MONASH University

# *The neurocognitive predictors of substance and non-substance related addictive behaviours*

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Bachelor of Psychology (Honours)

A thesis submitted for the degree of *Doctor of Philosophy* at
Monash University in 2023

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## *Copyright notice*

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# *Abstract*

Addiction is a chronic, and complex mental health disorder marked by persistent compulsive patterns of behaviour (like drug seeking or gambling), despite harmful consequences. Addictive behaviours are associated with a distinct pattern of neurocognitive functioning characterised by dysregulated reward processing and impaired cognitive control. However, empirical evidence articulating specific neurocognitive functions that predict addictive behaviours is lacking. Notably, most research is cross-sectional, lacks systematic examination of multiple neurocognitive functions, and rarely explores non-substance addictive behaviours. This thesis aimed to comprehensively evaluate the neurocognitive predictors of addiction and addictive behaviours across different addiction types, in both substance and non-substance-related behaviours.

Study one was a systematic review of the literature to assess whether cognitive control and reward-related processes predict the development and maintenance of addictive behaviours specifically, consumption, severity, and relapse. The findings expose a substantial lack of evidence, where less than half of the studies showed neurocognition significantly predicted addiction outcomes. Further, there was minimal research investigating the predictors of non-substance addiction. Studies two and three were derived from a large-scale longitudinal cohort study that evaluated the neurocognitive correlates (study two) and predictors (study three) of alcohol use and three common non-substance addictive behaviours: addictive-like eating (AE), problematic use of the internet (PUI) and problematic pornography use (PPU). Each study evaluated addictive behaviours dimensionally in a general population sample, using a comprehensive assessment battery selected specifically to measure theory-driven and expert endorsed neurocognitive constructs important to addiction.

Study two revealed addictive behaviours were associated with a unique profile of neurocognitive functioning (n=475), poorer performance monitoring was associated with more PPU and PUI (β=-0.10, $p$=.049; β=-0.09, $p$=.028), and less delay discounting was associated with higher PUI (β=-0.10, $p$=.025). None of the neurocognitive domains were found to be associated with AE or problematic alcohol use ($p$>.05). Study three involved a sub-sample of participants from study two who were followed longitudinally over a 3- (n=206) and 6-month (n=138) timeframe. Poorer

performance monitoring (β=-0.16, *p*=.004) and more reward-related attentional bias (β=0.14, *p*=.033) predicted higher AE. Less delay discounting predicted more PPU (β=-0.16, *p*=.014), and less reward-related attentional bias (β=-0.14, *p*=.003) and less risk-taking (β=-0.11, *p*=.029) predicted PUI. None of the neurocognitive variables were found to predict problematic alcohol use (*p*>.05).

This thesis reveals an absence of evidence for a consistent transdiagnostic neurocognitive correlate or predictor of addictive behaviours, challenging existing models of addiction. This underscores the importance of exploring differences as well as commonalities among addictive behaviours. These insights inform a deeper understanding of the types/profiles of individuals who may be at risk of developing different types of addiction and have key implications for early intervention, prognosis, and more accurate diagnosis. This thesis supports the need for tailored treatments targeting behaviour-related neurocognitive functions, acknowledging that cognitive dysfunction may vary across addictions.

# *Declaration*

This thesis is an original work of my research and contains no material which has been accepted for the award of any other degree or diploma at any university or equivalent institution and that, to the best of my knowledge and belief, this thesis contains no material previously published or written by another person, except where due reference is made in the text of the thesis.

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Date: 24/11/2023

# *Publications during enrolment*

***Publications***

**Christensen, E.,** Albertella, L., Chamberlain, S. R., Brydevall, M., Suo, C., Grant, J. E., Yücel, M., & Lee, R. S. C. (2023). The neurocognitive correlates of non-substance addictive behaviors. *Addictive Behaviors, 150, 107904*. https://doi.org/10.1016/j.addbeh.2023.107904

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# *Thesis including published works declaration*

I hereby declare that this thesis contains no material which has been accepted for the award of any other degree or diploma at any university or equivalent institution and that, to the best of my knowledge and belief, this thesis contains no material previously published or written by another person, except where due reference is made in the text of the thesis.

This thesis includes two original papers published in peer reviewed journals and one paper submitted for publication. The core theme of the thesis is evaluating the neurocognitive correlates and predictors of addictive behaviours. The ideas, development and writing up of all the papers in the thesis were the principal responsibility of myself, the student, working within the School of Psychological Sciences under the supervision of Dr. Rico Sze Chun Lee, Dr. Lucy Albertella and Professor Murat Yücel.

The inclusion of co-authors reflects the fact that the work came from active collaboration between researchers and acknowledges input into team-based research. In the case of *Chapters 2, 3 and 4* my contribution to the work involved the following:

| Thesis Chapter | Publication Title | Status | Nature and % of student contribution | Co-author name(s) Nature and % of Co-author's contribution* | Monash student Y/N* |
|---|---|---|---|---|---|
| 2 | Neurocognitive predictors of addiction-related outcomes: A systematic review of longitudinal studies | Published | 70% Concept & study design, data collection, data analysis, writing of manuscript | 1. Maja Brydevall – 8%<br>2. Lucy Albertella – 3%<br>3. Sashka K. Samarawickrama – 2%<br>4. Murat Yücel – 2%<br>5. Rico Sze Chun Lee – 15% | Yes<br>No<br>Yes<br>No<br>No |
| 3 | The neurocognitive correlates of non-substance addictive behaviors | Published | 70% Concept & study design, data collection, data analysis, writing of manuscript | 1. Lucy Albertella – 5%<br>2. Samuel R. Chamberlain – 3%<br>3. Maja Brydevall – 2%<br>4. Chao Suo – 2%<br>5. Jon E. Grant – 1%<br>6. Murat Yücel – 7%<br>7. Rico Sze Chun Lee - 10% | No<br>No<br>Yes<br>No<br>No<br>No<br>No |
| 4 | A comprehensive evaluation of the neurocognitive predictors of problematic alcohol use, eating, pornography, and internet use: A 6-month longitudinal study | Submitted | 70% Concept & study design, data collection, data analysis, writing of manuscript | 1. Lucy Albertella – 5%<br>2. Samuel R. Chamberlain – 3%<br>3. Chao Suo – 2%<br>4. Maja Brydevall – 2%<br>5. Jon E. Grant – 1%<br>6. Murat Yücel – 7%<br>7. Rico Sze Chun Lee – 10% | No<br>No<br>No<br>Yes<br>No<br>No<br>No |

I have not renumbered sections of submitted or published papers in order to generate a consistent presentation within the thesis.

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I hereby certify that the above declaration correctly reflects the nature and extent of the student's and co-authors' contributions to this work. In instances where I am not the responsible author I have consulted with the responsible author to agree on the respective contributions of the authors.

**Main Supervisor name:** RICO SZE CHUN LEE

**Main Supervisor signature:** **Date:** 24/11/2023

# *Acknowledgements*

First and foremost, I would like to sincerely thank my supervisory team – Dr. Rico Lee, Dr. Lucy Albertella and Professor Murat Yücel. I have the upmost respect and appreciation for each of you and am incredibly thankful to have had the opportunity to learn and grow under your guidance. Thank you for your kindness, encouragement, humour, and endless support. I am profoundly grateful for the opportunities you have afforded me. I can honestly say I have thoroughly enjoyed my candidature and am submitting this PhD feeling excited for what my next step on this academic path will hold. You have challenged me and inspired me. I am the researcher I am today because of your mentorship, thank you.

I would like to thank the members of BrainPark, BMS, and the wider MBI team past and present for an incredible six years. I have learnt so much from you all. You inspired me to commence this PhD journey and have supported me every step of the way. I have made friendships that will last a lifetime. I would also like to sincerely thank all the people who generously devoted their time to participate in my research studies. None of this work would have been possible without your commitment to helping further science and discovery. This research was supported through an Australian Government Research Training Program (RTP) Scholarship, as well as the National Health and Medical Research Council (NHMRC), for which I am sincerely grateful.

To my friends and family – thank you for always believing in me. Thank you for celebrating my wins and supporting me through my disappointments. Your love and endless faith in me is overwhelming, I am incredibly grateful to you all. Mum and Dad, thank you for the opportunities you have afforded me. I am the woman I am today because I stand on your shoulders.

Finally, to Jacob – the love of my life. Thank you for always believing I could do this. Your selfless support, kindness, and patience, especially over the past year, is deeply appreciated. Thank you for making me laugh when I failed to smile, for encouraging me forward when I felt stuck, and holding me tight when I needed safety and stillness. With you by my side, I feel like I can take on the world. I love you, thank you.

*Chapter 1*

# Introduction

Addictive behaviours, ranging from alcohol and other drug use to non-substance behaviours like gambling, excessive eating, and excessive internet use, pose significant health and societal risks. These behaviours can have serious consequences, impacting physical health, mental well-being, and relationships, causing distress to those affected (Carlson et al., 2018; Grant et al., 2004; Hunt et al., 2016, 2018; Kuss et al., 2014; Manning, 2006; Pedram et al., 2013; Raj et al., 2022). For some, addictive behaviours can transition into compulsions and full-threshold addiction. It is at least partially believed that addiction is underpinned by deficits in neurocognitive functions, specifically cognitive control, and risk-reward processes, making individuals more vulnerable to addiction and relapse (Domínguez-Salas et al., 2016; Goldstein & Volkow, 2011; Smith et al., 2014; Verdejo-García et al., 2008; Yücel et al., 2019; Yücel & Lubman, 2007). While cross-sectional work shows neurocognitive deficits have been implicated as transdiagnostic mechanisms of substance use disorders, their role in non-substance addictive behaviours has yet to be comprehensively evaluated. Further, there is limited empirical investigation into how these neurocognitive functions predict addiction risk. This thesis will determine the neurocognitive factors associated with a spectrum of non-substance addictive behaviours, seeking to identify both shared and unique neurocognitive mechanisms. These insights will allow for a deeper understanding of the potentially modifiable risk factors for addiction, informing prevention, early intervention, and targeted treatments.

## 1.1 Definition of addiction

Addiction has been defined as a compulsive disorder, with emphasis placed upon continued use or behaviour despite experiencing harmful consequences (American Society of Addiction Medicine, 2019; American Psychiatric Association, 2020). Accordingly, the core aspects of addiction can be distilled into three primary elements: craving, a conscious expression of desire to engage in the addictive behaviour (Tiffany, 1990); diminished control, manifested as an increasing inability to resist these cravings even when conflicting with personal goals (Grant et al., 2010); and continued

engagement in the behaviour despite negative consequences (Potenza, 2006), whether it is detrimental on physical health, psychological well-being, relationships, professional performance, or the ability to engage in social activities that were once enjoyed.

Formerly considered relevant only to substance use, addiction now encompasses non-substance use behaviours. This change was driven by a large body of evidence showing clinical, phenomenological, neurobiological and genetic similarities between substance-related disorders (Grant et al., 2010), which led to the inclusion of non-substance addictive behaviours as diagnosable disorders in the latest iteration of the Diagnostic Statistical Manual of Mental Health Disorders (5th edition, DSM-V). The DSM-V replaced the category of "Substance-related Disorders" with the more inclusive "Substance-related and Addictive Disorders" (American Psychiatric Association, 2013), including Gambling Disorder as a recognised disorder. Internet gaming disorder was also included as a condition for further study (Petry & O'Brien, 2013). This paradigm shift has brought non-substance addictive behaviours to the forefront, validating their significance as discrete phenomena.

## 1.2 Prevalence and societal impact

Addiction imposes a significant economic and health burden on society, affecting individuals and communities alike. One in four Australians will struggle with a substance use or gambling disorder at some point in their lifetime (Slade et al., 2009). The cost of addiction to the Australian economy is estimated at $80.3 billion (Rethink Addiction & KPMG, 2022). This includes, but is not limited to healthcare, workplace absenteeism and unemployment, law enforcement, and social services costs. Substance and gambling addiction are associated with higher risk of chronic medical health conditions (e.g. hypertension, diabetes, liver disease and stroke; Wadland & Ferenchick, 2004), unhealthy lifestyle choices and impaired quality of life (Black et al., 2013). Substance use and gambling problems also tend to co-occur with other chronic mental health problems, such as anxiety, depression and suicidal ideation (Grant et al., 2004; Hunt et al., 2016, 2018). In 2022, Australia experienced its highest rates of alcohol-induced deaths, with 1,742 recorded fatalities (Australian Bureau of Statistics, 2022). In the same year an additional 1,693 individuals died of drug overdose (Australian Bureau of Statistics, 2022). Over a similar time period, the total net financial losses in

Australia from gambling was 21.2 billion Australian dollars (Australian Institute of Health and Welfare, 2022). The impact and prevalence of these issues are amplified given alcohol, substance and gambling problems are highly co-morbid (Welte et al., 2001; Substance Abuse and Mental Health Services Administration, 2021).

## 1.3 The dimensional expression of addiction

Addiction exists on the severe end of a broad continuum of problematic use or behaviour. Despite the dimensional nature of addictive behaviours, the field continues to use categorical diagnostic frameworks to classify addiction, which although provides a reliable yardstick to compare across studies and samples, impedes our ability to fully capture its nuanced nature and varied expression. The landscape of mental health disorder classification has evolved significantly over time, with clinical criteria and diagnostic thresholds continuously debated and revised (Helzer & Hudziak, 2008), often making them appear somewhat arbitrary and lacking in underlying validity. In the case of addiction, diagnostic thresholds vary depending on the specific addictive behaviour under consideration. For instance, gambling disorder requires a minimum of four symptoms to meet diagnostic criteria, while Substance Use Disorder (SUD) necessitates a threshold of at least two symptoms (DSM-V; American Psychiatric Association, 2013), further contributing to the arbitrary nature of diagnosis. Utilising a dimensional approach when studying addictive behaviours allows researchers to move beyond these strict diagnostic thresholds.

Clinical presentations of addiction represent only the tip of the iceberg. Research conducted within clinical or treatment settings captures only a limited portion of the population impacted by addiction (Bőthe et al., 2023; Grant et al., 2015; Hasin et al., 2013). The trajectory from the onset of addiction problems to receiving clinical care can take a median of 18 years (Chapman et al., 2015). This time delay is likely driven in part by an individual's lack of insight into their problem (Raftery et al., 2020) preventing them from seeking help (Leavens et al., 2014; Slutske, 2006), stigma associated with addiction (Hammarlund et al., 2018), and poor accessibility of services that can provide appropriate diagnosis and treatment (Ritter et al., 2019). Further, clinically significant addiction is not a precondition to the harms associated with addictive behaviour. The majority of addiction problems

do not meet diagnostic thresholds (Grant et al., 2015; Hasin et al., 2013). Individuals engaging in addictive behaviours at sub-clinical levels experience poor psychosocial functioning and severe adverse mental health outcomes (Moghaddam et al., 2015; Neumark et al., 2000; Scherrer et al., 2005; Shankman et al., 2009; Weinstock et al., 2017). Examining addictive behaviours within general community samples offers an ideal opportunity to explore the dimensional aspects of addiction. This approach is particularly useful when evaluating early-stage phenotypes (i.e. before progression to clinical severity) that may be sensitive in identifying individuals at risk of developing an addictive disorder. However, utilising community samples does present a challenge in capturing a sufficient number of individuals exhibiting addictive behaviours at more severe levels, which is an important consideration to ensure there is enough variability to adopt a truly dimensional approach.

## 1.4 Alcohol and other drugs

Historically, addiction was primarily associated with drugs of abuse, such as alcohol, nicotine, and cocaine, to name a few. These drugs of abuse contain chemical or 'psychoactive' compounds that alter subjective experiences and/or behaviour through their interaction with the brain and spinal cord (the central nervous system; Müller & Schumann, 2011). These substances are often used to relieve negative affect, to enhance pleasure or to induce euphoria, and to improve self-confidence and social functioning (Bizzarri et al., 2009). While the majority of people who use drugs of abuse do not develop an addiction (Flórez-Salamanca et al., 2013; O'Brien, 2003), these substances are considered to possess addictive properties. The addictive nature of these substances lies in their ability to manipulate the brain's circuitry associated with behavioural reinforcement, known as the mesolimbic dopamine system (Everitt & Robbins, 2016; Koob & Volkow, 2010). The mesolimbic dopamine system comprises a collection of dopamine neurons in the midbrain of both humans and many other animals, playing a central role in reinforcement learning (Wise, 2004). These drugs are thought to create an imbalance in typical reward-seeking behaviours, leading to heightened reward-related learning in response to addiction-specific cues and valuation that perpetuates continued drug use despite negative consequences (Volkow & Morales, 2015).

Alcohol holds a prominent place in Australia's drug landscape, largely due to societal norms and its widespread availability. Seventy seven percent of Australians aged 14 and above report having consumed alcohol (Australian Institute of Health and Welfare, 2022), placing it as one of the most used drugs nationally. Frequently utilised as a 'social lubricant', or as a coping mechanism for stress and negative emotions (Bresin & Mekawi, 2021), 25.8% of Australian adults surpass the national guidelines for alcohol use (National Health and Medical Research Council, 2009) by either consuming more than 10 drinks per week, or 5 or more drinks on any day at least monthly (Australian Bureau of Statistics, 2022). Alcohol use and abuse poses a significant public health risk in Australia. Alcohol induced fatalities are increasing year by year (Australian Bureau of Statistics, 2022), and alcohol related issues place a significant burden on the country's healthcare system, with alcohol being the primary factor in drug related hospital admissions during 2019-2020 (Australian Institute of Health and Welfare, 2022). In 2021 alcohol related harm costs the Australian economy an estimated $22.6 billion, representing almost a third of tangible costs associated with addiction (Rethink Addiction & KPMG, 2022). Clearly, alcohol stands out as a primary concern among substance related health and financial challenges.

## 1.5 Non-substance addiction

Addiction can occur in the absence of psychoactive substances. Animal studies have shown natural rewards, such as food and sex, engage dopaminergic reward pathways in the brain and are capable of inducing addiction like changes to reward circuitry (Olsen, 2011). Human imaging studies further support this by revealing similarities in fMRI activation patterns associated with substance and non-substance rewards (Motoki et al., 2019), including gambling (Luijten et al., 2017), sexual desire and arousal (Bittoni & Kiesner, 2023), and palatable food (Volkow et al., 2013). However, classifying excessive non-substance related behaviours as addiction has been criticised for potentially blurring the line between risky behaviour and genuine addiction (Kardefelt-Winther et al., 2017). For example, pathologising "normal" and common behaviours. Some argue that these non-substance related addictive behaviours may also be secondary manifestations of other psychiatric illnesses, with individuals engaging in repetitive behaviours to alleviate discomfort (Karim & Chaudhri, 2012). For

example, comfort eating in those with depression (Willem et al., 2020). Further, qualitative analyses have revealed phenomenological disparities between non-substance and substance related addictive behaviours. For example, with the exception of gambling, tolerance and withdrawal does not seem to be present in most non-substance behaviours (Chamberlain et al., 2016; Starcevic, 2016). Despite this, there is consistent evidence showing non-substance addictive behaviours share phenomenological similarities with their substance use counterparts, including a loss of control over the behaviour, compulsive engagement, an urge or craving preceding the behaviour, and often an onset during adolescence or early adulthood (Brewer & Potenza, 2008; Chamberlain et al., 2016; Grant et al., 2013). The ongoing debate surrounding the conceptualisation of these recurring behaviours is crucial to further our etiological understanding and ultimately facilitate the development of more effective therapeutic approaches. This is particularly important considering these behaviours impose significant personal burdens on those who experience them (Grant & Chamberlain, 2016).

### 1.5.1 Addictive eating

The idea that certain foods hold addictive potential has been discussed since the 1950s (Avena et al., 2008; Randolph, 1956). Research has uncovered neuroendocrine pathways that link food and drug seeking behaviours (Leggio et al., 2011; Leggio, L., 2010; Von Der Goltz et al., 2010). However, it is important to note that the idea of food addiction remains a topic of debate (Hebebrand et al., 2014; Ziauddeen et al., 2012) and has not yet gained recognition as a distinct disorder in current diagnostic manuals (American Psychiatric Association, 2013; World Health Organisation, 2019). Despite this debate, the development of the Yale Food Addiction Scale (and its subsequent iterations; Gearhardt et al., 2009, 2016) has enabled over a decade of research into this phenomena. Food addiction has been defined by intense urges to eat, excessive and dysregulated eating behaviour, and eating despite harmful consequences (Gearhardt et al., 2009).

Food addiction "prevalence" is estimated at between 4-15% (Meule & Gearhardt, 2019) and has been linked to a number of adverse consequences, including obesity, depression, anxiety, stress, feelings of hopelessness, reduced sleep quality, and challenges in emotion regulation (Aguirre et al., 2018; Aloi et al., 2017; Brunault et al., 2019; Burrows et al., 2017; Carlson et al., 2018; Carter et al.,

2019; Müller et al., 2018; Pedram et al., 2013). Notably, food addiction includes elements of withdrawal or tolerance which sets it apart from other eating disorders, like binge eating disorder (BED; American Psychiatric Association, 2013). Eating disorders such as BED and Bulimia Nervosa also require the presence of bingeing episodes, defined as excessive eating during a discrete timeframe (American Psychiatric Association, 2013). Bingeing episodes are not a prerequisite of food, addiction which can also involve consistent uncontrolled snacking throughout the day. Both human and animal literature have shown that food addiction is a unique construct, consistent with criteria for other substance use disorder diagnoses, marked by neurobiological changes in reward systems, impaired control, and impulsivity (Gordon et al., 2018).

### 1.5.2 Problematic pornography use

In this modern age of unrestricted internet access on mobile devices, access to pornography has never been easier. Anywhere between 78% to over 90% of people have been exposed to pornography in their lifetime (Dwulit & Rzymski, 2019; Hald, 2006; Træen et al., 2004). The popular site Pornhub alone attracts 44,000 visitors per minute (Bőthe et al., 2018). Sexually explicit material such as pornography are highly rewarding and act as strong reinforcers (Georgiadis & Kringelbach, 2012; Kühn & Gallinat, 2011; Poeppl et al., 2014; Stoléru et al., 2012). As accessibility and exposure to explicit sexual content continue to grow, so have concerns about its potentially addictive nature, prompting the development of assessment scales aimed at quantifying problematic pornography use (PPU; Bőthe et al., 2018).

PPU has been defined by intense urges to use pornography, diminished control over use and use despite negative consequences (Wéry & Billieux, 2017). PPU comprises five key elements: excessive salience of pornography in one's life; mood-modifying properties; use for positive feelings/emotions such as sexual arousal or relaxation; conflict with other life domains such as relationships and work; tolerance requiring more intense stimuli for the same effects; and withdrawal resulting in negative emotional states when discontinuing use (Bőthe et al., 2018). Although, it should be noted that the use of the components model to define PPU has been criticised for not being able to appropriately distinguish between disordered and intense but non-problem sexual behaviours (e.g.

tolerance), and its failure to acknowledge etiological mechanisms from a process-based perspective (Castro-Calvo et al., 2022).

While elements of PPU align with characteristics commonly associated with substance and non-substance addictions, PPU has not yet been officially classified as an addictive disorder in current diagnostic manuals (American Psychiatric Association, 2013; World Health Organisation, 2019). Disordered sexual behaviour was recognised in the latest iteration of the International Classification of Diseases (ICD-11) in the form of compulsive sexual behaviour disorder (CSBD), encompassing pornography use and a range of other compulsive sexual behaviours. However, it has been argued that PPU in particular holds theoretical, clinical and mechanistic evidence to suggest consideration as an addictive disorder on its own (Brand et al., 2020). In the absence of a universally agreed upon diagnosis, assessing the prevalence of PPU is challenging, however estimates currently sit at between 7 and 12.5% (Kumar et al., 2021; Mennig et al., 2020). Most importantly, PPU is closely tied to adverse mental health outcomes, frequently co-occurring with severe levels of depression, anxiety and stress (Camilleri et al., 2021), as well as poorer sexual functioning, relationship dissatisfaction and loneliness (Manning, 2006; Yoder et al., 2005), underscoring its importance as a phenomenon worthy of recognition and study.

### 1.5.3 Problematic use of the internet

Access to the internet has become part and parcel of daily life, offering a multitude of benefits such as ease of communication, increased information sharing, more accessible education modalities, and improved work efficiency. Alongside these advantages, the increasing misuse of the internet has become a matter of growing concern. The internet serves as a platform where individuals can freely access a plethora of rewarding stimuli and experiences, providing feelings of pleasure, reducing negative emotions, and acting as an escape or stress relief, placing internet use at high risk of becoming an addictive behaviour (Brand et al., 2019). The public health and societal costs of excessive internet use behaviours is being increasingly recognised (Cheng & Li, 2014; Fineberg et al., 2018; Stein & Hartford, 2022; World Health Organization, 2015), and its associated psychological harms (Aboujaoude, 2010) are a growing concern for the field of mental health research (Ioannidis et

al., 2018). The European Union has recently funded a large-scale initiative to examine the harmful effects of digitization on mental health, particularly in young people, across the United Kingdom and Europe (European Commission, 2023).

Problematic use of the internet (PUI) describes the excessive and uncontrolled use of internet based activities such as online gaming, shopping, gambling, pornography and social media use (Fineberg et al., 2018). PUI is characterised by escalation and perceived loss of control over the use of the internet despite repeated attempts to control use, as well as preoccupation with, or urges to use, the internet that impacts daily life (Tiego et al., 2021). While the diagnostic criteria for PUI has yet to be agreed, a recent meta-analysis spanning 31 countries estimates the global prevalence of PUI to be approximately 7% (Pan et al., 2020). PUI is linked with a range of comorbid mental and physical health issues, encompassing depression, suicidal ideation, obsessive-compulsive disorder, lack of energy, poor vision, obesity, and excessive daytime sleepiness (Gecaite-Stonciene et al., 2021; Kuss et al., 2014). Notably, both mild and severe problematic internet use behaviour has been shown to be associated with equally adverse mental health consequences (Raj et al., 2022), supporting the need for a dimensional approach to studying PUI.

### 1.5.4 "Problematic" versus "addictive" behaviours

It is important to recognise that addictive behaviours can range from controlled and functional to uncontrolled and maladaptive (Fineberg et al., 2018; Tiego et al., 2021). To reflect this dimensionality, the present thesis labels each behaviour as "problematic" at mild to higher levels. While "PPU" and "PUI" are commonly used terms in the literature and are retained in this thesis, selecting a term to represent the dimensionality of food addiction presents a unique challenge. The term "problematic eating" is inadequate as it could potentially encompass a number of eating related disorders, which are not examined in this thesis (e.g. Binge Eating Disorder, Bulimia Nervosa). Therefore, the term "addictive eating" (AE) is adopted in this thesis to represent food addiction risk dimensionally.

## 1.6 Neurocognition and addiction

Neurocognition refers to cognitive processes that are underpinned by distinct neural pathways in the brain (American Psychological Association, 2015). Accordingly, addiction has been associated with a specific pattern of neurocognitive dysfunction (Ersche et al., 2013; Goldstein & Volkow, 2011; Smith et al., 2014; Verdejo-García et al., 2008; Yücel & Lubman, 2007). Neurocognitive models have proposed that addiction is a product of an imbalance between two distinct but interconnected neurocognitive systems that modulate decision-making: 'top-down' cognitive control processes and 'bottom-up' reward-related functioning (Bechara, 2005; Goldstein & Volkow, 2002, 2011). Cognitive control processes facilitate the pursuit of goals and are required for exercising 'willpower'. Reward-driven functions on the other hand, have the potential to weaken cognitive control processes in favour of reward-seeking behaviours. Addictive behaviours are by definition innately rewarding. Specifically, associative learning underpins how individuals attribute salience to reward-related cues that guide behaviour (Meyer et al., 2015). Repeated exposure to the addictive behaviour for vulnerable individuals can result in neuroadaptations that cause hypersensitised motivational response to reward cues which trigger an urge to engage in the addictive behaviour (Robinson & Berridge, 1993, 2008). Resisting that urge is difficult due to deficits in cognitive control (Goldstein & Volkow, 2002; Smith et al., 2014; Verdejo-García et al., 2008; Yücel et al., 2007), increasing the risk of bingeing.

Several theories have been developed to explain how neurocognitive deficits contribute to vulnerability to addiction. Two prominent perspectives are whether addiction is a product of domain-general neurocognitive functions, such as behavioural disinhibition (Verdejo-Garcia & Albein-Urios, 2021), or a result of domain-specific adaptions related to learning processes specific to the addictive stimulus or behaviour in question (Perales et al., 2020). The former describes a weakness in higher-order neurocognitive processes responsible for goal-directed behaviours, for example, top-down control of cortical systems to regulate general inhibitory behaviour and reward-driven responses (Verdejo-Garcia et al., 2008). The latter describes neurocognitive processes that are shaped according to interactions with the specific addictive behaviour or substance itself. For example, incentive-

sensitisation, a process in which repeated exposure to an addictive stimulus can lead to increased motivation to engage in or seek out that rewarding stimulus, even when it may work directly in opposition to other goals or result in negative consequences (Berridge & Robinson, 2016). While both approaches are valuable in contributing to our understanding of addiction, the present thesis adopts a domain-general approach. This approach aims to explore the neurocognitive processes that underlay addictive behaviours that cut across various addictive behaviour types, encompassing both substance and non-substance related behaviours.

### 1.6.1 The RDoC and addiction

Over a decade ago, the National Institute of Mental Health (NIMH) developed the Research Domain Criteria (RDoC) to promote research that integrates observable behavioural and biologically validated dimensional constructs into the study of mental illnesses. The RDoC reconceptualises mental health along continuums of observable constructs or domains of functioning (NIMH, 2021). Notably, RDoC is highly valuable as it promotes the utilisation of task-based cognitive assessments grounded in an understanding of their neurocircuitry and provides guidance for the use of specific validated tasks for measuring each construct. This approach proves particularly advantageous when tackling dimensional phenomena like neurocognition and addiction. In a recent three round Delphi consensus process, 37 leading international experts in addiction reviewed the RDoC domains to delineate the most critical factors driving addictions. It was concluded that addiction is underpinned by seven crucial neurocognitive constructs: reward valuation; reward learning; reward expectancy; action selection; response inhibition; habit; and compulsivity related neurocognition (Yücel et al., 2019). It is believed that these neurobiological constructs are mechanistically linked to motivation towards, and/or lack of self-regulation to limit, problematic addictive behaviours.

### 1.6.2 Going beyond the Delphi

While the seven constructs proposed by Yücel and colleagues (2019) provide a solid foundation for evaluating the neurocognitive functions underlying addictive behaviours, it is worth noting that the consensus study aimed to offer a concise list of essential constructs. This therefore resulted in the exclusion of functions that, while still potentially relevant to addiction, did not meet the

threshold for inclusion. The final selection of these seven Delphi constructs was based on two key criteria: attaining over 80% consensus and being categorised as 'very important' to 'essential' for understanding addiction. The original criteria appear to be overly reductionistic. When we broaden the second criterion to include constructs deemed 'moderately important' to 'essential,' an additional five constructs emerge as relevant: Sustained threat; initial responsiveness to reward attainment; goal selection, updating, representation and maintenance (hereafter referred to as goal selection/updating); performance monitoring; and flexible updating (i.e. subcomponent of working memory; see supplementary material of Yücel et al., 2019).

The usefulness of these neurocognitive constructs is constrained by our ability to assess and estimate them through behavioural means. This necessitates cognitive tasks with behavioural outputs capable of indicating an individual's functioning within a particular domain. Currently, there is no task designed to evaluate sustained threat. Reward responsiveness is typically assessed through physiological responses to favourable versus unfavourable stimuli, lacking a behavioural output metric. Similarly, the inclusion of compulsivity as one of the Delphi seven is perplexing. Compulsivity is a higher-order construct comprised of multiple diverse neurocognitive domains (e.g. inhibition, cognitive inflexibility, set shifting, attentional bias and habit learning; Lee et al., 2019), unable to be measured by neurocognitive task(s) alone.

Since the publication of the Delphi consensus study (Yücel et al., 2019) an updated RDoC matrix was introduced, in which "action selection" was omitted. Originally defined as “a process involving an evaluation of cost/benefits and occurring in the context of multiple potential choices being available for decision-making” (NIMH, 2011), it is now viewed as an integral component of reward valuation, encompassing the assessment of multiple options in light of their respective outcomes. After expanding the selection criteria, removing those constructs that could not be objectively measured via neurocognitive tasks, and using the updated RDoC matrix, eight construct emerged: response inhibition; goal selection/updating; performance monitoring; flexible updating; reward learning; habit; reward expectancy; and reward valuation. Each is discussed below.

### 1.6.3 Response inhibition

Response inhibition refers to the process in which automatic 'prepotent' responses are withheld to service a competing goal(s) (Verbruggen & Logan, 2008). For example, someone with the goal of reducing or ceasing their alcohol use may experience a strong craving for the substance, especially when faced with triggers such as being offered a glass of wine at a party. In such a situation, the individual must exercise response inhibition, continually resisting the urge to reach out and take the offered glass of wine, despite a strong desire and habitual tendency to accept such offerings. Response inhibition is commonly assessed via the Stop Signal Task (SST; Eagle & Robbins, 2003), which measures an individual's ability to interrupt or inhibit a pre-planned motor response (such as pressing a button) when presented with an unexpected stop signal (either a visual or auditory cue). One's ability to inhibit this prepotent response is quantified via their Stop Signal Reaction Time (SSRT), which is conceptualised as the mean time needed to successfully suppress an initiated "go" response. A longer SSRT indicates poorer inhibitory control. Neurobiological models of addiction increasingly recognise a general deficit in inhibitory control as a vulnerability marker for the development of addictive behaviours (Meng et al., 2015; Verdejo-García et al., 2008). Further, response inhibition as measured by the SST has been found to be associated with a range of addictive behaviours, including alcohol misuse, stimulant use, opioid dependence, problem gambling and PUI (Ioannidis et al., 2019; Lee et al., 2019; Smith et al., 2014).

### 1.6.4 Performance monitoring

Performance monitoring is the process in which an individual is able to monitor their actions and their subsequent consequences (Franken et al., 2018). Performance monitoring involves processing environmental feedback and errors associated with unexpected outcomes, and is important for flexible adaptation of behaviour (Ferdinand & Czernochowski, 2018). For example, error detection is important for insight into addiction problems; that is, catching a behaviour, such as driving to the liquor store, and encoding that this is not aligned with your goal of not drinking alcohol that day. Poor ability to monitor the consequences of engaging in addictive behaviour, or actions that facilitate addictive behaviour, can lead to a cycle of continued harmful actions reinforcing the

addictive behaviour. Performance monitoring can be assessed using the Error Awareness Task (EAT; Hester et al., 2005), a motor Go/No-go response inhibition task in which awareness of commission errors (i.e. responding on No-go trials) are assessed after each trial. The primary output metric of this task is the EAT score which represents the error awareness percentage across all commission errors made during the task. Poorer performance monitoring is associated with both substance dependence and some non-substance addictions (i.e. gambling, excessive eating and internet use; Franken et al., 2018; Luijten et al., 2014). However, it should be noted that this evidence is predominantly derived from neuroimaging findings (e.g. changes in amplitude of electroencephalography signal), not differences in cognitive task performance.

### 1.6.5 Goal selection/updating

Goal selection/updating are core components of cognitive control, referring to the process of choosing among potential actions, behaviours or outcomes and refreshing or updating information or cognitive representations related to specific outcomes of these actions or behaviours (NIMH, 2023). For example, an individual may wish to reduce their alcohol consumption with the goal of improving their mental and physical health (goal selection). They will then employ strategies to achieve this goal, often encountering challenges along the way which requires maintenance of the goal via flexible adaptation of these strategies (updating) in response to changing contexts. Goal selection/updating can be assessed using task switching paradigms that require the individual to switch between different rule sets such as the Category Switch Task (CST; Friedman et al., 2008). In this task, individuals are required to flexibly transition between different rule sets based on prompts. Each trial in the CST begins with the presentation of a word (e.g., "rabbit"). Depending on the trial type, signified by a cue presented above the word stimulus, participants must make one of two possible responses: determining whether the word describes something that is alive; or whether it is larger or smaller than a basketball. The presence of a heart icon signals that the trial necessitates a response regarding the stimulus' vitality, while trials with an arrow icon prompt individuals to respond based on the stimulus' size. A switch cost is calculated representing the difference in reaction time between trials that require the individual to switch rules from the previous trial and trials that did not. A larger

switch cost indicated poorer flexibility of goal selection/updating. Poorer goal selection/updating has been observed in individuals with problematic gambling (Lee et al., 2019; Odlaug et al., 2011), however evidence for these deficits in substance use disorders is inconsistent (Morris & Voon, 2016). Of the limited investigation into AE, there is no evidence for goal selection/updating deficits associated with AE (Iceta et al., 2021), and this cognitive domain has yet to be adequately evaluated in PUI (Ioannidis et al., 2019).

### 1.6.7 Flexible updating

Flexible updating refers to the neurocognitive process of adapting and revising information held in working memory to align with changing environmental conditions (Kessler & Meiran, 2008). Flexible updating promotes successful self-regulation by replacing outdated information with newly acquired knowledge, as well as accessing information in memory that may discourage addictive behaviours, thereby supporting goal-oriented decision-making and shielding against disruptive urges or temptations (Bledowski et al., 2010; D'Esposito & Postle, 2015; Hofmann et al., 2009). For example, individuals who are trying to reduce or cease a particular behaviour are required to use flexible updating to hold their commitment to sobriety in mind. Flexible updating can be assessed using tasks that manipulate working memory load, such as the N-back Task (Jaeggi et al., 2010). The N-back task is a go/no-go task that increases in difficulty, modulated by increasing interference and working memory load (Kirchner, 1958; Mackworth, 1959). In this task participants are presented with a sequence of letters. In the 0-back trials, participants are asked to respond when the stimuli presented is the letter "M". In 1-back trials participants are asked to respond when the letter is the same as the letter that proceeded it. In 2-back trials participants are asked to respond when the letter is the same as the one presented two trials before, and so on. The primary outcome metric of this task is the parametric measure of sensitivity value indicating the individual's ability to distinguish targets from non-targets. Higher values indicate better working memory performance and thus more flexible updating. Substance abuse has consistently been associated with deficits in working memory (Yücel et al., 2007). PUI and pathological gambling have both been found to be associated with impaired flexible updating (Zhou et al., 2016).

### 1.6.8 Reward valuation

Reward valuation is the process of evaluating the benefits of a reward in the context of external information, such as a social context, prior experiences and values (NIMH, 2023). It is influenced by stimulus characteristics, pre-existing biases, deprivation states, as well as learning and memory. For example, when deciding whether to have another alcoholic beverage on a night out, one may weigh up the value of immediate reward gratification of consuming that beverage now against the value of not feeling the effects of a hangover and being able to attend a social commitment the following day. Similarly, individuals who smoke tobacco may prioritise the immediate and certain rewarding effects of smoking a cigarette over the long-term health impacts of smoking, which are temporally unpredictable but further away. Reward valuation is a multifaceted process, that can be assessed using a variety of neurocognitive tasks. Two prominent tasks in the literature are the Delay Discounting Tasks (DDT; Kirby et al., 1999) and the Balloon Analogue Risk Task (BART; Lejuez et al., 2002).

The DDT (Kirby et al., 1999) measures temporal discounting, the tendency to seek a reward that is of lower value but quicker to obtain compared to a reward of higher value but takes more time to obtain. The individual must make a series of choices, each between two hypothetical reward options, a smaller reward now or a larger reward at some point in the future (e.g. "Would you prefer $15 today or $35 in 13 days"). A *k* function or value is calculated according to the individual's responses and represents the indifference point at which both rewards become equal in subjective value. Higher *k* values indicate steeper discounting, or a preference for more immediate gratification. Delay discounting has been found to be robustly associated with continuous measures of substance and non-substance addiction severity, as well as quantity/frequency of addictive behaviours (Amlung et al., 2017; Weinsztok et al., 2021).

The BART (Lejuez et al., 2002; Pleskac et al., 2008) evaluates one's risk-taking propensity under ambiguous situations. Individuals are presented with a series of balloons that can be inflated to potentially earn monetary rewards. The larger the balloon, the higher the potential earnings. However, each balloon will burst at a certain threshold, resulting in all potential earnings being lost. The task assesses reward valuation in the context of uncertainty, requiring one to evaluate the benefit of

inflating the balloon considering the probability of that balloon bursting. The primary outcome metric of this task is the unadjusted score which represents the mean pre-committed pumps averaged across all balloons (regardless of whether they burst or not; Pleskac et al., 2008). Higher mean pre-committed pumps indicate a propensity to value riskier choices. More risky decision-making assessed using the BART is associated with greater substance use (Hanson et al., 2014; Hopko et al., 2006), as well as substance and non-substance addictive behaviours (Lejuez et al., 2002). However, a recent evaluation of the relationship between BART performance and alcohol use specifically showed the findings are not always replicated (Canning et al., 2022).

### 1.6.9 Reward learning

Reward learning describes the process in which one acquires information that predict positive outcomes, leading to the adaption of behaviour in response to a novel reward (NIMH, 2023). This process encompasses probabilistic and reinforcement learning, reward prediction error (RPE), habit development (NIMH, 2023), as well as stimulus-outcome (Pavlovian) learning. In the context of addiction, an individual learns to associate the addictive behaviour with pleasurable sensations. Further, through classical conditioning, addiction-related cues or environments, such as the sound of a beer can opening, or walking past a bar, even in the absence of a closely followed addictive behaviour, become rewarding in and of themselves and act as powerful craving-inducing cues. RPE is also an important component of reward learning as it encodes the difference between the expected and the actual reward or a particular action (Glimcher, 2011). This can result in caching, in which an individual learns to associate a specific action with a given situation, and over time it is learned that in *this* specific situation it is best to do *that* specific action (Redish et al., 2008). Habitual decision-making can form when one relies on cached values of past RPE(s) instead of updating their expectations with new information (Daw et al., 2011).

It is difficult to isolate each aspect of reward learning, given their interactions and contributions to one another. Instead, tasks such as the Sequential Decision-Making Task (SDT; Kool et al., 2016, 2017) have been developed to evaluate reinforcement-based reward learning comprehensively, encompassing probabilistic learning, RPE, and habit development. The SDT is a

sequential choice task that measures the tendency to rely on model-based (goal-directed) versus model-free (non-goal-directed) learning. The task requires the individual to choose between two stimuli with the knowledge that each of these stimuli will either have a high (70%) or low (30%) chance of leading to a second stage associated with a reward payoff. The magnitude of this payoff changes gradually over time. People who rely more on their past experiences tend to switch their choices (model-free) if the reward payoff is low in the most recent trial. But those who think more about the overall strategy (model-based) know that sometimes probability can lead to no rewards, so they stick with their choice, even after not getting a reward, because it may still be the best (long-term) strategy for the task overall. A more detailed description of the task is published in Kool and colleagues (2016, 2017). The primary outcome metric of this task is mixing weight, which is calculated and represented by one's propensity to rely on model-free vs model-based decision-making.

