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FULL-LENGTH REPORT

A comprehensive evaluation of the neurocognitive predictors of problematic alcohol use, eating, pornography, and internet use: A 6-month longitudinal study

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ABSTRACT

Background and aims: Cognitive control and reward-related abnormalities are centrally implicated in addiction. However, findings from longitudinal studies addressing neurocognitive predictors of addictive behaviors are mixed. Further, little work has been conducted predicting non-substance-related addictive behaviors. Our study aimed to assess predictors of substance and non-substance addictive behaviors in a community sample, systematically evaluating each neurocognitive function's independent influence on addictive behavior. *Methods:* Australians (N = 294; 51.7% female; M[SD] age = 24.8 [4.7] years) 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. Linear regressions with bootstrapping assessed neurocognitive predictors for each addictive behavior across a 6-month period. *Results:* Neurocognition at baseline did not predict AE or PUI severity at 6-month follow-up. Less delay discounting at baseline predicted higher PPU at 6-month follow-up (β = −0.16, p = 0.005). Poorer performance monitoring at baseline predicted higher AE at 3-month follow-up (β = −0.16, p = 0.004), and more reward-related attentional capture at 3-months predicted higher AE at 6-month follow-up (β = 0.14, p = 0.033). Less reward-related attentional capture (β = −0.14, p = 0.003) and less risk-taking under ambiguity (β = −0.11, p = 0.029) at baseline predicted higher PUI at 3-month follow-up. All findings were of small effect size. None of the neurocognitive variables predicted problematic alcohol use. *Discussion and conclusions:* We were unable to identify a core set of specific neurocognitive functions that reliably predict multiple addictive behavior types. However, our findings indicate both cognitive control and reward-related functions predict non-substance addictive behaviors in different ways. Findings suggest that there may be partially distinct neurocognitive mechanisms contributing to addiction depending on the specific addictive behavior.

KEYWORDS

internet, pornography, eating, neurocognition, addiction, longitudinal

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INTRODUCTION

A profile of neurocognitive dysfunction has been proposed to be critical across addictive behaviors (Yücel et al., 2019, 2021); specifically, reward-related learning and higher-order executive functions such as cognitive control (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 behaviors 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 behaviors.

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 behaviors merit investigation. Addictive Eating (AE), Problematic Pornography Use (PPU), and Problematic Use of the Internet (PUI) are common behavioral problems, affecting 2.6%–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, Hinman, Koball, Hoffmann, & Carels, 2013; Burrows, Kay-Lambkin, Pursey, Skinner, & Dayas, 2018; Camilleri, Perry, & Sammut, 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, Sedgmond, Maizey, Chambers, & Lawrence, 2019; Albertella, Pelley, et al., 2019; Jeromin et al., 2016; Mechelmans et al., 2014; Nikolaidou et al., 2019), and steeper temporal discounting (Amlung, Vedelago, Acker, Balodis, & MacKillop, 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, Billieux, Gjerde, Eilertsen, & Landrø, 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 behaviors. 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, Slade, Hunt, & Teesson, 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 behavior severities. This is especially important as even individuals engaging in addictive behaviors at less severe levels also experience adverse outcomes (Shankman et al., 2009). Understanding the neurocognitive mechanisms that predict addictive behaviors 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 behaviors 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 behaviors. Neurocognitive functions were selected per expert-endorsed domains which are thought to drive addiction (Yücel et al., 2019), capturing cognitive control, working memory, reward learning, and reward valuation functions as defined by the Research Domain Criteria (RDoC: Insel et al., 2010). 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 behavior, before then modeling 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 behavior at 3-month follow-up, and then the relationship between neurocognitive functions assessed at 3-months and addictive behavior at 6-month follow-up. All models accounted for both trait impulsivity and compulsivity as well as key covariates (i.e. psychological distress, sex and age).

METHODS

Participants

This study was embedded within a larger cohort study (Christensen, Albertella, et al., 2023) which aimed to collect normative data in a demographically-stratified sample and to investigate the neurocognitive correlates of addictive behaviors. Nine-hundred-and-forty-four community members 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 behavior disorders is 25 years of age (Solmi et al., 2022). Participants were Australian residents, who were not color 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

All data were collected remotely via participants' laptops or desktop computers. Participants provided basic demographic information (i.e. age, sex, average household income, and education status), completed neurocognitive tasks and self-report surveys at baseline and 3-months, and underwent a brief assessment of self-report scales, including addictive behavior scales at 6-months. A detailed description of the study protocol and a full list of measures can be found in the supplementary materials for this manuscript.

