of age differences in reward processing. For example, at time 1 (ages 11-12), individuals with higher gaming disorder scores exhibited hyperactivity in System 1 during reward anticipation, but at time 2 (ages 13-14), we saw trending hyperactivity in System 1. These findings also highlight the model's lack of a clear operationalization for testing the neurological components it incorporates, which may hinder the empirical validation of the neurobiological mechanisms proposed. We operationalize System 1 and System 2 for our study based on previous gaming disorder research and the CNDS model. However, reproducibility and longitudinal research become difficult without explicitly stating the key systems and which fMRI tasks can best evoke the proposed areas. We suggest that future models of I-PACE incorporate CNDS, offering a dual system perspective and suggesting delayed discounting tasks for fMRI research. Lastly, the model does not adequately address the varying levels of addictive content present in different video games, which could influence both the severity and the rate of development of gaming disorder symptoms. While our study does not provide information to answer that question, future research should consider exploring more addictive content, which may advance gaming disorder symptoms more quickly, such as randomized reward systems like loot boxes -- especially in the unique developmental period of adolescents when reward sensitization is at a peak. Addressing these limitations would enhance the model's applicability and accuracy in explaining the intricate dynamics of gaming disorders.

The present study stands as the first to comprehensively examine the longitudinal risk and outcome factors of gaming disorder within an American adolescent population. Another strength stems from the rigor and incorporation of leading frameworks in gaming disorder research (e.g., I-PACE, GDAL, and CNDS). Leveraging the ABCD study dataset with a large semi-representative sample across the United States and validated measures strengthened our study.

Nonetheless, it is important to acknowledge several limitations of this study. The first limitation was introduced within the sample: only one-tenth of the total ABCD sample could be used, which could present potential exclusion biases. For example, many participants were excluded for not completing both fMRI scans with minimal movement, which may have excluded youth with ADHD or autism symptoms who would have difficulties staying still in the MRI scanner. Further, the exclusion of the baseline, 1-year follow-up, and 3-year follow-up visit data, which lacked measures of impulsivity and neuroimaging, limited the longitudinal depth of the analysis. Finally, the choice of neuroimaging task, the Monetary Incentive Delay (MID) task, may not be optimized for capturing a dual imbalance system associated with gaming disorder– proposed in I-PACE and CNDS. For future studies

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examining this system, we recommend a delayed discounting task and a functional network approach for analyses.

Expanding on these findings requires cross-checking with clinical samples of American adolescents, using more appropriate fMRI tasks, and examining inhibition and impulsivity through objective behavioral tasks. In-depth assessments considering clinical and socio-environmental contexts can provide a deeper understanding of the evolving dynamics of gaming disorder. Future research should explore predictors extensively across various populations, clinical and non-clinical, within the United States to offer a more nuanced and complete picture of how gaming disorder evolves over time.

Conclusion

This study delved into the complex landscape of gaming disorder, exploring its connections with adolescent development, predisposing factors, and neurobiological functioning over two years. Our cross-sectional review aligned with existing literature but uncovered contradictions when adjusting for demographic variables, highlighting relationship complexity. Crucially, sex and pubertal development emerged as influential factors, shaping the significance of predisposing elements. Our longitudinal results identified nine risk factors and two outcomes as significant. We verified two neurological systems— a hyperactive System 1 and System 2 during reward anticipation and a hyperactive System 2 during loss anticipation.

Despite strengths like a comprehensive exploration across gaming addiction frameworks and a large American adolescent sample, the study acknowledges limitations such as neuroimaging task selection and using a non-clinical population, calling for refined methodologies in future research. While the study boasts strengths such as comprehensive exploration across gaming addiction frameworks and a large American adolescent sample, it acknowledges limitations, urging refined methodologies in future research. The findings challenge debates on gaming disorder causality and outcomes, emphasizing caution in interpreting cross-sectional research due to potential confounding constructs. To mitigate spurious results in adolescents, popular models like I-PACE should integrate developmental components, clarify neurological processes, and propose standardized fMRI tasks. In essence, gaming disorder is recognized as a dynamic, complex phenomenon with overlapping factors and outcomes. The study encourages discussions to move beyond crosssectional studies, urging a focus on encompassing frameworks accounting for human development.