Pavlovian reward learning process can be evaluated using the Value-Modulated Attentional Capture Task (VMAC; Albertella, Pelley, et al., 2019; Le Pelley et al., 2015). This task measures the influence of learned value, through stimulus-outcome associations, on attentional capture (Le Pelley et al., 2016). The VMAC is a Pavlovian learning task that assesses one's ability to direct their attention in a goal-directed manner, as opposed to biasing attention toward reward-related cues that run counter to the task's objectives. In this task, participants look for a diamond among circles and must respond as fast as they can to earn points. The circles are sometimes coloured one of two colours. One of the colours signifies high-value trial, meaning correct performance will earn a high number of extra points. Some individuals will respond slower to the diamond when the high-reward colour is present compared to when the low-reward colour is there. This means that the promise of a big reward sometimes distracts them and slows them down, even though attending to the cue is counterproductive. The primary outcome metric of the VMAC task is the difference in response time between high and low value distractor trials. Longer reaction times indicate more reward-related attentional bias.

Performance on both the SDT and the VMAC tasks have been linked to addictive behaviours. Majority of the SDT literature has focused on alcohol use, showing that less adaptive reward or

model-based learning is associated with increased binge drinking (Doñamayor et al., 2018) and more adaptive reward learning may be linked to treatment outcomes such as relapse and abstinence (Sebold et al., 2017; Voon et al., 2015). However, model-based reward-learning has also been observed in relation to BED and methamphetamine dependence (Voon et al., 2015). Studies looking at VMAC have also shown reward-related attention bias being associated with compulsive addictive behaviours (Albertella, Pelley, et al., 2019).

## 1.7 Optimising measurement of neurocognition

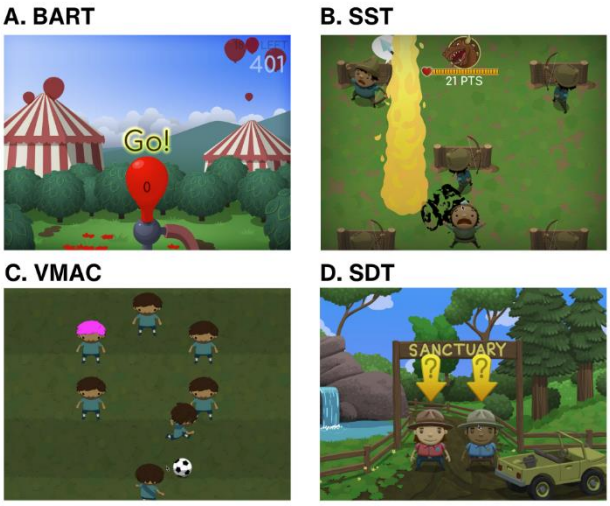
When collecting neurocognitive data, maintaining motivation of the participant is crucial to obtaining optimal task performance (DeRight & Jorgensen, 2015). Traditional neurocognitive assessment paradigms are frequently borrowed from those typically administered in conjunction with neuroimaging. They are often lengthy, repetitive, and effortful, making participants highly susceptible to boredom, mental fatigue, and general disengagement. Gamification has been proposed as a potential solution (Lumsden et al., 2016), incorporating game-like elements to traditional task paradigms such as utilising engaging visual displays, adding a narrative or utilising a points and scoreboard system. Gamified neurocognitive tasks have been shown to be more engaging and maintain positive affect during the course of a task (Bernecker & Ninaus, 2021). Gamification has already been successfully employed in a number of neurocognitive assessment paradigms, training tools and eHealth applications (Kirk et al., 2021; Sardi et al., 2017; Verdejo-Garcia et al., 2021). However, introducing game-like elements to such assessments does not come without risks, with the potential of increasing stress and/or cognitive demand which may impact performance (Katz et al., 2014). As such, it is important when adopting gamified neurocognitive assessments to interpret findings judiciously, and within the context of the additional gamified elements.

### 1.7.1 The BrainPark Assessment of Cognition application

The BrainPark Assessment of Cognition (BrainPAC; Lee et al., 2023) app was developed as a comprehensive and purpose-built assessment tool for addictions informed by the Delphi consensus study mentioned earlier. BrainPAC consists of a suite of gamified gold-standard neurocognitive paradigms including: the Stop Signal Task (SST; Eagle & Robbins, 2003), Balloon Analogue Risk

Task (BART, stretch variant; Lejuez et al., 2002), Value-Modulated Attentional Capture Task (VMAC; Albertella, Pelley, et al., 2019; Le Pelley et al., 2015), and Sequential Decision-Making Task (SDT; Daw et al., 2011). Each task taps into one or more neurocognitive domains identified as key to addiction (Yücel et al., 2019) (see Figure 1).

**Figure 1.** The BrainPAC application.




*Note: A) Balloon Analogue Risk Task (BART): risky decision-making, reward valuation; B) Stop Signal Task (SST): response inhibition; C) Value-Modulated Attentional Capture (VMAC) Task: reward-related attentional capture, reward learning; D) Sequential Decision-Making Task (SDT): reward learning.*

The BART game is presented as a carnival game in which the player must inflate a series of balloons to earn money. The larger the balloon, the more money that can be earned, but each balloon could burst at any moment (pseudorandomised burst threshold, with mean burst point set at 64 pumps), resulting in all the potential earnings on that trial being lost. The BrainPAC BART showed convergent validity with the standard BART paradigm (mean pre-committed pumps: $r = 0.58$, $p < .001$) and is positively associated with compulsive ($r = 0.23$, $p = .002$) and impulsive ($r = 0.15$, $p = .048$) behaviours (Lee et al., 2023).

The BrainPAC SST is set on a medieval battlefield in which the player is helping their fellow countrymen slay a dragon that has laid siege on their village. Players pass arrows to two archers (left/ right; go signal), as fast as they can while avoiding intermittent dragon fire (stop signal), to shoot

arrows and defend their village. The BrainPAC SST showed moderate convergent validity with the standard SST paradigm (SSRT: $r$ = 0.37, $p$ =.001) and adequate test-retest reliability (ICC = 0.72, $p$ <.001; Lee et al., 2023).

The BrainPAC VMAC uses a soccer game format in which a player must scan the pitch and pass the ball (left/right) to their teammate (signified by the same patterned jersey among visually similar distractor jerseys). The faster an accurate pass, the more points earned. Some trials have opposition players with different, bright hair colours acting as high and low distractors, indicating the potential reward value of that trial. Accurate and fast passes on high-value reward distractor trials earn ten times the points that can be earned on low-value distractor trials. The BrainPAC VMAC showed significant (albeit low) convergent validity with the standard VMAC paradigm (VMAC RT score: $r$ = 0.18, $p$ =.04; Lee et al., 2023).

The SDT is delivered in the form of an animal rescue game. A dangerous storm hits a wildlife sanctuary, and all the animals escape in fright. On each trial, the player must search the rainforest or the farmlands to rescue the animals and bring them back to the sanctuary. On each trial the player selects a ranger to help with their search. There are two pairs of rangers, one ranger per pair will always go to the farmlands and the other will always go to the rainforest. The number of animals that can be found in each environment gradually changes across the task. This requires the player to learn which rangers travel to which environments, as well as behind which objects in those environments, and adaptively change their ranger choice as the number of animals change, optimising the number of animals that can be found. The BrainPAC SDT has been validated against behavioural measures, and more model-free decision-making has been found to be associated with more impulsive behaviours ($r$ = -0.26, $p$ <.001; Lee et al., 2023).

### 1.7.2 Additional tasks

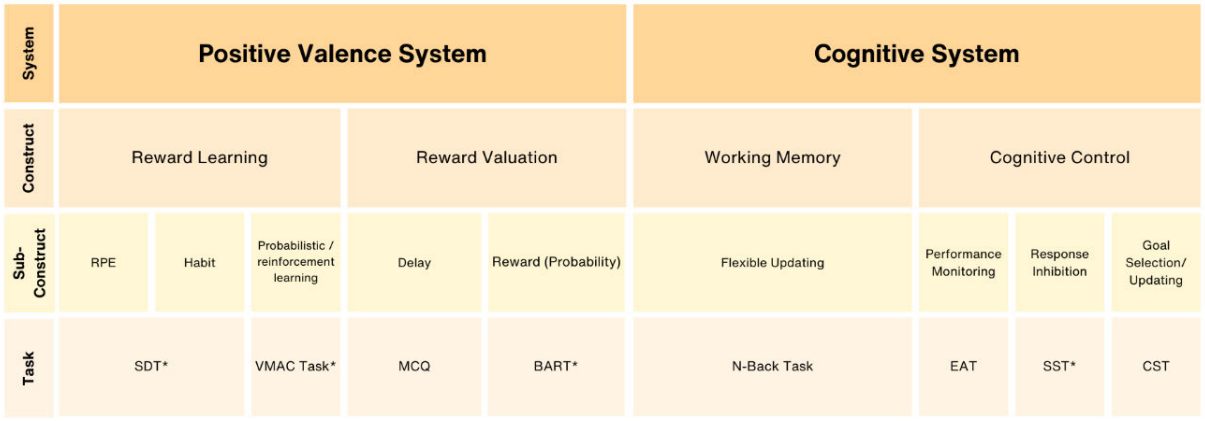
While the BrainPAC battery covers many of the neurocognitive constructs that are of interest to this thesis, there are several functions unassessed. Namely, performance monitoring, goal selection/updating, and flexible updating. Therefore, an additional three tasks were selected to cover these constructs (see Figure 2). The Error Awareness Task (EAT; Hester et al., 2007) was selected to

assess performance monitoring, the N-back Task with letter stimuli (Ragland et al., 2002) was chosen to assess flexible updating, and the Category Switch Task (CST; Friedman et al., 2008) was selected to assess goal selection/updating. While BrainPAC has a gamified version of a delay discounting task, I chose to use the original Monetary Choice Questionnaire (MCQ; Kirby et al., 1999) to assess temporal discounting instead. The BrainPAC DDT deviated significantly from the original MCQ in format and delivery, taking on the form of a Pac-Man game, in which the player must go through a maze to collect one of two coin options: a coin that was closer in distance but lower in value, or a coin that was higher in value but further away (so took longer to experientially to reach). The MCQ on the other hand (as described above), is a 27-item self-report questionnaire in which the individual is asked to make a choice between two hypothetical reward options, a smaller reward now or a larger reward at some point in the future. When comparing the k functions from both versions of the task, the BrainPAC DDT was not significantly associated with the MCQ, and steeper discounting was observed on the MCQ compared to the BrainPAC DDT (Lee et al., 2023). In essence, the gamified DDT was unable to replicate the "cost" associated with waiting for a larger reward as seen in the MCQ. That is, it was more costly to participants to hypothetically imagine waiting many days for monetary rewards, rather than expend additional seconds to earn more coins in the gamified paradigm. Further, when evaluating data obtained from the present thesis, preliminary analyses revealed the *k* function from the gamified DDT formed a bimodal distribution, while the *k* function from the MCQ displayed a normal distribution. This suggests individuals went into the gamified DDT with a predetermined strategy, opting to only go for the high-value (or low-value) coins on each trial. As such, this violates the assumption of determining *k* since each individual choice across the 27 items may not be independent decisions. Alternatively, the task may have been tapping into more than one function (e.g., temporal and effort discounting), which may explain the unexpected bimodal distribution.

An important consideration is that each of the neurocognitive tasks adopted in the present thesis are sensitive to multiple neurocognitive functions. Take the SDT for example, alongside reward learning, this task requires response inhibition when selecting the appropriate ranger, flexible updating to remember the ranger offering the most favourable outcome, and performance monitoring

to track the results of selections across consecutive trials. One method to disentangle the role of each of these functions and their relationship with addictive behaviours is to investigate performance on all tasks within the same model. This allows for a comprehensive analysis of the unique contribution of each specific function in predicting addictive behaviours.

**Figure 2.** Neurocognitive functions relevant to addiction assessed in the present thesis.




*Note: The figure is organised by RDoC construct categorisation, and including the tasks used to assess each function. RPE: Reward Prediction Error; SDT: Sequential Decision-Making Task; VMAC: Value-Modulated Attentional Capture; MCQ: Monetary Choice Questionnaire; BART: Balloon Analogue Risk Task; EAT: Error Awareness Task; SST: Stop Signal Task; CST: Category Switch Task. * Signifies gamified tasks taken from the BrainPAC battery, all remaining tasks were taken from the Inquisit assessment catalogue retrieved from https://www.millisecond.com.*

## 1.8 Demographic, clinical and trait-based factors

### 1.8.1 Demographic factors: Age and sex

When evaluating relationships between neurocognition and addictive behaviours several key demographic factors should be considered. Age is particularly important to adjust for when assessing neurocognitive processes. For instance, reaction time declines with age (Vallesi et al., 2021), as does flexible updating (Lugtmeijer et al., 2019), which are key processes that underpin many neurocognitive functions. A younger age also tends to be associated with heightened risky decision-making (Wilson et al., 2022), and a greater propensity to discount larger, later rewards for smaller, immediate gains (Bixter & Rogers, 2019). Sex differences have also been observed in some neurocognitive functions (Cross et al., 2011). For example, some studies have found females show

better response inhibition (Ribeiro et al., 2021; Sjoberg & Cole, 2018) and poorer selective attention than males (Stoet, 2010). By contrast, males tend to have a higher propensity to engage in risky behaviours (Charness & Gneezy, 2012; Pawlowski et al., 2008) and may make riskier decisions on the BART (Hunt et al., 2005). Further, males and females have been found to differ in their vulnerability to addiction (Quigley et al., 2021). Taking this into account, it is important to account for age and sex when investigating the relationship between neurocognition and addiction.

### 1.8.2 Psychological distress

Psychological distress (referred to in this thesis as stress) plays a crucial role in addiction (Ruisoto & Contador, 2019) and is associated with multiple addictive and compulsive behaviours (Albertella, Pelley, et al., 2019; Albertella, Rotaru, et al., 2021; Sepas et al., 2021). Stress can be conceptualised as an alteration in psychological homeostasis, accompanied by physiological arousal (e.g. increase heart rate, skin conductance, and perspiration) and neurobiological responses (Bohus et al., 1987), namely, activation of the hypothalamic-pituitary-adrenal (HPA) which acts as the body's primary stress response system. Chronic stress drives addictive behaviours through negative reinforcement and has long been established as a key factor associated with motivation to use rewarding substances, the development of addiction, and a primary contributor to risk of relapse (Burnatowska et al., 2022; Rodriguez et al., 2020; Sinha, 2008). Stress has also been shown to impact neurocognitive functions relevant to addiction. For example, stress promotes incentive salience of addictive behaviours and related stimuli (Ruisoto & Contador, 2019), impairs executive functioning (Ruisoto & Contador, 2019; Starcke et al., 2016), and impacts performance on tasks involving goal selection/updating (Renner & Beversdorf, 2010) and reward learning (Albertella, Pelley, et al., 2019). Accordingly, understanding the relationship between stress and both neurocognitive performance and addictive behaviours is important when trying to determine how neurocognitive processes predict addictive behaviours.

### 1.8.3 Trait impulsivity and compulsivity

Over and above neurocognitive functioning, it is well established that addiction is associated with related personality traits. Impulsivity is a tendency to react to a situation or trigger in a rapid,

unplanned and/or reward driven manner without considering the consequences of one's actions (Moeller et al., 2001). Impulsivity is a multifaceted construct, encompassing both personality trait-like domains, such as sensation seeking, lack of planning and emotion-driven rash behaviour (Cyders et al., 2014) as well as motor inhibition and impulsive action, or an inability to delay satisfaction (Brunner & Hen, 1997). Neurocognitive processes such as response inhibition and reward valuation (i.e. temporal discounting) are associated and overlap with impulsivity. While these facets of impulsivity are linked, trait-based measures and neurocognitive measures appear to tap into distinct aspects of what can be considered multidimensional constructs (Duckworth & Kern, 2011; Eisenberg et al., 2019) and may contribute uniquely to addictive behaviours (Christiansen et al., 2012).

Compulsivity describes automatic repetitive behaviours or cognitive processes that are inappropriate to the situation, have no clear connection to the overall goal and often result in negative consequences (Dalley et al., 2011). Compulsivity is also often accompanied by feeling of being compelled to perform an action or becoming stuck engaging in an action until the task is completed "just right" (Sica et al., 2015). Compulsivity has been shown to be associated with substance and non-substance related addictive behaviours (Forsén Mantilla et al., 2022) and is a hallmark feature of the transition from problematic use to addiction. Like impulsivity, compulsivity is a multidimensional construct (Tiego et al., 2023), and can be assessed via both trait scales and neurocognitive tasks. Several neurocognitive processes have been proposed to underpin compulsivity, such as task/attentional set shifting, attentional bias and habit learning (Fineberg et al., 2010; van Timmeren et al., 2018). While these functions have been found to correlate with trait-based measures (Albertella et al., 2020), they capture distinct aspects of compulsivity. For example neurocognitive tasks capture negative outcome expectancy driving compulsive behaviour, which can be evaluated using trait scales (Chamberlain & Grant, 2018; Tiego et al., 2023). Considering the addition of self-report trait scales alongside neurocognitive functions allows for us to determine the unique predictive value of neurocognitive processes over and above trait-based measures.

## 1.9 The importance of assessing neurocognitive constructs in conjunction

There is a lack of empirical evidence regarding how different neurocognitive domains are independently predictive of addictive behaviours since most studies to date focus on individual neurocognitive measures in isolation. This is problematic as the neurocognitive constructs that are thought to underpin addiction are correlated with one another and only by including a comprehensive battery of measures are we able to delineate unique contributions. This is a particularly critical area for investigation given both executive control and reward-related neurocognitive functions interact and in many cases overlap (Criaud & Boulinguez, 2013; Ridderinkhof et al., 2004). For example, reward valuation functions such as delay discounting tap into multiple neurocognitive processes. Primarily, delay discounting requires a reward valuation assessment, evaluating the cost versus benefit of immediate rewards against long-term objectives. This choice also relies on working memory functioning (Bobova et al., 2009; Shamosh et al., 2008), which allows individuals to keep important information in mind when making the decision. For example, remembering outcomes of previous similar decisions, and factoring in how each choice option matches up to their current goals.

## 1.10 The importance of a transdiagnostic approach

Taking a transdiagnostic approach by examining a spectrum of addictive behaviours is crucial to studying addiction. Neurocognitive models of addiction, characterised by an imbalance between a weak cognitive control system and hyperactive reward system (Bechara, 2005; Koob & Volkow, 2010; Verdejo-Garcia et al., 2018; Yücel et al., 2019), have been applied to both substance and non-substance-related addictive behaviours. Accordingly, it has been suggested that a common set of neurocognitive functions typically underlie addictions across various diagnostic categories (Yücel et al., 2019). By including a spectrum of addictive behaviours, we can discern whether various types of addictive behaviours share similar or distinct neurocognitive mechanisms. Addictive behaviours also tend to co-occur. For example, problematic gambling co-occurs with higher prevalence of nicotine dependence, substance use disorder, alcohol use disorder and PUI (Cunningham-Williams et al., 1998; Grant et al., 2006; Lobo & Kennedy, 2009; Lorains et al., 2011; Petry et al., 2005; Tozzi et al., 2013). PUI has also been found to be associated with increased harmful alcohol use (Hou et al., 2014;

Yen et al., 2009), as well as cannabis (Korkeila et al., 2010), and other substance use (Siomos et al., 2012). Notably, the strength of correlations between the aforementioned problematic behaviours varies depending on the combination of behaviours observed. Variations in comorbidity risk among behaviours may suggest significant distinctions in their underlying neurocognitive risk profiles. Identifying where different addictive behaviour types have shared or distinct neurocognitive mechanisms will progress etiological understanding of these behaviours, aid in the identification of individuals who might be susceptible to multiple forms of addiction, and facilitate the development of more targeted assessments and treatments.

## 1.11 The importance of longitudinal evaluations

Whist cross-sectional studies can provide valuable insights into the neurocognitive processes associated with different addictive behaviours, prospective/longitudinal designs that account for baseline addictive behaviour are required to identify those functions that have predictive value above and beyond current use. Addictive behaviours, whether they involve neurotoxic substances or are non-substance-related, impact reward regions of the brain evoking neurobiological changes that have the potential to impair neurocognitive functions (Clark et al., 2019; Fernández-Serrano et al., 2011; Weiss & Koob, 2001). It is therefore difficult to disentangle whether changes in neurocognitive functions predispose an individual towards addictive behaviours or whether repeated engagement in that addictive behaviour is what is driving neurocognitive changes. There exists a probable bidirectional relationship between neurocognition and addiction. As such, longitudinal study designs are essential to establishing predictive mechanisms of addictive behaviours.

## 1.12 Thesis aims

The present thesis has three core aims. First, to systematically synthesise and critically evaluate the current literature supporting the role of RDoC informed neurocognitive constructs predicting addiction and treatment-related outcomes in longitudinal studies (Chapter 2). Second, to empirically evaluate the neurocognitive cross-sectional correlates of addictive behaviours dimensionally in a general population sample (Chapter 3). Third, to empirically test whether these neurocognitive constructs predict addictive behaviours over a 6-month period using a longitudinal

study design in a general population sample (Chapter 4). All empirical chapters evaluate neurocognition using a comprehensive assessment battery selected specifically to measure constructs key to addiction, as well as in the context of other key demographic, clinical and trait-based factors (age, sex, stress, trait impulsivity and compulsivity).

*Chapter 2*

# A systematic review of longitudinal studies

Neuroscience and Biobehavioral Reviews 152 (2023) 105295

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

# Neurocognitive predictors of addiction-related outcomes: A systematic review of longitudinal studies

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ARTICLE INFO

*Keywords:*
Neurocognition
Cognition
Addiction
Substance use
Behavioral addiction
Relapse
RDoC
Cognitive control
Reward
Longitudinal
Prospective

ABSTRACT

It is well-established that addiction is typically associated with a distinct pattern of neurocognitive functioning with a consensus that it is typified by impaired top-down executive control and aberrant risk-reward processing. Despite a consensus that neurocognition plays an important role in characterizing and maintaining addictive disorders, there is a lack of systematic, bottom-up synthesis of quantitative evidence showing that neurocognition predicts addictive behaviors, and which neurocognitive constructs have the best predictive validity. This systematic review aimed to assess whether cognitive control and risk-reward processes as defined by the Research Domain Criteria (RDoC) predict the development and maintenance of addictive behaviors specifically, consumption, severity, and relapse. The findings from this review expose the substantial lack of evidence for neurocognition predicting addiction outcomes. However, there is evidence that suggests reward-related neurocognitive processes may be important for the detection of early risk for addiction, as well as a potentially viable target for designing novel, more effective interventions.

## 1. Introduction

Addiction is a chronic, remitting-relapsing mental health disorder defined by compulsive patterns of behavior (i.e. drug seeking, gambling), which persist despite harmful consequences (NIDA, 2020). Addictive behaviors are typically associated with a distinct pattern of neurocognitive functioning (for reviews see: Bechara, 2005; Goldstein and Volkow, 2011; Smith et al., 2014; Verdejo-García et al., 2008; Volkow et al., 2010; Yücel and Lubman, 2007) characterized by dysregulation of reward processes and decreased capacity to exert cognitive-control (Volkow and Morales, 2015). However, the specific mechanisms that precede the development of addiction and influence treatment outcomes are complex. Clarifying these mechanisms would inform the detection of risk and mechanism-targeted, personalized intervention.

Numerous models of addiction have been proposed over the years (Goldstein and Volkow, 2002, 2011; Redish et al., 2008; Robinson and Berridge, 1993; Verdejo-García and Bechara, 2009), all of which agree that addiction is associated with a hyperactive drive/reward salience network, which is paired with reduced executive functioning, or cognitive control over behavior. Behavioral disinhibition and reward-driven decision-making have also been put forward as key vulnerability markers for the development of substance use disorders (SUD) (Casey and Jones, 2010; Iacono et al., 2008; Verdejo-García et al., 2008), and predictive of treatment relapse (Domínguez-Salas et al., 2016). These cognitive functions are underpinned by neurobiological substrates, specifically corticostriatal projections and dopaminergic reward-related activity (Haber, 2016; Schultz, 2011; Shepherd, 2013). The corticostriatal pathway is crucial for higher-order executive functions including decision-making, working memory, and cognitive flexibility (Haber, 2016; Vaghi et al., 2017; Westbrook et al., 2021). Mesolimbic dopaminergic neurotransmission regulates reward-related functions such as reinforcement learning, motivation and goal-directed behaviour (Chong, 2018; Schultz, 2007, 2011; Schultz et al., 1997). Functional changes in corticostriatal circuitry and disruptions in mesolimbic dopamine release are associated with impairments in executive control and reward-related neurocognitive processes thought to contribute to the development and maintenance of addictive

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https://doi.org/10.1016/j.neubiorev.2023.105295
Received 3 March 2023; Received in revised form 9 June 2023; Accepted 23 June 2023
Available online 28 June 2023

behaviors (Ersche et al., 2020; Koob, 2001; Koob and Volkow, 2010). For a more detailed review of the neurobiological processes underpinning cognitive changes driving addiction, please see Koob and Volkow (2010). While current models of addiction elegantly articulate the general deficits associated with and predictive of addiction, it is still unclear which specific, measurable neurocognitive constructs drive these deficits. For example, a hyperactive motivational/reward salience network is associated with several reward-related neurocognitive functions (e.g. attentional bias, reward anticipation, reward valuation). Similarly, top-down cognitive control processes can be broken down into multiple specific executive functions (e.g. response inhibition, response selection, goal selection/updating). Isolating and interrogating the role of these constructs/subconstructs will better inform the mechanisms that underpin current addiction models.

Currently, there is a lack of standardized and validated methods for assessing vulnerability to addiction or determining treatment response (Yücel et al., 2019). Despite our growing understanding of the neurocognitive changes associated with addiction (Goldstein and Volkow, 2002, 2011; Redish et al., 2008; Robinson and Berridge, 1993; Verdejo-García and Bechara, 2009), there is a need for more comprehensive assessment frameworks that take into account these processes. Such assessments would enable the identification of early risk factors, monitoring of neurocognitive changes, and provide targets for early intervention. However, to develop these assessments, it is essential to gain a deeper understanding of the specific neurocognitive mechanisms involved in addiction.

For the past decade, there has been a surge of research into non-substance addictive disorders. Despite ongoing debate of whether compulsive behaviors should be classified as addiction (Chamberlain et al., 2016; Kardefelt-Winther et al., 2017) the similarities in their underlying mechanisms are evident (Grant et al., 2013; Rømer Thomsen et al., 2014). Behavioural addictions, such as pathological gambling, exhibit hallmark features of substance use disorders such as altered reward and motivational functions as well as diminished cognitive control (Brand et al., 2021; Goudriaan et al., 2014). Unlike previous reviews that have solely examined substance use (Dominguez et al., 2016), our objective in this review is to explore trans-addiction predictive mechanisms, with a focus on evaluating neurocognitive functions that serve as common risk indicators for addiction, regardless of the type of addiction.

Describing the neurocognitive mechanisms that underpin addiction is hindered by the lack of consistent language in the field. Similar constructs are often labelled with different names (e.g. delay discounting as 'choice impulsivity' versus a subconstruct of reward valuation, 'delay'), creating inconsistencies and confusion. The Research Domain Criteria (RDoC: Insel et al., 2010) provides an evidence-based framework to standardize existing findings, offering a comprehensive and evolving list of neurocognitive constructs that have neurobiological correlates. With the goal of synthesizing expert opinion on the key neurocognitive constructs and their associated paradigms pertinent to addiction, an international panel of experts in addiction reviewed the National Institute of Mental Health (NIMH) Research Domain Criteria (RDoC; Insel et al., 2010) and agreed upon seven key constructs. The Delphi concluded that response inhibition, response selection, reward valuation, reward expectancy, reward learning and action selection were the most crucial drivers of both a vulnerability to and chronicity of addiction, with habit and a new construct they termed 'compulsivity' most important for chronicity (Yücel et al., 2019). This list of addiction-specific RDoC domains serves as a compelling foundation for empirical investigation and underlies this review. One limitation of the Delphi process is that it is constrained by the diversity of experts involved. As such, a systematic, bottom-up synthesis of quantitative evidence is needed to corroborate the Delphi suggestions. Further, since the Delphi study was published, NIMH has updated their construct matrix. To make sense of the heterogeneity in the field of neurocognition and addiction, this review has adopted RDoC terminology and is structured according to the domains identified in the latest iteration of the domain criteria (NIMH: RDoC Snapshot, 2022).

The aim of this review was to identify and evaluate the evidence for the neurocognitive mechanisms that predict the development and maintenance of addictive disorders, as well as addiction treatment outcomes. The outcomes of interest were consumption, the severity of addictive behaviour and/or related problems, diagnosis of an addiction disorder, relapse, and treatment-related clinical change. These outcomes were selected to represent the stages of addiction (consumption through to post-intervention relapse). We acknowledge that this is not an exhaustive list of possible outcomes, however, we chose to select the most common outcomes that studies were most likely to include. Understanding the mechanisms that predict each of these addiction-related outcomes is important for the detection of risk and relapse, treatment matching based on prognosis and identification of treatment and early intervention targets.

## 2. Methods

The aims and methodology of this review were pre-registered via PROSPERO [CRD42020181560]. While the present review was initially intended to be a meta-analysis, we discovered the way studies defined and measured addiction-related outcomes in the literature was too heterogenous to perform an analytic evaluation. As such, a systematic review without meta-analysis was deemed more appropriate. Literature searches were performed using PubMed, PsychInfo and Web of Science (core collection) databases [16/05/2022], as well as reference lists of retrieved articles and relevant past reviews. The search included human studies written in English within the past 20 years. It would be ambitious to include search terms for all 49 RDoC neurocognitive domains, as such, we reduced our domains of interest to those identified by expert consensus as relevant to addiction (Yücel et al., 2019). Search terms were selected to include these key neurocognitive domains of interest:

"reward valuation", "expectancy", "action selection", "risk taking", "reward learning", "compuls* ", "habit", "reinforcement", "reward", "incentive salience", "inhibition", "impuls* ", "response selection", "decision making", "delay discounting", "set shifting", "task switching", "shifting", "goal selection", "goal maintenance", "goal updating", "goal representation", "performance monitoring", "goal performance monitoring", "behavior monitoring".

Outcomes of interest:

"addict* ", "relapse", "use", "treatment", "outcomes", "abstinence", "severity", "dependence", "abuse".

Key behavioural domains:

"heroin", "THC", "amphetamine", "cocaine", "stimulant", "marijuana", "cannabis", "internet", "alcohol", "gambling", "food", "methylenedioxymethamphetamine", "lysergic acid diethylamide", "LSD", "ketamine".

Additional search terms were included for study methodology: *"longitudinal", "prospective",* and exclusionary terms were also applied: *"review", "meta-analysis", "acute", "trait"*.

### 2.1. Selection criteria

Inclusion criteria were: 1) longitudinal study design, in which the addiction outcome measured was either not present at baseline (e.g. substance use had not commenced) or controlled/accounted for in analyses (e.g. using a change score or including a baseline measure of the addiction-related outcome as a covariate in the analyses); 2) all quantitative analysis methods were accepted as long as neurocognition was entered as a predictor variable and addiction outcomes were the dependent variable (see Table 2 for comprehensive list of all included inferential statistics); and 3) cognitive constructs assessed fell under one

of the ten determined by the Delphi expert consensus as: moderately important, important or essential for addictions, achieved > 80% consensus, and were measurable using a behavioural task (details in supplementary material of Yücel et al., 2019). To ensure we included studies that evaluated neurocognition as a phenotype for addiction we did not impose strict age range criteria, thus including cohort studies that span childhood through to adulthood.

Studies were excluded if they solely used neuroimaging as the predictor of addiction outcomes. Studies that involved neurologically invasive procedures (e.g., rTMS), or interventions with direct impact on neurocognition (i.e., cognitive training) were also excluded from the review.

### 2.2. Outcome measures

The primary outcomes of interests were: consumption, addiction severity, addiction diagnosis, treatment relapse, and treatment-related clinical change. Consumption was defined as the frequency/quantity of the behaviour (e.g the average number of alcoholic beverages consumed in the past week, or the number of days an individual gambled in the past month), addiction severity was defined as the severity of addiction-related symptoms (e.g total scores from standard self-report scales), and diagnosis of an addictive disorder is defined as a clinical diagnosis either from a relevant assessment tool or by a medical/mental health clinician. Relapse was defined as any post-resolution/-intervention engagement in the addictive behaviour of interest, and treatment-related clinical change was defined as clinically relevant change during or post-treatment as per an evaluation by a mental health clinician.

### 2.3. Data extraction and synthesis

Publications were initially imported into Covidence (a systematic review management web-based platform) and were screened according to eligibility criteria in order of title, abstract, and full body text. Two independent researchers screened the studies at the full body text stage (EC, MB), discrepancies between the researchers were resolved in the first instance by discussion, and in the instance where agreement could not be made, a third independent researcher (RSCL) screened the study and a consensus was achieved by agreement of at least two of the three parties. Data was extracted using a pro forma developed by the authors in accordance with the Cochrane Consumers and Communication Review Group data extraction template. The template was piloted on ten randomly selected studies and refined before being applied to all extracted studies.

### 2.4. Risk of bias assessment

The SIGN Checklist for Cohort Studies was used to formulate a risk of bias assessment (SIGN Checklist for Cohort Studies, 2012). Cognition was assigned as the exposure variable, i.e. the variable that is predicted to have an effect on the outcomes of interest, and addictive behaviours. Additional criteria were added to the SIGN checklist to develop a more comprehensive and specific assessment tool for the cohort studies included in this review (cf. clinical trials originally intended). Examples of items included were "for the analyses in this paper, was cognition measured prior to the outcome(s) being measured?", "was a sample size justification, power description, or variance and effect estimates provided?", "were the outcome variables either not present at baseline, or controlled for in analysis?", "was missing data described and accounted for". See Appendix I for the completed SIGN Checklist. Two researchers completed the risk of bias assessment independently (EC, MB), any disagreement was resolved by a third independent researcher (RSCL). Following the SIGN Checklist for Cohort Studies guidance, an overall risk of bias judgement was made for each study (*unacceptable*, *acceptable*, or *high quality)*. The results from studies that received an unacceptable risk of bias rating are qualified in the context of their rating and specific methodological concerns are discussed where necessary.

## 3. Results

### 3.1. Search results

Database searches retrieved 2080 studies initially (Fig. 1) and 7 studies were retrieved from reference lists of retrieved articles. Of these, 1924 studies were excluded during the title and abstract screening. The most common reasons for exclusion were that the study was not longitudinal, did not include the neurocognitive measures of interest, and the primary predictor variable was task-based fMRI BOLD signal change, rather than behavioural metrics. Inclusion and exclusion criteria were then applied to the remaining 163 studies through full-text screening. Three main study types were identified: prospective studies that follow a cohort of adolescence or young adults in the general community (i.e., not necessarily engaging in addictive behaviours), longitudinal studies that assess general community members who are currently engaging in an addictive behaviour followed up over time, and treatment/intervention studies that assess neurocognition prior to delivery of intervention and report treatment-related outcomes (e.g., relapse, clinical change).

Of the 33 studies included in this review, a total of 18 different neurocognitive tasks were identified (for a description of each task please see Table 1). We organised the literature by RDoC construct, identifying three key domains evaluated: cognitive control, reward valuation, and reward learning. Each of these constructs were broken down into their sub-constructs. Literature evaluating cognitive control primarily focussed on response selection/ inhibition and goal selection/ updating. Literature evaluating reward valuation focussed on reward predictability and delay. Literature evaluating reward learning focussed on probabilistic and reinforcement learning and reward bias. Fig. 2.

Despite being associated with common neurocognitive systems and frequently discussed together, response selection and response inhibition are considered distinct processes (Bender et al., 2016). Response inhibition refers to the process in which automatic responses are withheld to service goal-directed behaviour (Verbruggen and Logan, 2008). By contrast, response selection involves an evaluation and choice of the appropriate action to take when confronted with a given stimulus (Bender et al., 2016). As such, this review will evaluate the literature involving response selection and response inhibition separately.

Given the goal of this review was to determine the trans-addiction cognitive predictors of addictive behaviours (i.e. mechanisms that may be relevant across all substance and non-substance addictive behaviours), we chose not to include studies that looked at neurocognition in the context of specific addiction-related cues (e.g. substance-use related

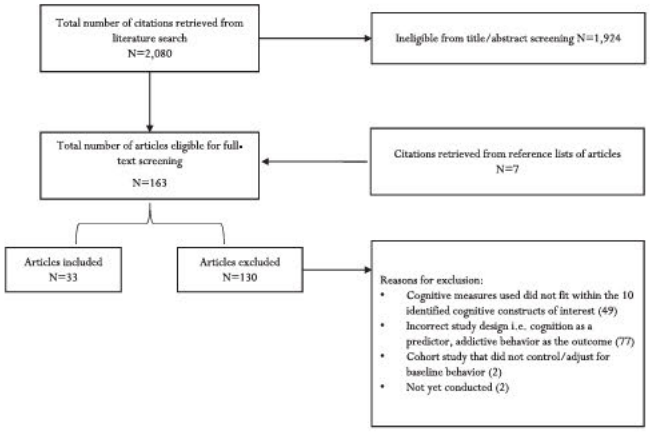



**Fig. 1.** Flow diagram of the review process.

**Table 1**
Description of neurocognitive tasks used in the literature.

| Task | Measures | Description | Key outcome variable (s) |
|---|---|---|---|
| Stop Signal Task (SST) (Logan and Cowan, 1984) | Response inhibition | On each trial, participants are presented with a stimulus (left or right facing arrow) and are required to respond via button press to that stimulus as quickly as possible. On a portion of trials, a stop signal will occur (e.g. an audio tone) which signifies to the participant that they must withhold their response. The delay between stimulus presentation and the stop signal is stair-cased so that the difficulty-level decreases or increases according to performance. | Stop Signal Reaction Time (SSRT): the estimated time it takes for a participant to inhibit their response. Longer SSRT indicates poorer inhibitory control. |
| Delay Task (Gordon and Mettelman, 1988) | Response inhibition | Participants are instructed to press a button, 'wait a while', and press the button again. Participants are not told how long they are required to wait. Those who wait longer than six seconds are rewarded with a point. If participants press the button too soon (before six seconds elapses), the trial resets and no points are earned. | Efficiency ratio: the number of responses longer than six seconds, divided by total responses. Larger ratios indicate better impulse control. |
| Go/No-Go Task (Fillmore et al., 2006) | Response inhibition | Participants are presented with a series of stimuli and must respond quickly when a 'go' stimulus is presented (i.e., pressing designated key), and withhold their response when a 'no-go' stimulus is presented (i.e., not pressing designated key). | Commission error rate: executing a 'go' response on a 'no-go' trial. Fewer errors signify better response inhibition. Omission error rate: withholding a response on 'go' trials. Higher number of errors suggest inattention. |
| Immediate Memory Task (IMT) (Dougherty et al., 2002) | Response inhibition | Participants are presented with a series of 5-digit numbers and instructed to respond when the current number is a repeat of the number in the preceding trial. Trials were equally divided into three trial types: target trials in which the 5-digit number was a repeated of that presented in the previous trial, foil trials in which every number differed from the previous trial, and catch trials in which only one of the 5 numbers differed from the previous trial. | Inhibition score: the ratio of catch trials to target trials responded to. Higher scores reflect poorer response inhibition. |
| Stroop Task (Stroop, 1935) | Response selection | The Stroop Task involves three subtasks. Task A (congruent task): Participants are presented with one of four words ('blue', 'green', 'red', or 'yellow'), and must read the words aloud as quickly as possible. Task B (congruent task): Participants are presented with colour patches (blue, green, red, or yellow) and must name the colour. Task C (incongruent task): Participants are presented with the same four words printed in incongruous colours (e.g., the word 'blue' printed in yellow ink) and must name the ink colour. | Interference score: the difference in average response time on incongruent trials and congruent trials (task A – task C). Higher scores indicate poorer cognitive control. |
| Delis–Kaplan Executive Function System Colour-Word Interference Test (D-KEFS CWIT) (Delis, Kaplan and Kramer, 2001) | Response selection Goal selection/ updating | The CWIT involves the same subtasks as the Stroop Task (above) but also includes an additional inhibition/ switching subtask. Participants are presented with a page containing the words "red," "green," and "blue" written in red, green, or blue ink. Half of these words are enclosed within boxes. Participants must say the ink colour for words not in boxes, and to read the word aloud (not name the ink colour) for words inside a box. | Interference score: same as above. Switching score (time/errors): time taken to complete the inhibition/ switching task; errors made during the inhibition/switching task. Faster completion times and fewer errors indicate better set shifting. |
| Shifting Attentional Set Visual Task (SAST) (De Sonneville, 1999) | Goal selection/ updating | Participants are presented with a red or green square, that jumps left and right, along a horizontal bar of | Shift attention score: computed by subtracting reaction time of compatible responses in Part 1 from reaction time of |

(continued on next page)

**Table 1** (*continued*)

| Task | Measures | Description | Key outcome variable (s) |
|---|---|---|---|
| | | grey squares. Part 1 (fixed compatible stimulus-response condition): If a green square jumps left, the participant must press the left button, and if it jumps right, they must press the right button. Part 2 (fixed incompatible stimulus-response condition): If a red square jumps left, the participant must press the right button and vice versa. Part 3 (random stimulus-response condition): A square will randomly jump left or right and will turn green or red. The participant must adjust their response based on the colour of the square, consistent with the rules learnt in the first two parts. | compatible responses in Part 3. Higher scores indicate poorer set shifting. |
| Wisconsin Card Sorting Task (WCST) (Heaton et al., 1993) | Goal selection/ updating | Participants are presented with stimulus cards, and are instructed to match the cards, but are not told how to match. Instead, they are told whether a match is correct or incorrect. After ten consecutive correct matches, the rule changes. The task involves six attempts to derive a rule. Testing continues until 128 cards are sorted. | Perseverative errors: the proportion of times a participant continues to use a previously successful sorting rule even after it is no longer valid. Higher scores indicate poorer set shifting. Non-perseverative errors: the proportion of times a participant makes an error in sorting that is not related to perseveration (i.e., not repeating a previously successful strategy). |
| Trail Making Test (TMT) (Reitan and Wolfson, 1985) | Goal selection/ updating | The task consists of two parts: Part A presents 25 circles numbered from 1 to 25 that are randomly distributed across a page (or screen). Participants are asked to connect the circles in numeric sequence starting from number 1 and finishing at number 25 as fast as they can. Part B presents 25 circles numbered 1–13 or lettered A to L that are randomly distributed across the page (or screen). Participants are required to connect the circles as quickly as possible alternating between numbers and letters (i.e. 1, A, 2, B, 3, C). | Time to complete part A: higher values represent poorer processing speed. Time to complete part B: higher values represent poorer flexibility. Time to complete part A – part B: higher values represent poorer flexibility accounting for processing speed. |
| Intra Extra Dimensional Shift Task CANTAB Cognitive Assessment Software (2019) | Goal selection/ updating | Participants are presented with two stimuli and are required to learn the rule that determines which stimuli is the correct one. After selecting the correct stimuli 6 consecutive times the stimuli and/or rule changes. This change first occurs intra-dimensionally (i.e. between shapes of the same colour) and then extra-dimensionally (i.e. between shapes and lines). The task has nine sets and ends either at the conclusion of the ninth set or after a participant fails to meet the learning criterion on 50 consecutive trials. | Set-shift errors: the number of errors made. Higher scores represent poorer flexibility. |
| Self-Ordered Pointing Test (SOPT) Petrides & Milner (2006) | Flexible updating | Participants are presented with a series of stimuli. On each presentation the same set of images are shown however their location varies randomly from trial to trial. Participants are required to point to a different item on each trial without pointing to one they have previously touched. | SOPT score: the mean proportion of error score across all trials. Higher scores indicate more errors and poorer flexible updating. |
| Delay Discounting Task / Monetary Choice Questionnaire (Kirby et al., 1999) | Reward valuation (delay) | Participants must respond to 27 questions, each requiring a choice between a smaller, immediate reward or larger, delayed reward, for example "Would you prefer $54 today, or $55 in 117 days?". A log transformed discounting rate is calculated according to the pattern of choices made by the participant across the questionnaire (or task). | Log *k*: higher values represent steeper discounting of delayed rewards and therefore a preference for smaller sooner rewards. |
| Delay Discounting Hypothetical Monetary | Reward valuation (delay) | Participants must choose between either an amount of money (between | Delay discounting score (range from 10 to 100): when reverse-scored, |