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).

Addictive behaviors. Problematic alcohol use was assessed by the Alcohol Use Disorder Identification Test total score (AUDIT; Saunders et al., 1993) which has shown excellent internal consistency: Cronbach's $\alpha = 0.96$ (Noorbakhsh et al., 2018). Addictive Eating (AE) was evaluated via the modified Yale Food Addiction Scale symptom count (mYFAS 2.0; Schulte & Gearhardt, 2017) which has shown good internal consistency: Cronbach's $\alpha = 0.88$ (Imperatori et al., 2019). Problematic Pornography Use (PPU) was assessed via the short version of the Problematic Pornography Consumption Scale total score (PPCS-6: Bőthe, Tóth-Király, Demetrovics, & Orosz, 2021) which has shown good internal consistency: Cronbach's $\alpha = 0.84$ (Bőthe et al., 2021). Problematic Use of the Internet (PUI) was evaluated by the abbreviated Young's Internet Addiction Test (IAT-10; Tiego et al., 2021) total score, which has shown good internal consistency: Cronbach's $\alpha = 0.80$ (Tiego et al., 2021). Participants reported addictive behaviors using the corresponding scale reflecting on the previous three months, with zero assigned if no engagement. All addictive behaviors were captured dimensionally.

Covariates. The total score of the Depression Anxiety Stress Scale was used to assess psychological distress (DASS-21; Szabó, 2010) which has shown excellent internal consistency: Cronbach's $\alpha = 0.93$ (Liu et al., 2021). The Cambridge-Chicago Compulsivity Trait Scale (CHI-T; Chamberlain & Grant, 2018; Tiego et al., 2023) assessed trait compulsivity, which has shown good internal consistency: Cronbach's $\alpha = 0.80$ (Chamberlain & Grant, 2018). The Short UPPS Impulsive Behavior 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). The SUPPS-P has shown acceptable internal consistency: Cronbach's $\alpha = 0.75$ (Liu et al., 2021).

Procedure

Assessments were delivered online using Qualtrics software (Version 08.23, 2023). 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 behavior scales). The order of task presentation was counterbalanced. At 6-month follow-up participants completed a single 30-min questionnaire that evaluated addictive behaviour engagement.

Statistical analysis

All data cleaning and analyses were performed using R Statistical Software (v4.1.2; R Core Team 2021). The data

Table 1. Neurocognitive tasks included in the study

Function	Task	Brief description
Response inhibition	The BrainPAC Stop Signal Taks (SST) 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 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) 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) 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 (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) (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) (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) 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.

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, Van Der Molen, & Logan, 2003]). Differences between individuals whose data were filtered out compared with included individuals were investigated using Welch's t-tests and when normality was violated Mann-Whitney U tests were used and reported in the supplementary materials. Similarly, Welch's t-tests and where appropriate 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 (Faul, Erdfelder, Lang, & Buchner, 2007) 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 (≥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 S1–4).

Four multiple regression models were generated for each outcome behavior of interest. The first model evaluated cross-sectional relationships between neurocognition, traits, covariates and addictive behavior at baseline. The second model included baseline variables as predictors of addictive behavior at 6-month follow-up, accounting for baseline addictive behavior severity, the third model included baseline variables as predictors of addictive behavior at 3-month follow-up, accounting for baseline addictive behavior severity, and the fourth model included variables measured at 3-months to predict addictive behavior at 6-month follow-up, accounting for 3-month addictive behavior severity (Tables 3–6). Visual inspection of residual plots and VIF values were evaluated independently for each regression model and revealed assumptions of linearity, independence, and no multicollinearity were met. VIF values less than 2.5 were taken to indicate no issue of multicollinearity (Johnston et al., 2018). Normality and homogeneity of variance were violated for each model, additionally, the distributions of all four outcome variables at each timepoint were positively skewed (Figure SII), constituting the choice of linear regression models with bootstrapping of residuals (5,000 samples; Hesterberg, 2015; Neal & Simons, 2007) using the boot.pval (Thulin, 2023) and car (Fox, 2019) packages. Age, sex, psychological distress, impulsivity, compulsivity, and the respective baseline addictive behavior 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 test the impact of serial correlation error the Heteroscedasticity and Autocorrelation Consistent (HAC) covariance matrix estimation was run for each longitudinal model (Andrews & Monahan, 1992), these results can be found in the supplementary materials (Tables S5–S8). 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; 10 imputations), these results are presented in the supplementary materials (Tables S9–S12).