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Appendix

Figure A1

Visualization of the I-PACE model from Brand et al., 2016

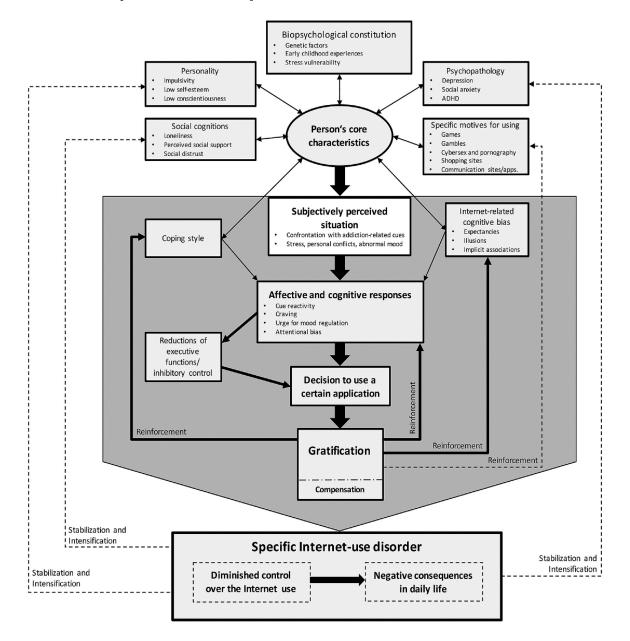


Figure A2

Time 1 Correlation Matrix

	addition for the sector
	1
adhd.y 0.6 0.350.09 0.03 120.160.520.470.010.090.140.25 0.10.160.060.020.160.03 53 0.050.030.060.050.150.24 aggression log.y 0.430.050.040.130.130.660.540.020.080.170.150.090.140.030.010.06 0 0.53 0.050.040.070.060.080.14	
aggression_109.9 0.430.050.040.130.130.840.540.020.840.140.150.040.140.040.040.040.040.040.040.040.04	- 0.8
basy 0.240.120.110.070.050.030.070.060.270.050.15 0 0.030.090.01 0 0.040.020.030.040.040.040.040.040.040.040.040.04	0.0
bis total.y 0.090.250.040.050.050.230.080.1 0.110.160.14 0 0.180.010.050.040.050.030.060.080.07	
bully agress log y 0.510.140.090.020.150.280.360.050.180.06 0 -0.050.060.070.01 0 -0.01 0 0.090.21	- 0.6
bully_victim_log.v 0.130.130.010.270.230.24 0 0.320.110.030.020.02.190.040.050.040.050.040.08 0.2	
conduct.y 0.42 0 0.060.180.210.140.14 0 -0.010.080.020.440.030.030.070.080.150.15	- 0.4
depression log y 0.020.18 0.10.090.050.1 0 0 -0.020.050.53 0.060.030.060.050.05 0.1	
education.y -0.040.030.020.3-0.110.090.130.020.070.040.070.080.060.060.060.03	
esteem_log.y 0.130.170.050.16 0.1 0.020.040.040.160.020.02 0 0 0.070.17	- 0.2
family_conflict.y 0.330.120.160.040.010.010.080.130.01 0 -0.050.020.130.19	
impulsivity.y -0.03.160.010.02 <mark>0.13</mark> 0.010.090.010.030.010.010.150.36	- 0
income.y -0.190.120.240.010.120.120.040.030.050.040.230.15	
negative_le_log.y 0.090.040.040.040.040.040.030.020.030.010.070.09	
puberty.y 0.090.510.040.02 0 0.01-0.040.020.010.12	0.2
race 0.04 <mark>0.22</mark> 0.010.070.060.070.060.050.05	
sex 0 0.020.010.010.030.020.240.39	0.4
site_int -0.01D.060.060.060.060.010.02	
social_log.y <mark>-0.04</mark> 0.010.040.020.040.11	0.6
system1_loss.y 0.880.850.76 <mark>0.030.04</mark>	-0.0
system1_reward.y 0.750.850.020.06	
system2_loss.y 0.890.040.02	0.8
system2_reward.y -0.030.04	
total_vg.y 0.42	-1

	diction	300 80 M
adhd.x 0.610.390.020.04.110.170.550.470.02 0.1 0.190.290.010.1-0.07 0 -0.070.04.540.050.050.050.040.120.15		1
aggression_log.x 0.430.050.010.120.180.660.530.040.120.250.250.040.150.050.02 0 -0.010.560.020.020.020.020.060.06		
anxiety_log.x - <mark>0.04</mark> 0.240.02 0.1 0.270.570.040.190.090.060.020.120.050.010.19 <mark>0.030.54</mark> 0.010.020.010.01 <mark>0.05</mark> 0.04	-	0.8
bas.x 0.210.080.140.07 <mark>0.07</mark> 0 -0.020.020.17 0 0.12-0.02 0 -0.020.020.02		
bis_total.x 0.050.21 <mark>0.06</mark> 0.140.070.330.130.050.160.170.170.010.310.010.090.010.03 0 0 - <mark>0.1</mark> 30.08		0.6
bully_agress_log.x 0.180.120.060.010.030.160.21-0.010.110.020.01 <mark>0.07</mark> 0.060.040.060.080.030.040.070.15		0.0
bully_victim_log.x 0.210.160.020.250.260.240.040.310.1140.010.13 0 0.17-0.030.020.040.030.010.12		
conduct.x 0.420.010.080.270.260.070.11 0 -0.020.040.030.480.030.040.040.120.07	F	0.4
depression_log.x 0.070.260.18 0.2 0.010.170.06 0 0.120.040.52 0.01 0 -0.020.020.020.13		
education.x 0.010.030.010.35 0 -0.040.150.030.05 0 -0.040.040.040.030.07 0		0.2
esteem_log.x 0.240.18 0 0.140.17 0 0.26 <mark>0.05</mark> 0.150.030.050.010.020.040.09		0.2
family_conflict.x 0.330.050.180.040.010.070.040.160.020.02 0 -0.010.070.17		
impulsivity.x 0.040.130.020.020.010.010.150.030.050.030.050.1 0.27	ŀ	0
income.x - <mark>0.110.040.26</mark> 0.040.1-0.060.020.030.030.050.270.05		
negative_le_log.x 0.080.070.110.040.090.01 0 -0.01 0 -0.020.05	-	-0.2
puberty.x 0.060.550.010.030.020.030.010.01-0.060.11		
race 0.06-0.20.010.020.010.020.020.110.06		
sex -0.030.060.010.010.010.0190.27	-	-0.4
site_int -0.040.030.040.030.040.030		
social_log.x -0.030.010.020.010.060.06	_	-0.6
system1_loss.x 0.890.840.720.010.06		
system1_reward.x 0.740.810.010.07		
system2_loss.x 0.880.020.05		-0.8
system2_reward.x 0.030.05		
total_vg.x 0.31		-1

Note. Variables are put into alphabetical order. The '.y' indicates Time 1, and the '.x'

indicates Time 2. 'Total_vg' = average time spent playing video games per day,

'vg_addiction_score' = continuous measure of gaming disorder based on the sum score of the

Video Game Addiction Questionnaire