(*continued on next page*)

**Table 1** (*continued*)

| Task | Measures | Description | Key outcome variable (s) |
|---|---|---|---|
| Choice Task (Green et al., 1994; Khurana et al., 2017) | | $10 and $90) received immediately or $100 to be received in six months time. The immediate value starts at $50 and iteratively increases or decreases in $10 increments. | higher scores indicate greater delay discounting and therefore a preference for smaller sooner rewards. |
| Experiential Discounting Task (EDT) (Reynolds and Schiffbauer, 2004) | Reward valuation (delay) | Participants must decide between a delayed and probabilistic 30 cents or an adjusting immediate and for sure amount of money. Each block of trials required choices between a standard amount that is delayed (by 0, 15, 40 or 60 s), and probabilistic (35% chance of receiving) and an adjusting amount of money delivered immediately and certainly. The aim is to earn as much money as possible. Choosing the standard amount increases the amount to be won with the adjusting option for the next round. Choosing the adjusting option decreases the amount to be won with the adjusting option for the next round. | Indifference point: the point at which the standard and adjusting option amounts were considered at equal subjective value. Higher indifference points represent more temporal discounting. |
| Probability Discounting Task (Pooseh et al., 2018) | Reward valuation (probability) | Participants are required to make a choice between smaller certain rewards and larger uncertain rewards (PDG: probability discounting for gains), or a choice between a smaller certain loss and a larger uncertain loss (PDL: probability discounting for losses). The probability of receiving the larger reward/loss varies over consecutive trials. An indifference point is calculated to form a discounting rate ($k$) which is log-transformed. | PDG log $k$: the indifference point on gain trials. Higher values represent steeper discounting of probabilistic gains, a bias toward risk seeking for gains.<br>PDL log $k$: the indifference point on loss trials. Higher values represent steeper discounting of probabilistic losses, a bias toward risk seeking for losses. |
| Spatial Orienting Task (SOT) (Derryberry and Reed, 1994) | Reward learning | Participants are presented with cues: a blue arrow pointing up indicating an 'easy' target (resulting in positive feedback 75% of the time), or a red arrow pointing down, indicating a 'difficult' target (resulting in negative feedback 75% of the time). Participants must respond (via keypress) to these cues quickly. After keypress, participants receive a feedback signal: a blue arrow pointing up indicates a fast response (positive feedback), and a red arrow pointing down indicates a slow response (negative feedback). The task consists of four positive and four negative trial blocks. Participants must score as many points as possible. On positive blocks, participants win 10 points for fast responses and do not gain points for slow responses. On negative blocks, participants lose 10 points for slow responses and do not lose points for fast responses. | Attentional bias for reward (engagement): faster reaction times toward cues of expected gain than reaction times toward cues of expected non-gain. Lower values represent more bias. Attentional bias for reward (disengagement): slower disengagement from expected gain than from expected non-gain. Higher values represent more bias. Attentional bias for non-punishment (engagement): faster engagement toward cues of expected non-loss than expected loss. Lower values represent more bias. Attentional bias for non-punishment (disengagement): slower disengagement from expected non-loss than from expected loss. Higher values represent more bias. |
| Value Modulated Attentional Capture (VMAC) Task (Albertella et al., 2019; Le Pelley et al., 2015) | Reward learning | Participants must search for a diamond target among grey circles, with faster responses earning more points. One (non-target) circle is coloured, referred to as the distractor. The distractor colour influences reward magnitude, so one colour signals availability of a large reward (high-reward distractor), and the other colour signals a small reward (low-reward distractor). Although distractors signal reward magnitude, these are not the target stimuli: the diamond must be | VMAC score: reaction time for high-reward trials minus reaction time for low-reward trials, during the training phase. Higher scores represent slower responding in the presence of the high-reward distractor, thus more reward-related attentional bias. |

(*continued on next page*)

**Table 1** (*continued*)

| Task | Measures | Description | Key outcome variable (s) |
|---|---|---|---|
| | | found to receive the reward. | |
| Sequential Decision-Making Task (SDT) (Daw et al., 2011) | Reward learning | Participants must make an initial choice between two options. Choices on the first stage probabilistically lead to a second stage, where another choice between two options must be made, each of which is associated with a different chance of monetary reward. The choice of one first-stage option largely leads (70% of the time) to one of the two second-stage options, with this relationship fixed throughout 201 trials (choices). Reward obtained in the second stage should inform first-stage choices on subsequent trials. Model-based learners are goal directed in their decision-making. Model free learners are habitual decision-makers, relying on cached values of past reward prediction errors. Most people engage in a hybrid of both model based and model free strategies, and the weighing parameter reflects the relative preference for either. | Weighting parameter ($w$): ranges from 0 to 1. Higher values indicate more model-based learning, lower values indicate more model-free learning. |

stimuli). This is particularly relevant for the attentional bias, a construct which is commonly assessed as bias towards specific drug or alcohol-related stimuli (for a review see Christiansen et al., 2015).

### 3.2. Cognitive control

#### 3.2.1. Response selection

*3.2.1.1. Consumption.* The literature search found one study that assessed the role of response selection in predicting consumption-related addiction outcomes. Fernández-Artamendi et al. (2018) showed response selection, measured by the Stroop task (interference score), at 13 years of age did not predict the number of alcohol intoxication episodes two years later.

*3.2.1.2. Severity.* The literature search revealed two studies that looked at response selection to predict alcohol use and cannabis use severity respectively. Fernández-Artamendi et al. (2018) found response selection in adolescence, measured by the Stroop task (interference score), did not predict alcohol use problems (Rutgers Alcohol Problem Index [RAPI] score) two years later. Cousijn and colleagues (2015) also found Stroop interference score did not predict cannabis use or cannabis use disorder severity (Cannabis Use Disorder Identification Test) 6 months post-treatment for adults with cannabis use disorder. However, the authors indicated that their small sample size may have impacted their ability to achieve appropriate power to detect an effect, they were also unable to include key covariates in their model, subsiquently the study was considered to have an unacceptable risk of bias (for details see Appendix I).

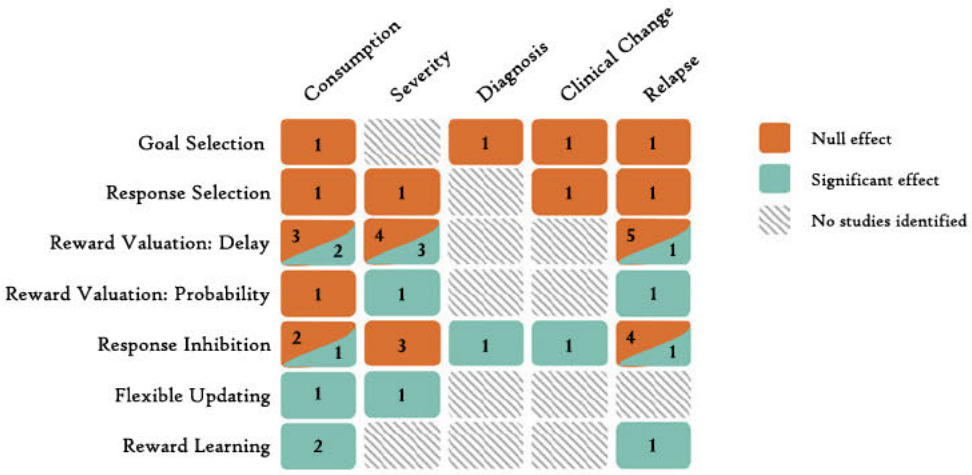
*3.2.1.3. Relapse.* The literature search revealed three studies that investigated response selection and relapse. None of the three studies found evidence that response selection, measured by the Stroop, predicted relapse for substance use disorder or gambling disorder. Lima et al. (2019) found there was no difference in Stroop performance (task C time; task C errors) between individuals who relapsed and those who did not 3 months after receiving inpatient treatment for cocaine use disorder. However, the authors did not use the standard metric for Stroop (interference score), which takes into account reading speed when calculating the inhibition score. Without controlling for processing speed it is incorrect to assume Stroop performance is representative of response selection. As such, this study was classified as having an unacceptable risk of bias. Barreno et al. (2019) also did not find Stroop performance predicted relapse of cannabis, cocaine, alcohol, or opioid use in a sample of inpatients receiving treatment for SUD (treatment duration ranged from approximately 1 month to 1 year). However, this study was also considered as having an unacceptable risk of bias due to a small sample size. In a sample of individuals diagnosed with pathological gambling disorder, Goudriaan and colleagues (2008) found Stroop performance was not associated with a higher likelihood of relapse after one year.

*3.2.1.4. Clinical change.* One study by Vergara-Moragues and colleagues (2017) looked at treatment-related clinical change over the course of inpatient treatment for individuals diagnosed with cocaine use disorder. Delis-Kaplan Executive Function System (D-KEFS) Color-Word Interference Test (CWIT) performance did not predict whether therapeutic goals were met over the course of an individual's treatment.

In summary, of the six studies reviewed, none of them showed response selection predicted addiction-related outcomes, although three of the six studies did not pass the risk of bias assessment due to methodological concerns (Barreno et al., 2019; Cousijn et al., 2015; Lima et al., 2019). The current evidence suggests response selection does not predict alcohol consumption (Fernández-Artamendi et al., 2018), alcohol (Fernández-Artamendi et al., 2018) or cannabis use severity (Cousijn et al., 2015), relapse for substance use disorder (Barreno et al., 2019; Lima et al., 2019) and gambling disorder (Goudriaan et al., 2008), or treatment-related clinical change for cocaine use disorder (Vergara-Moragues et al., 2017).

#### 3.2.2. Response inhibition

*3.2.2.1. Consumption.* Of the three studies identified from the literature search, only one showed response inhibition predicted consumption-related outcomes. Goudriaan et al. (2011) found response inhibition, measured by the Stop Signal Task (SST) did not predict the average alcohol intake (quantity x frequency) of college students over a two-year period. Bø et al. (2017) also found SST performance did not predict frequency/quantity of alcohol use or change in consumption over an 18-month period for regular drinking university students. By contrast, in a sample of alcohol-naïve teenagers followed over an 8-year period, Jones et al. (2021) found response inhibition, measured by the Immediate Memory Task (IMT), significantly predicted the quantity of alcohol




*Note: The value in each box reflects the number of studies that found an effect, the nature of this effect is indicated via the color of the box. Some studies are reported more than once given they assessed more than one neurocognitive domain and/or addiction-related outcome. Only studies that achieved an acceptable risk of bias or higher were included in this figure.*

**Fig. 2. Summary of review findings.**

consumed per drinking episode (25% increase in drinks consumed), but not frequency of alcohol intoxication.

*3.2.2.2. Severity.* None of the three studies identified in the literature showed evidence that response inhibition is predictive of alcohol use problem severity. Jones et al. (2021) found IMT performance did not predict the number of alcohol-related problems (see Table 2 for authors definition of problems) over an eight year follow-up of alcohol naïve teens. Goudriaan et al. (2011) found the SST did not predict alcohol use problem severity (proxy DSM-IV measure) for a sample of college students. Whelan et al. (2014) also did not find SST predicted classification of binge drinking at 16 years of age. However, the authors used the mean and standard deviation reaction time on Go trials which is not the standard metric for the SST and does not index response inhibition.

*3.2.2.3. Diagnosis.* The literature search identified only one study that investigated response inhibition as a predictor of alcohol dependence. Rubio et al. (2008) found poorer response inhibition at baseline predicted increased odds of alcohol dependence at follow-up (small effect size) in a sample of heavy-drinking adults without an AUD diagnosis or treatment history.

*3.2.2.4. Relapse.* None of the four studies identified showed that response inhibition predicted relapse for substance use disorders, however one study showed response inhibition does predict relapse for gambling disorder. López-Torrecillas et al. (2014) found response inhibition, measured by the Go/No-Go Task was not predictive of tobacco relapse after one year, for individuals receiving outpatient treatment for tobacco smoking. Passetti et al., (2008, 2011) found Go/No-Go performance did not predict relapse after three months, for individuals seeking either outpatient or inpatient treatment for opiate use disorder. Stevens et al. (2015) also did not find SST performance predicted substance use relapse after three months for individuals enrolled in an inpatient detoxification service. In a sample of individuals diagnosed with gambling disorder, Goudriaan and colleagues (2008) found poorer SST performance was associated with a higher likelihood of post-treatment relapse (medium effect size).

*3.2.2.5. Clinical change.* The literature search revealed one study that found an association between response inhibition and treatment-related clinical change (defined above). Vergara-Moragues and colleagues (2017) found poorer response inhibition, measured by the Go/No-Go Task at baseline predicted a decreased likelihood of meeting treatment goals (a small effect size).

In summary, we identified 11 studies that looked at response inhibition as a predictor of addiction-related outcomes. One study found poorer response inhibition prior to alcohol exposure predicts increased alcohol consumption (quantity per drinking session) (Jones et al., 2021). However, response inhibition assessed after regular alcohol use does not predict increased consumption (Bø et al., 2017; Goudriaan et al., 2011). Further, the evidence suggests response inhibition does not predict the severity of alcohol use problems in samples that are not currently heavily drinking (Goudriaan et al., 2011; Jones et al., 2021; Whelan et al., 2014) but does predict the development of alcohol dependence for already heavily drinking individuals (Rubio et al., 2008). Response inhibition does not predict relapse for substance use disorders (López-Torrecillas et al., 2014; Passetti et al., 2008, 2011; Stevens et al., 2015), but does predict relapse for gambling disorder (Goudriaan et al., 2008) and treatment-related clinical change for individuals with cocaine use disorder (Vergara-Moragues et al., 2017).

### *3.2.3. Goal selection; updating, representation and maintenance*

*3.2.3.1. Consumption.* Only one study was identified that assessed the predictive relationship between goal selection/updating and consumption. Bø and colleagues (2017) did not find goal selection/updating, measured by the Intra-Extra Dimensional Set Shift Task (IED), predicted frequency/quantity of alcohol use or change in frequency/quantity of alcohol consumption over an 18-month follow-up period.

*3.2.3.2. Diagnosis.* Only one study was identified that looked at whether goal selection/updating predicted addiction diagnosis. Boelema and colleagues (2016) found goal selection/updating, measured by the Shifting Attentional Set Task (SAST), at age 11 did not predict the diagnosis of alcohol abuse or dependence eight years later.

*3.2.3.3. Relapse.* Of the four studies identified by the literature search, only one study showed goal selection/updating predicted relapse. Czapla and colleagues (2016) found performance on the IED task did not predict alcohol relapse six months post-inpatient treatment. Desfosses and colleagues (2014) also found goal selection/updating, measured by the Wisconsin Card Sorting Task (WCST) and Trail Making Test (TMT), was not correlated with alcohol relapse three months after inpatient treatment. Further, Lima et al. (2019) found TMT performance was not predictive of self-reported cocaine relapse three months post-discharge from an inpatient treatment program for individuals with cocaine use

**Table 2**
Summary of cohort studies.

| Consumption, Severity and Diagnosis | | | | | | | |
|---|---|---|---|---|---|---|---|
| **Author/s** | **Task: metric** | **Outcome (s)** | **Sample** | **FU** | **Analysis method and covariates** | **Findings** | **RoB rating** |
| **Alcohol** | | | | | | | |
| Bernhardt et al. (2017) (part A) | Delay Discounting Task (DDT): log k<br>Probability Discounting for Losses (PDL): log k<br>Probability Discounting for Gains (PDG): log k | Δ Alcohol consumption (z-standardized CIDI alcohol consumption items; FU-BL) | Social drinking* young adults.<br>N = 155<br>Age= 18<br>Gender(F): 0% | 12 mos | Regression<br>IV: DD log(k), PDG log(k), PDL log(k), MG<br>CV: NA | **No effect** | Acceptable |
| Bø et al. (2017) | Intra-Extra Dimensional Shift Task (IED): errors, reversal and set shifting<br>Stop Signal Task: stop signal reaction time (SSRT), post error slowing | Frequency/quantity of alcohol use (last three questions of the AUQ^)<br>Δ Frequency/quantity of alcohol use | Regular drinking** university students.<br>N = 103<br>Age M(SD) = 21.7 (2.1)<br>Gender(F): 48.5% | 18 mos | Random intercepts multilevel model<br>IV: reversal errors, set shifting errors, SSRT, post error slowing, IST, LNS, IGT<br>CV: sex, time | **No effect** | Acceptable |
| Boelema et al. (2016) | Shifting Attentional Set Task (SAST): reaction time | Alcohol abuse (DSM-IV)<br>Alcohol dependence (DSM-IV) | N = 1596<br>Age M(SD) = 11.3 (0.6)<br>Gender(F): 54% | 8 yrs | Logistic hierarchical regression<br>IV: reaction time, Sustained Attention, Memory Search<br>CV: gender, SES, quantity of alcohol intake at age 13 and age 16 | **No effect** | High quality |
| Fernández-Artamendi et al. (2018) | Delay Discounting: area under the curve (AUC)<br>Stroop: interference score | Alcohol intoxication episodes<br>Alcohol related problems (RAPI) | N = 1430<br>Age M(SD) = 13.0 (0.5)<br>Gender(F): 46.1% | 2 yrs | Random intercepts cross-lagged panel model<br>IV: AUC, interference score<br>CV: NA | **No effect** | Acceptable |
| Goudriaan et al. (2011) | Stop Signal Task (SST): stop signal reaction time (SSRT) | Alcohol quantity/ frequency measure (average quantity of alcohol used per drinking occasion x average frequency of drinking occasions per week)<br>Alcohol-related problems (DSM-IV)# | College students.<br>N = 176<br>Age M(SD) = 20.0 (0.4) Gender(F): 51% | 2 yrs | Multivariate analysis of covariance<br>IV: SSRT, IGT<br>CV: gender, BL alcohol use, precollege quantity/ frequency of drinking and ACT scores<br>Analysis of covariance<br>IV: SSRT, IG<br>CV: gender, BL alcohol use, precollege heavy drinking and ACT scores | **No effect** | Acceptable |
| Rubio et al. (2008) | Stop Signal Task (SST): stop signal reaction time (SSRT)<br>The Delay Task: efficiency score | Alcohol consumption (alcohol units drunk over follow-up period)<br>Problem alcohol use (AUDIT) | Heavy drinking*** individuals.<br>N = 380<br>Age M(SD) = 37.7 (7.4)<br>Gender(F): 37.2% | 4 yrs | Correlations<br>Logistic regression<br>IV: efficiency score, SSRT<br>CV: alcohol family history, lifetime comorbidity for ADHD or impulse control disorders, impulsivity score (BIS-11), years of education, anxiety, age, gender, marriage status | **Effect**<br>SST predicted increased risk of alcohol dependence<br>r = 0.11<br>∋·<br>**No effect**<br>The Delay Task did not predict increased risk of alcohol dependence | Acceptable |
| Whelan et al. (2014) | Monetary Choice Questionnaire (MCQ): log k<br>Stop Signal Task (SST): mean Go RT; standard deviation Go RT | Classification of binge drinker (minimum of three lifetime binge drinking episodes) | N = 271<br>Age M(SD) = 14.5 (0.4)<br>Gender(F): 48% | 2 yrs | Machine learning logistic regression with elastic net regularization.<br>IV: demographic variables (e.g. sex, age, handedness), past events (e.g. alcohol uses by age 14, smoking at age 14, family history of drug misuse, gestational alcohol exposure), personality traits, genetics, brain volume and function, and neurocognition (e.g. log k, mean Go RT, standard deviation Go RT, CGT, WISC-IV) | **No effect** | Acceptable |

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**Table 2** (*continued*)

| Consumption, Severity and Diagnosis | | | | | | | |
|---|---|---|---|---|---|---|---|
| **Author/s** | **Task: metric** | **Outcome (s)** | **Sample** | **FU** | **Analysis method and covariates** | **Findings** | **RoB rating** |
| Worhunsky at al. (2016) | Experiential Discounting Task: area under the curve (AUC), total score | Escalating drinking (increase in number of maximum drinks consumed per session; FU-BL) | Individuals who drink alcohol<br>N = 36,<br>Age M(SD) = 18.4 (0.7)<br>Gender(F): 69.4% | 1 yr | Multivariate analysis of variance<br>IV: latent variables: impulsive choice (AUC, total score), impulsive action (BIS-11,ZSSS), approach motivation (BIS/BAS), impulsivity/ compulsivity (SPSRQ, Padua compulsivity score), thrill seeking (ZSSS, BIS/BAS), risky choice (BART)<br>CV: NA | **No effect** | Unacceptable |
| Chen et al. (2021) | Sequential Decision-Making Task (SDT): *w* | Alcohol quantity (grams of alcohol per drinking occasion)<br>Alcohol consumption (AUDIT-C) | N = 201<br>Age: 18<br>Gender(F): 0% | 3 yrs | Linear growth curve model<br>IV: *w*, neural activation, working memory capacity, processing speed and trait impulsivity<br>CV: NA | **Effect**<br>SDT predicted quantity of alcohol consumed per drinking session<br>r = 0.48<br>**No effect**<br>SDT did not predict AUDIT-C | Acceptable |
| Fröhner et al. (2022) | Monetary Choice Questionnaire (MCQ); log k | Problematic alcohol use (AUDIT)<br>Cumulative alcohol use (grams-per-week; AUC) | N = 1350<br>Age M(SD) = 14.4 (0.4)<br>Gender(F): 51.2% | 8 yrs | Correlation<br>Linear growth curve model<br>IV: log k<br>CV: NA<br>Additional analysis added gender as a grouping variable and found this did not change the original effects found. | **Effect**<br>MCQ correlated with cumulative alcohol use<br>r = 0.09 and predicted problematic alcohol use<br>β = 0.10 | Acceptable |
| Jones et al. (2021) | Immediate Memory Task (IMT): inhibition score | Alcohol quantity (maximum drinks consumed in 24 h period)<br>Heavy episodic drinking (frequency of consuming ≥4 / ≥5 (women/men) drinks per occasion in past year)<br>Alcohol-related problems (DIS and YAAPST) | Alcohol naïve youths.<br>N = 249<br>Age M(SD) = 12.18(1.5)<br>Gender(F): 49% | 8 yrs | Structural equation model<br>IV: inhibition score, trait impulsivity<br>CV: age, sex, race/ ethnicity, parental education, family history of AUD | **Effect**<br>IMT predicted maximum drinks in a day<br>IRR= 1.27<br>**No effect**<br>IMT did not predict heavy drinking episodes or lifetime number of alcohol-related problems | High quality |
| Peeters et al. (2014) | Self Ordered Pointing Task (SOPT): SOPT score | Latent variable representing past month alcohol frequency, quantity and problem use (CRAFFT) | N = 381<br>Age M(SD) = 13.6 (0.9)<br>Gender(F): 11.8% | 1.5 mos | Cross-lagged model<br>IV: BL latent alcohol use, SOPT score<br>CV: correlations between IVs and confounding variables (cannabis, extasy and cocaine use) were not significant. Thus, not included in the model. | **Effect**<br>SOPT predicted the latent alcohol variable<br>β = 0.05 | High quality |
| Tschorn et al. (2021) | Monetary Choice Questionnaire (MCQ): metric unspecified | Problem alcohol use (AUDIT) | N = 1376,<br>Age M(SD) = 14.6 (0.4)<br>Gender(F): 52% | 2 yrs | Regression<br>IV: MCQ, sexuality and deviance, hopelessness, impulsivity, extraversion, WISC-IV, CGT, MCQ, SNP risk score, neural activation during Monetary Incentive Delay Task, neural activation during SST<br>CV: SES, baseline smoking, baseline drinking, familial history of alcohol and drugs, prenatal cigarette and alcohol consumption | **No effect** | Acceptable |
| **Cannabis** | | | | | | | |

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**Table 2** (*continued*)

| Consumption, Severity and Diagnosis | | | | | | | |
|---|---|---|---|---|---|---|---|
| **Author/s** | **Task: metric** | **Outcome (s)** | **Sample** | **FU** | **Analysis method and covariates** | **Findings** | **RoB rating** |
| Cousijn et al. (2015) | Stroop: interference score | Cannabis use frequency<br>CUD severity (CUDIT) | N = 57<br>Age M(SD) = 19.6 (2.0)<br>Gender(F): 24.6%<br>Treatment:<br>CBT<br>Outpatient | 6 mos | Hierarchical linear and logistic regression<br>IV: interference score, cannabis attentional bias, approach bias, craving<br>CV: BL CUDIT | **No effect** | Unacceptable |
| **Tobacco** | | | | | | | |
| Audrain-McGovern et al. (2009) | Monetary Choice Questionnaire (MCQ): log k | Δ Daily tobacco use (number of cigarettes smoked)<br>Membership of tobacco smoking trajectory class | N = 800<br>Age = 15<br>Gender(F): 53% | 5 yrs | Linear growth curve model<br>IV: log k<br>CV: sex, ethnicity, academic performance, depression symptoms, novelty seeking, attention, hyperactivity, alcohol use, cannabis use, peer smoking, household smoking<br>Growth mixture model<br>IV: log k.<br>CV: sex, ethnicity, academic performance, depression symptoms, novelty seeking, attention, hyperactivity, alcohol use, cannabis use, peer smoking, household smoking | **Effect**<br>DDT predicted change in daily tobacco use<br>r = 0.03<br><br>∋<br>**No effect**<br>DDT did not discriminate between individuals who were classed as slow progressors and those classified as fast smoking adopters | High quality |
| **SUD (unspecified)** | | | | | | | |
| Khurana et al. (2017) | Delay Discounting Hypothetical Monetary Choice Task: DD score | SUD criteria: alcohol, cannabis, tobacco (DSM-IV) | N = 387<br>Age M(SD) = 12.1 (0.9)<br>Gender(F): 52% | 7 yrs | Correlations<br>Structural equation model<br>IV: log k, working memory factor score, acting without thinking.<br>CV: early drug use experimentation, early drug use progression, alcohol dependence symptoms, cannabis dependence symptoms, tobacco dependence symptoms, sex, age | **Effect**<br>DD score correlated with tobacco dependence symptoms<br>r = 0.12<br>**No effect**<br>DD score did not predict SUD | High quality |
| van Hemel-Ruiter (2015) | Spatial Orienting Task (SOT): engagement toward nonpunishment, engagement toward reward, disengagement toward nonpunishment, disengagement toward reward | Substance use quantity & frequency: alcohol, tobacco and cannabis | N = 715<br>Age M(SD) = 16.1 (0.6)<br>Gender(F): 52.3% | 3 yrs | Hierarchical regression<br>IV: engagement and disengagement scores<br>CV: age, gender, baseline substance use<br>Post hoc: regression in sample of substance use naïve participants<br>IV: engagement and disengagement scores<br>CV: age, gender, baseline substance use | **No effect**<br>Attentional engagement toward reward<br>**Effect**<br>Stronger engagement toward longer presented cues of non-punishment predicted higher substance use<br>$r^2 = 0.24$ | Acceptable |
| Kräplin et al. (2020) | Delay Discounting Task (DDT): log k<br>Probability Discounting for Losses (PDL): log k<br>Probability Discounting for Gains (PDG): log k | SUD: z-standardized sum of the fulfilled diagnostic criteria for alcohol or tobacco use disorder (DSM-V)<br>AD: Non-Substance Addictive Disorder: z-standardized sum of the fulfilled diagnostic criteria for gambling, internet use, gaming, or shopping addictive disorder (adapted DSM-V)<br>SUD QFI (z-standardized score: | Community members meeting criteria for SUD (tobacco/alcohol) or a non-substance AD (gambling, shopping, internet, gaming).<br>N = 218<br>Age M(SD) = 21.8 (1.7)<br>Gender(F): 59% | 1 yr | Bayesian linear regression<br>IV: DDT logk, PDL logk, PDG logk, MGT.<br>CV: BL SUD, BL AD, BL QIF (SUD, AD), age, gender, IQ, income, school graduation | **Effect**<br>DDT predicted increased SUD criteria (probability of 99%) and AD criteria (probability of 72%).<br>DDT predicted increased SUD (probability of 83%) and AD (probability of 93%) QFIs.<br>PDG predicted | Acceptable |

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**Table 2** (*continued*)

| Consumption, Severity and Diagnosis | | | | | | | |
|---|---|---|---|---|---|---|---|
| **Author/s** | **Task: metric** | **Outcome (s)** | **Sample** | **FU** | **Analysis method and covariates** | **Findings** | **RoB rating** |
| | | quantity and frequency of substance use)<br>AD QFI (z-standardized score: quantity and frequency of addictive behaviour) | | | | increased SUD (probability of 84%) and AD criteria (probability of 90%).<br>PDL predicted increased AD criteria (probability of 98%)<br>**No effect**<br>PDG did not substantially predict SUD or AD QIF<br>PDL did not predict SUD criteria. Nor SUD or AD QFIs | |

Note: All studies reported binary sex and/or gender, they did not report on gender fluidity. RoB: Risk of Bias, adapted from SIGN Checklist for Cohort Studies; FU: Follow up; BL: Baseline; CUD: Cannabis Use Disorder; ACT: Academic achievement score, USA; SNP: single nucleotide polymorphisms; SES: social economic status; CIDI: German Composite International Diagnostic Interview (Wittchen and Pfister, 1997); DSM-IV: Diagnostic and Statistical Manual for Mental Disorders 4th Edition (American Psychiatric Association, 2000); RAPI: Rutgers Alcohol Problem Index (White and Labouvie, 1989); AUDIT: Alcohol Use Disorder Identification Test (Saunders et al., 1993); AUDIT-C: Alcohol Use Disorder Identification Test consumption questions; CUDIT: Cannabis Use Disorder Identification Test (Adamson and Sellman, 2003); AUQ: Alcohol Use Questionnaire (Bø et al., 2017); CRAFFT: Car, Relax, Alone, Forget, Friends, Trouble Scale for problem drinking in adolescence (Knight et al., 1999); DIS: Diagnostic Interview Schedule (Robins, 1981); YAAPST: Young Adults Alcohol Problems Screening Test (Hurlbut, Sher, 1992); MG: Mixed Gambles Task (Bernhardt et al., 2017); LNS: Letter Number Sequencing (Wechsler, 2003); MST: Matrix Span Task (Kane et al., 2004); IST: Information Sampling Task (CANTAB, 2019); IGT: Iowa Gambling Task (Bechara et al., 1994); Sustained Attention, Memory Search (Amsterdam Neuropsychological Tasks; De Sonneville, 1999); CGT: Cambridge Gambles Task (CANTAB, 2019); WISC-IV: Wechsler Intelligence Scale for Children (Wechsler, 2003). BIS-11: Barrat Impulsiveness Scale (Patton et al., 1995); BIS/BAS: Behavioural Inhibition/Activation Scale (Carver and White, 1994); Padua Inventory (Sanavio, 1988); ZSSS: Zuckerman Sensation-Seeking Scale (Zuckerman and Neeb, 1979); SPSRQ: Sensitivity to Punishment and Sensitivity to Reward Questionnaire (Torrubia et al., 2001); BART: Balloon Analogue Risk Task (Lejuez et al., 2002);

* At least 2 drinking occasions in the past 3 months.

* * AUDIT $\geq$ 1

* ** AUDIT score $\geq$ 8

#Proxy measure of DSM-IV alcohol dependence developed by Grekin and Sher (2006)

ˆ Q.10) Pace of consumption when drinking i.e. number of drinks per hour; Q.11) Number of times intoxicated by alcohol; Q.12) Percentage of time drunk when going out drinking

⇛ Converted from Odds Ratio

disorder. However, both Desfosses et al. and Lima at al.'s study were deemed to have an unacceptable risk of bias due to methodological concerns (see Appendix I for details). In a sample of individuals seeking in-patient detoxification treatment for alcohol use disorder, Morrison (2011) found poorer performance on the TMT at baseline predicted more frequent alcohol consumption (days of use) in the three months post treatment, with a large effect. However, Morrison evaluated goal selection/updating via time to complete part B of the TMT, failing to account for baseline levels of processing speed (i.e., part A of the TMT). As such, we are unable to determine whether shifting predicted alcohol relapse, or whether this was driven instead by impaired processing speed. Further, neurocognitive assessments were conducted anywhere between 5 and 10 days following drinking cessation, thus performance is likely to have been impacted by detoxification and not representative of actual functioning. This study was deemed to have an unacceptable risk of bias. Table 3.

*3.2.3.4. Clinical change.* The literature search identified only one study that evaluated goal selection/updating and treatment-related clinical change. Vergara-Moragues and colleagues (2017) found performance on the D-KEFS CWIT (switching score) did not predict the achievement of therapeutic goals during inpatient treatment.

In sum, only one of the seven studies identified in the literature search found goal selection/updating predicted addiction-related outcomes (Morrison, 2011), however as articulated above, this study did not pass the risk of bias assessment and it is therefore inappropriate to make any formal conclusions from their results. The evidence suggests goal selection/updating does not predict alcohol consumption (Bø et al., 2017), the diagnosis of an alcohol use disorder (Boelema et al., 2016), relapse of alcohol and cocaine use disorder (Czapla et al., 2016; Desfosses et al., 2014; Lima et al., 2019), or treatment-related clinical change for cocaine use disorder (Vergara-Moragues et al., 2017).

### 3.2.4. Flexible updating

*3.2.4.1. Consumption and severity.* The review found one study that assessed the relationship between flexible updating, measured by the Self-Ordered Pointing Task (SOPT), and alcohol use and severity in adolescents (Peeters et al., 2014). The study involved four time points that spanned 18 months. The authors found poorer SOPT performance at time point two predicted more severe alcohol use and problems (latent score, see Table 2) six months later (small effect). The same was found for SOPT performance at time point three. However, baseline SOPT did not predict future alcohol use outcomes, instead baseline alcohol use and problems predicted time point two SOPT performance.

**Table 3**
Summary of studies involving treatment/intervention.

**Treatment-Related Outcomes**

| Author/s | Task:metric | Outcome (s)/ metric | Treatment / Intervention | Sample | FU | Analysis method and covariates | Findings | RoB rating |
|---|---|---|---|---|---|---|---|---|
| **Alcohol** | | | | | | | | |
| Albertella et al. (2021) | Value-Modulated Attentional Capture Task (VMAC): VMAC score | Alcohol abstinence (self-report) | Community abstinence challenge | N = 683<br>Age M(SD) = 52.3 (12.1)<br>Gender(F): 64% | 1 mo | Logistic regression<br>CV: age and gender | **Effect**<br>VMAC predicted abstinence<br>r = 0.0008 | Acceptable |
| Czapla et al. (2016) | Intra-Extra Dimensional Shift Task (IED): number of stages completed, total number of errors, reversal learning errors | Alcohol relapse (TLFB; urine and blood test) | Detoxification, medication, CBT, psychoeducation, individual and group therapy<br>Inpatient | N = 81<br>Age M(SD) = 48.1 (9.3)<br>Gender(F): 19% | 6 mos | Logistic regression<br>IV: latent IED variable<br>CV: number of previous detoxification efforts, utilisation of treatment offers in post-inpatient follow-up period | **No effect** | Acceptable |
| Bernhardt et al. (2017) (part B) | Delay Discounting Task (DDT): log k<br>Probability Discounting for Losses (PDL): log k<br>Probability Discounting for Gains (PDG): log k | Alcohol relapse (TLFB; PEth levels) | Unspecified | N = 114<br>Age M(SD) = 45.1 (11.64)<br>Gender(F): 16.5% | 12 mos | Cox proportional hazard regression<br>IV: DDT (logk), PDL (logk), PDG (logk), MGT<br>CV: alcohol dependence severity, obsessive-compulsive drinking and craving, anxiety, depression | **Effect**<br>PDL predicted relapse<br>HR= 0.67<br>**No effect**<br>DD<br>PDG | Acceptable |
| Tucker et al. (2016) | Delay Discounting Task (DDT): log k | Alcohol resolution status (TLFB): resolved abstinent (RA), resolved non abstinent (RNA), unstable resolution (UR)* ** | NA | N = 175<br>Age M(SD) = 50.65 (11.83)<br>Gender(F): 24.6% | 12 mos | Logistic regression<br>IV: log k, Alcohol-Savings Discretionary Expenditure Index, APT, MM<br>CV: age, pre-resolution year days well-functioning | **No effect** | Acceptable |
| Morrison (2011) | Trail Making Test (TMT): time to complete part B | Number of days drunk alcohol (TLFB) | Detoxification and treatment<br>Inpatient | N = 34<br>Age M(SD) = 47.28 (10.03)<br>Gender(F): 41% | 3 mos | Regression<br>IV: time to complete part B, RAVLT, LNS<br>CV: NA | **Effect**<br>TMT predicted the number of days of drinking post-treatment<br>$r^2 = 0.44$ | Unacceptable |
| Desfosses et al. (2014) | Wisconsin Card Sorting Test (WCST): total correct responses; persev erative errors; non-perseverative errors<br>Trail Making Test (TMT): time to complete part B – part A | Alcohol relapse (self-report: return to use at pre-treatment levels) | Counselling services<br>Inpatient | N = 21<br>Age M(SD) = 42.2 (9.0)<br>Gender(F): NA | 3 mos | Correlations<br>CV: NA | **No effect** | Unacceptable |
| **SUD (non-specific)** | | | | | | | | |
| Barreno et al. (2019) | Stroop: interference score<br>Monetary Choice Questionnaire (MCQ): area under the curve (AUC) | SUD relapse: cannabis cocaine, alcohol and opioids (positive result of urine/blood test) | CBT, psycho-education, occupational therapy, pharmacological treatment<br>Inpatient | N = 68<br>Age M(SD) = 37.4 (11.35)<br>Gender(F): 8.4% | End of treatment M = 148.36 days, range 22–289 days | Cox proportional hazard regression<br>IV: interference score, AUC, affective Go/No-Go, IGT<br>CV: age, gender, disability, employment status, education history, cannabis use | **No effect** | Unacceptable |
| de Wilde et al. (2013) | Delay Discounting Task (DDT): log k | Substance use relapse (self-reported any use of substances other than | Unspecified<br>Inpatient | N = 37<br>Age M(SD) = 31.9 (6.9)<br>Gender(F): 18.9% | 3 mos | Analysis of variance<br>IV: log k<br>CV: age, age of onset, duration, BIS-11, SPSRQ | **No effect** | Unacceptable |

(*continued on next page*)

**Table 3** (*continued*)

| Treatment-Related Outcomes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Author/s** | **Task:metric** | **Outcome (s)/ metric** | **Treatment / Intervention** | **Sample** | **FU** | **Analysis method and covariates** | **Findings** | **RoB rating** |
| | | caffeine or nicotine during FU period) | | Poly-substance dependent | | | | |
| Stevens et al. (2015) | Stop Signal Task (SST): stop signal reaction time (SSRT)<br>Delay Discounting Task (DDT): log k | Substance use relapse (self-report any use of an illicit substance during FU period) | Detoxification, medical management of withdrawal symptoms, crisis support (medical, social, administrative), motivation techniques<br>Inpatient | N = 70<br>Age M(SD) = 28.5 (6.1)<br>Gender(F): 10% | 3 mos | Logistic regression<br>IV: log K, SSRT, UPPS sensation seeking and urgency<br>CV: individuals who relapsed vs abstained did not differ in terms of gender, age, education, age of onset, duration of drug use, past month use, poly-drug use, prior treatment episodes, treatment retention, depression, ADHD | **Effect**<br>DDT predicted relapse r = 0.22<br>∋<br>**No effect**<br>SSRT | Acceptable |
| **Cocaine** | | | | | | | | |
| Lima et al. (2019) | Trail Making Test (TMT): time to complete part B – part A<br>Stroop Colour-Word Test (part C time to complete; part C errors) | Cocaine relapse (self and family member reports of any cocaine use) | Cognitive behavioural group therapy, occupational activities, medication<br>Inpatient | N = 65<br>Age M(SD) = 18.45 (2.9)<br>Gender(F): NA | 3 mos post discharge | Student's *t*-test and Mann-Whitney U tests<br>IV: time to complete part B – part A, part C time to complete; part C errors<br>CV: NA | **No effect** | Unacceptable |
| Vergara-Moragues et al. (2017) | Stroop: interference score, inhibition score<br>Go/No-go Task: total errors (omission + commission) | Cocaine use disorder clinical change* | Drug-free treatment, replacement therapy, social and educational activities, medical and psychological treatments offered<br>Inpatient | N = 226<br>Age M(SD) = 35(7.10)<br>Gender(F): 7.5% | At discharge | *t* -test<br>IV: interference score, inhibition score, total errors<br>CV: NA | **Effect**<br>Go/No-go predicted clinical change r = 0.20#<br>**No effect**<br>Stroop | Acceptable |
| **Tobacco** | | | | | | | | |
| López-Torrecillas et al.2014 | Go/No-Go Task: commission errors<br>Monetary Choice Questionnaire (MCQ): area under the curve (AUC) | Tobacco relapse (self-report and co-oximetry hemoglobin levels > 10 ppm) | Behaviour change and pharmacological treatment<br>Outpatient | N = 112<br>Age M(SD) = 47.36 (8.19)<br>Gender(F): 58% | 1 yr | Regression<br>IV: commission errors, AUC, temperament scores, BIS-11, IGT<br>CV: NA | **No effect** | Acceptable |
| **Opiates** | | | | | | | | |
| Passetti et al. (2008) | Delay Discounting Task (DDT): log k<br>Go/No-Go Task: commission errors<br>* *Neurocognitive task performance was classified as "impaired" and "unimpaired" based upon predetermined cut offs. | Opiate relapse (self-report and urine analysis) | Group-based treatment, psychological, pharmacological<br>Outpatient | N = 37<br>Age M(SD) = 37.5 (2.3)<br>Gender(F): 20.7% | 3 mos | Stepwise linear discriminant function analysis<br>IV: log k, commission errors, IGT, IST, CGT, SOC<br>CV: Methadone dose<br>No significant differences between those who abstained vs relapsed in any demographic variables, drug histories, psychological or physical health. | **No effect** | Acceptable |
| Passetti et al. (2011) | Delay Discounting Task (DDT): log k<br>Go/No-Go Task: commission errors | Opiate relapse (self-report and urine analysis) | Assisted withdrawal, individual and group-based therapy<br>Inpatient (n = 32)<br>Assisted detoxification programme, access to psychology, psychiatry, social work<br>Outpatient (n = 48) | N = 80<br>Age M(SD) = 36.6 (7.1)<br>Gender(F): 28.8% | 3 mos | Logistic regression<br>IV: log k, commission errors, IGT, IST, CGT, SOC<br>CV: ethnicity<br>This analysis was repeated using cut-off scores which defined 'unimpaired' vs 'impaired' neuropsychological | **No effect** | Acceptable |

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**Table 3** (*continued*)

| Treatment-Related Outcomes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Author/s** | **Task:metric** | **Outcome (s)/ metric** | **Treatment / Intervention** | **Sample** | **FU** | **Analysis method and covariates** | **Findings** | **RoB rating** |
| | | | | | | performance at baseline. No significant differences between those who abstained vs relapsed in any demographic variables, drug histories, psychological or physical health. | | |
| **Gambling** | | | | | | | | |
| Goudriaan et al. (2008) | Stop Signal Task (SST): stop signal reaction time (SSRT) Stroop: interference score | Gambling relapse (self-report**) | 10 × 2-hour sessions of CBT | N = 46 Age M(SD) = 38(9.3) Gender(F): 26.5% | 1 yr | Logistic regression IV: SSRT, interference score, BIS-11, BAS, IGT CV: duration of pathological gambling | **Effect** SST predicted relapse β = 1.11 **No effect** Stroop | Acceptable |

Note: All studies reported binary sex and/or gender, they did not report on gender fluidity. RoB: Risk of Bias, adapted from SIGN Checklist for Cohort Studies; PEth – phosphatidylethanol; CBT: Cognitive Behavioural Therapy; MG: Mixed Gambles Task (Bernhardt et al., 2017); Alcohol-Savings Discretionary Expenditure Index (Rachlin et al., 1981); APT: Alcohol Purchase Task (Murphy and MacKillop, 2006); MM: Melioration-Maximization Task (Heyman and Dunn, 2002). RAVLT: Rey Auditory Verbal Learning Test (Rey, 1958); LNS: Letter Number Sequencing (Wechsler, 2003); BIS-11: Barrat Impulsiveness Scale (Patton et al., 1995); SPSRQ: Sensitivity to Punishment and Sensitivity to Reward Questionnaire (Torrubia et al., 2001); UPPS: Urgency Premeditation, Perseveration, Sensation Seeking Scale (Whiteside and Lynam, 2014); IGT: Iowa Gambling Task (Bechara et al., 1994); IST: Information Sampling Task (CANTAB, 2019); CGT: Cambridge Gambles Task (CANTAB, 2019); SOC: Stockings of Cambridge (Owen et al., 1990); BAS: Behavioural Activation Scale (Carver and White, 1994); TLFB: Timeline Follow Back (Sobell and Sobell, 1992).