Ethics

The study was approved by the Monash University Human Research Ethics Committee [26088]. All subjects were informed about the study and all provided informed consent.

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 (Fig. 1) the final sample sizes included in analyses were as follows: $n = 198$ baseline predicting 6-month outcomes, $n = 206$ baseline predicting 3-month outcomes, and $n = 138$ for 3-months predicting 6-month outcomes. Participant demographics are displayed in Table 2. When comparing individuals lost to 6-month follow-up and those who were retained on characteristic at baseline, participants lost to follow-up were younger (mean age = 23.7 vs 25.4 years; $r_{pb} = -0.22$, $p = 0.002$), had higher PPU scores (mean score = 11.2 vs 9.5; $r_{pb} = 0.18$, $p = 0.010$), had higher delay discounting (mean log $k = -4.2$ vs -4.7; Hedges' $g = 0.28$, $p = 0.025$), and more males did not return for the 6-month follow-up ($n = 110$) than females ($n = 42$). 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; $r_{pb} = -0.26$, $p < 0.001$) and showed better response inhibition (mean SSRT = 0.32 vs 0.34s; Hedges' $g = -0.32$, $p = 0.005$). 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 than those retained (mean score = 9.8 vs 8.6; Hedge's $g = 0.47$, $p < 0.001$), and more males did not come back for 6-month follow-up ($n = 20$) than females ($n = 17$).

Cross-sectional model

Steeper delay discounting (preference for sooner smaller rewards; $\beta = 0.15$, $p = 0.007$) was associated with greater AE. No further relationships were found between neurocognition and addictive behaviors.

Longitudinal multivariate models

Predicting problematic alcohol use. None of the neurocognitive variables predicted problematic alcohol use for any of the longitudinal models. These results held after correcting for serial correlation error and were replicated in regression models using multiple imputation (Tables S5 and S9).

Predicting addictive eating (AE). The 6-month longitudinal model did not find any neurocognitive variables significantly predicted AE. The 3-month longitudinal models showed poorer performance monitoring ($\beta = -0.16$, $p = 0.004$) significantly predicted higher AE at 3-month follow-up and greater reward-related attentional capture ($\beta = 0.14$, $p = 0.033$) at 3-month follow-up significantly predicted higher AE at 6-month follow-up. All of these results held after correcting for serial correlation error (Table S6) except the relationship between reward-related attentional capture and AE at 6-month follow-up reduced to trend levels ($p = 0.059$). Findings were also replicated in the regression models using multiple imputation (Table S10), although the performance monitoring and reward-related attentional capture effects reduced to trend levels (95% CI [−0.01, 0.00], $p = 0.066$; 95% CI [−0.86, 10.33], $p = 0.097$).

Predicting problematic pornography use (PPU). The 6-month longitudinal model showed less delay discounting ($\beta = -0.16$, $p = 0.005$) predicted higher PPU at 6-month follow-up. The 3-month longitudinal models showed neurocognition at baseline did not predict PPU at 3-month follow-up, but less delay discounting ($\beta = -0.16$, $p = 0.014$)

Table 2. Demographic and behavioral 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
% met diagnostic threshold *	5.4	6.2	9.4
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). * The mYFAS also provides a diagnostic score adapted from the DSM-V criteria for substance use disorder. To further characterize the sample, the percentage of individuals at each time point who met this diagnostic threshold is reported, however, these values were not used for statistical analyses.

at 3-month follow-up predicted higher PPU at 6-month follow-up. After correcting for serial correlation error delay discounting remained a significant predictor for the 6-month model but was no longer a significant predictor for the 3-month model (Table S7), these findings were also reflected by the regression models using multiple imputation (Table S11).