*A reference clinician evaluated whether the individual has met the goals set out at admission of treatment for three therapeutic targets: socio-educational, psychological and biomedical.

* *"Do you think you have a gambling problem again?"

* ** Resolved abstinent (RA): continuous abstinence throughout follow-up period. Resolved non-abstinent (RNA): defined as any drinking post resolution that was below the threshold for risky drinking, that is < 4/3 standard drinks for men/women, no symptoms on the Alcohol Dependence Scale (Skinner and Horn, 1984) and no negative consequences measured by the Drinking Problems Scale (Cahalan, 1970). Unstable resolution (UR): one or more relapse events of risky drinking.

# Converted from Cohen's d

∋Converted from an Odds Ratio

### 3.3. Reward valuation

#### 3.3.1. Delay

*3.3.1.1. Consumption.* The literature search found six studies that investigated delay discounting and consumption outcomes. Four of these studies looked specifically at alcohol consumption. Bernhardt et al. (2017) found that delay discounting did not predict a change in alcohol consumption in young male adults over a 12-month follow-up period. Worhunsky et al. (2016) also did not find a relationship between baseline delay discounting and change in the quantity of alcohol consumed per session over a 12-month period, however the study was found to have an unacceptable risk of bias due to a limited sample size. Fernández-Artamendi et al. (2018) found delay discounting at 13 years of age did not predict the number of alcohol intoxication episodes at age 15. By contrast, Fröhner and colleagues (2022) found a weak positive correlation between delay discounting assessed at 14 years of age and cumulative alcohol use (measured as grams consumed per week) over an eight-year follow-up period. However, this relationship did not hold in the final Linear Growth Curve Model.

Two studies found delay discounting predicted non-alcohol related substance use. Audrain-McGovern et al. (2009) reported that steeper delay discounting was associated with a change in daily tobacco use in high school students over a five year period. For every one standard deviation increase in baseline delay discounting there was a corresponding 11% increase in daily tobacco use. Kräplin and colleagues (2020) evaluated community members who met the criteria for SUD (tobacco, alcohol) or a non-substance addiction disorder (gambling, shopping, internet, gaming), they found delay discounting predicted increased consumption (quantity and frequency) for both substance use (83% probability) and non-substance addictive behaviour (93% probability) with a small effect.

*3.3.1.2. Severity.* The literature search revealed six studies that looked at delay discounting and addiction severity. Five studies investigated alcohol use outcomes and four of the five studies showed delay discounting did not predict the severity of problem alcohol use. Whelan et al. (2014) found delay discounting in adolescence did not predict the classification of binge drinking two years later. Khurana et al. (2017) found delay discounting at age 12 did not correlate with alcohol dependence criteria (measured by DSM-IV) seven years later. Fernández-Artamendi et al. (2018) and Tschorn et al. (2021) both investigated adolescents over a 2-year period and found delay discounting was not predictive of the severity of alcohol-related problems (RAPI) or problem alcohol use (measured by Alcohol Use Disorder Identification Test [AUDIT]) respectively. By contrast, one study by Fröhner and colleagues (2022) found steeper temporal discounting at age 14 was associated with increasing AUDIT scores over an eight year period. However, the effect size was very small, accounting for only 1% of the variance in the model. Khurana et al. (2017) also investigated the relationship between delay discounting and tobacco and cannabis use disorder symptoms. The authors found discounting was weakly correlated with tobacco use disorder symptoms (DSM-IV) but not cannabis dependence symptoms at follow-up.

The literature search revealed one study that looked at predicting addiction severity in a sample of individuals with either substance use

(alcohol, tobacco) or non-substance (gambling, internet, gaming, or shopping) addiction disorder (Kräplin et al., 2020). Kräplin and colleagues found steeper delay discounting assessed at 21 years of age predicted an increase in SUD (99% probability) and non-substance use disorder (72% probability) criteria one year later (with a small effect).

*3.3.1.3. Relapse.* The literature search revealed eight studies that investigated delay discounting's role in predicting relapse. Only one of the eight studies found an effect. Tucker and colleagues (2016) found delay discounting did not predict relapse to risky drinking for individuals engaging in unassisted abstinence over the course of the following year. Bernhardt et al. (2017) also found that delay discounting did not predict alcohol relapse for treatment-seeking individuals with alcohol dependence (treatment modality was unspecified). Barreno et al. (2019) found delay discounting did not predict substance use relapse during 48 week inpatient treatment. Similarly, for individuals with poly-substance use disorder undergoing inpatient treatment, de Wilde et al. (2013) found delay discounting did not predict self-reported relapse over a three month period. However, due to limited sample size, both Barreno et al. and de Wilde et al.'s studies were deemed to have an unacceptable risk of bias (see Appendix I for details). López-Torrecillas et al. (2014) found, for individuals receiving outpatient treatment for tobacco smoking, delay discounting did not predict tobacco relapse after one year. Passetti et al., (2008, 2011) found delay discounting did not predict opiate relapse, for individuals seeking either outpatient or inpatient treatment for opiate use disorder. By contrast, Stevens et al. (2015) found for individuals enrolled in an inpatient detoxification service for substance use disorder, delay discounting predicted relapse over a subsequent three month period.

In summary, the literature search revealed 17 studies that looked at the role of delay discounting in predicting addiction-related outcomes. Four studies showed weak or no evidence that delay discounting predicts alcohol consumption (Bernhardt et al., 2017; Fernández-Artamendi et al., 2018; Fröhner et al., 2022; Worhunsky et al., 2016). Two studies showed delay discounting predicted tobacco use (Audrain-McGovern et al., 2009) and the frequency of engaging in non-substance-related addictive behaviours (Kräplin et al., 2020). When assessed in adolescence, prior to the development of an addictive disorder, only one of five studies found delay discounting predicts substance use severity (Fernández-Artamendi et al., 2018; Fröhner et al., 2022; Khurana et al., 2017; Tschorn et al., 2021; Whelan et al., 2014). When assessed after the onset of substance/non-substance addictive disorder, one study showed delay discounting did predict addiction severity for problematic substance/ non-substance addictive behaviours (Kräplin et al., 2020). The majority of the evidence (seven out of eight studies) suggests delay discounting does not predict substance use relapse (Barreno et al., 2019; Bernhardt et al., 2017; de Wilde et al., 2013; López-Torrecillas et al., 2014; Passetti et al., 2008, 2011; Tucker et al., 2016).

### 3.3.2. Probability

*3.3.2.1. Consumption and severity.* The literature search revealed one study that evaluated probability discounting and substance-/non-substance-related consumption and problem severity. Kräplin and colleagues (2020) found, for community members who met the criteria for SUD (tobacco/alcohol) or a non-substance addiction disorder (gambling, internet, gaming, or shopping), probability discounting (either for losses or for gains) did not predict consumption. However, lower probability discounting for losses predicted more severe non-substance addiction one year later (98% probability) with a small effect. Further, steeper probability discounting for gains at baseline predicted an increase in both SUD (84% probability) and AD (90% probability) severity with a small effect.

*3.3.2.2. Relapse.* One study showed probability discounting predicted alcohol relapse. Bernhardt et al. (2017) found, in a sample of treatment-seeking individuals with alcohol dependence, probabilistic discounting for losses but not for gains predicted alcohol relapse. Lower probability discounting for losses was associated with a 43% risk of relapse within one year.

In summary, we found two studies that investigated probability discounting and addiction-related outcomes. These studies showed probability discounting did not predict substance-/non-substance-related consumption but did predict increased substance and non-substance use severity (Kräplin et al., 2020) and alcohol relapse (Bernhardt et al., 2017), but in different ways. Lower probability discounting for losses predicts increased non-substance use severity, as well as increased likelihood of alcohol relapse, and steeper probability discounting for gains, predicts increased substance and non-substance addictive disorder severity.

## 3.4. Reward learning

### 3.4.1. Consumption

Two studies investigated reward learning and substance use consumption. Van Hemel-Ruiter et al. (2015) evaluated the role of reward bias, measured via the Spatial Orienting Task (SOT), in predicting substance use consumption. Reward bias was defined as a stronger engagement towards cues of non-punishment. Van Hemel-Ruiter and colleagues (2015) found reward bias predicted substance use quantity and frequency (alcohol, tobacco, and cannabis) three years later for substance naïve adolescents (large effect). No effect was found when including individuals who had already commenced substance use at baseline. Chen et al. (2021) used the Sequential Decision-Making Task (SDT) to investigate whether model-based versus model-free learning in young adult males predicted alcohol consumption over a three year follow up period. The authors found model-based learning predicted quantity of alcohol consumption per session (with a medium effect) but not AUDIT consumption at follow up.

### 3.4.2. Relapse

The literature search revealed one study that assessed the role of reward-related attentional bias, measured by the Value-Modulated Attentional Capture task (VMAC), in predicting alcohol abstinence. Albertella and colleagues (2021) found greater baseline reward-related attentional bias was associated with a reduced ability to remain abstinent (with a small effect) during a month-long, community-based, socialised abstinence initiative (IkPas).

In sum, all of the three studies that emerged from the literature showed different aspects of reward learning predicts substance use consumption and abstinence. There is evidence suggesting reward bias and model-based reward learning predicts substance use consumption (Chen et al., 2021; van Hemel-Ruiter et al., 2015) and attentional bias to reward cues predicts the ability to abstain from alcohol use (Albertella et al., 2021).

## 4. Discussion

To the authors' knowledge, this is the first review to systematically evaluate the neurocognitive predictors of addiction-related outcomes across longitudinal studies, encompassing consumption and severity, to diagnosis and relapse. A strength of the current review is that it takes a standardized approach (RDoC) to define specific addiction-related neurocognitive constructs and evaluate their role in predicting a broad range of addiction outcomes. Further, using an expert-guided approach (Yücel et al., 2019), we selected addiction-specific neurocognitive domains and evaluated their predictive utility across multiple substance use and non-substance addictive behaviours. A total of seven neurocognitive constructs were investigated, thus comprehensively evaluating functions that span cognitive control and reward-related systems.

Thirty-three studies were included in the final analysis and reviewed in full. Seven of the studies reviewed, due to methodological concerns, were deemed as having unacceptable risk of bias (Barreno et al., 2019; Cousijn et al., 2015; de Wilde et al., 2013; Desfosses et al., 2014; Lima et al., 2019; Morrison, 2011; Worhunsky et al., 2016). All but one of these studies (Morrison, 2011) reported a null effect for neurocognition predicting addiction outcomes which was in line with findings from studies that had acceptable-high quality risk of bias ratings. Alcohol use studies were overwhelmingly represented across the literature (Albertella et al., 2021; Bernhardt et al., 2017; Bø et al., 2017; Boelema et al., 2016; Chen et al., 2021; Czapla et al., 2016; Desfosses et al., 2014; Ellingson et al., 2019; Fernández-Artamendi et al., 2018; Fröhner et al., 2022; Goudriaan et al., 2011; Harvanko et al., 2013; Heinrich et al., 2016; Jones et al., 2021; Morrison, 2011; Peeters et al., 2014; Rubio et al., 2008; Tschorn et al., 2021; Tucker et al., 2016; Whelan et al., 2014; Worhunsky et al., 2016; Xiao et al., 2009). By contrast, non-substance addictive behaviors were under-researched, with only two studies meeting inclusion criteria (Goudriaan et al., 2008; Kräplin et al., 2020). Across all studies reviewed, the mean follow-up period ranged from one month to eight years. Of the studies that evaluated treatment-related outcomes, the most common follow up period was three months and the average follow up period for cohort studies was three and a half years.

The evidence suggests most of the neurocognitive functions reviewed herein do not predict addiction outcomes; less than half of the studies reviewed showed neurocognition significantly predicted addiction outcomes. Firstly, all the evidence for response selection (Barreno et al., 2019; Cousijn et al., 2015; Fernández-Artamendi et al., 2018; Goudriaan et al., 2011; Lima et al., 2019; Vergara-Moragues et al., 2017) and almost all the evidence (all but one study: Morrison et al., 2011) for goal setting/updating suggest neither subconstructs predict substance use consumption, severity, relapse, or clinical change (Bø et al., 2017; Boelema et al., 2016; Czapla et al., 2016; Desfosses et al., 2014; Lima et al., 2019; Vergara-Moragues et al., 2017). Secondly, across the four substance use studies, no studies found response inhibition predicted relapse of use (López-Torrecillas et al., 2014; Passetti et al., 2008, 2011; Stevens et al., 2015). Thirdly, the majority of the evidence suggests delay discounting does not predict substance use consumption (Bernhardt et al., 2017; Fernández-Artamendi et al., 2018; Worhunsky et al., 2016), severity of substance use prior to onset of a SUD (Fernández-Artamendi et al., 2018; Khurana et al., 2017; Tschorn et al., 2021; Whelan et al., 2014), or substance use relapse (Barreno et al., 2019; Bernhardt et al., 2017; López-Torrecillas et al., 2014; Passetti et al., 2008, 2011; Tucker et al., 2016; de Wilde et al., 2013).

Conversely, some studies did support the role of neurocognition in predicting addiction outcomes. In adolescence, early assessment of response inhibition, prior to alcohol use onset, predicted increased quantities of alcohol consumption in early adulthood (Jones et al., 2021), poorer flexible updating for those who have already engaged in alcohol use predicted increased alcohol use and related problems (Peeters et al., 2014), and a preference for sooner smaller rewards in adolescence predicted increased daily tobacco use (Audrain-McGovern et al., 2009) and dependence (Khurana et al., 2017). For adults already consuming alcohol, higher levels of model-based learning (ie, goal-directed choices) was associated with a lower likelihood of binge drinking in men (Chen et al., 2021). Poorer response inhibition predicted the transition from problematic drinking to dependence (Rubio et al., 2008). Steeper delay discounting and probability discounting for gains and losses predicted increased SUD and non-substance addiction severity respectively (Kräplin et al., 2020). For treatment-seeking individuals, poorer response inhibition predicted poorer clinical improvement for cocaine use disorder (Vergara-Moragues et al., 2017), as well as relapse for gambling disorder (Goudriaan et al., 2008).

Existing studies suggest that reward-related processes are important risk factors for both early and late-stage addiction outcomes. Attentional bias to reward was found to be a key vulnerability marker for early substance use progression (van Hemel-Ruiter et al., 2015) and maladaptive reward learning predicted alcohol use progression (Chen et al., 2021). In the later stages of addiction (i.e. post-diagnosis of an addiction disorder), increased risk-seeking for reward predicted increased severity of substance and non-substance addictive disorders (Kräplin et al., 2020), and increased reward-related attentional capture predicted the likelihood of alcohol "relapse" (Albertella et al., 2021). This heightened motivational pull for reward-seeking is paired with a devaluation of the potential impact of loss. Less probability discounting for losses was found to predict increased severity of non-substance use disorder (Kräplin et al., 2020) as well as relapse for alcohol use disorder (Bernhardt et al., 2017). Taken together, the evidence suggests an increased valuation of reward and a devaluation of the impact of loss may be key neurocognitive risk factors for the progression of addiction and poor intervention outcomes.

Neurocognition may differentially predict substance-/non-substance-related outcomes. This review revealed two key instances where the relationship between neurocognition and addiction depended on the addictive behaviour in question. The first was that delay discounting did not reliably predict alcohol and illicit substance use outcomes (Bernhardt et al., 2017; Fernández-Artamendi et al., 2018; Khurana et al., 2017; Tschorn et al., 2021; Whelan et al., 2014; Worhunsky et al., 2016), but did predict tobacco use and dependence (Audrain-McGovern et al., 2009; Khurana et al., 2017). Steeper delay discounting has been found to preceded tobacco use uptake and is not influenced by tobacco smoking (Audrain-McGovern et al., 2009). It is possible that individuals with a stronger preference for immediate rewards may also prefer substances with more immediate effects. Tobacco, for instance, has a rapid mode of action (approximately 10–20 s) (Le Houezec, 2003), while other substances like alcohol are comparatively slower to take effect (Tabakoff and Hoffman, 2013). The second example of how neurocognition may differentially predict substance-related outcomes involves response inhibition and relapse. The review showed that response inhibition consistently did not predict relapse for substance use disorder (López-Torrecillas et al., 2014; Passetti et al., 2008, 2011; Stevens et al., 2015), but has been found to predict relapse for pathological gambling (Goudriaan et al., 2008). In line with evidence that the ability to inhibit automatic responses is crucial to pathological gambling (Brevers and Noël, 2013), these findings suggest perhaps response inhibition is more sensitive in predicting gambling versus substance use relapse. However, further replication is required.

Adolescence is a period of development in which the individual experiences maturation of key neurocognitive processes and these processes are particularly vulnerable to the impact of substance use (Blakemore and Choudhury, 2006; Luna et al., 2010; Paus, 2005; Squeglia et al., 2009; Van Leijenhorst et al., 2010). In particular, there is a mismatch between top-down executive control processes, which is underdeveloped and bottom-up heighted rereward-seeking and risk taking (Paus, 2005). Variability in maturation rate, as well as the impact of neurotoxic substance use, may disproportionately cloud the predictive relationship of neurocognition on addictive behaviour. Jones and colleagues (2021) found early assessment of response inhibition, prior to alcohol use onset may be valuable in identifying adolescents that are likely to engage in higher quantities of alcohol consumption. This suggests that there may be value in assessing neurocognition before initiating substance use. However, Jones at el. did not find response inhibition predicted the frequency of heavy drinking episodes or lifetime number of alcohol-related problems, so we have still yet to evidence a direct link between early response inhibition ability and future problematic alcohol use behavior.

Perhaps neurocognition is a better predictor of outcomes in later stages of addiction. Response inhibition has been shown to predict the transition from problematic drinking to dependence in already heavy-drinking adults (Rubio et al., 2008). Further, steeper delay discounting was associated with increased addiction severity for individuals with an existing substance or non-substance addictive disorder (Kräplin et al.,

2020). This is likely a result of clinical samples having more variability in substance use than general population samples. Those studies that looked at non-clinical populations generally showed low levels of problem alcohol use in their samples, for example a mean AUDIT score of between 1.5 and 10 (Bø et al., 2017; Fröhner et al., 2022; Tschorn et al., 2021) representing low-medium risk use. However, these findings also highlight the bidirectional relationship between neurocognition and addiction. Research suggests that neuroadaptations resulting from further engagement in addictive behaviour, not to mention the neurotoxic effects of past substance use, impacts neurocognitive functioning (López-Caneda et al., 2014). For example, Khurana and colleagues' (2017) showed early substance use progression to greater severity of use was an important mediator of the relationship between delay discounting and meeting criteria for SUD. This is further evidenced by Peeters et al.'s (2014) findings that alcohol use/problems at baseline predicted flexible updating performance which then subsequently predicted future alcohol use outcomes. Neither Rubio et al. (2008) nor Kräplin et al. (2020) accounted for previous alcohol/substance use history in their analysis models, so it is possible that past substance use/repeated exposure to the addictive behaviour is what is driving the effect of neurocognition observed. As such, we cannot conclude that neurocognition is a robust predictor of later stage addiction outcomes.

### 4.1. Limitations and future directions

This review revealed some substantial shortcomings in the field. There was a lack of diversity of substances and very little investigation of behavioral addictions in the literature. For some of the subconstructs (e.g. response inhibition) the literature reviewed herein has pertained only to alcohol use, which limits our understanding to just one substance. To truly determine predictive trans-addiction mechanisms requires more longitudinal research that compares different types of substance and non-substance addictive behaviours within each study. In particular, this review highlighted the need for more research on behavioral addictions beyond gambling disorder, such as problematic eating, problematic usage of the internet, problematic pornography use, and compulsive shopping. Another limitation of the current work in the field is that some studies do not take into account past substance use in their predictive models. Early substance use initiation is a powerful predictor of future drug use and abuse (Grant and Dawson, 1998; Griffin et al., 2010; Odgers et al., 2008) and has been shown to mediate the predictive effect of neurocognition on the development of SUDs (Khurana et al., 2017). We recommended the assessment of neurocognition prior to substance use initiation or to account for age-of-first-use in analysis. Given substance use initiation commonly occurs in mid-adolescence (Richmond-Rakerd et al., 2017), adolescent cohort studies are an ideal approach to investigate the mechanisms that predict addiction progression. However, it is important to consider key neurocognitive maturation processes that occur during adolescence and early adulthood (Blakemore and Choudhury, 2006; Luna et al., 2010; Paus, 2005; Van Leijenhorst et al., 2010) as well as the sensitivity of this period to the neurotoxic effects of substance use (Squeglia et al., 2009). It is therefore integral when predicting addiction outcomes in adolescence, to not only account for substance use over the study period but also change in neurocognitive function over time. Change scores (calculated by subtracting a variable captured at one time point from another) are commonly used but are inherently problematic (see Tennant et al., 2021 for detailed description), and if used, need to be handled appropriately in analysis models. That is, baseline score needs to be entered as covariates in the model. Alternatively, growth curve models which estimate between-person differences in within-person change (Curran et al., 2010) can more powerfully model both neurocognitive functioning and substance use trajectories over time (Duncan and Duncan, 2004).

In summary, we recommend the following essential methodological considerations for future longitudinal research in this space: 1) include key variables as covariates in analyses, at the bare minimum past use (substance or engagement in behaviour), age and gender should be taken into consideration either in the method or analysis; 2) clearly define the outputs used for neurocognitive tasks, including how it was calculated; 3) do not use change scores for outcome variables, or when using change scores make sure to also include a measure of the outcome at baseline as well; 4) assess neurocognition over time and factor any changes into the analysis model, this is particularly relevant for cohort studies that span adolescence to early adulthood, but also for treatment studies in which the treatment may improve neurocognitive functioning for some, but not others. Utilising latent growth curve and/or structural equation models will allow for any variance in the neurocognition over time to be captured; 5) Assess multiple neurocognitive domains in the same model, this will allow for the evaluation of which domains hold the best predictive value when held accountable to each other.

## 5. Conclusion

In conclusion, the present study aimed to investigate the evidence for neurocognition predicting addiction and treatment-related outcomes. Results of the review revealed the majority of the literature did not find neurocognition predicted consumption, severity, or relapse of addictive behaviours. In particular, cognitive control processes do not seem to be relevant to the prediction of the development of problematic use and addiction-related outcomes. However, there is some evidence that reward-related neurocognitive dysfunction is a sensitive predictor of both early and late-stage addiction outcomes, as well as treatment-related outcomes. These findings suggest that reward-related processes may be important for the detection of early risk for addiction, as well as treatment response, and are worthy mechanisms for further investigation. Overall, this review contributes to the growing body of literature on the relationship between neurocognition and addiction. We have exposed the substantial gaps in knowledge of the field and made recommendations for how to better conduct future research. We urge researchers to conduct more prospective and longitudinal investigations into the key neurocognitive domains that predict both substance and non-substance-related addiction.

## Funding sources

RSCL was supported by an NHMRC Investigator Grant funded by the Medical Research Future Fund (APP1193946). L.A has received funding from the David Winston Turner Endowment Fund. MY receives funding from: government funding bodies such as the NHMRC, Australian Research Council (ARC), Australian Defence Science and Technology (DST), the Department of Industry, Innovation and Science (DIIS), the National Institutes of Health (NIH, USA); philanthropic donations from the David Winston Turner Endowment Fund, Wilson Foundation; sponsored Investigator-Initiated trials including Incannex Healthcare Ltd; and payments in relation to court-, expert witness-, and/or expert review-reports. These funding sources had no role in the data analysis, presentation, or interpretation and write-up of the data. Murat Yücel also sits on the Advisory Boards of: Centre of The Urban Mental Health, University of Amsterdam; Monash Biomedical Imaging Centre; and Enosis Therapeutics.

## Competing interests statement

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2023.105295.

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*Chapter 3*

# The cross-sectional neurocognitive correlates of addictive behaviours

Addictive Behaviors 150 (2024) 107904

Contents lists available at ScienceDirect

Addictive Behaviors

journal homepage: www.elsevier.com/locate/addictbeh

# The neurocognitive correlates of non-substance addictive behaviors

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ARTICLE INFO

*Keywords:*
Addiction
Cognition
Neurocognition
Alcohol
Internet use
Pornography

ABSTRACT

Neurocognitive deficits have been implicated as transdiagnostic risk markers of substance use disorders. However, these have yet to be comprehensively evaluated in other, non-substance addictions. In a large, general community sample (N 475) the present study evaluated the neurocognitive correlates of problem alcohol use and three non-substance-related addictive behaviors: addictive eating (AE), problematic pornography use (PPU), and problematic use of the internet (PUI), to identify potential shared and distinct neurocognitive correlates. A sample of Australian residents (54.4 % female M[SD] age 32.4[11.9] years) completed a comprehensive online assessment of neurocognitive tasks tapping into eight distinct expert-endorsed domains purportedly associated with addiction. Multiple linear regressions with bootstrapping were used to examine associations among each addictive behavior of interest and neurocognition, trait impulsivity, and compulsivity, as well as key covariates. Neurocognition was differentially associated with each addictive behavior. None of the neurocognitive domains were significantly associated with problematic alcohol use or AE ($p > .05$), poorer performance monitoring was significantly associated with higher levels of PPU and PUI (β −0.10, $p$ .049; β −0.09, $p$ .028), and a preference for delayed gratification was associated with more severe PUI (β −0.10, $p$ .025). Our findings have theoretical implications for how we understand non-substance addiction and suggest the need for a more nuanced approach to studying addictive behaviors that take into account the underlying neurocognitive mechanisms associated with each type of addiction.

## 1. Introduction

Addiction is a complex condition characterized by repeated engagement in substance use or other behavior despite negative consequences (Diagnostic Statistical Manual of Mental Disorders [DSM-5]: American Psychiatric Association, 2013). The literature has predominantly centered on substance use disorders, such as alcohol use disorder (AUD), whereas there has been an increasing focus on non-substance (behavioral) addictions (Chamberlain et al., 2016). Recent such efforts include addictive eating (AE), problematic pornography use (PPU), and problematic use of the internet (PUI) due to their associated psychological distress and poorer quality of life (Burmeister et al., 2013; Burrows et al., 2018; Camilleri et al., 2021; Fineberg et al., 2018; Floros & Ioannidis, 2021; Kuss et al., 2014; Raj et al., 2022). It is essential to acknowledge that none of these addictive behaviors have agreed-upon clinical criteria. Consequently, it is more appropriate to view them as problem behaviors positioned on a severity continuum. AE, PPU, and PUI may share some important features with substance addiction, including loss of control, craving, and emotional distress (Adams et al., 2017; Fineberg et al., 2018; Grubbs et al., 2015; Tiego et al., 2021). Addictive behaviors can be conceptualized using current neurocognitive 'dual-process' models of addiction, typified by excessive drive and reward-seeking coupled with impaired executive 'top-down' control (Volkow et al., 2019; Volkow & Morales, 2015). Dual process models have been further extended in non-substance addictive behaviors (Brand, 2022; Brand et al., 2019; Wei et al., 2017). However, addiction-specific neurocognitive functions have yet to be comprehensively evaluated in non-substance addictions. For example, it is unclear whether

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https://doi.org/10.1016/j.addbeh.2023.107904
Received 2 August 2023; Received in revised form 19 October 2023; Accepted 2 November 2023
Available online 7 November 2023

non-substance addictive behaviors share neurocognitive features. Substance use research has also shown inconsistent neurocognitive correlates (Ekhtiari et al., 2017; Lundqvist, 2005; Smith et al., 2014). Identifying the neurocognitive correlates of addictive behaviors is essential to progressing etiological understanding and facilitating the development of effective prevention and intervention strategies. This study investigates the *trans*-behavioral neurocognitive correlates of non-substance addictive behaviors; namely, AE, PPU, and PUI, in comparison to problematic alcohol use, to identify potential shared and distinct neurocognitive mechanisms.

Current consensus is that some neurocognitive deficits can serve as transdiagnostic markers of addiction, regardless of the specific type of behavior (Yücel et al., 2019). Self-regulation is often defined as being comprised of: *Inhibition*, the ability to stop or inhibit an undesirable automatic response (Chambers et al., 2009); *Shifting*, the ability to flexibly shift between tasks, mental sets, or demands (Kiesel et al., 2010); and *Performance monitoring*, the ability to monitor and evaluate action outcomes to adjust performance (Franken et al., 2018). Poorer executive functioning in individuals with addiction is marked by reduced inhibitory control, poorer performance monitoring, and inflexible task shifting (Franken et al., 2018; Odlaug et al., 2011; Rodrigue et al., 2018; Smith et al., 2014; Zhou et al., 2013). By contrast, the 'bottom-up' motivational processes associated with addiction are underpinned by heightened reward-seeking; chief among this is an increased preference for immediate over larger but delayed rewards or poor 'delay discounting' (Amlung et al., 2017), and heightened incentive attribution towards addiction-related cues (Berridge & Robinson, 2016). Unfortunately, most studies to date have focused on individual neurocognitive factors in isolation. Therefore, there is a lack of empirical evidence regarding how each domain is independently associated with addictive behaviors. This is a critical area of further investigation since neurocognitive functions associated with executive control and heightened reward-seeking significantly overlap with each other (Criaud & Boulinguez, 2013; Ridderinkhof et al., 2004).

Few studies have examined neurocognitive functioning associated with AE, PPU, and PUI. Existing studies have generally identified neurocognitive markers implicated in problematic alcohol use, including heightened attentional bias toward reward cues (Adams et al., 2019; Jeromin et al., 2016; Mechelmans et al., 2014; Nikolaidou et al., 2019) and a preference for immediate gratification over larger, later rewards (Cheng et al., 2021; Kowalewska et al., 2017; VanderBroek-Stice et al., 2017). Individuals with AE, PPU, and PUI also present with executive dysfunction, but in differing domains. PUI but neither AE or PPU has been shown to be associated with impaired response inhibition (Antons & Brand, 2018; Antons & Matthias, 2020; Hardee et al., 2020; Ioannidis et al., 2022; Meule et al., 2012; VanderBroek-Stice et al., 2017), and both AE and PUI have been shown to be associated with impaired performance monitoring (Franken et al., 2018; Rodrigue et al., 2018; Zhou et al., 2013). It is unclear whether these differences arise from methodological differences, for example selection of neurocognitive measures, sample characteristics, and inclusion (or lack there-of) of covariates. Because many studies only look at a single neurocognitive measure, it is entirely plausible that differing executive control profiles among AE, PPU, and PUI may be due to common neurocognitive function(s) that drive performance across measures. Further, sample characteristics often vary, including differing illness severities, and demographic features (e.g. sex, age). These studies also do not address comorbidities of other addictive behaviors. Assessing the relationship between neurocognition and multiple addictive behaviors in the same sample would account for these disparities, and help us evaluate whether AE, PPU, and PUI share common underlying neurocognitive processes.

Examining addictive behaviors in general community samples using a dimensional approach can lead to a more nuanced understanding of the neurocognitive functions underlying addictive behaviors. Clinical presentations of addiction represent only the tip of the iceberg, as the majority of addiction problems do not meet strict diagnostic thresholds (Grant et al., 2015; Hasin et al., 2013). Further, despite not meeting diagnostic thresholds, these addictive behaviors still contribute to burden of disease and are important to detect early. By adopting a dimensional approach and examining addictive behaviors across the full spectrum of severities, we can detect often more subtle effects, with direct implications for the development of early intervention strategies.

In addition to neurocognition, addictive behaviors are associated with trait impulsivity and compulsivity (Forsén Mantilla et al., 2022; Kuss et al., 2014; Murphy et al., 2014). Impulsivity can be defined as the tendency to rapidly react to a situation in a reward-driven manner, without forethought or consideration of the consequences (Moeller et al., 2001). Impulsivity is a multifaceted construct, commonly conceptualized and assessed in addiction research as being made up of four key facets: lack of planning, lack of perseverance, sensation seeking and emotion-driven rash action (urgency; Cyders et al., 2014). Compulsivity can be defined as repeated actions that are inappropriate to a given situation and lacks a clear connection to an overarching goal, often resulting in negative consequences (Dalley et al., 2011). Although previous research has found neurocognitive functions correspond with trait impulsivity (Christiansen et al., 2012) and compulsivity (Albertella et al., 2020), when assessed concurrently it is clear that they are not measuring the same underlying construct (Eisenberg et al., 2019). For example, convergent validity among task-related executive function and self-report questionnaires was only moderate in a large *meta*-analysis on self-regulation ability (Duckworth & Kern, 2011). Therefore, both trait-based and neurocognitive-based measures of impulsivity and compulsivity appear to contribute to addiction vulnerability in different ways.

This study sought to investigate the neurocognitive correlates of addictive behaviors. Using a tailored assessment battery that measures a comprehensive range of addiction-specific neurocognitive functions, the present study aims to evaluate the shared and distinct neurocognitive correlates of problematic alcohol use, AE, PPU, and PUI, adjusting for key covariates. A demographically targeted, general community sample was recruited to capture a broad spectrum of behavioral severity.

## 2. Methods

### 2.1. Participants and procedure

This study was embedded in a larger normative study for the BrainPark Assessment of Cognition (BrainPAC) neurocognitive battery (see supplementary material). Australian residents were recruited via Prolific, social media advertisements, and local community newsletters. Inclusion criteria were: 18 to 65 years old, not color blind, self-reported absence of a neurological disorder (i.e. stroke, brain injury, and dementia) or history of a psychotic disorder. Participants completed all measures online via the Qualtrics survey platform (https://www.qualtrics.com). The neurocognitive tasks were separated by self-report surveys (trait and behavior scales) and the order of task presentation was counterbalanced. The study was approved by the Monash University Human Research Ethics Committee [26088].

### 2.2. Neurocognitive measures

The neurocognitive battery (Table 1) was selected to measure expert-endorsed neurocognitive domains as associated with addiction and addiction-related outcomes (Yücel et al., 2019).

### 2.3. Self-report scales

*Trait Compulsivity* – The Cambridge-Chicago Compulsivity Trait Scale (CHI-T; Chamberlain & Grant, 2018; Tiego et al., 2023) is a 15-item scale. Items are summed into an overall score. Higher scores indicate higher compulsivity.

*Trait Impulsivity* – The Short UPPS Impulsive Behavior Scale (SUPPS-

**Table 1**
Neurocognitive assessment battery.

| Function | Task | Brief description | Trials (practice) | Primary metric |
|---|---|---|---|---|
| Response inhibition | The BrainPAC Stop Signal Taks (SST) (Lee et al., 2023) | A gamified visual cue stop signal paradigm (Verbruggen et al., 2019) in a medieval war game format, with the goal of defeating a dragon. Players pass arrows to two archers (left/ right) as fast as they can (go signal) whilst avoiding the dragon's fire (stop signal) so they can shoot the arrows and defend their village. | 150 (10) | Stop signal reaction time (SSRT). Higher RT indicates poorer response inhibition. |
| Reward learning (reward-related attentional bias) | The BrainPAC Value Modulated Attentional Capture (VMAC) Task (Lee et al., 2023) | A gamified version of the original VMAC task (Albertella et al., 2019; Le Pelley et al., 2015) following a soccer format. Players must kick the ball (left/right), with speed and accuracy to earn points. Some trials have players with different hair colors acting as distractors and indicating the potential reward value of that trial. | 5x24 (6) | VMAC score averaged across the last two blocks of the task. Higher values reflect more reward-related attentional capture. |
| Reward learning (goal-directed vs habitual) | The BrainPAC Sequential Decision-Making Task (SDT) (Lee et al., 2023) | A gamified two-step choice task (Kool et al., 2016, 2017), presented in the form of an animal rescue game. Participants select a ranger to search two environments (the forest or the farmland) for lost animals. Model-based decision-makers learn which rangers are most effective at finding the maximum number of animals. | 125 (25) | Mixing weight (*w*). Higher scores indicate more goal-directed (model-based) decision-making. |
| Reward valuation (risky decision-making under uncertainty) | The BrainPAC Balloon Analogue Risk Task (BART) (Lee et al., 2023) | A gamified version of the BART stretch variant (Lejuez et al., 2002) paradigm in which players inflate a series of balloons to earn hypothetical money (maximum earning of $128 AUD p/ balloon). Each balloon has a pseudorandomized burst threshold (mean burst point at $64 AUD), resulting in any potential earnings being lost. | 30 (10) | Mean pre-committed pumps across all balloons. Higher values indicate riskier choice in the face of uncertainty. |
| Flexible updating | N-back Task (Ragland et al., 2002; Inquisit 5, 2018) | A letter sequencing go/no-go task. Participants respond to "M" in 0-back trials, to the previous letter in 1-back trials, and to the letter two trials back in 2-back trials, and so on. | 3x60 (9) | 3-back d' (parametric measure of sensitivity). Higher d' values indicate more flexible updating/ better working memory performance. |
| Goal selection; updating, representation and maintenance | Category Switch Task (CST) (Friedman et al., 2008; Inquisit 5, 2018) | Participants are presented with a word they must categorize in terms of A) 'living', or B) 'size'. Each trial is accompanied by a cue that indicates to the participant whether they are required to categorize the object according to conditions A or B. | 65 (80) | Latency switch cost. Higher values indicate poorer task switching. |
| Performance monitoring | Error Awareness Task (EAT) (Hester et al., 2007; Inquisit 5, 2018) | A visual go/no-go paradigm in which participants indicate their error awareness following any commission error. | 2x150 (70) | Percentage error awareness (commission errors). Higher values indicate better error awareness. |
| Temporal discounting | Monetary Choice Questionnaire (MCQ) (Kirby et al., 1999) | A 27-item questionnaire asks the participant to choose between two hypothetical reward options, a smaller reward now, or a larger reward at some point in the future e.g. "Would you prefer $15 today or $35 in 13 days". | 27 (NA) | Log *k* Higher values indicate a preference for sooner but smaller rewards. |

P; Cyders et al., 2014) is a 20-item scale. Three scores were calculated: urgency (combining negative and positive urgency subscales), lack of perseverance and premeditation (combining lack of perseverance and premeditation subscales), and sensation seeking (SS: sensation seeking subscale). Higher values indicate greater impulsivity.

*Psychological Distress* – The Depression Anxiety Stress Scale (DASS-21; Szabó, 2010) is a 21-item scale used to assess current psychological distress. Items were summed into form a total score. Higher scores indicate greater distress.

### 2.4. Dependent variables

Participants were asked to respond reflecting on the past three months.

*Problematic Alcohol Use* – The Alcohol Use Disorder Identification Test (AUDIT; Saunders et al., 1993) is a 10-item scale. A total score is summed, ranging from 0 to 40.

*Addictive Eating* – The modified Yale Food Addiction Scale version 2 (mYFAS 2.0; Schulte & Gearhardt, 2017) is a 13-item scale. A symptom count is computed, ranging from 0 to 11.

*Problematic Pornography Use (PPU)* - The Problematic Pornography Consumption Scale (PPCS-6: Bőthe et al., 2021) is a 6-item scale. A total score is calculated, ranging from 6 to 42.

*Problematic Use of the Internet* – The abbreviated Young's Internet Addiction Test (IAT-10; Tiego et al., 2021) is a 10-item scale. A total score ranging from 10 to 50 is computed.

### 2.5. Data cleaning

To ensure data quality, online neurocognitive assessment requires comprehensive screening protocols and post-hoc data cleaning. Bots and fraudulent responses were identified and excluded using features embedded in the Qualtrics survey platform. Implausible responses and poor performance presumedly due to lack of effort were identified and removed via a) attention check questions (e.g. "Please select the option *Piano Keys*"); b) neurocognitive task performance at less than chance levels (as per Lee et al., 2023; Albertella, Watson, et al., 2019); c) task-specific cleaning procedures (e.g. SST go trial accuracy, stop trial accuracy, [Verbruggen et al., 2019] and Independent Race Model check [Band et al., 2003]). Mann Whitney U and t-tests were used to investigate differences between individuals whose data was filtered out compared with included individuals on all variables (Table A1).

### 2.6. Data analysis

Individuals who reported not engaging in an addictive behavior in the past 3 months were assigned a zero for the corresponding problem behavior scale. Statistical outliers on neurocognitive measures $\geq 3$ standard deviations from the mean were removed (Field et al., 2012). All analyses were conducted on complete data sets (i.e. participants who provided data for all variables of interest). Bivariate Spearman correlations, adjusting for multiple comparisons (Holm method: Holm, 1979), investigated relationships among all variables (Table 3). The

distributions of all four addictive behavior scales were positively skewed, constituting the choice of linear regression models with bootstrapping (5,000 samples) (Neal & Simons, 2007). Multicollinearity was assessed for each model independently, with VIF values less than 2.5 indicating no issue of multicollinearity (Johnston et al., 2018). Age, sex, psychological distress, trait impulsivity, and compulsivity were included as covariates in each regression model (Eisenberg et al., 2019; Sjoberg & Cole, 2018; Starcke et al., 2016). G-Power 3.1 (Faul et al., 2007) was used to calculate the minimum sample size required for multiple regression analyses with 15 predictors and an alpha error probability set to 0.05. A sample of N = 139 was deemed sufficient to find a medium effect ($f^2 = 0.15$, Power = 0.80).

## 3. Results

### 3.1. Participants

Nine-hundred-and-forty-four participants were enrolled. Sixty-three withdrew during the assessment sessions, and 78 were identified as fraudulent responses (i.e. spam bots/not genuine responses). Eight-hundred-and-three participants completed the assessment, with 311 removed due to missing data on one or more variables of interest (i.e. those included in the regression models), failed attention checks, or poor neurocognitive task performance. After removing outliers based on neurocognitive task performance, a final sample of 475 individuals had complete datasets (see Fig. 1). Participant demographics are displayed in Table 2.

### 3.2. Multiple regression models

Correlation analysis findings are presented in Table 3. The multivariate models (Tables 4-7) showed the neurocognitive variables were not significantly associated with problematic alcohol use or AE. Poorer error awareness was significantly associated with greater PPU (β = 0.10, $p$ =.049) and PUI (β = 0.09, $p$ =.028), and less steep delay discounting was significantly associated with higher PUI (β = 0.10, $p$ =.025). Higher levels of psychological distress were significantly associated with more problematic alcohol use (β = 0.22, $p$ =.003), AE (β = 0.26, $p$ <.001), PPU (β = 0.20, $p$ <.001), and PUI (β = 0.37, $p$ <.001). Higher levels of urgency were significantly associated with more PPU (β = 0.14, $p$ =.013) and PUI (β = 0.15, $p$ =.003). Sensation seeking was positively associated with problematic alcohol use (β = 0.17, $p$ =.003) and negatively associated with AE (β = 0.12, $p$ =.004) and PUI (β = 0.09, $p$ =.014). Higher levels of trait compulsivity were significantly associated with more AE (β = 0.18, $p$ =.002) and PUI (β = 0.12, $p$ =.010). Age was positively associated with problematic alcohol use (β = 0.16, $p$ =.002), and negatively associated with PUI (β = 0.17, $p$ <.001). Being male was significantly associated with more PPU (β = 0.46, $p$ <.001), and PUI (β = 0.15, $p$ <.001) whilst being female was significantly associated with greater AE (β = 0.19, $p$ <.001). None of the multivariate models showed problems with multicollinearity.

## 4. Discussion

This is the first study to comprehensively investigate and control for the neurocognitive correlates of non-substance addictive behaviors across AE, PPU, PUI, and problem alcohol use. We took a dimensional approach to identify correlates associated with these addictive behaviors at varying degrees of severity in the general community. Our findings indicate, in this sample, addictive behaviors are associated with a unique profile of neurocognitive functioning. Our multivariate models showed none of the neurocognitive domains were associated with AE or problematic alcohol use. Poorer performance monitoring was independently associated with more PPU and PUI, and a higher preference for delayed gratification was also independently associated with higher PUI. Importantly, these findings are identified whilst adjusting for known confounds (i.e. age, sex, psychological distress), as identified as a key methodological shortcoming of prior research (Christensen et al., 2023). Our findings suggest the need for a more nuanced approach to studying addictive behaviors that take into account the underlying neurocognitive mechanisms associated with each type of addiction.

Our performance monitoring findings are consistent with prior research (Zhou et al., 2013). However, this is the first study to show a link between poorer performance monitoring and PPU. Very little work has been done evaluating the potential neurocognitive mechanisms associated with PPU. Of the extant work, findings are mixed. For example, PPU has been associated with both improved and poorer inhibitory control (Antons & Brand, 2018; Antons & Matthias, 2020). Considerable research has investigated PUI, with studies showing PUI is associated with poorer working memory, response inhibition, and risk-

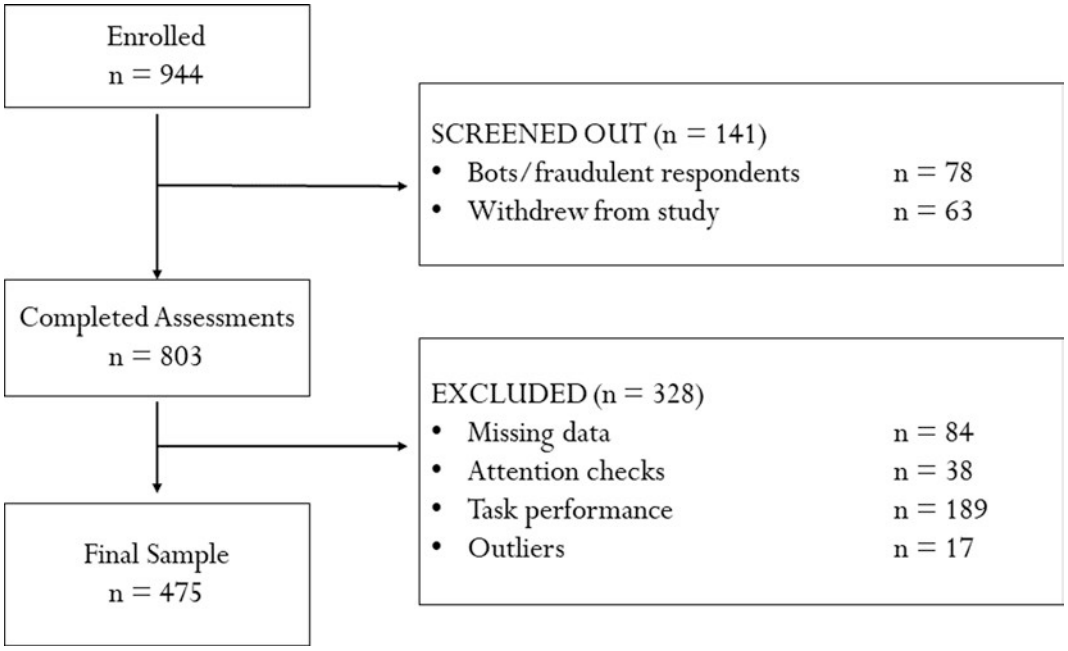


**Fig. 1.** Flow diagram mapping data collection, cleaning, and reasons for exclusion.

**Table 2**
Description of demographic characteristics of the sample.

| Variables | N | |
|---|---|---|
| Sample | 475 | |
| Mean age (SD) | 32.3 (11.8) | |
| Sex, N (%) | | |
| Male | 217 (45.7) | |
| Female | 258 (54.3) | |
| Gender, N (%)Man | 216 (45.5) | |
| Woman | 254 (53.5) | |
| Non-binary | 4 (0.8) | |
| Not listed/ Prefer not to say | 1 (0.2) | |
| Race/Ethnicity, N | | |
| Aboriginal or Torres Strait Islander | 2 | |
| African | 2 | |
| Asian | 101 | |
| Black or African American | 2 | |
| Hispanic or Latino | 4 | |
| Middle Eastern | 4 | |
| South Asian | 30 | |
| White or Caucasian | 317 | |
| Other | 13 | |
| Household income in AUD, N | | |
| < $10,000 | 15 | |
| $10,000 – $20,000 | 17 | |
| $20,000 – $40,000 | 48 | |
| $40,000 – $60,000 | 74 | |
| $60,000 – $80,000 | 58 | |
| $80,000 – $100,000 | 66 | |
| > $100,000 | 197 | |
| **Addictive behaviors** | **M (SD), range** | **Classified as problematic: N (%)** |
| AUDIT | 5.09 (4.82), 0–30 | Harmful/hazardous: 47 (10)<br>Suspected dependence: 15 (3) |
| mYFAS | 0.71 (1.78), 0–10 | Mild: 28 (6)<br>Moderate: 15 (3)<br>Severe: 22 (5) |
| PPCS | 9.61 (5.89), 6–42 | 31 (7) |
| IAT | 16.31 (6.67), 10–48 | 163 (34) |

Note: Sex was defined as biological sex. AUDIT: Alcohol Use Identification Test, no/low problem use (0–7), harmful/ hazardous use (8–14), likely alcohol dependence (≥15); mYFAS: modified Yale Food Addiction Scale 2.0, mild (2–3), moderate (4–5) severe (≥6) symptoms. PPCS: Problematic Pornography Consumption Scale, problematic use (≥20). IAT: an abbreviated version of Young's Internet Addiction Test, problematic use (≥17).

taking behaviors, as measured by the BART (for a *meta*-analysis: Ioannidis et al., 2019). Our study did not find these associations, potentially because the previous studies compared individuals with a PUI "diagnosis" to controls, while our sample included individuals with varying PUI severity. Further, if such deficits are related to vulnerability, they may be more strongly associated with presence vs absence of a disorder rather than its severity. Relatedly, it may be that deficits in these domains are evident in those with high levels of PUI, whereas the current study was conducted in a relatively normative sample. Performance monitoring is necessary to support higher-order functions such as cognitive control (Ferdinand & Czernochowski, 2018), and so reduced performance monitoring may be detectible before other cognitive functions, acting as an early *trans*-behavioral risk indicator for PPU and PUI. However, it is important to note the concurrence of PUI and PPU in our sample (Table 3). Given pornography is predominantly accessed via the internet, our PPU findings may instead be driven by problematic internet use to watch pornography, rather than specifically pornography-related functions.

Some of our findings diverge from that of previous work. For instance, the finding that more severe PUI was associated with less steep delay discounting, or a preference for later larger rewards, is contrary to substantial literature showing individuals with internet addiction are significantly steeper discounters compared to controls (for *meta*-analysis: Cheng et al., 2021). A potential interpretation relates to the fact that we are looking at PUI dimensionally, thus including individuals at both low and high levels of problem severity. Tiego et al. (2021) have argued that PUI as measured by the IAT has a unipolar distribution in which meaningful variance is found at the higher end of the severity spectrum. Given this, including individuals at lower PUI severity may have disrupted the expected effect. Further, the finding that the severity of AE symptoms was not associated with any neurocognitive variables contrasts with previous studies (Franken et al., 2018; Rodrigue et al., 2018; VanderBroek-Stice et al., 2017). One explanation for this may be that neurocognitive functions are more likely to be implicated in more severe AE. The aforementioned studies included samples with 35–100 % of participants endorsing mild-severe food addiction, compared to 18 % currently. Similarly, contrary to expectations, the present study did not find any of the neurocognitive variables were significantly associated with problematic alcohol use. The majority of our sample had no/low alcohol use problems, making it difficult to compare with previous work which has mostly been conducted in hazardous-severe alcohol use cohorts (Stavro et al., 2013). It is possible that neurocognitive risk or sequelae may not be detectable at mild levels of severity. However, our findings may be attributed to the study sample, with PUI being the most prevalent addictive behavior endorsed. This sample may differ significantly from traditional alcohol-focused research cohorts, suggesting a unique group of individuals in this study.

Psychological distress was a *trans*-behavioral correlate across all four addictive behaviors. This is consistent with previous research showing increased psychological distress is associated with multiple addictive and compulsive behaviors (Albertella et al., 2021; Albertella, Pelley, et al., 2019; Sepas et al., 2021). This finding is likely to present a bi-directional relationship between psychological distress and addictive behaviors, a) that addictive behaviors are motivated by way of coping with psychological distress (Burnatowska et al., 2022; Rodriguez et al., 2020), and b) addictive behaviors may themselves enhance distress (Yang et al., 2022).

The present study suggests that different problematic behaviors may be associated with differing trait impulsivity and compulsivity thresholds. Higher SS was significantly associated with more problematic alcohol use, and less AE and PUI. Our alcohol findings are in keeping with the literature in that SS is consistently associated with higher levels of alcohol use, albeit with a small effect (Hittner & Swickert, 2006). Further, our AE findings replicate that of Burrows et al. (2017) who showed a negative relationship between SS and food addiction. Less SS has also been linked with more time spent online (Müller et al., 2016). We also found AE and PUI were significantly positively associated with trait compulsivity, which is in line with previous findings (Albertella et al., 2021). Taken together we see a pattern emerge according to behavior type: after controlling for key covariates, substance-related addictive behaviors (i.e. alcohol use) may be more driven by the desire for sensation, presumably the reinforcing effects of the substance; whilst non-substance addictive behaviors may be more compulsively driven. It is important to note that this does not refute the role of impulsivity in non-substance-related addictive behaviors, nor compulsivity in substance addiction both of which have been well evidenced elsewhere (Everitt & Robbins, 2016; Lee et al., 2019). Rather, the present findings may speak to the relative contributions of these constructs per addictive behavior type (Tiego et al., 2019), particularly in less severe, non-clinical general population cohorts.

### 4.1. Limitations and future directions

Despite the strengths of our study, namely, a large sample size, the assessment of multiple addictive behaviors, and a comprehensive evaluation of addiction-specific neurocognitive correlates for each behavior, our findings should be considered in light of several limitations. The most notable limitation is the relatively low levels of severity (see appendix Fig. A1) in the study sample preventing the generalizability of

**Table 3**
Spearman correlations corrected for multiple comparisons.

| Variable | *M* | *SD* | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1.mYFAS | 5.09 | 4.82 | – | | | | | | | | | | | | | | | | | | |
| 2.PPCS | 9.61 | 5.89 | 0.00 | – | | | | | | | | | | | | | | | | | |
| 3.IAT | 16.31 | 6.67 | **0.29***** | **0.37***** | – | | | | | | | | | | | | | | | | |
| 4.AUDIT | 0.71 | 1.78 | 0.07 | 0.10 | −0.02 | – | | | | | | | | | | | | | | | |
| 5. SST: SSRT | 0.36 | 0.09 | 0.02 | **−0.17*** | **−0.17*** | −0.04 | – | | | | | | | | | | | | | | |
| 6. VMAC: VMAC score | 0.01 | 0.05 | −0.02 | 0.04 | −0.01 | −0.03 | −0.06 | – | | | | | | | | | | | | | |
| 7. BART: *M* pre-committed pumps | 57.29 | 16.51 | −0.08 | 0.11 | 0.01 | 0.06 | −0.12 | 0.05 | – | | | | | | | | | | | | |
| 8. CST: Switch cost latency | 0.30 | 0.20 | 0.01 | 0.00 | −0.03 | −0.05 | 0.13 | −0.03 | −0.06 | – | | | | | | | | | | | |
| 9. DDT: Log *k* | −4.60 | 1.67 | 0.01 | 0.08 | −0.06 | 0.03 | −0.04 | 0.03 | 0.01 | 0.04 | – | | | | | | | | | | |
| 10. EAT: Error awareness | 67.00 | 34.88 | −0.03 | −0.04 | −0.02 | 0.04 | −0.14 | −0.04 | 0.02 | −0.09 | −0.15 | – | | | | | | | | | |
| 11. SDT: *w* | 0.51 | 0.36 | −0.07 | 0.10 | −0.01 | −0.03 | −0.11 | 0.05 | 0.00 | −0.03 | **−0.18*** | 0.15 | – | | | | | | | | |
| 12. N-Back: 3-back d' | 2.15 | 1.16 | −0.07 | 0.02 | −0.02 | 0.06 | −0.04 | 0.03 | 0.02 | 0.05 | −0.12 | **0.19*** | 0.14 | – | | | | | | | |
| 13. SUPPS-P: Lack of perseverance and premeditation | 7.50 | 1.76 | 0.02 | 0.04 | 0.13 | 0.12 | −0.10 | 0.02 | 0.06 | −0.12 | 013 | −0.04 | −0.14 | −0.04 | – | | | | | | |
| 14. SUPPS-P: Urgency | 8.42 | 2.45 | **0.20***** | **0.23***** | **0.34***** | 0.08 | −0.03 | 0.01 | −0.04 | 0.01 | 0.12 | −0.10 | −0.07 | −0.07 | **0.32***** | – | | | | | |
| 15. SUPPS-P: Sensation seeking | 9.41 | 2.79 | −0.13 | 0.14 | −0.01 | **0.25***** | 0.01 | −0.01 | **0.17*** | −0.04 | 0.13 | −0.06 | −0.05 | 0.04 | 0.11 | **0.25***** | – | | | | |
| 16. CHI-T: Trait compulsivity score | 27.23 | 6.19 | **0.27***** | 0.11 | **0.33***** | −0.03 | −0.05 | 0.03 | −0.05 | 0.02 | 0.02 | 0.02 | −0.03 | −0.03 | **−0.28***** | **0.34***** | −0.02 | – | | | |
| 17. DASS: Total score | 12.39 | 11.36 | **0.35***** | 0.15 | **0.53***** | 0.15 | −0.08 | −0.07 | −0.08 | 0.00 | −0.03 | 0.03 | −0.01 | −0.04 | 0.01 | **0.35***** | −0.02 | **0.40***** | – | | |
| 18.Age | 32.32 | 11.85 | −0.06 | **−0.21***** | **−0.39***** | 0.00 | **0.34***** | −0.01 | 0.03 | **0.21***** | −0.06 | −0.09 | −0.04 | −0.06 | **−0.17*** | −0.08 | **−0.25***** | −0.09 | **−0.17*** | **−0.27***** | – |
| 19. Sex (F) | – | – | **0.25***** | **−0.52***** | −0.08 | 0.00 | 0.09 | −0.01 | **−0.18*** | −0.03 | −0.11 | 0.02 | −0.08 | −0.08 | −0.11 | 0.03 | −0.08 | **−0.24***** | 0.02 | 0.12 | 0.03 – |

Note: *p* values were adjusted for multiple comparisons using Holm method; **p* <.05, ***p* <.01, ****p* <.001.

**Table 4**
Linear regression model with bootstrapping for problematic alcohol use.

| | β | *SE* | 95 % CI | | *p* |
|---|---|---|---|---|---|
| | | | Lower | Upper | |
| Demographics | | | | | |
| Age | 0.16 | 0.02 | 0.02 | 0.10 | 0.002** |
| Sex (F) | 0.01 | 0.51 | 1.09 | 0.94 | 0.921 |
| Neurocognition | | | | | |
| SST: SSRT | 0.06 | 2.87 | 8.39 | 3.14 | 0.332 |
| VMAC: VMAC score | 0.01 | 4.10 | 7.55 | 9.05 | 0.912 |
| BART: *M* pre-committed pumps | 0.04 | 0.02 | 0.02 | 0.04 | 0.458 |
| CST: Switch cost latency | 0.02 | 0.00 | 0.00 | 0.00 | 0.666 |
| EAT: Error awareness | 0.03 | 0.01 | 0.01 | 0.02 | 0.606 |
| SDT: *w* | 0.07 | 0.56 | 2.05 | 0.15 | 0.088 |
| N-Back: 3-back d' | 0.04 | 0.15 | 0.15 | 0.45 | 0.291 |
| DDT: Log *k* | 0.00 | 0.12 | 0.24 | 0.21 | 0.948 |
| Covariates | | | | | |
| DASS: Total score | 0.22 | 0.03 | 0.03 | 0.15 | 0.003** |
| SUPPS-P: Lack of perseverance and premeditation | 0.04 | 0.16 | 0.20 | 0.40 | 0.475 |
| SUPPS-P: Urgency | 0.05 | 0.11 | 0.14 | 0.31 | 0.440 |
| SUPPS-P: Sensation seeking | 0.17 | 0.09 | 0.11 | 0.44 | 0.003** |
| CHI-T: Trait compulsivity score | 0.05 | 0.05 | 0.13 | 0.06 | 0.503 |

Note. β: Unstandardized coefficient; SE: Standard error; **p* <.05, ***p* <.01, ****p* <.001.

**Table 5**
Linear regression model with bootstrapping for addictive eating.

| | β | *SE* | 95 % CI | | *p* |
|---|---|---|---|---|---|
| | | | Lower | Upper | |
| Demographics | | | | | |
| Age | 0.05 | 0.01 | 0.01 | 0.02 | 0.310 |
| Sex (F) | 0.19 | 0.14 | 0.41 | 0.96 | <0.001*** |
| Neurocognition | | | | | |
| SST: SSRT | 0.06 | 0.93 | 0.79 | 2.94 | 0.257 |
| VMAC: VMAC score | 0.02 | 1.54 | 2.40 | 3.77 | 0.698 |
| BART: *M* pre-committed pumps | 0.04 | 0.00 | 0.00 | 0.01 | 0.350 |
| CST: Switch cost latency | 0.02 | 0.00 | 0.00 | 0.00 | 0.714 |
| EAT: Error awareness | 0.01 | 0.00 | 0.00 | 0.00 | 0.918 |
| SDT: *w* | 0.03 | 0.23 | 0.58 | 0.32 | 0.579 |
| N-Back: 3-back d' | 0.03 | 0.06 | 0.06 | 0.16 | 0.391 |
| DDT: Log *k* | 0.07 | 0.05 | 0.01 | 0.17 | 0.109 |
| Covariates | | | | | |
| DASS: Total score | 0.26 | 0.01 | 0.02 | 0.06 | <0.001*** |
| SUPPS-P: Lack of perseverance and premeditation | 0.03 | 0.06 | 0.08 | 0.14 | 0.621 |
| SUPPS-P: Urgency | 0.08 | 005 | 0.03 | 0.14 | 0.195 |
| SUPPS-P: Sensation seeking | 0.12 | 0.03 | 0.14 | 0.02 | 0.004** |
| CHI-T: Trait compulsivity score | 0.18 | 0.02 | 0.02 | 0.09 | 0.002** |

Note. β: Unstandardized coefficient; SE: Standard error; **p* <.05, ***p* <.01, ****p* <.001.

our findings to populations with more severe addictive behaviors. Although rates of AE and PPU corresponded with what has previously been estimated in general population samples (4–15 % for AE and 7 % for PPU; Mennig et al., 2020; Meule & Gearhardt, 2019), rate of hazardous alcohol use was well below Australian population estimates (22 %; O'Brien et al., 2020). We recommend that future research in community samples focus recruitment efforts to target individuals at more severe levels of addictive behavior. Contrary to this, the rate of PUI in the study sample was much higher than global estimates (6 %; Cheng & Li, 2014), suggesting our sample experienced more PUI than what would typically be observed in the general population. An additional limitation was the unsupervised nature of data collection. While online remote-access data collection is beneficial when wanting to target demographically diverse samples at scale, it also requires a rigorous data cleaning protocol which necessitated the removal of just under 40 % of the

**Table 6**
Linear regression model with bootstrapping for problematic pornography use.

| | β | *SE* | 95 % CI | | *p* |
|---|---|---|---|---|---|
| | | | Lower | Upper | |
| Demographics | | | | | |
| Age | 0.04 | 0.02 | 0.06 | 0.03 | 0.450 |
| Sex (F) | 0.46 | 0.53 | 6.47 | 4.40 | <0.001*** |
| Neurocognition | | | | | |
| SST: SSRT | 0.04 | 2.26 | 6.71 | 0.97 | 0.287 |
| VMAC: VMAC score | 0.03 | 4.09 | 3.54 | 12.20 | 0.291 |
| BART: *M* pre-committed pumps | 0.01 | 0.01 | 0.03 | 0.03 | 0.883 |
| CST: Switch cost latency | 0.00 | 0.00 | 0.00 | 0.00 | 0.924 |
| EAT: Error awareness | 0.10 | 0.01 | 0.03 | 0.00 | 0.049* |
| SDT: *w* | 0.03 | 0.67 | 0.75 | 1.88 | 0.400 |
| N-Back: 3-back d' | 0.05 | 0.24 | 0.70 | 0.21 | 0.298 |
| DDT: Log *k* | 0.05 | 0.16 | 0.49 | 0.15 | 0.327 |
| Covariates | | | | | |
| DASS: Total score | 0.20 | 0.03 | 0.04 | 0.16 | <0.001*** |
| SUPPS-P: Lack of perseverance and premeditation | 0.04 | 0.20 | 0.26 | 0.53 | 0.581 |
| SUPPS-P: Urgency | 0.14 | 0.13 | 0.06 | 0.59 | 0.013* |
| SUPPS-P: Sensation seeking | 0.03 | 0.09 | 0.24 | 0.13 | 0.542 |
| CHI-T: Trait compulsivity score | 0.05 | 0.06 | 0.06 | 0.16 | 0.414 |

Note. β: Unstandardized coefficient; SE: Standard error; *$p$ <.05, **$p$ <.01, ***$p$ <.001.

**Table 7**
Linear regression model with bootstrapping for problematic use of the internet.

| | β | *SE* | 95 % CI | | *p* |
|---|---|---|---|---|---|
| | | | Lower | Upper | |
| Demographics | | | | | |
| Age | 0.17 | 0.02 | 0.14 | 0.05 | <0.001*** |
| Sex (F) | 0.15 | 0.54 | 3.07 | 0.98 | <0.001*** |
| Neurocognition | | | | | |
| SST: SSRT | 0.01 | 2.90 | 4.81 | 6.37 | 0.801 |
| VMAC: VMAC score | 0.00 | 5.13 | 10.66 | 9.10 | 0.886 |
| BART: *M* pre-committed pumps | 0.03 | 0.02 | 0.02 | 0.04 | 0.444 |
| CST: Switch cost latency | 0.02 | 0.00 | 0.00 | 0.00 | 0.597 |
| EAT: Error awareness | 0.09 | 0.01 | 0.03 | 0.00 | 0.028* |
| SDT: *w* | 0.07 | 0.76 | 2.81 | 0.22 | 0.093 |
| N-Back: 3-back d' | 0.02 | 0.22 | 0.55 | 0.31 | 0.555 |
| DDT: Log *k* | 0.10 | 0.18 | 0.75 | 0.04 | 0.025* |
| Covariates | | | | | |
| DASS: Total score | 0.37 | 0.03 | 0.15 | 0.29 | <0.001*** |
| SUPPS-P: Lack of perseverance and premeditation | 0.08 | 0.19 | 0.06 | 0.71 | 0.102 |
| SUPPS-P: Urgency | 0.15 | 0.14 | 0.13 | 0.67 | 0.003** |
| SUPPS-P: Sensation seeking | 0.09 | 0.09 | 0.40 | 0.04 | 0.014* |
| CHI-T: Trait compulsivity score | 0.12 | 0.05 | 0.03 | 0.24 | 0.010* |

Note. β: Standardized coefficient; SE: Standard error; *$p$ <.05, **$p$ <.01, ***$p$ <.001.

dataset. Post-hoc comparisons of our sample for those whose data was screened out found that the individuals who were removed from the analysis had significantly higher AE, PUI, and lack of perseverance/premeditation than included participants (see appendix). As such, our sample may be biased towards less impulsive individuals which may not be representative of the wider community. Future research should consider the trade-off associated with unsupervised data collection.

The use of gamified neurocognitive tasks to assess aspects of neurocognitive function may also have impacted our findings. Whilst gamification can enhance engagement and motivation (Lumsden et al., 2016), the addition of game-like elements did cause some of the tasks to deviate from their traditional counterparts. For instance, enhanced complexity of the visual display which may affect the salience of visual cues, potentially impacting paradigms that rely on distractor cues such as the VMAC task. Further, the BrainPAC SST utilized a points element in which faster responses earned more points, potentially encouraging faster but less accurate responding and thus impacting SSRT calculations.

Finally, the cross-sectional nature of the present study limits our ability to determine causal relationships between the variables of interest. The next natural step would be to conduct a longitudinal evaluation of the same predictors to determine the key mechanisms that predict the development of AE, PPU, and PUI over time. This would better inform intervention and prevention targets for non-substance addictive behaviors and could shed light on the relative contribution of impulsivity and compulsivity (in terms of cognition or traits) at different stages of addiction (e.g. as relates to duration of addictive symptoms).

In conclusion, the present study revealed that different addictive behaviors may have unique neurocognitive mechanisms. There are likely partly distinct mechanisms or pathways to addiction depending on the addictive behavior in question. This has key implications for early intervention, in particular, our study supports the need for tailored treatments that focus on the specific behavior-related neurocognitive functions rather than assuming cognitive dysfunction is necessarily the same across addictions.

## CRediT authorship contribution statement

**Erynn Christensen:** Conceptualization, Methodology, Software, Formal analysis, Investigation, Data curation, Writing – original draft, Writing – review & editing. **Lucy Albertella:** Conceptualization, Supervision, Writing – review & editing. **Samuel R. Chamberlain:** Writing – review & editing. **Maja Brydevall:** Software, Writing – review & editing. **Chao Suo:** Software. **Jon E. Grant:** Writing – review & editing. **Murat Yücel:** Funding acquisition, Supervision, Writing – review & editing. **Rico Sze Chun Lee:** Funding acquisition, Conceptualization, Supervision, Writing – review & editing.

## Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## Data availability

Data will be made available on request.

## Acknowledgement

Funding for this study was provided by the National Health and Medical Research Council (NHMRC) Project Grant [APP1162031] and NHMRC Medical Research Future Fund Investigator Grant [APP1193946]. This research was also supported by an Australian Government Research Training Program (RTP) Scholarship. Author disclosures are as follows: LA and MY have received funding from the David Winston Turner Endowment Fund. MY and RSCL have received funding from the NHMRC. MY has received funding from the Australian Research Council (ARC), Australian Defence Science and Technology (DST), the Department of Industry, Innovation and Science (DIIS), the National Institutes of Health (NIH, USA), the Wilson Foundation, and sponsored Investigator-Initiated trials including Incannex Healthcare Ltd. MY has also received payments in relation to court-, expert witness-, and/or expert review-reports. MY also sits on the Advisory Boards of: Centre of The Urban Mental Health, University of Amsterdam; Monash Biomedical Imaging Centre; and Enosis Therapeutics. JEG has received research grant support from NIDA, and from Biohaven, Janssen, and Boehringer Ingelheim Pharmaceuticals. He receives yearly

compensation from Springer Publishing for acting as Editor-in-Chief of the Journal of Gambling Studies and has received royalties from Oxford University Press, American Psychiatric Publishing, Inc., Norton Press, and McGraw Hill. SRC and LA receive honoraria for editorial work at Elsevier. The above funding sources have had no role in the present study design, collection, analysis or interpretation of the data, writing the manuscript, or the decision to submit this paper for publication.

## Appendix

**Table A1**
Variables that significantly differed between the sample retained in the study and the sample removed during data cleaning.

| | Mean(SD) | | |
|---|---|---|---|
| **Variable** | **Sample retained** | **Sample removed** | **Test statistic** |
| mYFAS symptom count | 0.72 (1.79) | 2.39 (2.95) | 47366*** |
| IAT score | 16.3 (6.68) | 22.46 (8.86) | 45768*** |
| Lack of perseverance and premeditation | 7.52 (1.77) | 8.84 (2.19) | 6.02*** |

Note: ***$p$ <.001; Mann-Whitney $U$ test was used for non-parametric comparisons (mYFAS and IAT); $t$-test was used for parametric comparison (lack of perseverance and premeditation).

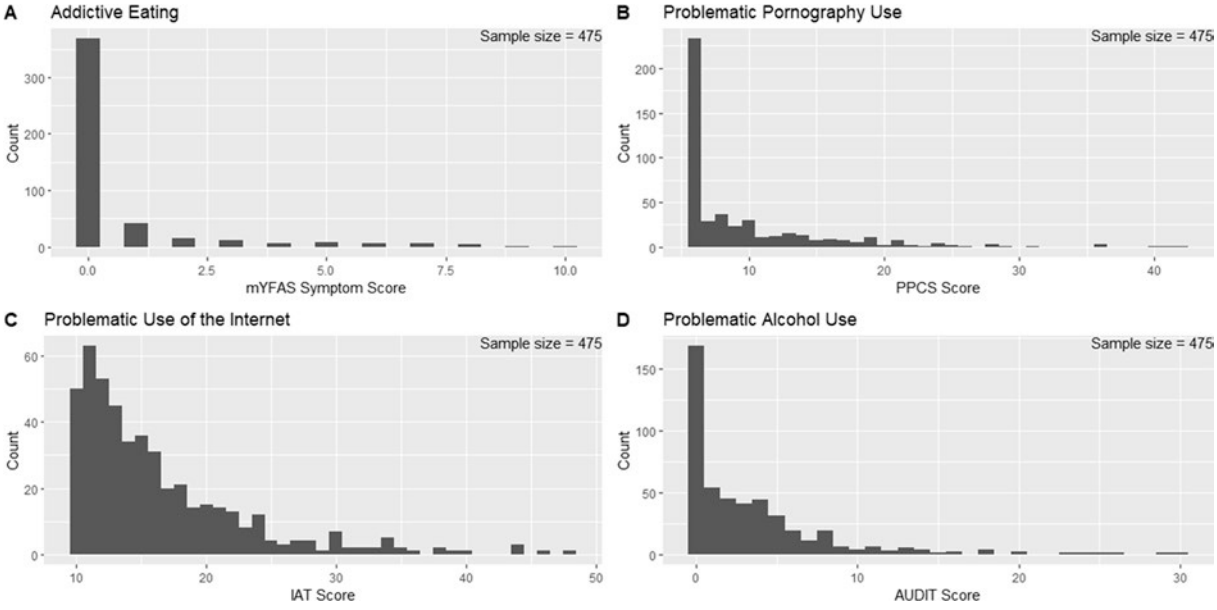


**Fig. A1.** Histograms of addictive behaviors in the sample.

## Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.addbeh.2023.107904.

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*Chapter 4*

# The longitudinal neurocognitive predictors of addictive behaviours

**Christensen**, **E**., Albertella, L., Chamberlain, S.R., Suo, C., Brydevall, M., Grant, J.E., Yücel, M., & Lee, R.S.C. A comprehensive evaluation of the neurocognitive predictors of problematic alcohol use, eating, pornography, and internet use: A 6-month longitudinal study. Submitted to *Journal of Behavioral Addictions.*

## 4.1 Abstract

Cognitive control and reward-related abnormalities are centrally implicated in addiction. However, findings from longitudinal studies addressing neurocognitive predictors of addictive behaviours are mixed. Further, little work has been conducted predicting non-substance related addictive behaviours. Our study aimed to assess predictors of substance and non-substance addictive behaviours in a community sample, systematically evaluating each neurocognitive function's independent influence on addictive behaviour. Australian residents completed online neurocognitive tasks and surveys at baseline and 3-month follow-up. Self-report scales assessed problematic alcohol use, addictive eating (AE), problematic pornography use (PPU), and problematic internet use (PUI) at 3- and 6-month follow-ups. N=294(51.7% female; M[SD] age=24.8[4.7] years) were included in analyses. Linear regressions with bootstrapping assessed neurocognitive predictors for each addictive behaviour (baseline predicting 3-month and 3-month predicting 6-month follow-up). Poorer performance monitoring at baseline predicted higher AE at 3-month follow-up (β=-0.16, *p*=.004), and more reward-related attentional capture at 3-months predicted higher AE at 6-month follow-up (β=0.14, *p*=.033). Less delay discounting at 3-months predicted higher PPU at 6-month follow-up (β=-0.16, *p*=.014). Less reward-related attentional capture (β=-0.14, *p*=.003) and less risk taking under ambiguity (β=-0.11, *p*=.029) at baseline predicted higher PUI at 3-month follow-up. All findings were of small effect size. Results failed to show that any of the neurocognitive variables predicted problematic alcohol use. Our findings indicate both specific cognitive control and reward-related neurocognitive functions predict non-substance addictive behaviours. Our findings also suggest that there may be partially distinct neurocognitive mechanisms contributing to addiction, depending on the specific addictive behaviour under consideration.

## 4.2 Introduction

A core set of neurocognitive dysfunction has been proposed to be critical across addictive behaviours (Yücel et al., 2019, 2021), namely deficits in cognitive control and aberrant reward-related processes (Bechara, 2005; Smith et al., 2014; Verdejo-Garcia & Albein-Urios, 2021; Volkow & Boyle, 2018; Volkow & Morales, 2015; Yücel & Lubman, 2007). However, a recent systematic review revealed that individual differences in these cognitive control and reward-related processes do not consistently predict addiction outcomes in longitudinal samples (Christensen, Brydevall, et al., 2023). This finding was in part attributed to methodological disparities in the literature, such as the inconsistent selection of neurocognitive domains, choice of tasks, and choice of confounders (or lack thereof) to include in models. There has also been very little literature investigating the neurocognitive predictors of non-substance addictive disorders, making it difficult to determine whether there are trans-addiction neurocognitive mechanisms or if different addictive behaviours have unique neurocognitive predictors. These insights would allow for the development of more targeted and effective interventions that address distinct pathways involved in specific addictive behaviours.

Longitudinal research on the neurocognitive mechanisms of addiction has predominantly focussed on substance use disorders (SUDs) and problem gambling (PG). However, several non-substance related addictive behaviours merit investigation. Addictive Eating (AE), Problematic Pornography Use (PPU), and Problematic Use of the Internet (PUI) are common behavioural problems, affecting 2.6% to 15% of the population globally (Kumar et al., 2021; Mennig et al., 2020; Meule & Gearhardt, 2019; Pan et al., 2020). In keeping with SUDs and PG, AE, PPU and PUI have been linked to significantly poorer psychosocial outcomes (Burmeister et al., 2013; Burrows et al., 2018; Camilleri et al., 2021; Fineberg et al., 2018; Floros & Ioannidis, 2021; Kuss et al., 2014; Raj et al., 2022; Rodrigue et al., 2018).

Similar to SUDs, neurocognitive changes have been observed in individuals with AE, PUI, and PPU, such as decreased response inhibition, poorer performance monitoring ability, inflexible task shifting, and risky decision-making (Franken et al., 2018; Ioannidis et al., 2019; Müller et al., 2023; Odlaug et al., 2011; Rodrigue et al., 2018; Smith et al., 2014; Zhou et al., 2013), alongside enhanced

attentional bias towards addiction or reward-related cues (Adams et al., 2019; Albertella, Pelley, et al., 2019; Jeromin et al., 2016; Mechelmans et al., 2014; Nikolaidou et al., 2019), steeper temporal discounting (Amlung et al., 2017; Antons et al., 2019), and riskier decision-making (Ioannidis et al., 2019). However, little longitudinal research exists expressly testing predictive factors. Only a handful of studies have evaluated the neurocognitive predictors of non-substance addictions and, of these, none have looked at AE, PUI, or PPU specifically (Christensen, Brydevall, et al., 2023). This is especially important given work in SUDs suggests cross-sectional correlates of addiction do not necessarily imply predictive mechanisms, for example, delayed discounting and decreased response inhibition have been shown to be key correlates of substance addiction (Amlung et al., 2017; Smith et al., 2014), yet these relationships have not been consistently replicated longitudinally (Christensen, Brydevall, et al., 2023).

The current lack of evidence suggesting neurocognitive functions predict consumption (i.e. frequency/quantity), severity, or diagnosis of addiction (Christensen, Brydevall, et al., 2023), may be due to the heterogeneous nature of past longitudinal studies, making them difficult to compare. In a recent systematic review of the literature by Christensen and colleagues (2023), 44% of studies focused on a single, specific neurocognitive function (Audrain-McGovern et al., 2009; Chen et al., 2021; Cousijn et al., 2015; Fröhner et al., 2022; Jones et al., 2021; Peeters et al., 2014; van Hemel-Ruiter et al., 2015). Those that looked at multiple functions predominantly evaluated a single domain, for example reward valuation, assessed via delay discounting and probability discounting tasks (Bernhardt et al., 2017; Kräplin et al., 2020), or multiple tasks that evaluated aspects of cognitive control (Bø et al., 2017; Rubio et al., 2008), for example response inhibition and set shifting. Rarely were multiple neurocognitive tasks that independently tapped into both reward-related and cognitive control functions included in the same model (Fernández-Artamendi et al., 2018; Whelan et al., 2014). This is a critical area for investigation given both cognitive control and reward-related neurocognitive processes interact and in many cases overlap (Criaud & Boulinguez, 2013; Ridderinkhof et al., 2004). Interrogating the unique role of specific cognitive control and reward-related functions in the same model, will allow us to determine each function's relative contribution to addictive behaviours. This

knowledge can be used to identify individuals at higher risk of developing addiction and inform novel treatment targets and more tailored therapeutic strategies.

A further issue with current studies is the preponderance of clinical samples. The trajectory from the onset of addiction problems to receiving clinical care can take a median of 18 years (Chapman et al., 2015). Studies that assess individuals in clinical treatment settings only capture a small subset of those affected by addiction (Grant et al., 2015; Hasin et al., 2013). Investigating general community samples affords the opportunity to identify and study these individuals before they would typically be accessible in treatment settings. We can also adopt a dimensional approach that encompasses a spectrum of addictive behaviour severities. This is especially important as even individuals engaging in addictive behaviours at less severe levels also experience adverse outcomes (Shankman et al., 2009). Understanding the neurocognitive mechanisms that predict addictive behaviours dimensionally will better identify early risk indicators for addiction and provide insights into key treatment targets at different stages of illness.

The aim of this study was to evaluate the extent to which neurocognitive measures predict subsequent substance (alcohol) and non-substance (AE, PPU and PUI) addictive behaviours in a general community sample. This study took a systematic and comprehensive approach to neurocognitive assessment (Lee et al., 2023) by evaluating the independent influence of multiple individual neurocognitive functions on addictive behaviours. Given literature looking at the neurocognitive functions associated with AE, PPU and PUI is still sparse, we first evaluated the cross-sectional correlates of each addictive behaviour, before then modelling these relationships longitudinally over a 6-month period. To determine whether these relationships were replicable, we evaluated the relationship between neurocognitive functions assessed at baseline and addictive behaviour at 3-month follow-up, and then the relationship between neurocognitive functions assessed at 3-months and addictive behaviour at 6-month follow-up. All models accounted for both trait impulsivity and compulsivity as well as key covariates (i.e. stress, sex and age).