Predicting problematic use of the internet (PUI). The 6-month longitudinal model did not find neurocognition

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

	Baseline (N = 294)					Baseline predicting 6-months (N = 198)					Baseline predicting 3-months (N = 206)					3-months predicting 6-months (N = 138)				
			95% CI					95% CI					95% CI					95% CI		
Variable	β	*SE*	LL	UL	*p*	β	*SE*	LL	UL	*p*	β	*SE*	LL	UL	*p*	β	*SE*	LL	UL	*p*
Demographics																				
Age	0.04	0.05	−0.06	0.13	0.498	0.15	0.04	0.03	0.20	0.008**	0.04	0.03	−0.03	0.10	0.276	0.06	0.05	−0.06	0.16	0.372
Sex (F)	0.14	0.44	0.19	1.87	0.020*	−0.05	0.37	−1.10	0.38	0.334	0.02	0.29	−0.46	0.71	0.676	0.01	0.46	−0.81	1.01	0.821
Neurocognition																				
SST: SSRT	−0.03	3.17	−7.66	4.73	0.645	−0.10	2.75	−10.43	0.57	0.079	−0.03	2.25	−5.19	2.51	0.494	−0.07	3.41	−10.46	3.13	0.257
VMAC: VMAC score	−0.10	5.26	−19.66	1.34	0.079	−0.06	4.63	−14.26	4.02	0.273	−0.01	4.51	−8.25	5.99	0.768	0.02	5.39	−9.15	12.60	0.761
BART: *M* pre-committed pumps	−0.05	0.01	−0.04	0.02	0.417	−0.08	0.01	−0.04	0.01	0.154	0.01	0.01	−0.02	0.02	0.824	−0.04	0.01	−0.03	0.02	0.478
CST: Switch cost latency	−0.08	0.00	−0.00	0.00	0.189	−0.04	0.00	−0.00	0.00	0.416	0.02	0.00	−0.00	0.00	0.574	0.05	0.00	−0.00	0.00	0.357
EAT: Error awareness	0.07	0.01	−0.01	0.02	0.231	0.02	0.01	−0.01	0.01	0.694	0.01	0.00	−0.01	0.01	0.735	0.01	0.01	−0.02	0.02	0.867
SDT: *w*	−0.03	0.61	−1.37	0.93	0.692	−0.06	0.51	−1.54	0.43	0.285	−0.02	0.40	−0.98	0.57	0.601	−0.03	0.58	−1.45	0.81	0.572
N-Back: 3-back d'	0.11	0.19	−0.02	0.72	0.060	0.01	0.16	−0.30	0.35	0.900	0.02	0.11	−0.18	0.30	0.630	0.02	0.17	−0.25	0.42	0.647
DDT: Log *k*	0.02	0.13	−0.21	0.31	0.701	−0.03	0.11	−0.28	0.17	0.612	0.01	0.07	−0.15	0.20	0.722	−0.02	0.13	−0.31	0.21	0.719
Covariates																				
AUDIT	–	–	–	–	–	0.68	0.06	0.53	0.74	<0.001***	0.84	0.06	0.76	0.92	<0.001***	0.78	0.06	0.70	0.96	<0.001***
DASS: Total score	0.22	0.02	0.03	0.12	<0.001***	0.07	0.02	−0.02	0.06	0.248	0.04	0.01	−0.02	0.04	0.458	0.07	0.02	−0.02	0.07	0.245
SUPPS-P: Lack of perseverance and premeditation	0.07	0.14	−0.13	0.42	0.273	−0.13	0.12	−0.47	0.00	0.051	0.00	0.12	−0.19	0.19	0.995	−0.04	0.14	−0.38	0.21	0.508
SUPPS-P: Urgency	0.00	0.11	−0.23	0.22	0.985	0.17	0.10	0.06	0.44	0.013*	0.09	0.08	−0.01	0.29	0.064	−0.06	0.12	−0.34	0.13	0.406
SUPPS-P: Sensation seeking	0.27	0.08	0.18	0.51	<0.001***	−0.00	0.10	−0.14	0.14	0.978	0.02	0.05	−0.08	0.13	0.660	0.04	0.08	−0.11	0.21	0.567
CHI-T: Trait compulsivity	−0.04	0.05	−0.11	0.06	0.552	−0.08	0.04	−0.12	0.03	0.231	−0.06	0.04	−0.10	0.03	0.269	0.07	0.05	−0.06	0.16	0.360

Note. β: Standardized coefficient; SE: Standard error; $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

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

	Baseline (N = 294)					Baseline predicting 6-months (N = 198)					Baseline predicting 3-months (N = 206)					3-months predicting 6-months (N = 138)				
			95% CI					95% CI					95% CI					95% CI		
Variable	β	*SE*	LL	UL	*p*	β	*SE*	LL	UL	*p*	β	*SE*	LL	UL	*p*	β	*SE*	LL	UL	*p*
Demographics																				
Age	0.01	0.02	−0.03	0.05	0.799	0.06	0.03	−0.03	0.07	0.364	0.08	0.02	−0.01	0.07	0.175	−0.01	0.03	−0.06	0.05	0.891
Sex (F)	0.18	0.19	0.22	0.96	0.002**	−0.13	0.23	−0.95	−0.05	0.027*	−0.01	0.19	−0.42	0.34	0.849	−0.06	0.24	−0.69	0.28	0.398
Neurocognition																				
SST: SSRT	0.03	1.35	−1.89	3.52	0.579	0.03	1.67	−2.67	4.03	0.689	−0.05	1.24	−3.51	1.35	0.355	−0.01	1.83	−3.89	3.49	0.862
VMAC: VMAC score	0.03	2.22	−3.10	5.68	0.610	0.01	2.78	−5.12	5.85	0.894	−0.02	2.31	−5.31	3.82	0.740	0.14	2.86	0.53	11.62	0.033*
BART: *M* pre-committed pumps	0.01	0.01	−0.01	0.01	0.896	−0.06	0.01	−0.02	0.01	0.321	−0.00	0.01	−0.01	0.01	0.953	−0.11	0.01	−0.02	0.00	0.088
CST: Switch cost latency	0.02	0.00	−0.00	0.00	0.778	0.10	0.00	−0.00	0.00	0.104	−0.05	0.00	−0.00	0.00	0.421	0.01	0.00	−0.00	0.00	0.934
EAT: Error awareness	0.03	0.00	−0.00	0.01	0.640	−0.06	0.00	−0.01	0.00	0.306	−0.16	0.00	−0.01	−0.00	0.004**	−0.01	0.00	−0.01	0.01	0.970
SDT: *w*	−0.06	0.26	−0.76	0.24	0.303	0.04	0.31	−0.43	0.79	0.535	−0.03	0.24	−0.62	0.36	0.600	−0.07	0.31	−0.93	0.26	0.253
N-Back: 3-back d'	0.01	0.08	−0.14	0.17	8.29	−0.01	0.10	−0.21	0.17	0.863	0.04	0.08	−0.09	0.20	0.457	−0.07	0.09	−0.26	0.08	0.275
DDT: Log *k*	0.15	0.06	0.05	0.26	0.007**	0.10	0.07	−0.03	0.24	0.139	−0.01	0.06	−0.12	0.10	0.829	0.09	0.07	−0.04	0.23	0.148
Covariates																				
mYFAS	–	–	–	–	–	0.41	0.08	0.30	0.60	<0.001***	0.60	0.06	0.50	0.74	<0.001***	0.67	0.07	0.60	0.89	<0.001***
DASS: Total score	0.32	0.01	0.03	0.06	<0.001***	0.17	0.01	0.00	0.05	0.023*	0.08	0.01	−0.01	0.03	0.219	0.12	0.01	−0.00	0.04	0.094
SUPPS-P: Lack of perseverance and premeditation	−0.05	0.06	−0.16	0.07	0.454	0.09	0.07	−0.05	0.23	0.235	0.05	0.06	−0.07	0.17	0.419	0.07	0.08	−0.08	0.22	0.378
SUPPS-P: Urgency	0.05	0.05	−0.05	0.14	0.406	0.13	0.06	−0.01	0.22	0.083	0.09	0.05	−0.03	0.16	0.200	−0.05	0.06	−0.15	0.09	0.556
SUPPS-P: Sensation seeking	−0.13	0.03	−0.14	−0.01	0.023*	−0.01	0.04	−0.08	0.08	0.902	0.07	0.03	−0.03	0.11	0.251	0.03	0.04	−0.06	0.11	0.671
CHI-T: Trait compulsivity	0.13	0.02	−0.00	0.07	0.052	0.08	0.02	−0.02	0.07	0.296	0.08	0.02	−0.02	0.06	0.267	0.13	0.03	−0.02	0.09	0.140