## 4.3 Methods

### Participants

This study was embedded within a larger cohort study (Christensen, Albertella, et al., 2023). Nine-hundred-and-forty-four participants were recruited from Prolific and online advertisements via popular social media sites and enrolled in the wider study; 400 of which, aged between 18 and 35 years, were invited to take part in the longitudinal protocol and made up the study sample for the present paper. This age band was chosen given the median age of onset for substance use/addictive behaviour disorders is 25 years of age (Solmi et al., 2022). Participants were Australian residents, who were not colour blind and self-reported an absence of a neurological disorder (i.e. stroke, brain injury, and dementia) and an absence of a history of a psychotic disorder.

### Measures

A detailed description of the measures included in this study can be found elsewhere (Christensen, Albertella, et al., 2023; Lee et al., 2023). Participants provided basic demographic information (i.e. age, sex, average household income and education status) and completed the following tasks and self-report surveys.

*Neurocognitive Tasks:*

Tasks were selected to assess neurocognitive functions linked to addiction (Yücel et al., 2019), see Table 1 for details of each task. Four of the eight tasks were delivered by the BrainPark Assessment of Cognition application (BrainPAC), a novel expert endorsed digital assessment tool for addictive disorders (refer to Lee et al., 2023 for psychometric information). The remaining four were delivered via Inquisit 5 (2018).

**Table 1.** Neurocognitive tasks included in the study

| Function | Task | Brief description |
|---|---|---|
| Response inhibition | The BrainPAC Stop Signal Taks (SST)<br>(Lee et al., 2023) | A gamified visual cue stop signal paradigm. The primary outcome metric is stop signal reaction time (SSRT). Higher SSRT indicated poorer inhibitory control. |
| Reward learning (reward-related attentional bias) | The BrainPAC Value Modulated Attentional Capture (VMAC) Task<br>(Lee et al., 2023) | A gamified version of a standard VMAC task (Albertella, Pelley, et al., 2019; Le Pelley et al., 2015). The primary outcome metric is the VMAC score, the difference in reaction time between trials with a high versus low value distractor present. The VMAC score is averaged across the last two blocks of the task. Higher values indicate more reward-related attentional capture. |
| Reward learning (goal-directed vs habitual) | The BrainPAC Sequential Decision-Making Task (SDT)<br>(Lee et al., 2023) | A gamified two-stage choice task. The primary outcome metric is mixing weight (*w*). Higher scores indicate more goal-directed (model-based) decision-making. |
| Reward valuation (risky decision-making under uncertainty) | The BrainPAC Balloon Analogue Risk Task (BART)<br>(Lee et al., 2023) | A gamified version of the BART stretch variant. The primary outcome metric is the mean pre-committed pumps across all trials. Higher values indicate riskier choice in the face of uncertainty. |
| Flexible updating | N-back Task<br>(Ragland et al., 2002; Inquisit 5, 2018) | A letter sequencing go/no-go task that progressively increases working memory load. The primary outcome metric is the parametric measure of sensitivity (d'), which in this study was calculated from 3-back trials. Higher d' values indicate more flexible updating. |
| Goal selection; updating, representation and maintenance | Category Switch Task (CST)<br>(Friedman et al., 2008; Inquisit 5, 2018) | A task switching paradigm. The primary outcome metric is the latency switch cost, calculated as the difference in reaction time on switch versus non-switch trials. Higher values indicate poorer task switching. |
| Performance monitoring | Error Awareness Task (EAT)<br>(Hester et al., 2007; Inquisit 5, 2018) | A visual go/no-go paradigm in which participants indicate their error awareness following any commission error. The primary outcome metric is percentage awareness of commission errors. Higher values indicate better performance monitoring. |
| Temporal discounting | Monetary Choice Questionnaire (MCQ)<br>(Kirby et al., 1999) | A 27-item questionnaire asks the participant to choose between two hypothetical reward options, a smaller reward now or a larger reward at some point in the future. The primary outcome measure is discounting rate (log *k*). Higher values indicate preference for sooner but smaller rewards. |

*Addictive behaviours:*

Problematic alcohol use was assessed by the Alcohol Use Disorder Identification Test (AUDIT; Saunders et al., 1993), AE was assessed via the modified Yale Food Addiction Scale (mYFAS 2.0; Schulte & Gearhardt, 2017), PPU was assessed by the abbreviated Young's Internet Addiction Test (IAT-10; Tiego et al., 2021), and PPU was assessed via the Problematic Pornography Consumption Scale (PPCS-6: Bőthe et al., 2021). Participants reported addictive behaviours using the corresponding scale reflecting on the previous three months, with zero assigned if no engagement.

*Covariates:*

The total score of the Depression Anxiety Stress Scale was used to assess psychological distress (DASS-21; Szabó, 2010). The Cambridge-Chicago Compulsivity Trait Scale (CHI-T; Chamberlain & Grant, 2018; Tiego et al., 2023) assessed trait compulsivity. The Short UPPS Impulsive Behaviour Scale (SUPPS-P; Cyders et al., 2014) assessed trait impulsivity, with three scores: urgency, lack of planning/persistence, and sensation seeking, as validated by factor analyses. (Billieux et al., 2012, 2021).

**Procedure**

The study was approved by the Monash University Human Research Ethics Committee [26088]. All subjects were informed about the study and provided informed consent. Assessments were delivered online using Qualtrics (https://www.qualtrics.com). At baseline and 3-month follow-up, participants completed three one-hour assessment sessions across three consecutive days. During each session, the neurocognitive tasks were separated by self-report surveys (trait and behaviour scales). The order of task presentation was counterbalanced. At 6-month follow-up participants completed a single 30-minute questionnaire that evaluated addictive behaviour engagement.

**Statistical analysis**

The data underwent a cleaning procedure to ensure data quality. The cleaning procedures were specified in advance of data curation by the study team. Implausible responses and poor performance presumedly due to lack of effort were identified and removed via attention check questions, neurocognitive task performance at less than chance levels (as per Lee et al., 2023; Albertella, Watson, et al., 2019), as well as task-specific cleaning procedures (e.g. SST go trial

accuracy, stop trial accuracy, [Verbruggen et al., 2019] and Independent Race Model check [Band et al., 2003]). Differences between individuals whose data was filtered out compared with included individuals were investigated using t-tests and Mann Whitney U tests and found to be non-significant. Similarly, t-tests and Mann Whitney U tests were used to compare individuals who returned for each longitudinal assessment, and those lost to follow-up and are reported below.

Power calculations using G-Power (3.1) deemed n=190 was the minimum sample size required for multiple regression analyses with 16 predictors to find a small effect ($f^2$=0.10, power=0.80). Statistical outliers on neurocognitive measures ($\geq$3 standard deviations from the mean) were removed (Field, 2012). All analyses were conducted on complete data sets (i.e. participants who provided data for all variables of interest). Bivariate Spearman correlations (adjusted for multiple comparisons using the Holm method: Holm, 1979), investigated relationships among variables of interest at each time point (Table S1-3).

Three multiple regression models were generated for each outcome behaviour of interest. The first model evaluated cross-sectional relationships between neurocognition, traits, covariates, and addictive behaviour at baseline. The second model included baseline variables as predictors of addictive behaviour at 3-month follow-up, accounting for baseline addictive behaviour severity, and the third model included variables measured at 3-months to predict addictive behaviour at 6-month follow-up, accounting for 3-month addictive behaviour severity (Tables 3-6). To prevent the impact of Type I error rate inflation on the longitudinal analyses, we implemented a Bonferroni correction to adjust the alpha levels indicating a significant effect. Consequently, a significance level of $p$ <.025 was established to represent a significant effect. The distributions of all four outcome variables at each timepoint were positively skewed (Figure S1), constituting the choice of linear regression models with bootstrapping (5,000 samples; Neal & Simons, 2007). Multicollinearity was assessed for each model independently, with VIF values less than 2.5 indicating no issue of multicollinearity (Johnston et al., 2018). Age, sex, stress, impulsivity, compulsivity, and the respective baseline addictive behaviour scale (measured at 3-month assessment for regressions predicting 6-month outcome) score were included as covariates in each regression model (Eisenberg et al., 2019; Sjoberg & Cole, 2018; Starcke et al., 2016; Tennant et al., 2021). To counter potential bias of our longitudinal

models due to drop-out over the course of the study, as secondary analyses we ran each longitudinal regression using MICE multiple imputation for missing data (bootImpute::bootMice in R; 10 imputations), these results are presented in the supplementary materials (Tables S4-7).

## 4.4 Results

Four hundred participants completed the baseline assessment, 283 completed the 3-month follow-up and 262 completed the 6-month follow-up. After data cleaning (Figure 1) the final sample sizes included in analyses were as follows: n=294 cross-sectional analyses, n=206 predicting 3-month outcomes, and n=138 predicting 6-month outcomes. Participant demographics are displayed in Table 1. When comparing individuals lost to follow-up and those retained at 3-month follow-up, participants lost to follow-up were younger (mean age=23.3 vs 25.4 years; $r_{pb}$=0.26, $p$<.001) and showed increased response inhibition (mean SSRT=0.32 vs 0.34s; Cohen's $d$=0.32, $p$=.005). Further, more females (n=46) were lost to follow up than males (n=38). Individuals lost to follow up at 6-months had higher urgency scores at 3-months (M=9.8) than those retained (M=8.6), Cohen's $d$=-0.47, $p$<.001, and more males did not come back for 6-month follow up (n=20) than females (n=17).

**Figure 1.** Flow diagram mapping data collection, cleaning, and reasons for exclusion.

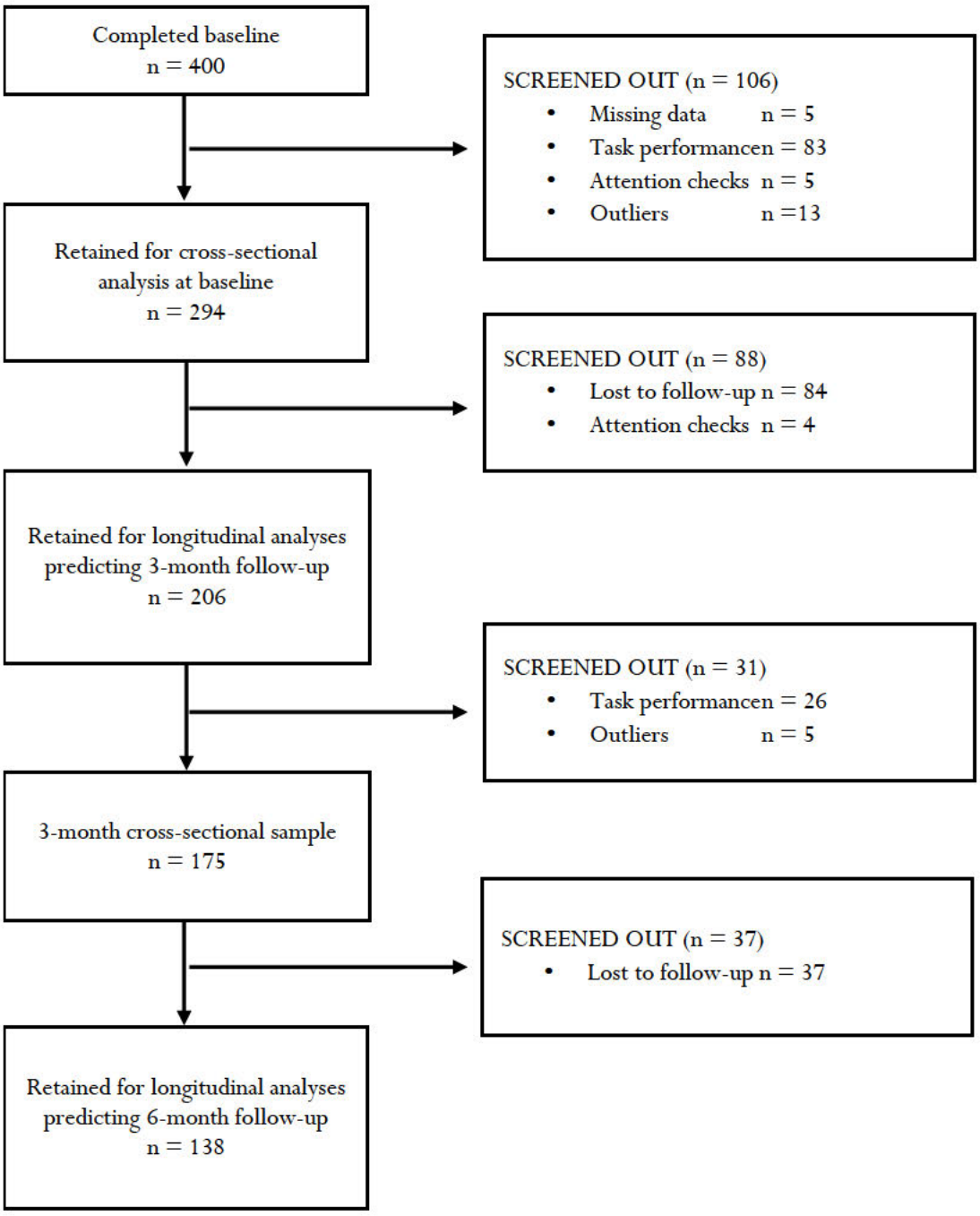


**Table 2.** Demographic and behavioural characteristics

| Variables | BL | 3-mths | 6-mths |
|---|---|---|---|
| N | 294 | 175 | 138 |
| Mean age (SD) | 24.8 (4.7) | 24.7 (4.5) | 25.0 (4.4) |
| Sex, N (%) | | | |
| Female | 152 (51.7) | 90 (51.4) | 73 (52.9) |
| Gender, N (%) | | | |
| Man | 141 (48.0) | 85 (48.6) | 65 (47.1) |
| Woman | 149 (50.7) | 88 (50.3) | 71 (51.4) |
| Non-binary | 3 (1.0) | 1 (0.6) | 1 (0.7) |
| Not listed/ Prefer not to say | 1 (0.3) | 1 (0.6) | 1 (0.7) |
| Ethnicity, N | | | |
| Aboriginal or Torres Strait Islander | 1 | 0 | 0 |
| African | 2 | 0 | 0 |
| Asian | 84 | 57 | 49 |
| Black or African American | 1 | 1 | 0 |
| Hispanic or Latino | 3 | 2 | 1 |
| Middle Eastern | 3 | 3 | 3 |
| South Asian | 23 | 13 | 9 |
| White or Caucasian | 166 | 91 | 69 |
| Other | 11 | 8 | 7 |
| Household income in AUD, N | | | |
| < $10,000 | 13 | 9 | 6 |
| $10,000 – $20,000 | 12 | 10 | 9 |
| $20,000 – $40,000 | 25 | 18 | 12 |
| $40,000 – $60,000 | 55 | 32 | 26 |
| $60,000 – $80,000 | 38 | 25 | 21 |
| $80,000 – $100,000 | 45 | 24 | 19 |
| > $100,000 | 106 | 57 | 45 |
| AUDIT | | | |
| Mean (SD) | 3.0 (3.7) | 2.7 (4.3) | 2.2 (3.8) |
| Range | 0-24 | 0-30 | 0-21 |
| % classified as problematic (hazardous use \| dependence) | 10.2 \| 1.4 | 8.6 \| 1.7 | 2.9 \| 3.6 |
| mYFAS | | | |
| Mean (SD) | 0.7 (1.7) | 0.7 (1.6) | 0.7 (1.8) |
| Range | 0-10 | 0-10 | 0-11 |
| % classified as problematic (mild \| moderate \| severe) | 5.8 \| 3.1 \| 4.1 | 8.0 \| 4.7 \| 3.4 | 6.5 \| 5.1 \| 2.9 |
| PPCS | | | |
| Mean (SD) | 10.1 (5.8) | 10.3 (5.6) | 9.9 (5.9) |
| Range | 6-40 | 6-30 | 6-37 |
| % classified as problematic | 7.1 | 9.1 | 6.5 |
| IAT | | | |
| Mean (SD) | 17.4 (6.8) | 17.3 (6.7) | 16.8 (5.7) |
| Range | 10-48 | 10-42 | 10-37 |
| % classified as problematic | 41.2 | 41.1 | 39.9 |

*Note: Sex was defined as biological sex. Gender was defined as the participant's gender identity at the time of the baseline assessment. AUDIT: Alcohol Use Identification Test, harmful/ hazardous use (≥8), likely alcohol dependence (≥15); mYFAS: modified Yale Food Addiction Scale 2.0, mild (2-3), moderate (4-5) severe (≥6) symptoms. PPCS: Problematic Pornography Consumption Scale, problematic use (≥20). IAT: an abbreviated version of Young's Internet Addiction Test, problematic use (≥17).*

**Cross-sectional model**

Steeper delay discounting (preference for sooner smaller rewards; β=0.16, *p*=.008) was associated with greater AE. No further relationships were found between neurocognition and addictive behaviours.

**Longitudinal multivariate models**

*Predicting problematic alcohol use:*

None of the neurocognitive variables predicted problematic alcohol use at 3- or 6-month follow-up. These results were replicated in regression models using multiple imputation (Table S4).

*Predicting AE:*

Poorer performance monitoring (β=-0.16, *p*=.004) significantly predicted higher AE at 3-month follow-up. Greater reward-related attentional capture (β=0.14, *p*=.036) at 3-month follow-up significantly predicted higher AE at 6-month follow-up. However, this effect did not hold after Bonferroni correction was applied. These results were replicated in the regression models using multiple imputation (Table S5), although the performance monitoring and reward-related attentional capture effects reduced to trend levels (95% CI [-0.01,0.00], *p*=.065; 95% CI [-0.86,10.26], *p*=.097).

*Predicting PPU:*

Neurocognition at baseline did not predict PPU at 3-month follow-up. Less delay discounting (β=-0.16, *p*=.010) at 3-month follow-up predicted higher PPU at 6-month follow-up. These results were not replicated in regression models using multiple imputation which showed neurocognition was no longer a significant predictor of PPU at either time-point (Table S6).

*Predicting PUI:*

Less reward-related attentional capture (β=-0.14, *p*=.003) significantly predicted higher PUI at 3-month follow-up. Less risk taking under uncertainty (β=-0.10, *p*=.030) also significantly predicted higher PUI at 3-month follow-up. However, this effect reduced to trend level after Bonferroni correction was applied. Neurocognition at 3-moth follow-up did not predict PUI at 6-months. Regressions using multiple imputation replicated these findings (Table S7), although the baseline risk-taking effect reduced to trend level (95% CI [-0.09,0.00], *p*=.075).

**Table 3.** Cross-sectional and longitudinal multiple regression models of problematic alcohol use

| Variable | Baseline (N=294) | | | | | Baseline predicting 3-months (N=206) | | | | | 3-months predicting 6-months (N=138) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | β | *SE* | 95% CI | | *p* | β | *SE* | 95% CI | | *p* | β | *SE* | 95% CI | | *p* |
| | | | LL | UL | | | | LL | UL | | | | LL | UL | |
| Demographics | | | | | | | | | | | | | | | |
| Age | 0.04 | 0.05 | -0.06 | 0.13 | .498 | 0.04 | 0.03 | -0.03 | 0.10 | .276 | 0.06 | 0.05 | -0.06 | 0.16 | .372 |
| Sex (F) | 0.14 | 0.44 | 0.19 | 1.87 | .020* | 0.02 | 0.29 | -0.46 | 0.71 | .676 | 0.01 | 0.46 | -0.81 | 1.01 | .821 |
| Neurocognition | | | | | | | | | | | | | | | |
| SST: SSRT | -0.03 | 3.17 | -7.66 | 4.73 | .645 | -0.03 | 2.25 | -5.19 | 2.51 | .494 | -0.07 | 3.41 | -10.46 | 3.13 | .257 |
| VMAC: VMAC score | -0.10 | 5.26 | -19.66 | 1.34 | .079 | -0.01 | 4.51 | -8.25 | 5.99 | .768 | 0.02 | 5.39 | -9.15 | 12.60 | .761 |
| BART: *M* pre-committed pumps | -0.05 | 0.01 | -0.04 | 0.02 | .417 | 0.01 | 0.01 | -0.02 | 0.02 | .824 | -0.04 | 0.01 | -0.03 | 0.02 | .478 |
| CST: Switch cost latency | -0.08 | 0.00 | -0.00 | 0.00 | .189 | 0.02 | 0.00 | -0.00 | 0.00 | .574 | 0.05 | 0.00 | -0.00 | 0.00 | .357 |
| EAT: Error awareness | 0.07 | 0.01 | -0.01 | 0.02 | .231 | 0.01 | 0.00 | -0.01 | 0.01 | .735 | 0.01 | 0.01 | -0.02 | 0.02 | .867 |
| SDT: *w* | -0.03 | 0.61 | -1.37 | 0.93 | .692 | -0.02 | 0.40 | -0.98 | 0.57 | .601 | -0.03 | 0.58 | -1.45 | 0.81 | .572 |
| N-Back: 3-back d’ | 0.11 | 0.19 | -0.02 | 0.72 | .060 | 0.02 | 0.11 | -0.18 | 0.30 | .630 | 0.02 | 0.17 | -0.25 | 0.42 | .647 |
| DDT: Log *k* | 0.02 | 0.13 | -0.21 | 0.31 | .701 | 0.01 | 0.07 | -0.15 | 0.20 | .722 | -0.02 | 0.13 | -0.31 | 0.21 | .719 |
| Covariates | | | | | | | | | | | | | | | |
| AUDIT | - | - | - | - | - | 0.84 | 0.06 | 0.76 | 0.92 | <.001*** | 0.78 | 0.06 | 0.70 | 0.96 | <0.001*** |
| DASS: Total score | 0.22 | 0.02 | 0.03 | 0.12 | <.001*** | 0.04 | 0.01 | -0.02 | 0.04 | .458 | 0.07 | 0.02 | -0.02 | 0.07 | .245 |
| SUPPS-P: Lack of perseverance and premeditation | 0.07 | 0.14 | -0.13 | 0.42 | .273 | 0.00 | 0.12 | -0.19 | 0.19 | .995 | -0.04 | 0.14 | -0.38 | 0.21 | .508 |
| SUPPS-P: Urgency | 0.00 | 0.11 | -0.23 | 0.22 | .985 | 0.09 | 0.08 | -0.01 | 0.29 | .064 | -0.06 | 0.12 | -0.34 | 0.13 | .406 |
| SUPPS-P: Sensation seeking | 0.27 | 0.08 | 0.18 | 0.51 | <.001*** | 0.02 | 0.05 | -0.08 | 0.13 | .660 | 0.04 | 0.08 | -0.11 | 0.21 | .567 |
| CHI-T: Trait compulsivity | -0.04 | 0.05 | -0.11 | 0.06 | .552 | -0.06 | 0.04 | -0.10 | 0.03 | .269 | 0.07 | 0.05 | -0.06 | 0.16 | .360 |

*Note. β: Standardised coefficient; SE: Standard error; *p < .05, **p < .01, ***p < .001*

**Table 4.** Cross-sectional and longitudinal multiple regression models of AE

| Variable | Baseline (N=294) | | | | | Baseline predicting 3-months (N=206) | | | | | 3-months predicting 6-months (N=138) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | β | *SE* | 95% CI | | *p* | β | *SE* | 95% CI | | *p* | β | *SE* | 95% CI | | *p* |
| | | | LL | UL | | | | LL | UL | | | | LL | UL | |
| Demographics | | | | | | | | | | | | | | | |
| Age | 0.01 | 0.02 | -0.03 | 0.05 | .799 | 0.08 | 0.02 | -0.01 | 0.07 | .175 | -0.01 | 0.03 | -0.06 | 0.05 | .891 |
| Sex (F) | 0.18 | 0.19 | 0.22 | 0.96 | .002** | -0.01 | 0.19 | -0.42 | 0.34 | .849 | -0.06 | 0.24 | -0.69 | 0.28 | .398 |
| Neurocognition | | | | | | | | | | | | | | | |
| SST: SSRT | 0.03 | 1.35 | -1.89 | 3.52 | .579 | -0.05 | 1.24 | -3.51 | 1.35 | .355 | -0.01 | 1.83 | -3.89 | 3.49 | .862 |
| VMAC: VMAC score | 0.03 | 2.22 | -3.10 | 5.68 | .610 | -0.02 | 2.31 | -5.31 | 3.82 | .740 | 0.14 | 2.86 | 0.53 | 11.62 | .033* |
| BART: *M* pre-committed pumps | 0.01 | 0.01 | -0.01 | 0.01 | .896 | -0.00 | 0.01 | -0.01 | 0.01 | .953 | -0.11 | 0.01 | -0.02 | 0.00 | .088 |
| CST: Switch cost latency | 0.02 | 0.00 | -0.00 | 0.00 | .778 | -0.05 | 0.00 | -0.00 | 0.00 | .421 | 0.01 | 0.00 | -0.00 | 0.00 | .934 |
| EAT: Error awareness | 0.03 | 0.00 | -0.00 | 0.01 | .640 | -0.16 | 0.00 | -0.01 | -0.00 | .004** | -0.01 | 0.00 | -0.01 | 0.01 | .970 |
| SDT: *w* | -0.06 | 0.26 | -0.76 | 0.24 | .303 | -0.03 | 0.24 | -0.62 | 0.36 | .600 | -0.07 | 0.31 | -0.93 | 0.26 | .253 |
| N-Back: 3-back d’ | 0.01 | 0.08 | -0.14 | 0.17 | 8.29 | 0.04 | 0.08 | -0.09 | 0.20 | .457 | -0.07 | 0.09 | -0.26 | 0.08 | .275 |
| DDT: Log *k* | 0.15 | 0.06 | 0.05 | 0.26 | .007** | -0.01 | 0.06 | -0.12 | 0.10 | .829 | 0.09 | 0.07 | -0.04 | 0.23 | .148 |
| Covariates | | | | | | | | | | | | | | | |
| mYFAS | - | - | - | - | - | 0.60 | 0.06 | 0.50 | 0.74 | <.001*** | 0.67 | 0.07 | 0.60 | 0.89 | <.001*** |
| DASS: Total score | 0.32 | 0.01 | 0.03 | 0.06 | <.001*** | 0.08 | 0.01 | -0.01 | 0.03 | .219 | 0.12 | 0.01 | -0.00 | 0.04 | .094 |
| SUPPS-P: Lack of perseverance and premeditation | -0.05 | 0.06 | -0.16 | 0.07 | .454 | 0.05 | 0.06 | -0.07 | 0.17 | .419 | 0.07 | 0.08 | -0.08 | 0.22 | .378 |
| SUPPS-P: Urgency | 0.05 | 0.05 | -0.05 | 0.14 | .406 | 0.09 | 0.05 | -0.03 | 0.16 | .200 | -0.05 | 0.06 | -0.15 | 0.09 | .556 |
| SUPPS-P: Sensation seeking | -0.13 | 0.03 | -0.14 | -0.01 | .023* | 0.07 | 0.03 | -0.03 | 0.11 | .251 | 0.03 | 0.04 | -0.06 | 0.11 | .671 |
| CHI-T: Trait compulsivity | 0.13 | 0.02 | -0.00 | 0.07 | .052 | 0.08 | 0.02 | -0.02 | 0.06 | .267 | 0.13 | 0.03 | -0.02 | 0.09 | .140 |

*Note. β: Standardised coefficient; SE: Standard error; *p < .05, **p < .01, ***p < .001*

**Table 5.** Cross-sectional and longitudinal multiple regression models of PPU

| Variable | Baseline (N=294) | | | | | Baseline predicting 3-months (N=206) | | | | | 3-months predicting 6-months (N=138) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | β | *SE* | 95% CI | | *p* | β | *SE* | 95% CI | | *p* | β | *SE* | 95% CI | | *p* |
| | | | LL | UL | | | | LL | UL | | | | LL | UL | |
| Demographics | | | | | | | | | | | | | | | |
| Age | -0.05 | 0.07 | -0.20 | 0.07 | .348 | 0.02 | 0.06 | -0.09 | 0.14 | .647 | -0.07 | 0.09 | -0.26 | 0.09 | .306 |
| Sex (F) | -0.51 | 0.62 | -7.03 | -4.64 | <.001*** | -0.16 | 0.62 | -3.01 | -0.51 | .004* | -0.14 | 0.89 | -3.38 | 0.13 | .074 |
| Neurocognition | | | | | | | | | | | | | | | |
| SST: SSRT | -0.02 | 4.59 | -10.56 | 7.17 | .630 | 0.01 | 3.64 | -6.04 | 8.18 | .797 | -0.04 | 5.77 | -14.24 | 7.78 | .524 |
| VMAC: VMAC score | 0.00 | 7.57 | -14.79 | 14.30 | .993 | 0.06 | 6.77 | -4.64 | 22.63 | .186 | -0.02 | 9.05 | -20.57 | 16.08 | .782 |
| BART: *M* pre-committed pumps | 0.02 | 0.02 | -0.03 | 0.05 | .753 | -0.00 | 0.02 | -0.04 | 0.03 | .966 | 0.00 | 0.02 | -0.04 | 0.04 | .986 |
| CST: Switch cost latency | 0.04 | 0.00 | -0.00 | 0.00 | .476 | 0.07 | 0.00 | -0.00 | 0.00 | .176 | 0.04 | 0.00 | -0.00 | 0.01 | .448 |
| EAT: Error awareness | -0.03 | 0.01 | -0.02 | 0.01 | .647 | 0.06 | 0.01 | -0.01 | 0.02 | .268 | 0.03 | 0.01 | -0.02 | 0.03 | .632 |
| SDT: *w* | 0.06 | 0.84 | -0.72 | 2.59 | .300 | 0.07 | 0.74 | -0.40 | 2.46 | .136 | 0.05 | 0.96 | -1.25 | 2.58 | .417 |
| N-Back: 3-back d’ | -0.08 | 0.26 | -0.95 | 0.08 | .104 | 0.02 | 0.22 | -0.35 | 0.53 | .681 | 0.03 | 0.29 | -0.40 | 0.69 | .664 |
| DDT: Log *k* | -0.02 | 0.18 | -0.44 | 0.27 | .638 | -0.02 | 0.16 | -0.38 | 0.26 | .729 | -0.16 | 0.22 | -0.97 | -0.12 | .014* |
| Covariates | | | | | | | | | | | | | | | |
| PPCS | - | - | - | - | - | 0.67 | 0.05 | 0.56 | 0.77 | <.001*** | 0.68 | 0.08 | 0.61 | 0.93 | <.001*** |
| DASS: Total score | 0.13 | 0.03 | 0.01 | 0.13 | .034* | -0.01 | 0.03 | -0.06 | 0.05 | .919 | 0.11 | 0.03 | -0.01 | 0.12 | .120 |
| SUPPS-P: Lack of perseverance and premeditation | 0.02 | 0.20 | -0.31 | 0.47 | .704 | -0.00 | 0.17 | -0.35 | 0.33 | .944 | -0.02 | 0.24 | -0.54 | 0.41 | .809 |
| SUPPS-P: Urgency | 0.12 | 0.16 | -0.03 | 0.60 | .075 | 0.06 | 0.14 | -0.13 | 0.42 | .281 | -0.02 | 0.19 | -0.41 | 0.35 | .871 |
| SUPPS-P: Sensation seeking | -0.04 | 0.11 | -0.30 | 0.14 | .451 | -0.05 | 0.10 | -0.28 | 0.10 | .345 | 0.05 | 0.14 | -0.16 | 0.38 | .447 |
| CHI-T: Trait compulsivity | 0.12 | 0.06 | -0.01 | 0.25 | .071 | 0.03 | 0.06 | -0.09 | 0.15 | .603 | -0.02 | 0.09 | -0.20 | 0.14 | .744 |

*Note. β: Standardised coefficient; SE: Standard error; *p < .05, **p < .01, ***p < .001*

**Table 6.** Cross-sectional and longitudinal multiple regression models of PUI

| Variable | Baseline (N=294) | | | | | Baseline predicting 3-months (N=206) | | | | | 3-months predicting 6-months (N=138) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | β | *SE* | 95% CI | | *p* | β | *SE* | 95% CI | | *p* | β | *SE* | 95% CI | | *p* |
| | | | LL | UL | | | | LL | UL | | | | LL | UL | |
| Demographics | | | | | | | | | | | | | | | |
| Age | -0.12 | 0.08 | -0.31 | -0.02 | .027* | -0.00 | 0.08 | -0.15 | 0.15 | .997 | 0.09 | 0.08 | -0.04 | 0.27 | .144 |
| Sex (F) | -0.20 | 0.72 | -4.05 | -1.27 | <.001*** | -0.06 | 0.70 | -2.21 | 0.50 | .237 | -0.05 | 0.71 | -1.87 | 0.84 | .456 |
| Neurocognition | | | | | | | | | | | | | | | |
| SST: SSRT | 0.07 | 5.03 | -3.34 | 16.87 | .192 | -0.01 | 4.68 | -10.20 | 7.82 | .803 | -0.01 | 5.37 | -11.35 | 8.83 | .804 |
| VMAC: VMAC score | -0.04 | 8.29 | -23.04 | 10.08 | .437 | -0.14 | 8.75 | -43.89 | -9.16 | .003** | -0.07 | 8.16 | -26.60 | 5.95 | .223 |
| BART: *M* pre-committed pumps | 0.07 | 0.02 | -0.01 | 0.07 | .180 | -0.11 | 0.02 | -0.10 | -0.01 | .029* | 0.07 | 0.02 | -0.01 | 0.06 | .236 |
| CST: Switch cost latency | 0.05 | 0.00 | -0.00 | 0.01 | .365 | -0.00 | 0.00 | -0.00 | 0.00 | .913 | -0.01 | 0.00 | -0.00 | 0.00 | .845 |
| EAT: Error awareness | -0.10 | 0.10 | -0.04 | 0.00 | .052 | -0.09 | 0.01 | -0.04 | 0.00 | .068 | 0.03 | 0.01 | -0.02 | 0.03 | .643 |
| SDT: *w* | -0.08 | 0.96 | -3.31 | 0.48 | .143 | -0.04 | 0.92 | -2.51 | 1.13 | .465 | -0.07 | 0.85 | -2.73 | 0.69 | .238 |
| N-Back: 3-back d’ | -0.04 | 0.29 | -0.83 | 0.34 | .390 | -0.00 | 0.29 | -0.59 | 0.55 | .909 | 0.04 | 0.25 | -0.31 | 0.68 | .484 |
| DDT: Log *k* | -0.07 | 0.21 | -0.72 | 0.11 | .158 | -0.04 | 0.21 | -0.60 | 0.21 | .365 | 0.00 | 0.20 | -0.38 | 0.40 | .936 |
| Covariates | | | | | | | | | | | | | | | |
| IAT | - | - | - | - | - | 0.72 | 0.06 | 0.65 | 0.89 | <.001*** | 0.77 | 0.06 | 0.54 | 0.75 | <.001*** |
| DASS: Total score | 0.31 | 0.04 | 0.11 | 0.25 | <.001*** | -0.05 | 0.04 | -0.10 | 0.05 | .477 | 0.04 | 0.03 | -0.04 | 0.08 | .520 |
| SUPPS-P: Lack of perseverance and premeditation | 0.09 | 0.22 | -0.07 | 0.80 | .107* | 0.10 | 0.22 | -0.02 | 0.84 | .063 | -0.05 | 0.22 | -0.57 | 0.27 | .492 |
| SUPPS-P: Urgency | 0.11 | 0.18 | -0.03 | 0.68 | .076 | -0.01 | 0.18 | -0.38 | 0.32 | .850 | 0.06 | 0.17 | -0.21 | 0.48 | .439 |
| SUPPS-P: Sensation seeking | -0.14 | 0.13 | -0.57 | -0.08 | .015* | 0.01 | 0.13 | -0.22 | 0.28 | .784 | -0.06 | 0.12 | -0.35 | 0.11 | .305 |
| CHI-T: Trait compulsivity | 0.22 | 0.07 | 0.12 | 0.40 | <.001*** | 0.09 | 0.08 | -0.04 | 0.27 | .143 | 0.05 | 0.08 | -0.10 | 0.21 | .460 |

*Note. β: Standardised coefficient; SE: Standard error; *p < .05, **p < .01, ***p < .001*

## 4.5 Discussion

The aim of the present study was to empirically test proposed trans-addiction, longitudinal mechanisms of addictive behaviours, namely problematic alcohol use, AE, PPU and PUI. We did this by first investigating the cross-sectional correlates of each addictive behaviour, then evaluated the predictors of each behaviour longitudinally. Overall, it is evident that in a general community sample, different addictive behaviours were predicted by different neurocognitive functions.

**Cross-sectional correlates of addictive behaviours**

Our cross-sectional analyses, accounting for key covariates, found steeper delay discounting was associated with more AE symptoms which is in line with previous literature (VanderBroek-Stice et al., 2017). However, neurocognition was not significantly associated with any of the other addictive behaviours: problematic alcohol use; PPU; and PUI. This contrasts with previous literature that has consistently shown problematic alcohol use and PUI are associated with neurocognitive deficits, namely poorer response inhibition and less flexible updating (Ioannidis et al., 2019; Stavro et al., 2013) and steeper discounting (Amlung et al., 2017; Cheng et al., 2021). Our findings may differ from other studies given we looked at a general community sample, compared to more severe and/or clinical samples investigated previously (Cheng et al., 2021; Ioannidis et al., 2022; Stavro et al., 2013). This is particularly of note for alcohol use, given the majority of the sample engaged in no or low problem use.

**Longitudinal predictors of addictive behaviours**

Only a minority of neurocognitive functions assessed predicted addictive behaviours longitudinally, and effect sizes were generally of small magnitude. Poorer performance monitoring at baseline predicted greater AE at 3-month follow-up. Whilst similar relationships have been found cross-sectionally (Franken et al., 2018), this is the first study to demonstrate this relationship longitudinally. Previous research on performance monitoring deficits associated with AE compared samples classified with food addiction to those who were not (Franken et al., 2018; Rodrigue et al., 2018; VanderBroek-Stice et al., 2017). In contrast, the present study identified these effects

dimensionally in a community sample, underscoring the potential significance of performance monitoring as an antecedent for AE dimensionally.

We also found that greater reward-related attentional bias at 3-months predicted more AE symptoms at 6-month follow-up. However, this effect narrowly fell short of reaching the required level of significance following adjustments for the Type I error rate. The VMAC Task, evaluates how cues indicating high-value rewards affect overall task performance. Greater reward-related attentional bias indicates the allure of a substantial reward is so distracting that it hinders one's ability to pursue the goal of the task. Individuals prone to developing more severe AE are perhaps more susceptible to distraction by highly rewarding food cues, even when it may work against their goals, or satiety.

Reward-related attentional bias was also found to predict (with a small effect) PUI but in the opposite direction. Greater freedom from distraction by reward-related cues, thus more goal-directed performance was associated with higher PUI at 3-month follow-up. Addictive behaviours can be purposeful, primarily driven by relief based motivations (Köpetz et al., 2013; Liu et al., 2021). More goal-directed VMAC performance may predict more goal-directed motives to engage in internet use (Albertella, Vd Hooven, et al., 2021). We also found less risky decision-making in the face of ambiguity (BART) was shown to predict more PUI at 3-month follow-up. However, this effect reduced to trend levels after Type I error adjustment was applied. Whilst studies have shown problematic alcohol and other substance use is associated with more risky BART performance (Fernie et al., 2010; Hopko et al., 2006), a recent meta-analysis showed this was not the case for PUI (Müller et al., 2023). Less risk taking under ambiguity seen here is akin to the patterns observed in individuals with obsessive-compulsive disorder (Pushkarskaya et al., 2015), and perhaps signifies a distinct difference between PUI and other addictive behaviours, suggesting PUI could be more characteristic of compulsive disorders.

The only relationship we found between neurocognition and PPU severity was delay discounting. This finding was unexpected and contradicts those observed in a study by Antons and colleagues (2019) that showed individuals who problematically use internet pornography had steeper delay discounting compared to those engaging in recreational use (Antons et al., 2019). It is unclear why we found a negative relationship between delay discounting and PPU. While our research

participants exhibited a prevalence of PPU similar to that found in other general population samples (7%; Mennig et al., 2020), the majority of the individuals in our current study demonstrated either no PPU or low severity. Such a low occurrence of more severe levels of PPU may have impacted our ability to observe meaningful associations with 'problematic' pornography use (cf. non-problematic use). As such, the relationship between PPU and neurocognition is still poorly established, and additional research is warranted to replicate our findings, perhaps in higher severity samples, to better understand the neurocognitive predictors of PPU.

We did not find neurocognition predicted future problematic alcohol use. This aligns with prior longitudinal studies conducted in community settings, which have failed to consistently demonstrate neurocognition predicting future problem use (Goudriaan et al., 2011; Jones et al., 2021; Whelan et al., 2014). Instead, neurocognition is seemingly more relevant at more severe levels of alcohol use. For example, poorer response inhibition has been shown to predict the development of alcohol dependence in a sample of already heavily drinking individuals (Rubio et al., 2008). It may be that neurocognitive impairments arising from exposure to alcohol use, or an interaction between the two, predict problematic future use. For example, Peeters and colleagues (2014) found that alcohol use at baseline predicted flexible updating performance, which, in turn, predicted further alcohol use outcomes. Rubio and colleague's sample were already at risk of transitioning to dependence (i.e. currently drinking heavily), general population samples without any risk indictors (i.e. family history, current regular use etc.) may never go on to have problems. Finally, neurocognitive processes may be more relevant when individuals are attempting to change or control their drinking behaviour (Albertella, Vd Hooven, et al., 2021). In the current study, participants were not required to change their drinking. Without the motivation to change, it is unlikely an individual's alcohol use would be predicted by cognitive disposition (Albertella, Vd Hooven, et al., 2021).

A strength of this study lies in our broad, longitudinal evaluation of neurocognitive functions. Each neurocognitive domain was selected based on theoretical frameworks and expert endorsement that attest to their relevance in addiction. This approach allowed for both a comprehensive and targeted evaluation of neurocognitive functions and their role in predicting addictive behaviours. Another strength of this study is that we investigated multiple addictive behaviour types in the same

sample. Given addictive behaviours often co-occur (Christensen, Albertella, et al., 2023; Ford & Håkansson, 2020), investigating influence of neurocognitive predictors on various addictive behaviours within the same group, helps us understand whether there are common factors that predict addictive behaviours across different types or if these predictors are specific to each behaviour.

**Limitations and future directions**

While our choice to focus on a community sample enhances the dimensional evaluation of addictive behaviour, it is important to recognise that the selected sample exhibited relatively low levels of engagement in addictive behaviours, specifically in the areas of AE, alcohol use, and PPU. This limitation may have impeded our ability to identify subtle associations. Importantly, our failure to identify trans-diagnostic neurocognitive predictors does not mean they do not exist. Rather, these functions may be more pertinent at later stages of addictive behaviour and could play a crucial role in treatment success (Domínguez-Salas et al., 2016), although this line of reasoning remains speculative. Notably, no prior studies have conducted such an extensive neurocognitive evaluation in higher severity samples or within the context of treatment success. It is also likely that the 3-month timeframe was too short to see sufficient change in addictive behaviours. We suggest future studies should extent our work by looking at how neurocognition may predict addictive behaviours across several years to increase the likelihood of capturing transitions to problem use.

An additional limitation is the complexity of our regression models in relation to the sample size. This is particularly relevant to our 3-month predicting 6-month longitudinal model which, due to participant drop-out across the course of the study, was underpowered to detect small effects like those observed herein. Another notable limitation is that participant drop-out resulted in distinct differences between those individuals who completed the baseline assessment and those who attended the 3-month follow-up. This made it difficult to compare the 3- and 6-month longitudinal analyses given the baseline sample was slightly different in each analysis, and likely explains our inability to replicate the longitudinal results. Participant drop-out also has the potential to bias longitudinal models, however, our multiple imputation analyses largely replicated the findings obtained from the raw data, mitigating the significance of this concern. Further, the effect size differences comparing individuals who were retained versus lost to follow-up were only small.

Finally, once-off assessments of neurocognitive functions are perhaps not the most appropriate method of capturing what are dynamic processes, sensitive to both intrinsic (physiological processes) and extrinsic (environmental) factors (Schmitter-Edgecombe et al., 2020). Ecological Momentary Assessment (EMA) paradigms have been suggested to account for this, providing multiple daily snapshots of neurocognition, able to track within-person fluctuations in different contexts and under different psychological states and environmental situations (Sliwinski et al., 2018).

**Conclusion**

In conclusion, we did not identify a core set of specific neurocognitive functions that reliably predicted addictive behaviours across multiple behaviour types. However, it is important to acknowledge that an absence of evidence is not evidence of absence. We did show reward-related neurocognitive processes were implicated across each non-substance addictive behaviour, but in different ways. Our findings suggest more work should be done to interrogate these differences, particularly focussing on reward-related functions. Our findings would also benefit from being replicated in studies that observe samples across a longer follow-up period (i.e. multiple years). This could lead to a deeper understanding of the types/profiles of individuals who may be at risk of developing specific types of addiction and inform the development of early identification and intervention strategies for specific addictive behaviours.

## 4.6 Supplementary material

**Table S1.** Spearman correlations corrected for multiple comparisons at baseline

| Variable | *M* | *SD* | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1.mYFAS | 0.69 | 1.69 | - | | | | | | | | | | | | | | | | | | |
| 2.PPCS | 10.06 | 5.78 | -0.02 | - | | | | | | | | | | | | | | | | | |
| 3.IAT | 17.43 | 6.79 | 0.28*** | 0.32*** | - | | | | | | | | | | | | | | | | |
| 4.AUDIT | 2.97 | 3.73 | 0.08 | 0.08 | -0.03 | - | | | | | | | | | | | | | | | |
| 5.SST | 332.32 | 68.23 | 0.04 | -0.06 | -0.02 | -0.02 | - | | | | | | | | | | | | | | |
| 6.VMAC | 8.97 | 40.62 | 0.00 | 0.01 | -0.07 | -0.08 | 0.02 | - | | | | | | | | | | | | | |
| 7.BART | 56.61 | 14.99 | -0.09 | 0.14 | 0.06 | -0.02 | -0.17 | 0.00 | - | | | | | | | | | | | | |
| 8.CST | 268.05 | 181.10 | 0.01 | 0.08 | 0.09 | -0.12 | 0.07 | 0.01 | -0.09 | - | | | | | | | | | | | |
| 9.DDT | -4.52 | 1.69 | 0.05 | 0.09 | -0.02 | 0.04 | -0.02 | 0.04 | 0.02 | 0.06 | - | | | | | | | | | | |
| 10.EAT | 69.28 | 34.07 | -0.02 | -0.03 | -0.06 | 0.07 | -0.16 | -0.11 | -0.02 | -0.11 | -0.19 | - | | | | | | | | | |
| 11.SDT | 0.51 | 0.36 | -0.06 | 0.13 | -0.06 | -0.04 | -0.07 | 0.07 | 0.00 | -0.04 | -0.14 | 0.14 | - | | | | | | | | |
| 12.N-Back | 2.24 | 1.15 | -0.12 | 0.00 | -0.07 | 0.07 | 0.02 | -0.02 | 0.01 | 0.11 | -0.10 | 0.09 | 0.10 | - | | | | | | | |
| 13.Lack of persev/premed | 7.61 | 1.77 | 0.00 | -0.03 | 0.08 | 0.14 | -0.05 | -0.04 | 0.01 | -0.08 | 0.16 | -0.02 | -0.18 | -0.06 | - | | | | | | |
| 14.Urgency | 8.71 | 2.33 | 0.18 | 0.21* | 0.28*** | 0.11 | 0.09 | -0.02 | 0.01 | 0.08 | 0.13 | -0.10 | -0.09 | -0.06 | 0.31*** | - | | | | | |
| 15.SS | 9.53 | 2.89 | -0.15 | 0.12 | -0.09 | 0.28*** | 0.08 | -0.03 | 0.13 | -0.05 | 0.19 | -0.11 | -0.02 | 0.11 | 0.09 | 0.26** | - | | | | |
| 16.CHI-T | 27.87 | 5.83 | 0.26*** | 0.08 | 0.33*** | 0.02 | 0.02 | 0.06 | -0.01 | 0.06 | -0.02 | 0.00 | -0.02 | -0.01 | -0.29 | 0.29*** | -0.05 | - | | | |
| 17.Psychological distress | 13.64 | 11.49 | 0.36*** | 0.10 | 0.49*** | 0.21* | -0.02 | -0.14 | -0.10 | 0.04 | -0.05 | 0.01 | -0.04 | -0.07 | 0.08 | 0.30*** | -0.09 | 0.40*** | - | | |
| 18.Age | 24.82 | 4.66 | -0.02 | -0.07 | -0.20 | -0.11 | 0.23** | 0.15 | 0.03 | 0.07 | -0.08 | -0.07 | -0.08 | 0.11 | -0.15 | -0.17 | -0.06 | -0.05 | -0.17 | - | |
| 19.Sex | - | - | 0.27*** | -0.50*** | -0.08 | 0.13 | 0.03 | 0.01 | -0.22* | -0.07 | -0.16 | 0.05 | -0.06 | -0.14 | 0.04 | -0.09 | -0.25** | 0.07 | 0.15 | -0.07 | - |

*Note: p values were adjusted for multiple comparisons using Holm method; SST, VMAC, and CST means and standard deviations are reported in milliseconds. mYFAS: modified Yale Food Addiction Scale; PPCS: Problematic Pornography Consumption Scale; IAT: Young's Internet Addiction Test; AUDIT: Alcohol Use Disorder Identification Test; SST: Stop Signal Task; VMAC: Value-Modulated Attentional Capture Task; BART: Balloon Analogue Risk Task; CST: Category Switch Task; DDT: Delay Discounting Task; EAT: Error Awareness Task; SDT: Sequential Decision-Making Task; N-Back: N-Back Task; Lack of persev/premed: SUPPS-P lack of perseverance and premeditation score; Urgency: SUPPS-P urgency score; SS: SUPPS-P sensation seeking score; CHIT: CHI-T trait compulsivity score; Psychological distress: DASS-21 total score; *p < .05, **p < .01, ***p < .001*

**Table S2.** Spearman correlations corrected for multiple comparisons between baseline predictors and addictive behaviours at 3 months

| Variable | *M* | *SD* | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1.mYFAS | 0.68 | 1.62 | - | | | | | | | | | | | | | | | | | | |
| 2.PPCS | 9.77 | 5.41 | 0.07 | - | | | | | | | | | | | | | | | | | |
| 3.IAT | 17.22 | 6.60 | 0.36*** | 0.22 | - | | | | | | | | | | | | | | | | |
| 4.AUDIT | 2.88 | 3.71 | 0.02 | 0.03 | -0.03 | - | | | | | | | | | | | | | | | |
| 5.SST | 337.55 | 73.36 | 0.05 | 0.04 | -0.02 | -0.05 | - | | | | | | | | | | | | | | |
| 6.VMAC | 9.95 | 37.82 | 0.01 | 0.10 | -0.07 | -0.01 | 0.00 | - | | | | | | | | | | | | | |
| 7.BART | 56.83 | 14.82 | -0.04 | 0.13 | -0.04 | -0.08 | -0.13 | 0.02 | - | | | | | | | | | | | | |
| 8.CST | 272.14 | 184.20 | 0.04 | 0.06 | -0.02 | -0.04 | 0.07 | 0.04 | -0.06 | - | | | | | | | | | | | |
| 9.DDT | -4.63 | 1.66 | 0.01 | 0.02 | -0.04 | 0.10 | -0.04 | 0.05 | 0.05 | 0.03 | - | | | | | | | | | | |
| 10.EAT | 69.73 | 33.82 | -0.12 | -0.01 | -0.11 | 0.02 | -0.14 | -0.14 | -0.07 | -0.14 | -0.21 | - | | | | | | | | | |
| 11.SDT | 0.52 | 0.37 | -0.13 | 0.17 | -0.15 | -0.05 | -0.09 | 0.11 | 0.03 | -0.08 | -0.14 | 0.09 | - | | | | | | | | |
| 12.N-Back | 2.27 | 1.16 | -0.02 | 0.02 | -0.12 | 0.11 | 0.05 | 0.01 | -0.02 | 0.15 | -0.07 | 0.05 | 0.10 | - | | | | | | | |
| 13.Lack of persev/premed | 7.52 | 1.76 | 0.12 | -0.06 | 0.13 | 0.17 | -0.07 | 0.02 | 0.03 | -0.06 | 0.16 | -0.09 | -0.19 | -0.03 | - | | | | | | |
| 14.Urgency | 8.73 | 2.34 | 0.28** | 0.19 | 0.27* | 0.15 | 0.10 | -0.07 | 0.01 | 0.13 | 0.17 | -0.16 | -0.12 | -0.05 | 0.31*** | - | | | | | |
| 15.SS | 9.38 | 2.91 | -0.03 | 0.05 | -0.10 | 0.32** | 0.12 | -0.02 | 0.12 | 0.00 | 0.25 | -0.13 | 0.00 | 0.12 | 0.12 | 0.25 | - | | | | |
| 16.CHI-T | 27.69 | 5.62 | 0.27* | 0.02 | 0.29** | -0.02 | 0.02 | -0.01 | 0.01 | 0.07 | -0.10 | 0.09 | -0.02 | -0.02 | -0.25* | 0.31** | -0.08 | - | | | |
| 17.Psychological distress | 13.31 | 11.31 | 0.37*** | 0.07 | 0.36*** | 0.10 | 0.00 | -0.07 | -0.15 | 0.00 | -0.11 | 0.05 | -0.04 | -0.06 | -0.04 | 0.23 | -0.15 | 0.47*** | - | | |
| 18.Age | 25.48 | 4.70 | -0.01 | -0.02 | -0.15 | -0.04 | 0.26* | 0.08 | 0.02 | 0.07 | -0.03 | -0.08 | -0.15 | 0.12 | -0.09 | -0.19 | -0.07 | -0.11 | -0.11 | - | |
| 19.Sex | - | - | 0.13 | -0.51*** | -0.07 | 0.12 | 0.00 | -0.03 | -0.24 | -0.07 | -0.20 | 0.13 | -0.07 | -0.06 | 0.04 | -0.08 | -0.23 | 0.12 | 0.25* | -0.08 | - |

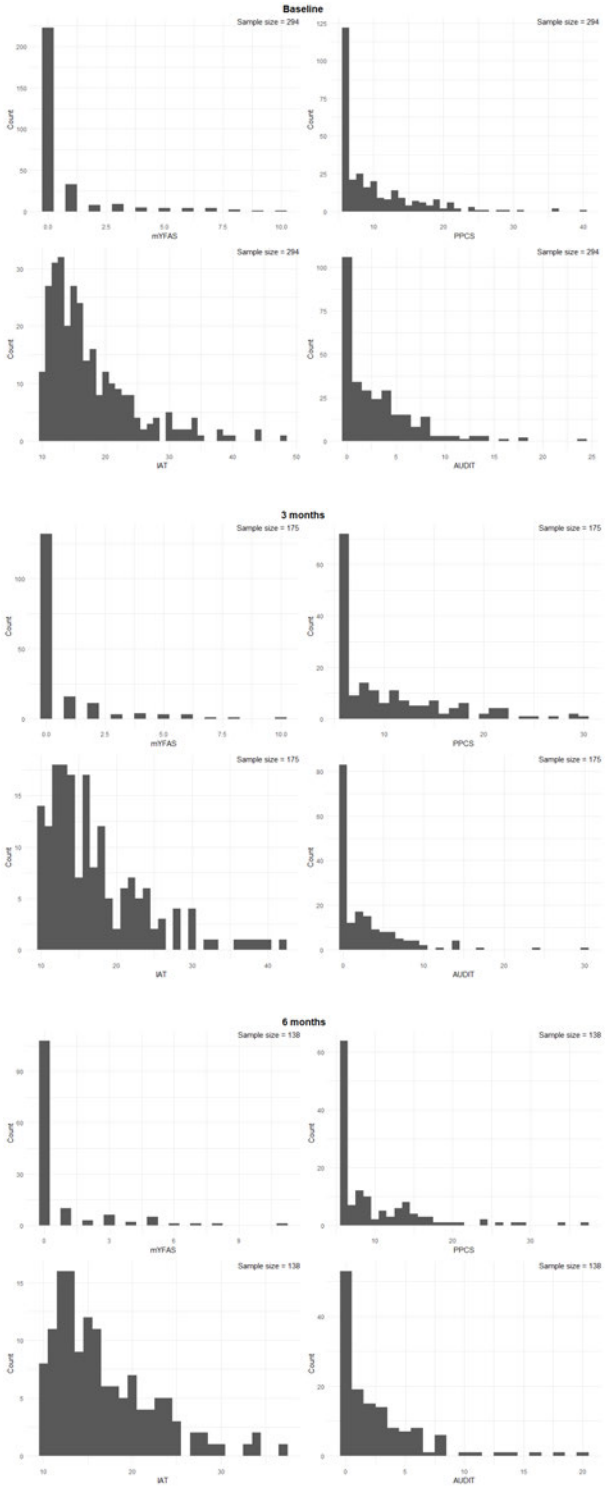
*Note: p values were adjusted for multiple comparisons using Holm method; SST, VMAC, and CST means and standard deviations are reported in milliseconds. mYFAS: modified Yale Food Addiction Scale; PPCS: Problematic Pornography Consumption Scale; IAT: Young's Internet Addiction Test; AUDIT: Alcohol Use Disorder Identification Test; SST: Stop Signal Task; VMAC: Value-Modulated Attentional Capture Task; BART: Balloon Analogue Risk Task; CST: Category Switch Task; DDT: Delay Discounting Task; EAT: Error Awareness Task; SDT: Sequential Decision-Making Task; N-Back: N-Back Task; Lack of persev/premed: SUPPS-P lack of perseverance and premeditation score; Urgency: SUPPS-P urgency score; SS: SUPPS-P sensation seeking score; CHIT: CHI-T trait compulsivity score; Psychological distress: DASS-21 total score; *p < .05, **p < .01, ***p < .001*

**Table S3.** Spearman correlations corrected for multiple comparisons between 3-month predictors and addictive behaviours at 6 months

| Variable | *M* | *SD* | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1.mYFAS | 0.70 | 1.64 | - | | | | | | | | | | | | | | | | | | |
| 2.PPCS | 10.07 | 5.26 | -0.02 | - | | | | | | | | | | | | | | | | | |
| 3.IAT | 17.26 | 6.82 | 0.35** | 0.21 | - | | | | | | | | | | | | | | | | |
| 4.AUDIT | 2.48 | 3.57 | -0.04 | 0.01 | -0.04 | - | | | | | | | | | | | | | | | |
| 5.SST | 324.20 | 64.49 | -0.12 | -0.13 | -0.17 | -0.09 | - | | | | | | | | | | | | | | |
| 6.VMAC | 2.96 | 40.20 | 0.01 | -0.03 | -0.07 | -0.11 | 0.03 | - | | | | | | | | | | | | | |
| 7.BART | 55.72 | 16.91 | -0.02 | 0.05 | -0.02 | 0.00 | -0.06 | -0.04 | - | | | | | | | | | | | | |
| 8.CST | 212.78 | 145.13 | 0.09 | 0.03 | -0.07 | -0.06 | 0.14 | -0.07 | 0.05 | - | | | | | | | | | | | |
| 9.DDT | -4.88 | 1.75 | 0.02 | -0.06 | -0.17 | 0.05 | -0.09 | -0.02 | -0.08 | -0.05 | - | | | | | | | | | | |
| 10.EAT | 76.75 | 31.20 | 0.04 | 0.03 | -0.07 | 0.15 | -0.18 | 0.07 | 0.04 | -0.11 | -0.17 | - | | | | | | | | | |
| 11.SDT | 0.55 | 0.38 | -0.08 | 0.06 | -0.10 | 0.09 | -0.01 | 0.07 | 0.15 | -0.06 | -0.18 | 0.22 | - | | | | | | | | |
| 12.N-Back | 2.48 | 1.26 | -0.06 | 0.01 | -0.06 | 0.07 | 0.00 | 0.06 | 0.08 | -0.11 | 0.16 | 0.16 | 0.06 | - | | | | | | | |
| 13.Lack of persev/premed | 7.48 | 1.73 | 0.09 | 0.16 | 0.04 | -0.09 | 0.02 | 0.02 | -0.03 | -0.12 | 0.06 | 0.02 | -0.02 | 0.06 | - | | | | | | |
| 14.Urgency | 8.65 | 2.44 | 0.18 | 0.04 | 0.34** | 0.06 | 0.02 | -0.14 | 0.01 | 0.00 | -0.06 | -0.15 | -0.03 | -0.01 | 0.17 | - | | | | | |
| 15.SS | 9.24 | 2.98 | -0.09 | 0.07 | 0.01 | 0.09 | 0.07 | -0.16 | 0.09 | 0.10 | 0.05 | -0.15 | -0.15 | -0.07 | 0.02 | 0.33* | - | | | | |
| 16.CHI-T | 28.09 | 5.42 | 0.20 | -0.02 | 0.32* | 0.04 | -0.02 | -0.20 | 0.02 | 0.09 | -0.13 | 0.00 | -0.03 | -0.08 | -0.36** | 0.41*** | -0.02 | - | | | |
| 17.Psychological distress | 13.97 | 11.79 | 0.29 | 0.01 | 0.27 | 0.09 | -0.10 | -0.06 | -0.07 | -0.07 | -0.03 | 0.12 | -0.02 | -0.01 | 0.08 | 0.28 | -0.15 | 0.33* | - | | |
| 18.Age | 25.01 | 4.42 | 0.01 | -0.07 | -0.06 | 0.12 | 0.17 | 0.07 | 0.00 | 0.09 | -0.08 | -0.11 | 0.02 | 0.10 | -0.19 | -0.30 | -0.12 | -0.08 | -0.15 | - | |
| 19.Sex | - | - | 0.10 | -0.47*** | -0.04 | 0.07 | 0.14 | -0.04 | -0.09 | 0.04 | -0.17 | 0.16 | 0.10 | -0.07 | -0.10 | 0.07 | -0.19 | 0.19 | 0.33* | -0.10 | - |

*Note: p values were adjusted for multiple comparisons using Holm method; SST, VMAC, and CST means and standard deviations are reported in milliseconds. mYFAS: modified Yale Food Addiction Scale; PPCS: Problematic Pornography Consumption Scale; IAT: Young's Internet Addiction Test; AUDIT: Alcohol Use Disorder Identification Test; SST: Stop Signal Task; VMAC: Value-Modulated Attentional Capture Task; BART: Balloon Analogue Risk Task; CST: Category Switch Task; DDT: Delay Discounting Task; EAT: Error Awareness Task; SDT: Sequential Decision-Making Task; N-Back: N-Back Task; Lack of persev/premed: SUPPS-P lack of perseverance and premeditation score; Urgency: SUPPS-P urgency score; SS: SUPPS-P sensation seeking score; CHIT: CHI-T trait compulsivity score; Psychological distress: DASS-21 total score; *p < .05, **p < .01, ***p < .001*

**Figure S1.** Distributions of problematic behaviour engagement at each study time point




*Note: AUDIT: Alcohol Use Identification Test, no/low problem use (0-7), harmful/ hazardous use (8-14), likely alcohol dependence (≥15); mYFAS: modified Yale Food Addiction Scale 2.0, mild (2-3), moderate (4-5) severe (≥6) symptoms. PPCS: Problematic Pornography Consumption Scale, problematic use (≥20); IAT: an abbreviated version of Young's Internet Addiction Test, problematic use (≥17).*

**Table S4.** Longitudinal multiple regression model with imputed data of problematic alcohol use

| Variable | Baseline predicting 3-months (N=294) | | | | | 3-months predicting 6-months (N=206) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | **β** | ***SE*** | **95% CI** | | ***p*** | **β** | ***SE*** | **95% CI** | | ***p*** |
| | | | **LL** | **UL** | | | | **LL** | **UL** | |
| Demographics | | | | | | | | | | |
| Age | 0.04 | 0.03 | -0.02 | 0.10 | .228 | 0.05 | 0.06 | -0.06 | 0.16 | .367 |
| Sex (F) | 0.15 | 0.27 | -0.37 | 0.67 | .580 | -0.03 | 0.42 | -0.86 | 0.80 | .952 |
| Neurocognition | | | | | | | | | | |
| SST: SSRT | -1.36 | 2.05 | -5.38 | 2.66 | .506 | -3.72 | 2.25 | -8.13 | 0.69 | .100 |
| VMAC: VMAC score | 0.33 | 4.02 | -7.54 | 8.20 | .935 | -0.28 | 4.18 | -8.47 | 7.92 | .947 |
| BART: *M* pre-committed pumps | 0.00 | 0.01 | -0.01 | 0.02 | .719 | -0.01 | 0.01 | -0.03 | 0.02 | .551 |
| CST: Switch cost latency | 0.00 | 0.00 | -0.00 | 0.00 | .443 | 0.00 | 0.00 | -0.00 | 0.00 | .625 |
| EAT: Error awareness | 0.00 | 0.00 | -0.01 | 0.01 | .536 | 0.00 | 0.01 | -0.01 | 0.02 | .579 |
| SDT: *w* | -0.11 | 0.38 | -0.86 | 0.63 | .769 | -0.27 | 0.62 | -1.48 | 0.95 | .667 |
| N-Back: 3-back d’ | 0.04 | 0.10 | -0.16 | 0.24 | .701 | 0.05 | 0.11 | -0.16 | 0.26 | .661 |
| DDT: Log *k* | 0.02 | 0.07 | -0.11 | 0.15 | .767 | -0.01 | 0.11 | -0.22 | 0.20 | .948 |
| Covariates | | | | | | | | | | |
| AUDIT | 0.83 | 0.06 | .710 | 0.94 | <.001*** | 0.68 | 0.10 | 0.49 | 0.88 | <.001*** |
| DASS-TS | 0.01 | 0.01 | -0.01 | 0.04 | .264 | 0.02 | 0.02 | -0.01 | 0.05 | .277 |
| SUPPS-P: Lack of perseverance and premeditation | 0.00 | 0.11 | -0.21 | 0.22 | .982 | -0.15 | 0.10 | -0.35 | 0.05 | .147 |
| SUPPS-P: Urgency | 0.13 | 0.08 | -0.02 | 0.28 | .094 | -0.06 | 0.18 | -0.42 | 0.30 | .744 |
| SUPPS-P: Sensation seeking | 0.03 | 0.05 | -0.07 | 0.13 | .567 | 0.02 | 0.08 | -0.12 | 0.17 | .744 |
| CHI-T: Trait compulsivity | -0.04 | 0.03 | -0.11 | 0.02 | .163 | 0.03 | 0.05 | -0.07 | 0.13 | .510 |

*Note. β: Unstandardised coefficient; SE: Standard error; *p < .05, **p < .01, ***p < .001*

**Table S5.** Longitudinal multiple regression model with imputed data of AE

| Variable | Baseline predicting 3-months (N=294) | | | | | 3-months predicting 6-months (N=206) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | **β** | ***SE*** | **95% CI** | | ***p*** | **β** | ***SE*** | **95% CI** | | ***p*** |
| | | | **LL** | **UL** | | | | **LL** | **UL** | |
| Demographics | | | | | | | | | | |
| Age | 0.03 | 0.02 | -0.12 | 0.06 | .183 | -0.00 | 0.02 | -0.05 | 0.04 | .886 |
| Sex (F) | 0.02 | 0.16 | -0.30 | 0.34 | .907 | -0.24 | 0.21 | -0.66 | 0.17 | .253 |
| Neurocognition | | | | | | | | | | |
| SST: SSRT | -0.13 | 1.03 | -2.15 | 1.89 | .898 | -0.11 | 1.39 | -2.85 | 2.62 | .935 |
| VMAC: VMAC score | 0.20 | 2.21 | -4.14 | 4.53 | .929 | 4.73 | 2.85 | -0.86 | 10.33 | .097 |
| BART: *M* pre-committed pumps | 0.00 | 0.01 | -0.01 | 0.01 | .841 | -0.01 | 0.01 | -0.02 | 0.00 | .164 |
| CST: Switch cost latency | -0.00 | 0.00 | -0.00 | 0.00 | .998 | 0.00 | 0.00 | -0.00 | 0.00 | .829 |
| EAT: Error awareness | -0.01 | 0.00 | -0.01 | 0.00 | .066 | 0.00 | 0.00 | -0.01 | 0.01 | .889 |
| SDT: *w* | -0.13 | 0.22 | -0.57 | 0.31 | .556 | -0.26 | 0.23 | -0.71 | 0.19 | .262 |
| N-Back: 3-back d’ | 0.02 | 0.07 | -0.12 | 0.15 | .822 | -0.08 | 0.09 | -0.25 | 0.10 | .375 |
| DDT: Log *k* | 0.01 | 0.05 | -0.08 | 0.10 | .795 | 0.08 | 0.06 | -0.04 | 0.20 | .210 |
| Covariates | | | | | | | | | | |
| mYFAS | 0.56 | 0.12 | 0.33 | 0.80 | <.001*** | 0.73 | 0.12 | 0.49 | 0.97 | <.001*** |
| DASS-TS | 0.01 | 0.01 | -0.01 | 0.04 | .302 | 0.02 | 0.01 | -0.01 | 0.04 | .170 |
| SUPPS-P: Lack of perseverance and premeditation | 0.05 | 0.05 | -0.05 | 0.16 | .342 | 0.05 | 0.09 | -0.12 | 0.21 | .586 |
| SUPPS-P: Urgency | 0.03 | 0.04 | -0.04 | 0.11 | .402 | -0.04 | 0.05 | -0.14 | 0.07 | .500 |
| SUPPS-P: Sensation seeking | 0.04 | 0.03 | -0.02 | 0.09 | .201 | 0.01 | 0.04 | -0.06 | 0.08 | .689 |
| CHI-T: Trait compulsivity | 0.02 | 0.03 | -0.03 | 0.08 | .371 | 0.03 | 0.02 | -0.02 | 0.08 | .218 |

*Note. β: Unstandardised coefficient; SE: Standard error; *p < .05, **p < .01, ***p < .001*

**Table S6.** Longitudinal multiple regression model with imputed data of PPU

| Variable | Baseline predicting 3-months (N=294) | | | | | 3-months predicting 6-months (N=206) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | **β** | ***SE*** | **95% CI** | | ***p*** | **β** | ***SE*** | **95% CI** | | ***p*** |
| | | | **LL** | **UL** | | | | **LL** | **UL** | |
| Demographics | | | | | | | | | | |
| Age | 0.02 | 0.05 | -0.08 | 0.11 | .714 | -0.10 | 0.07 | -0.23 | 0.03 | .148 |
| Sex (F) | -1.55 | 0.59 | -2.71 | -0.39 | .010* | -1.42 | 0.95 | -3.28 | 0.45 | .136 |
| Neurocognition | | | | | | | | | | |
| SST: SSRT | 0.90 | 2.97 | -4.92 | 6.71 | .762 | -3.06 | 4.74 | -12.35 | 6.22 | .518 |
| VMAC: VMAC score | 7.57 | 5.68 | -3.57 | 18.71 | .183 | -0.38 | 8.06 | -16.19 | 15.43 | .963 |
| BART: *M* pre-committed pumps | 0.00 | 0.02 | -0.03 | 0.04 | .889 | -0.00 | 0.02 | -0.04 | 0.04 | .982 |
| CST: Switch cost latency | 0.00 | 0.00 | -0.00 | 0.00 | .381 | 0.00 | 0.00 | -0.00 | 0.01 | .402 |
| EAT: Error awareness | 0.01 | 0.01 | -0.01 | 0.03 | .367 | 0.01 | 0.02 | -0.03 | 0.05 | .713 |
| SDT: *w* | 0.94 | 0.72 | -0.47 | 2.35 | .193 | 0.61 | 1.13 | -1.61 | 2.83 | .589 |
| N-Back: 3-back d’ | 0.05 | 0.16 | -0.26 | 0.37 | .735 | 0.04 | 0.42 | -0.77 | 0.86 | .920 |
| DDT: Log *k* | -0.06 | 0.15 | -0.36 | 0.23 | .664 | -0.43 | 0.35 | -1.12 | 0.26 | .220 |
| Covariates | | | | | | | | | | |
| PPCS | 0.64 | 0.10 | 0.45 | 0.82 | <.001*** | 0.76 | 0.10 | 0.56 | 0.96 | <.001*** |
| DASS-TS | -0.00 | 0.02 | -0.05 | 0.05 | .975 | 0.04 | 0.05 | -0.05 | 0.14 | .360 |
| SUPPS-P: Lack of perseverance and premeditation | -0.07 | 0.17 | -0.40 | 0.25 | .657 | -0.09 | 0.20 | -0.48 | 0.30 | .661 |
| SUPPS-P: Urgency | 0.14 | 0.11 | -0.08 | 0.37 | .212 | -0.04 | 0.15 | -0.34 | 0.27 | .811 |
| SUPPS-P: Sensation seeking | -0.08 | 0.07 | -0.23 | 0.06 | .273 | 0.08 | 0.11 | -0.14 | 0.31 | .472 |
| CHI-T: Trait compulsivity | 0.01 | 0.05 | -0.10 | 0.11 | .918 | -0.01 | 0.07 | -0.16 | 0.13 | .875 |

*Note. β: Unstandardised coefficient; SE: Standard error; *p < .05, **p < .01, ***p < .001*

**Table S7.** Longitudinal multiple regression model with imputed data of PUI

| Variable | Baseline predicting 3-months (N=294) | | | | | 3-months predicting 6-months (N=206) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | β | *SE* | 95% CI | | *p* | β | *SE* | 95% CI | | *p* |
| | | | LL | UL | | | | LL | UL | |
| Demographics | | | | | | | | | | |
| Age | 0.01 | 0.07 | -0.13 | 0.15 | .884 | 0.08 | 0.07 | -0.06 | 0.22 | .259 |
| Sex (F) | -0.67 | 0.68 | -2.00 | 0.65 | .319 | -0.63 | 0.77 | -2.13 | 0.88 | .414 |
| Neurocognition | | | | | | | | | | |
| SST: SSRT | -0.10 | 4.05 | -8.03 | 7.84 | .981 | -0.64 | 4.83 | -10.11 | 8.83 | .894 |
| VMAC: VMAC score | -21.58 | 9.05 | -39.32 | -3.84 | .017* | -9.95 | 7.96 | -25.55 | 5.65 | .211 |
| BART: *M* pre-committed pumps | -0.04 | 0.02 | -0.09 | 0.00 | .062 | 0.03 | 0.02 | -0.01 | 0.06 | .161 |
| CST: Switch cost latency | 0.00 | 0.00 | -0.00 | 0.01 | .586 | -0.00 | 0.00 | -0.00 | 0.00 | .761 |
| EAT: Error awareness | -0.01 | 0.01 | -0.04 | 0.01 | .211 | 0.01 | 0.01 | -0.01 | 0.03 | .484 |
| SDT: *w* | -0.40 | 0.92 | -2.20 | 1.41 | .667 | -1.10 | 0.91 | -2.88 | 0.68 | .226 |
| N-Back: 3-back d’ | -0.21 | 0.28 | -0.77 | 0.34 | .454 | 0.18 | 0.27 | -0.35 | 0.70 | .512 |
| DDT: Log *k* | -0.16 | 0.20 | -0.55 | 0.23 | .421 | 0.01 | 0.20 | -0.38 | 0.39 | .973 |
| Covariates | | | | | | | | | | |
| IAT | 0.74 | 0.09 | 0.56 | 0.92 | <.001*** | 0.65 | 0.06 | 0.53 | 0.77 | <.001*** |
| DASS-TS | -0.01 | 0.04 | -0.08 | 0.07 | .832 | 0.02 | 0.03 | -0.05 | 0.08 | .589 |
| SUPPS-P: Lack of perseverance and premeditation | 0.36 | 0.19 | -0.01 | 0.72 | .056 | -0.24 | 0.22 | -0.67 | 0.19 | .271 |
| SUPPS-P: Urgency | -0.07 | 0.16 | -0.40 | 0.25 | .652 | 0.09 | 0.17 | -0.25 | 0.43 | .614 |
| SUPPS-P: Sensation seeking | 0.04 | 0.11 | -0.18 | 0.26 | .699 | -0.14 | 0.11 | -0.35 | 0.08 | .213 |
| CHI-T: Trait compulsivity | 0.08 | 0.07 | -0.07 | 0.22 | .293 | 0.06 | 0.08 | -0.10 | 0.22 | .474 |

*Note. β: Unstandardised coefficient; SE: Standard error; *p < .05, **p < .01, ***p < .001*

*Chapter 5*

# Discussion and concluding remarks

## 5.1 General discussion of key findings

The aim of this thesis was to critically evaluate the neurocognitive correlates and predictors of addictive behaviours, first by systematically reviewing the current prospective literature, and then empirically evaluating relationships using a comprehensive, expert consensus-driven assessment of neurocognition in a longitudinal cohort study. A systematic review of the current literature (Chapter 2) did not find evidence for a reliable neurocognitive predictor across addictions. Further, less than half of studies showed neurocognition significantly predicted addiction outcomes. This may be contributed to, in part, by inconsistencies in the neurocognitive domains assessed, the choice of assessment paradigms and the failure to include essential covariates in respective models. The review further revealed that most of our current understanding is based on alcohol use literature and there is very little research on the neurocognitive predictors of non-substance addiction. To address this gap in the literature, I conducted a 6-month longitudinal study using a suite of neurocognitive measures tapping into expert-endorsed constructs and accounting for key covariates (sex, age, psychological distress); evaluating cross-sectional associations (Chapter 3) and longitudinal predictors (Chapter 4) of both problematic alcohol use and three common non-substance addictive behaviours: eating, pornography use, and internet use. While originally intending on also evaluating the neurocognitive corelates and predictors of problem gambling, there were not enough people who engaged in gambling in the sample to justify investigation. Findings from Chapter 3 showed neurocognition was differentially associated with addictive behaviour type, suggesting that neurocognitive dysfunction is not necessarily equally implicated across addictions. I then revealed similar findings longitudinally in Chapter 4 revealing different addictive behaviours are predicted by different neurocognitive functions. The implications of these findings to both understanding the mechanisms of addictive behaviours as

well as potential clinical implications of the work is discussed in detail below. The limitations of the present thesis and an exploration of the potential future directions of this work follows.

## 5.2 The cross-sectional correlates of addictive behaviours

The first empirical chapter of this thesis (Chapter 3) aimed to evaluate the role of a broad range of neurocognitive functions and how they relate to both substance (alcohol use) and non-substance addictive behaviours (AE, PPU and PUI). This was done in the context of self-reported impulsive and compulsive traits, psychological distress, age and sex, so as to identify whether specific neurocognitive functions play an independent role above and beyond these known confounds (and each other) that have previously been linked with addiction (Albertella, Pelley, et al., 2019; Albertella, Rotaru, et al., 2021; Charness & Gneezy, 2012; Lugtmeijer et al., 2019; Sepas et al., 2021; Sjoberg & Cole, 2018; Stoet, 2010; Vallesi et al., 2021). Chapter 3 findings indicated that each addictive behaviour was associated with a unique profile of neurocognitive functioning. Surprisingly, the regression models showed that none of the neurocognitive domains were associated with problematic alcohol use or AE. Poorer performance monitoring was independently associated with more PPU and PUI, and a higher preference for delayed gratification was also independently associated with higher PUI. This suggests that while these addictive behaviours share neurobiological and phenomenological similarities such as craving, tolerance, and withdrawal (Brewer & Potenza, 2008; Chamberlain et al., 2016; Grant et al., 2010, 2013), they may have different neurocognitive mechanisms and likely different pathways to addiction.

The second empirical chapter of this thesis (Chapter 4) took a sub-sample of that included in Chapter 3 and investigated both cross-sectional associations at baseline, as well as longitudinal predictors of addictive behaviours. The sample included in Chapter 4 was aged between 18 and 35 years, an age range specifically selected to target individuals who may be at higher risk of developing addictive behaviours given the median age of onset for addiction is 25 years (Solmi et al., 2022). In keeping with Chapter 3 findings, baseline cross-sectional analyses revealed no evidence that any of the neurocognitive functions were significantly associated with problematic alcohol use. However, Chapter 4 regression models showed steeper delay discounting was independently associated with

higher AE, and none of the neurocognitive functions were associated with PPU or PUI. The discrepancy between Chapter 3 and 4 cross-sectional findings is discussed below.

Significantly, the approach adopted in Chapters 3 and 4 distinguishes this research from previous studies. Specifically, this thesis provides a comprehensive assessment of neurocognitive functions within the same statistical model, encompassing both cognitive control and reward-related functions, while exploring their correlations with various addictive behaviours in a single, large community sample. Previous studies have typically concentrated on a single addictive behaviour and a limited number of neurocognitive functions (Amlung et al., 2017; Stavro et al., 2013). As a result, directly comparing the findings of this thesis with existing literature is challenging. Nonetheless, such comparisons are a valuable expansion to the existing body of knowledge.

The lack of evidence found for associations between any of the neurocognitive functions assessed and problematic alcohol use in Chapter 3 and 4 was unexpected. Deficits in neurocognitive functions have been consistently linked with AUD, including inflexible updating and shifting (Manning et al., 2016), poorer response inhibition and executive function (Naim-Feil et al., 2014; Stavro et al., 2013), and steeper delay discounting (Amlung et al., 2017). As discussed in Chapter 3, a reason for our lack of findings may be the relatively low frequency of severe problematic alcohol use in the sample, compared to previous studies identifying these differences in AUD participants (Manning et al., 2016; Naim-Feil et al., 2014; Stavro et al., 2013). A similar study that looked at a measure of cognitive control and a measure of reward-related attentional bias controlling for trait impulsivity in a non-clinical sample, Albertella and colleagues (2017), also failed to find an association between neurocognition and alcohol use frequency. Further, a recent study found that in a high-severity sample, problematic alcohol use was not linked to impairments in inhibitory control, flexible updating, and processing speed (Meredith et al., 2020). However, the study sample size was conservative and the authors acknowledged that the effect sizes may be smaller than the medium effect they were powered to detect.

It was also unexpected that a relationship between neurocognitive function and AE severity was not found in Chapter 3. AE has been found to be associated with poorer inhibitory control and inflexible task switching (Rodrigue et al., 2018), impaired performance monitoring (Franken et al.,

2018) and steeper delay discounting (VanderBroek-Stice et al., 2017). Similar to the alcohol use findings, this lack of association might be attributed to the relatively low AE severity level in the sample. However, when analysing cross-sectional associations in Chapter 4, steeper temporal discounting was significantly positively associated with AE. The primary difference between the samples included in Chapter 3 and 4 was age. The participants in Chapter 4 were aged between 18 and 35, whereas those in Chapter 3 spanned from 18 to 65. Subsequent post hoc analyses revealed that there was a significant interaction between age and discounting rate. Individuals under 30 demonstrated a positive relationship between AE and discounting, while those over 30 exhibited a negative relationship [Appendix A]. This discovery clarifies why a significant positive relationship emerged in Chapter 4, but not in the broader and more diverse age range analysed in Chapter 3. Further, this finding suggests that different neurocognitive functions may be relevant for AE at different stages of life. For instance, steeper discounting may be a key factor underpinning problematic eating in younger adults (aged 18-30 years) specifically.

The findings in Chapter 3 revealed a significant association between poorer performance monitoring and higher PUI. Contrary to expectations, our study did not reproduce previous findings that link PUI with less flexible updating, poorer response inhibition and more risky decision-making (Ioannidis et al., 2019). This may in part stem from the approach taken; we examined PUI dimensionally, while earlier studies compared individuals diagnosed with PUI to control groups, therefore these samples were engaging in PUI at higher levels than the present study. However, considering that performance monitoring is essential for supporting higher-order functions like cognitive control (Ferdinand & Czernochowski, 2018), it may signify that performance monitoring deficits are detectable above and beyond other higher-order neurocognitive functions. The relationship between performance monitoring and PUI was also evident in the baseline analyses in Chapter 4, although this relationship was only observed at a trend level. This discrepancy is likely due to the reduced sample size in Chapter 4 analyses.

Contrary to findings from a systematic review and meta-analysis that showed PUI is consistently associated with steeper delay discounting (Cheng et al., 2021), the results from Chapter 3 revealed the opposite findings. Less steep delay discounting was found to be associated with more

severe PUI. This unexpected relationship might be attributed, once again, to our examination of PUI across a spectrum in which a substantial proportion of the sample had PUI severities that did not meet the threshold for a "diagnosis". In contrast, Cheng and colleagues (2021) systematic review compared individuals with a "diagnosis" of PUI and those without. Upon conducting additional exploratory correlation analyses solely among individuals meeting the threshold for a "diagnosis" of PUI in our Chapter 3 sample, no contradictory relationship was found [Appendix Figure B]. When conducting the same analysis in individuals who did not meet the PUI diagnostic threshold, we found a near significant negative relationship ($p$=.05; [Appendix Figure B]). This suggests that the presence of less severe cases might have influenced the observed effect by diluting the potential findings. Additionally, given we observed varying associations depending on whether PUI is examined categorically or dimensionally, may suggest PUI does not necessarily exist on a linear continuum as do other addictions (i.e. alcohol). It has been found that internet use at mild levels is more beneficial to mental health than no use at all (Lee et al., 2016). Further, Tiego et al. (2021) have argued that PUI (as measured by the IAT-10) only reflects meaningful variance when above the classification cut-off (a score of $\geq$ 17). Taken together, it is reasonable to suggest that internet use can be beneficial to a certain extent before then becoming problematic.

Similar to our performance monitoring PUI findings, the results from Chapter 3 showed poorer performance monitoring was also associated with higher PPU. These findings imply performance monitoring may be a common neurocognitive predictor across different addictive behaviours. However, considering the co-occurrence of PPU and PUI in the sample, and given that accessing pornography usually occurs online, our findings may be driven by an overlap between individuals who reported higher PUI scores because of their online pornography use. Additional analyses demonstrate that when adjusting for PUI in the statistical model, performance monitoring was no longer significantly associated with the severity of PPU [Appendix C]. In a systematic review addressing the relationship between neurocognitive functioning and PPU, it was concluded that PPU was characterised by impaired inhibitory control, poorer working memory and steeper delay discounting (Castro-Calvo et al., 2021). However, the majority of the studies reviewed looked at neurocognition in the context of sexual stimuli rather than "pure" neurocognitive functions as

evaluated in this thesis. Further, those studies that did evaluate stimuli-nonspecific neurocognitive deficits did not look at PPU specifically (Au & Tang, 2019; Lawyer, 2008). Accordingly, it would be inappropriate to compare our Chapter 3 findings with the findings from this review. However, our absence of neurocognitive findings aligns with a recent study comparing individuals diagnosed with Compulsive Sexual Behavior Disorder (CSBD) and controls (Draps et al., 2021). In their study, Draps and colleagues conducted an extensive assessment using various neurocognitive tasks to evaluate cognitive control and reward-related functions. Interestingly, they discovered no significant differences in performance across tasks measuring risk and ambiguity, incentive delay, reward learning, or response inhibition (Draps et al., 2021). When considered alongside the results from Chapter 3, it appears that the neurocognitive profile associated with problematic sexual behaviours, such as PPU, may diverge from what is typically observed in other forms of addiction.

## 5.3 The longitudinal predictors of substance-related addictive behaviours

In Chapters 2 and 4 of the thesis, the objective was to evaluate the longitudinal neurocognitive predictors of addictive behaviours. This was done through a systematic review of existing literature and a subsequent 6-month empirical longitudinal study. The following discussion synthesises the findings from both chapters, revealing that the neurocognitive predictors of addiction vary based on characteristics of the sample, the severity, and the specific type of addictive behaviour under consideration.

### 5.3.1 Adolescent cohort studies

The findings from Chapter 2 (systematic review) of the thesis highlight that the age and life stage at which neurocognitive functions are evaluated impact whether they are relevant predictors of substance use. Systematic synthesis of the current literature revealed that functions assessed in adolescence predicted increased substance use in adulthood. Response inhibition assessed at age 12 (Jones et al., 2021), and delay discounting at age 15 (Audrain-McGovern et al., 2009) predicted increased substance use (alcohol consumption and tobacco use respectively) at age 20. However, response inhibition and delay discounting assessed in adolescence at age 14 did not predict alcohol consumption at age 16 (Whelan et al., 2014). This suggests that adolescent cohort studies may

necessitate a more extended follow-up period, spanning from adolescence to early adulthood, to observe the enduring impact of neurocognitive function. It is plausible that more time is needed for discernible differences in substance use to emerge.

### 5.3.2 Different neurocognitive functions may be relevant at different stages of addiction

The findings from Chapter 2 also revealed that in adult cohorts, different neurocognitive functions may be relevant at different stages of substance use severity. For example, when assessed in adults recreationally engaging in alcohol use (i.e. college students) response inhibition was not found to predict increased alcohol consumption (Bø et al., 2017; Goudriaan et al., 2011). However, when neurocognition is evaluated post onset of heavy or problematic use (i.e. reaching the clinical threshold), poorer response inhibition, steeper delay discounting and steeper discounting for rewards (i.e. preference for larger but uncertain versus smaller certain rewards) predicted worsening of problematic drinking and SUD severity (Kräplin et al., 2020; Rubio et al., 2008). It may well be that college student cohorts capture individuals who are able to drink moderately and in control and have a lower risk of developing problems, while when assessed in high-risk samples (i.e. already heavily engaging in alcohol use) neurocognitive functions can more easily distinguish who go onto have more severe problems. These findings suggest neurocognitive functions are important predictors of substance use progression at later stages of use.

The results from Chapter 2 indicate that most evidence seems to refute the idea that any particular neurocognitive function reliably predicts clinical treatment outcomes. Specifically, neither response inhibition (López-Torrecillas et al., 2014; Passetti et al., 2008, 2011; Stevens et al., 2015), delay discounting (Bernhardt et al., 2017; López-Torrecillas et al., 2014; Passetti et al., 2008, 2011; Tucker et al., 2016) nor goal selection/updating (Czapla et al., 2016) consistently predicted alcohol or other substance use relapse. However, two distinct findings did emerge. First, steeper probability discounting for losses (i.e. preference for larger but uncertain versus smaller certain loss; Bernhardt et al., 2017) predicted alcohol use relapse. Second, less reward-related attentional capture (i.e. more goal-directed learning) predicted increased likelihood of alcohol abstinence during a community abstinence challenge (Albertella et al., 2021). These findings extend those of Domínguez-Salas and

colleagues (2016), who, in a similar systematic review, showed decision-making assessed via the Iowa Gambling Task (IGT) consistently predicted risk of substance use relapse. The IGT is a decision-making task requiring the individual to evaluate future rewards and losses in the context of uncertain risk (Bechara et al., 2002). Poor performance on this task is indicative of two distinct deficits: insensitivity to future consequences of an action; and hypersensitivity to reward outcomes (Bechara et al., 2002). The findings from Chapter 2 further delineate this distinction, showing that insensitivity to potential loss (probability discounting for losses) and hypersensitivity to reward-related cues (reward-related attentional capture), independently predict relapse and abstinence.

Collectively, the results from Chapter 2 demonstrate that not only different neurocognitive functions are important at different stages of problematic use, but also the neurocognitive functions linked to predicting substance use severity are different again from those that predict relapse. For example, response inhibition and delay discounting have been found to predict substance use progression in both early (Audrain-McGovern et al., 2009; Jones et al., 2021) and late (Kräplin et al., 2020; Rubio et al., 2008) stages of use, but are not predictive of relapse. These findings indicate different cognitive functions should be targeted depending on whether the priority is to evaluate risk of developing problematic substance use, or to identify mechanisms that can be targeted to improve addiction outcome.

### 5.3.3 Predicting problematic alcohol use in community samples

The findings in Chapter 2 revealed that while several studies have examined the neurocognitive predictors of alcohol consumption, (i.e. frequency and/or quantity of use), less attention was paid to investigating the predictors of the severity of problematic alcohol use, as measured by instruments like the AUDIT (Goudriaan et al., 2011; Kräplin et al., 2020; Rubio et al., 2008). This aspect is crucial because measures of consumption do not fully encompass critical elements of problematic alcohol use, such as the lack of control over drinking, the impact of alcohol use on daily functioning, or the negative emotional associations with drinking. Exploring predictors of problematic use is especially important in general community samples where individuals might engage in frequent or heavy drinking without necessarily developing addiction-related issues.

Among the existing studies that explored the severity of problematic alcohol use, most used a targeted recruitment approach to gather samples engaged in heavy use (Rubio et al., 2008) or already meeting criteria for SUD (Cousijn et al., 2015; Kräplin et al., 2020). Only one study investigated addictive behaviour dimensionally, recruiting individuals across low to high severity of problem alcohol use (i.e. low, moderate, and high binge drinking; Goudriaan et al., 2011) and they did not find neurocognition predicted problem use. Chapter 4 of the thesis addressed this gap in the literature by comprehensively examining whether neurocognitive functions predicted problematic alcohol use dimensionally in a community sample. In keeping with Goudriaan and colleagues' (2011) findings, neither response inhibition nor any other neurocognitive functions of interest were found to predict future problematic alcohol use. Collectively, the findings from Chapters 2 and 4 suggest that in general community samples not already engaged in high levels of problematic use, neurocognition might not be a reliable predictor of future problematic alcohol use.

## 5.4 The longitudinal predictors of non-substance addictive behaviours

The findings from Chapter 2 and 4 indicate that substance and non-substance addictive behaviours are predicted by different neurocognitive functions. Despite only two studies investigating non-substance related addictive behaviours, both found distinct relationships compared to substance use outcomes. Kräplin et al. (2020) found probability discounting for losses predicted severity of non-substance but not substance addictive behaviour. Further, Goudriaan et al. (2008) showed delay discounting predicted gambling relapse, which has not been shown in studies looking at substance use relapse (Bernhardt et al., 2017; López-Torrecillas et al., 2014; Passetti et al., 2008, 2011; Tucker et al., 2016). Similarly, the results from Chapter 4 of this thesis revealed different neurocognitive functions predicted different behaviours. In fact, not only were there differences between substance versus non-substance addictive behaviours, but also amongst the non-substance addictive behaviours themselves. This highlights the heterogeneity of potential neurocognitive mechanisms underpinning these addictive behaviours and suggests that it is equally as important to pursue research further articulating the differences as well as the similarities between neurocognitive mechanisms of specific behaviours.

### 5.4.1 Addictive eating

Findings from Chapter 4 revealed poorer performance monitoring predicted higher AE symptoms at 3-month follow-up. This implies that individuals facing challenges in self-monitoring their task performance, may experience similar difficulties in real-world monitoring of behaviours such as eating. They may therefore be more susceptible to developing problematic eating issues in the future. This might indicate a difficulty in recognising when one is overeating, for example, ignoring signals of fullness. These findings carry significance for treatment strategies for AE, as unlike other addictive behaviours, eating is essential for life. Abstinence from eating is impossible, and even reducing certain foods is challenging given the widespread availability of tempting, palatable foods in daily life. For some individuals, employing treatment strategies that focus on awareness of behaviour and physical sensations, such as mindfulness, might be more effective in addressing deficits in performance monitoring. This could potentially lead to better control overeating behaviour in those experiencing problematic eating patterns.

Independent of the relationship between performance monitoring and AE, Chapter 4 also revealed greater reward-related attentional capture predicted higher AE symptoms albeit at trend levels. Research has demonstrated that neural activation in brain regions associated with visual attention in response to food packaging can influence subsequent food choice behaviour (Van Der Laan et al., 2012). Further, it has been observed that more visually appealing packaging tends to enhance the intention to purchase a particular food item (Van Der Laan et al., 2012). These combined findings indicate that for certain individuals, problematic eating behaviour may be driven by a hypersensitivity to reward-related cues. Consequently, these individuals might be more susceptible to food-related cues, including attractive packaging and visual advertising, thus potentially perpetuating AE severity. This finding has important implications for food packaging and public health policies more broadly, particularly concerning unhealthy food products. Moreover, it suggests the potential effectiveness of attention training techniques such as Approach-Avoidance Training (AAT). Studies have demonstrated AAT using food cues can reduce approach bias to unhealthy foods (Ferentzi et al., 2018; Kakoschke et al., 2017, 2018) and increase healthier food choice behaviours (Kakoschke et al., 2017, 2018).

### 5.4.2 Problematic pornography use

Chapter 4 was the first study to evaluate the longitudinal neurocognitive predictors of PPU. Results revealed that shallower delay discounting predicted higher PPU at follow-up. While seemingly counterintuitive, similar findings have previously been observed and presented by Kowalewska and colleagues (2017), who showed preliminary evidence that individuals classified as 'sex addicts' (a phenomena that falls under the same umbrella as PPU of 'hypersexuality'; Karila et al., 2014) exhibited a preference for larger, later rewards compared to a control group. However, these findings were presented in conference proceedings and published in the form of an abstract, thus have not been subject to sufficient peer review. More importantly, our findings contrast with a large-scale study of over 1,000 males that showed individuals who problematically use internet pornography had steeper delay discounting compared to those engaging in recreational use (Antons et al., 2019). It is unclear why we found this negative relationship between delay discounting and PPU. Although the study sample showed a comparable prevalence of PPU as other general population samples (7%; Mennig et al., 2020), most of the current sample showed no or low PPU severity. Such a low occurrence of more severe levels of PPU may have impacted our ability to observe meaningful associations with 'problematic' pornography use. It is suggested that future PPU research should use targeted recruitment strategies, such as porn-specific online forums, to obtain a more varied spread of problematic use.

### 5.4.3 Problematic use of the internet

Chapter 4 also revealed an unexpected relationship between seemingly more adaptive neurocognitive functioning and higher levels of PUI. Less reward-related attentional capture and less risk-taking under ambiguity predicted higher PUI at follow-up. These findings are contrary to that expected, given addictive behaviour is typically thought to be marked by more risky decision-making (Fernie et al., 2010; Hopko et al., 2006) and reward-related attentional bias (Albertella, Pelley, et al., 2019). Follow-up analyses in individuals who met full-threshold for a PUI "diagnosis" revealed the same relationships between reward-related attentional capture and risky decision-making [Appendix D]. Further, when re-running the 3-month predicting 6-month follow-up model in the PUI only

sample, the relationship between reward-related attentional bias and PUI was retained [Appendix D], suggesting a reliable association between more goal-directed reward learning and higher levels of problem use. Addictive behaviours can be goal-directed, driven by relief-based motivations (Köpetz et al., 2013; Liu et al., 2021). For example, Hogarth conceptualised addiction as excessive goal-directed decisions driven by extreme negative affect. As such, more goal-directed learning may reflect more goal-directed motives to engage in internet use (Albertella, Vd Hooven, et al., 2021), which aligns with previous research proposing that excessive internet use is a response to fulfilling psychosocial needs and managing emotional distress through compensatory strategies (Caplan, 2003; Davis, 2001; Kardefelt-Winther, 2014; Scerri et al., 2019; Van Rooij & Prause, 2014).

An alternate interpretation of these neurocognitive findings is that they suggest PUI is better conceptualised as a compulsive rather than an 'addiction disorder'. Considering the BART findings in Chapter 4, less risk-taking under ambiguity seen here is similar to that observed in individuals with obsessive-compulsive disorder (Pushkarskaya et al., 2015). However, these findings may instead reflect a failure of the tool used to measure PUI to appropriately distinguish between high commitment to internet use from problematic involvement (Perales et al., 2020). The tool selected to assess problematic internet use in the present thesis (IAT-10) has been shown to be less sensitive to more severe or clinical levels of PUI, due to the omission of more severe items during scale formation (Tiego et al., 2021). For example, the IAT-10 does not fully capture crucial aspects of addiction-like behaviours, such as loss of control and continued use despite negative consequences (Tiego et al., 2021). Further, the definition of PUI based on the IAT-10 lacks fundamental features integral to the traditional understanding of addiction, such as withdrawal and tolerance, as noted by other studies (Tao et al., 2010; Weinstein & Lejoyeux, 2010). Therefore, the findings presented in this thesis may reflect more on the limitations of the behavioural scale used to measure PUI rather than being inherent characteristics of the concept of PUI itself.

Another important aspect to consider is that gamified cognitive tasks may not be suitable for populations experiencing PUI due to the probability that those with PUI are accustomed to, and proficient in playing online games. This is particularly relevant to the reward-related attentional bias findings. In internet gaming, many games demand that players concentrate on the game's objective

without getting distracted by colourful, gamified elements. For instance, first-person shooter games like Call of Duty, strategy fighting games such as World of War Craft, and Role-Playing Games like Skyrim or The Witcher all involve quests culminating in dynamic fighting sequences, requiring focused attention amongst distracting visual stimuli. It is plausible that individuals in our sample referred to their gaming behaviour as one of their primary problematic internet use behaviours. Consequently, our sample may be more adept at developing goal-directed strategies for performing neurocognitive tasks such as the VMAC task. However, this theory is less relevant to individuals whose PUI behaviours do not involve online gaming (i.e. social media use). Given the study did not differentiate between specific types of PUI in the sample, this assumption is untested.

## 5.5 Neurocognitive mechanisms of addictive behaviours

Taken together, Chapters 2 to 4 demonstrate that each addictive behaviour is both associated with (Chapter 3) and predicted by (Chapter 2 and Chapter 4) different neurocognitive functions. Findings from Chapter 2 revealed that differences are evident across different substance classes, for example, delay discounting predicted tobacco (Audrain-McGovern et al., 2009; Kräplin et al., 2020) but not alcohol use outcomes (Bernhardt et al., 2017; Fernández-Artamendi et al., 2018; Fröhner et al., 2022; Khurana et al., 2017; Tschorn et al., 2021; Whelan et al., 2014; Worhunsky et al., 2016). Similarly, findings from Chapter 3 and 4 revealed different neurocognitive functions were relevant to different addictive behaviours, for example reward-related attentional bias predicted AE and PUI but in the opposite direction. Our findings suggest not only do the mechanisms underlying risk of addiction need to be qualified by the specific substance (Lee et al., 2019), but also the specific behaviour in question. Further, these findings support the notion that non-substance addictions are more appropriately conceptualised as part of a spectrum of related, but distinct, conditions (Perales et al., 2020). It is recommended that future research on the neurocognitive mechanisms of addiction further interrogate these differences, understanding how each distinct addictive behaviour may be characterised by unique and specific neurocognitive patterns, as well as continuing to identify shared predictive mechanisms.

Over the past decade, there has been growing interest in the concept of precision psychiatry, an approach for prevention and treatment of mental health disorders taking into account an individual's unique characteristics, such as genetics, biomarkers, neurocognitive profile, and lifestyle factors (Fernandes et al., 2017; Passos et al., 2022; Zanardi et al., 2021). The current findings of this thesis support the central premise motivating this school of thinking. Specifically, there is significant heterogeneity within addiction and addictive behaviours. As such, treatment approaches need to be tailored according to the behaviour type, which will allow for a more targeted approach to ameliorating underlying neurocognitive deficits, and may result in more successful treatment outcomes. For example, delivering attentional bias modification interventions (e.g. AAT) to individuals with AE, but not PUI.

## 5.6 Limitations and future directions

### 5.6.1 The pitfalls of gamification

While gamification putatively served to boost participant motivation and engagement in completing unsupervised neurocognitive tasks (Bernecker & Ninaus, 2021; Lumsden et al., 2016), it is important to consider its limitations. Chief among these is that the association between the BrainPAC gamified tasks and the original paradigms was significantly less than expected, with each primary outcome metric showing only small (VMAC Task) to large (BART) associations (Lee et al., 2023). Further, the less a task was gamified, the greater the association between the original and the gamified paradigm. For example, the total points for the original VMAC Task was significantly higher than the gamified paradigm (Lee et al., 2023), which suggests the gamified elements were perhaps more distracting or salient, impairing speed and accuracy of performance and therefore may have compromised the purity of the paradigm. These findings potentially underscore the likelihood of task-specific effects influencing the results. However, validation of the BrainPAC tasks found the gamified paradigms showed stronger correlations with real-world impulsive and compulsive behaviours than the original paradigms, for example the SST was significantly positively associated with impulsive behaviours, and the BART was significantly associated with more impulsive behaviours, compulsive behaviours, and problematic alcohol use (Lee et al., 2023). This suggests

gamification of standard paradigms may improve the validity of these tasks in evaluating real-world actions. An ongoing question in the field is how to balance the benefits of incorporating gamified elements to enhance engagement without compromising the validity of indexing a particular neurocognitive paradigm. It remains crucial to continue to enhance these gamified neurocognitive assessments, given their utility in collecting data at scale, ability to be delivered remotely, and reach populations that would typically be challenging to assess.

### 5.6.2 Challenges with remote data collection

Both empirical studies in this thesis were conducted using online samples. While this approach facilitated the recruitment of participants nationwide, and enabled testing of a larger sample size than that which is feasible when conducting face-to-face assessments, this approach does come with some limitations. The utilisation of online samples and remote data collection, especially for task-based assessments, increases the risk that participants are careless in their responses or do not provide sufficient effort. Further, the unsupervised administration of neurocognitive tasks, particularly without standardisation across time and context, poses an increased risk of inflated measurement error. However, efforts were made to mitigate these risks through both pre-planned and post hoc data cleaning procedures. Further the study protocols were specifically designed to reduce participant burden and enhance motivation and engagement, for example utilising gamified neurocognitive tasks and ensuring assessment sessions were no longer than one hour at a time.

### 5.6.3 Limitations of single task assessments of neurocognition

A potential critique of this thesis is the use of single neurocognitive tasks to assess potentially latent mechanisms. When aiming to characterise individual differences, measurement reliability is crucial. However, the BrainPAC task paradigms show inconsistent test-retest reliability. Although the SST stop signal reaction time showed adequate test-retest reliability and the VMAC total points showed excellent test-retest reliability, other task metrics such as the VMAC score, BART pre-committed pumps and SDT weighting parameter showed reduced reliability (Lee et al., 2023). However, this problem is not specific to the tasks used in the present thesis, rather a widespread problem in the field in general as research indicates that the test-retest reliability of neurocognitive

tasks varies significantly (Enkavi et al., 2019) and task-based assessments are subject to inherent measurement error (Hedge et al., 2018).

One potential solution to address these challenges lies in using latent variable models, as they allow for the evaluation of neurocognitive functions on addictive behaviours at the level of latent constructs rather than individual task performance (Goschke, 2014). Utilising multiple tasks that assess the same underlying construct (e.g. Go/No-Go task and SST for inhibitory control) and deriving latent variables that represent convergent evidence from both measures (Goschke, 2014; Verdejo-Garcia & Albein-Urios, 2021), may provide a more robust and comprehensive way to measure and understand cognitive processes. Latent variable models can partition out measurement error. Further, many neurocognitive constructs are broad and cannot be fully captured by a single task (Ruiz et al., 2023), while latent factors allow for the measurement of variables comprehensively using multiple tasks. However, this approach would increase participant burden in an already time-consuming and detailed research protocol. Further, while latent variable models have been shown to produce more stable variables (Enkavi et al., 2019), latent representations of neurocognitive functions have not proven to be superior predictors of real-world behaviours (Eisenberg et al., 2019).

### 5.6.4 The problem with one-time only neurocognitive assessments

In some studies, neurocognition has been shown to be dynamic and highly context-dependent (Schmitter-Edgecombe et al., 2020). For instance, factors such as fatigue (Guo et al., 2018), sleep quality (Drummond et al., 2006), and stress (Chang et al., 2020) have been shown to impact neurocognitive performance. As such, single or one-time assessments of neurocognition are influenced by both random and systematic intra-individual variability that is difficult to control (Sliwinski et al., 2018). Another crucial aspect to consider is the temporal relationships between neurocognition and addictive behaviour. Assessing neurocognition during specific "at-risk" times (e.g. under stress) or situations (e.g. cue exposure) might provide the best insight into ecologically valid behaviours.

Utilising Ecological Momentary Assessment (EMA) paradigms presents an attractive solution to address the above issues. EMA involves multiple assessments of an individual’s functioning over

time in varied contexts, enhancing the precision and reliability of measurements by accounting for within-person variability, and establishing a more accurate estimation of an individual's average level of function (Shiffman et al., 2008; Sliwinski, 2008). Moreover, EMA enables the assessment of context-specific neurocognitive functioning, offering insights into the relationships between context, neurocognition, and behaviour. However, EMA studies are time consuming and substantially enhance participant burden (Burke et al., 2017), which may act as a barrier to study entry (i.e. deter potential participants from signing up) and lead to significant drop-out across the course of the study. Additionally, concise neurocognitive measures, specifically validated for administration through EMA within the domains examined in this thesis, have not yet been developed or validated.

### 5.6.5 Pathways to addiction, do different roads lead to the same destination?

An underlying assumption in the present thesis is that there are a core set of neurocognitive functions that predict addictive behaviours across all individuals. However, this perspective fails to acknowledge inter-individual variability or heterogeneity of mechanisms which may be driving inconsistencies in findings across both the field and the empirical chapters of this thesis. It is probable that there are multiple subtypes of individuals each with a distinct profile of neurocognitive functioning but share risk of developing addiction (Drossel et al., 2023; Mallorquí-Bagué et al., 2018). Specific neurocognitive factors might individually confer a certain level of addiction risk, but it is their combination, permutation, and potential interactive influence that truly matters. Studies employing clustering approaches have been successful in extracting neurocognitive profiles associated with addiction. For example, Dacosta-Sánchez and colleagues (2021) identified three distinct profiles of neurocognitive and affective functioning associated with SUD, one of which carried a higher risk of relapse. Similarly, different neurocognitive profiles have been identified in gambling (Devos et al., 2020) and three distinct routes to problem gambling have been proposed (Blaszczynski & Nower, 2002). Though beyond the aims and scope of this thesis, characterising individual neurocognitive heterogeneity in predicting addictive behaviours is crucial. This presents a promising avenue for future research, potentially leading to the identification of specific groups of individuals who can benefit from tailored treatments, moving towards personalised medicine (George

& Koob, 2017). So far, there have been no studies aimed at delineating *longitudinal* neurocognitive subgroups or trajectories that predict addiction. Such investigations would contribute to disentangling how neurocognitive changes interact over time in different individuals to increase addiction risk.

### 5.6.6 Neurocognition in the context of addiction-specific cues

An important aspect of this thesis is the deliberate selection of neurocognitive tasks free from specific substance or behaviour-related stimuli or cues, which allowed the examination of processes that transcend specific addictive behaviour types. However, Perales and colleagues (2020) assert that both domain-general and domain-specific functions are important in the development of addiction. They suggest that certain domain-general characteristics may make individuals more likely to develop addictive behaviours. However, whether this tendency actually leads to addiction depends on other factors, such as the inherent addictive qualities of the substance or activity involved, including its neurochemical or structural properties.

Further, the literature suggests that studies might yield more insights if cognitive functions were assessed in the context of addiction-specific cues (Brand et al., 2016), thus more closely replicating real-world decision-making scenarios. For instance, one's ability to exhibit inhibitory control in response to neutral stimuli (e.g., arrows) might differ significantly when faced with highly salient addiction-related cues (e.g., a glass of beer). This has been evidenced in studies looking at alcohol, cocaine and heroin use, showing impaired response inhibition in the context of substance-related cues but not neutral stimuli (Czapla et al., 2016; Pike et al., 2013; Su et al., 2020). Similar findings are observed in specific internet use disorders, showing poorer inhibitory control (Nie et al., 2016; Yao et al., 2015; Zhou et al., 2012), impaired goal selection/updating (Zhou et al., 2012), and inflexible updating (Nie et al., 2016) on tasks that employ internet-specific cues.

Addiction-related stimuli may also be particularly relevant when evaluating reward-related neurocognitive domains. For example, studies evaluating delay discounting have shown addiction-related outcomes (i.e. drugs, food and sex) are discounted more steeply than monetary outcomes (Odum et al., 2020). Moreover, it's more challenging to evoke a robust reward-related response from neutral and recently learned cues (such as those in the VMAC Task) in comparison to addiction-

related stimuli which have been linked to rewards over years of associative learning. However, assessing neurocognition within the context of addiction-related stimuli poses a risk that task performance is altered by an individual's past learning experiences. Task performance in the context of substance-related cues will be biased to one's prior exposure to the substance, making it difficult to disentangle what may be underlaying differences in neurocognitive functioning versus learned reward-response behaviours. Importantly, tasks that incorporate addiction-specific stimuli cannot be used transdiagnostically to compare across addictions, which was a primary goal of this thesis.

### 5.6.7 The impact of the COVID-19 pandemic

An important consideration when interpreting the results from the empirical chapters of this thesis is the influence of the COVID-19 pandemic. The data collection began in 2020 and extended through iterative recruitment and testing waves until mid-2022. Throughout a considerable proportion of this period, Australians faced repeated strict lockdowns and various levels of restrictions that affected travel, social interactions, and work. Consequently, behaviours during this time might not accurately reflect those in normal circumstances. This is particularly significant for internet use behaviours. With the internet serving as the primary means for work, socialising, and entertainment during the pandemic, higher than usual levels of PUI in our sample may be attributed to the increased online activity by everyone. Further, the PUI severity in our sample may also be a reflection of avoidant coping with the realities of the pandemic (Mota et al., 2021). However, whether increased time spent online during the pandemic tangibly resulted in an increased prevalence of PUI is unclear (Burkauskas et al., 2022). Our lower than usual problematic alcohol use in the sample was also likely contributed to by the pandemic. Australians showed a reduction in alcohol consumption during the pandemic, which was thought to be due to the closure of licenced venues and social distancing measures put in place during this time (Callinan et al., 2021). To mitigate the pandemic's potential impact on our collected data, we staggered recruitment and assessments. However, this approach limited our ability to control for the pandemic's effects on our findings. Nevertheless, the inclusion of psychological distress in all analysis models can serve as an indirect assessment of the pandemic's potential impact.

## 5.7 Concluding thoughts

This thesis offers a comprehensive and critical evaluation of the neurocognitive mechanisms underpinning both substance and non-substance-related addictive behaviours. Employing a deliberate selection of theory-driven and expert-endorsed neurocognitive constructs (Yücel et al., 2019), assessed through biologically validated paradigms (Insel et al., 2010), has shown behaviour-specific neurocognitive correlates and longitudinal predictors of addictive behaviours. This thesis suggests that different neurocognitive functions may play a role at various stages of problem use and, most notably, across different substance and non-substance addictive behaviour types.

To date, this thesis is the most comprehensive evaluation of neurocognitive predictors of non-substance addictive behaviours. The review is the first to systematically evaluate the neurocognitive predictors of addiction-related outcomes across longitudinal studies. The empirical studies are the first large-scale and comprehensive investigations of the neurocognitive correlates and predictors of substance and multiple non-substance addictive behaviours. Importantly, this thesis focusses on general population samples, allowing for addictive behaviours to be investigated dimensionally across a broad spectrum of risk, rather than solely focussing on clinical presentations.

The findings from this thesis lay a solid foundation for future research, suggesting several potential pathways. These include investigating the effectiveness of treatments that target specific neurocognitive mechanisms, such as performance monitoring and reward-bias for AE. Moreover, there is an opportunity to investigate whether motivation to engage in PUI, specifically goal-directed use, may influence PUI outcomes. Additionally, it is evident that more research articulating the neurocognitive underpinnings of PPU is essential. Replicating this work in higher risk samples (e.g. individuals already engaging in more severe addictive behaviours) and following such cohorts over a longer period of time (i.e. several years) would be valuable to allow for a deeper understanding of how the neurocognitive mechanisms identified in this thesis predict the trajectory to the development of an addiction disorder.

Finally, this thesis demonstrates the potential of a novel neurocognitive assessment battery (Lee et al., 2023) in predicting addictive behaviours. Although further refinement of this tool is

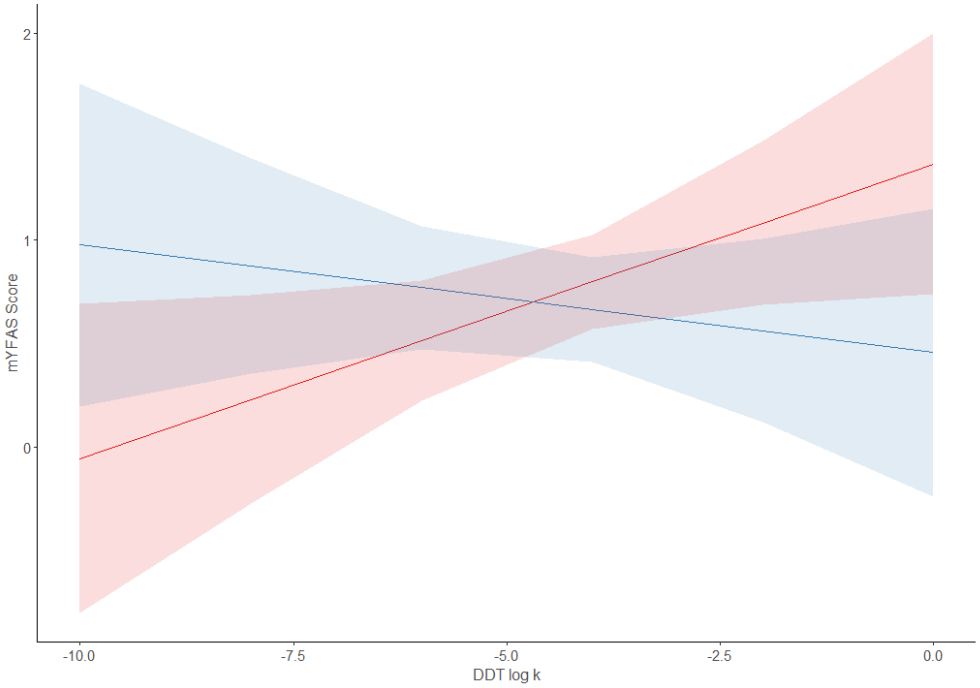
critical for future work, the findings underscore the value of using BrainPAC to gauge addiction-related neurocognitive functions, especially in the context of remote and large-scale data collection.

In conclusion, this thesis reveals that there is a disparity between expert consensus and empirical evidence regarding the transdiagnostic neurocognitive underpinnings of addiction. It is recommended that future research should focus on interrogating the reasons behind this disconnect, aiming to both enhance empirical investigations by adopting techniques discussed herein, and also refine theoretical understanding in accordance with emerging empirical evidence.

# Appendices

## Appendix A

**Figure A1.** The relationship between delay discounting and AE mediated by age




*Note: Colour denotes age bracket: 30 years and over (Blue), less than 30 years of age (Red)*

**Table A1.** Mediation regression model of AE

| Variables | β | *SE* | *p* |
|---|---|---|---|
| DDT | 0.14 | 0.07 | .033 |
| Age | -0.91 | 0.48 | .058 |
| DDT*Age | -0.19 | 0.10 | .047 |

*Note. β: Unstandardised coefficient; SE: Standard error.*

## Appendix B

**Figure B1.** Spearman correlation between delay discounting and PUI in individuals not meeting the threshold for PUI

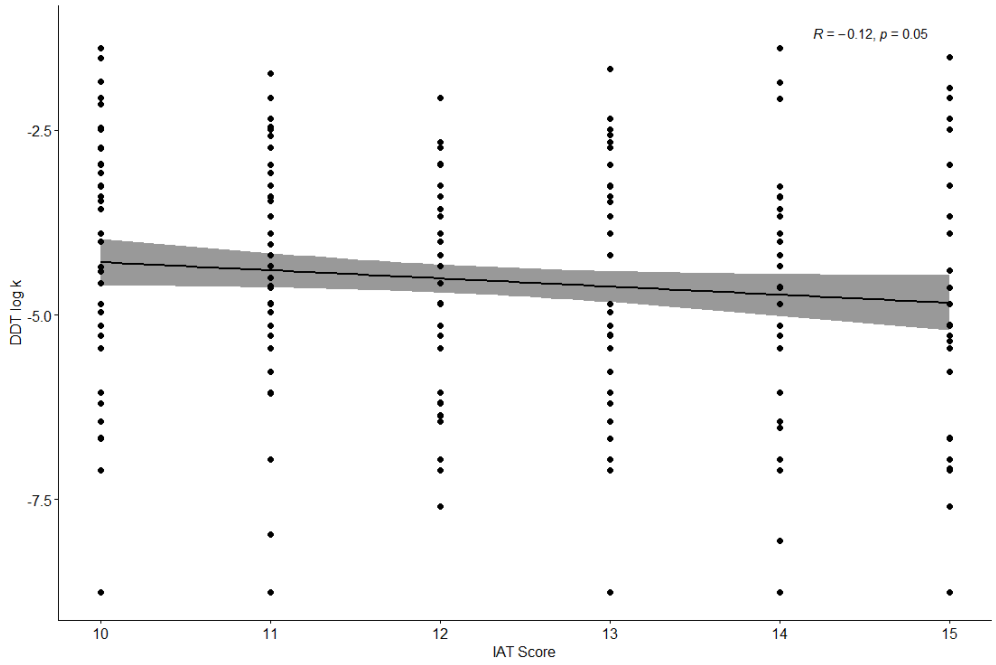
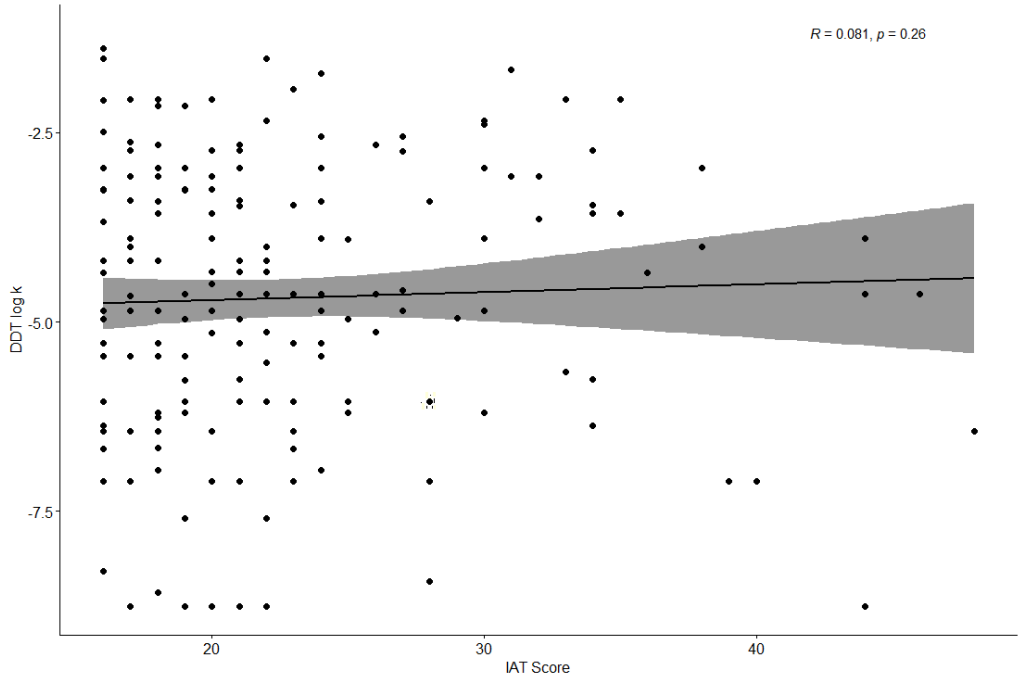



**Figure B1.** Spearman correlation between delay discounting and PUI in individuals meeting the threshold for PUI



# Appendix C

**Table D1.** Multiple regression model of PPU severity in Chapter 3 adjusted for PUI

| Variable | Baseline predicting 3-months (N=206) | | | | |
|---|---|---|---|---|---|
| | **β** | ***SE*** | **95% CI** | | ***p*** |
| | | | **LL** | **UL** | |
| Demographics | | | | | |
| Age | 0.04 | 0.02 | -0.03 | 0.06 | .467 |
| Sex (F) | -0.40 | 0.47 | -5.59 | -3.73 | <.001*** |
| Neurocognition | | | | | |
| SST: SSRT | -0.04 | 2.09 | -6.79 | 1.25 | .197 |
| VMAC: VMAC score | 0.04 | 4.06 | -3.20 | 12.63 | .258 |
| BART: *M* pre-committed pumps | -0.01 | 0.01 | -0.03 | 0.02 | .890 |
| CST: Switch cost latency | -0.00 | 0.00 | -0.00 | 0.00 | .943 |
| EAT: Error awareness | -0.06 | 0.01 | -0.02 | 0.00 | .187 |
| SDT: *w* | 0.06 | 0.60 | -0.12 | 2.23 | .076 |
| N-Back: 3-back d’ | -0.04 | 0.24 | -0.65 | 0.26 | .381 |
| DDT: Log *k* | -0.00 | 0.13 | -0.28 | 0.25 | .948 |
| Covariates | | | | | |
| IAT | 0.42 | 0.06 | 0.24 | 0.49 | <.001*** |
| DASS-TS | 0.04 | 0.03 | -0..03 | 0.07 | .444 |
| SUPPS-P: Lack of perseverance and premeditation | 0.00 | 0.18 | -0.34 | 0.34 | .994 |
| SUPPS-P: Urgency | 0.08 | 0.12 | -0.05 | 0.42 | .138 |
| SUPPS-P: Sensation seeking | 0.01 | 0.08 | -0.14 | 0.19 | .785 |
| CHI-T: Trait compulsivity | -0.00 | 0.05 | -0.10 | 0.10 | .963 |

*Note. β: Standardised coefficient; SE: Standard error; *p < .05, **p < .01, ***p < .001*

# Appendix D

**Table D1.** Longitudinal multiple regression models of PUI in individuals meeting the threshold for PUI

| Variable | Baseline predicting 3-months (N=206) | | | | | 3-months predicting 6-months (N=138) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | β | *SE* | 95% CI | | *p* | β | *SE* | 95% CI | | *p* |
| | | | LL | UL | | | | LL | UL | |
| Demographics | | | | | | | | | | |
| Age | 0.14 | 0.17 | -0.09 | 0.56 | .152 | 0.15 | 0.26 | -0.19 | 0.63 | .283 |
| Sex (F) | -0.04 | 1.58 | -3.62 | 2.34 | .655 | -0.28 | 1.89 | -6.20 | -0.46 | .026* |
| Neurocognition | | | | | | | | | | |
| SST: SSRT | -0.07 | 10.16 | -28.60 | 13.72 | .521 | -0.02 | 15.06 | -23.56 | 19.71 | .880 |
| VMAC: VMAC score | -0.20 | 18.56 | -71.73 | -5.08 | .023* | -0.25 | 20.29 | -73.18 | -3.60 | .033* |
| BART: *M* pre-committed pumps | -0.27 | 0.06 | -0.25 | -0.03 | .017* | 0.05 | 0.06 | -0.08 | 0.12 | .669 |
| CST: Switch cost latency | 0.03 | 0.00 | -0.01 | 0.01 | .793 | 0.12 | 0.01 | -0.00 | 0.02 | .326 |
| EAT: Error awareness | -0.11 | 0.30 | -0.07 | 0.02 | .245 | 0.06 | 0.03 | -0.03 | 0.06 | .642 |
| SDT: *w* | 0.10 | 1.90 | -1.89 | 5.66 | .337 | -0.13 | 2.10 | -5.95 | 1.92 | .297 |
| N-Back: 3-back d’ | -0.00 | 0.73 | -1.48 | 1.35 | .975 | -0.04 | 0.60 | -1.29 | 0.91 | .717 |
| DDT: Log *k* | -0.10 | 0.52 | -1.47 | 0.56 | .368 | 0.01 | 0.44 | -0.75 | 0.81 | .977 |
| Covariates | | | | | | | | | | |
| IAT | 0.63 | 0.19 | 0.53 | 1.02 | <.001*** | 0.65 | 0.14 | 0.36 | 0.82 | <.001*** |
| DASS-TS | -0.14 | 0.08 | -0.24 | 0.07 | .283 | 0.12 | 0.07 | -0.07 | 0.18 | .377 |
| SUPPS-P: Lack of perseverance and premeditation | 0.22 | 0.56 | -0.10 | 2.16 | .076 | 0.01 | 0.63 | -0.99 | 1.09 | .927 |
| SUPPS-P: Urgency | -0.08 | 0.40 | -1.10 | 0.54 | .505 | -0.01 | 0.51 | -0.91 | 0.86 | .953 |
| SUPPS-P: Sensation seeking | 0.11 | 0.34 | -0.33 | 0.95 | .358 | -0.21 | 0.41 | -1.15 | 0.26 | .217 |
| CHI-T: Trait compulsivity | 0.20 | 0.17 | -0.07 | 0.66 | .127 | 0.02 | 0.18 | -0.30 | 0.32 | .893 |

*Note. β: Standardised coefficient; SE: Standard error; *p < .05, **p < .01, ***p < .001*

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