Note. β: Standardized coefficient; SE: Standard error; $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

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

	Baseline (N = 294)					Baseline predicting 6-months (N = 198)					Baseline predicting 3-months (N = 206)					3-months predicting 6-months (N = 138)				
			95% CI					95% CI					95% CI					95% CI		
Variable	β	*SE*	LL	UL	*p*	β	*SE*	LL	UL	*p*	β	*SE*	LL	UL	*p*	β	*SE*	LL	UL	*p*
Demographics																				
Age	−0.05	0.07	−0.20	0.07	0.348	−0.06	0.06	−0.19	0.05	0.267	0.02	0.06	−0.09	0.14	0.647	−0.07	0.09	−0.26	0.09	0.306
Sex (F)	−0.51	0.62	−7.03	−4.64	<0.001***	−0.17	0.63	−2.99	−0.52	0.003**	−0.16	0.62	−3.01	−0.51	0.004*	−0.14	0.89	−3.38	0.13	0.074
Neurocognition																				
SST: SSRT	−0.02	4.59	−10.56	7.17	0.630	0.01	4.17	−7.08	9.09	0.851	0.01	3.64	−6.04	8.18	0.797	−0.04	5.77	−14.24	7.78	0.524
VMAC: VMAC score	0.00	7.57	−14.79	14.30	0.993	0.01	6.62	−11.90	14.54	0.840	0.06	6.77	−4.64	22.63	0.186	−0.02	9.05	−20.57	16.08	0.782
BART: *M* pre-committed pumps	0.02	0.02	−0.03	0.05	0.753	−0.03	0.02	−0.05	0.03	0.625	−0.00	0.02	−0.04	0.03	0.966	0.00	0.02	−0.04	0.04	0.986
CST: Switch cost latency	0.04	0.00	−0.00	0.00	0.476	0.07	0.00	−0.00	0.00	0.197	0.07	0.00	−0.00	0.00	0.176	0.04	0.00	−0.00	0.01	0.448
EAT: Error awareness	−0.03	0.01	−0.02	0.01	0.647	−0.06	0.01	−0.03	0.01	0.298	0.06	0.01	−0.01	0.02	0.268	0.03	0.01	−0.02	0.03	0.632
SDT: *w*	0.06	0.84	−0.72	2.59	0.300	0.02	0.79	−1.25	1.74	0.734	0.07	0.74	−0.40	2.46	0.136	0.05	0.96	−1.25	2.58	0.417
N-Back: 3-back d'	−0.08	0.26	−0.95	0.08	0.104	0.06	0.24	−0.21	0.72	0.267	0.02	0.22	−0.35	0.53	0.681	0.03	0.29	−0.40	0.69	0.664
DDT: Log *k*	−0.02	0.18	−0.44	0.27	0.638	−0.16	0.17	−0.82	−0.16	0.005**	−0.02	0.16	−0.38	0.26	0.729	−0.16	0.22	−0.97	−0.12	0.014*
Covariates																				
PPCS	–	–	–	–	–	0.63	0.05	0.49	0.70	<0.001***	0.67	0.05	0.56	0.77	<0.001***	0.68	0.08	0.61	0.93	<0.001***
DASS: Total score	0.13	0.03	0.01	0.13	0.034*	−0.00	0.03	−0.06	0.06	0.974	−0.01	0.03	−0.06	0.05	0.919	0.11	0.03	−0.01	0.12	0.120
SUPPS-P: Lack of perseverance and premeditation	0.02	0.20	−0.31	0.47	0.704	0.04	0.17	−0.23	0.47	0.480	−0.00	0.17	−0.35	0.33	0.944	−0.02	0.24	−0.54	0.41	0.809
SUPPS-P: Urgency	0.12	0.16	−0.03	0.60	0.075	0.05	0.14	−0.17	0.40	0.459	0.06	0.14	−0.13	0.42	0.281	−0.02	0.19	−0.41	0.35	0.871
SUPPS-P: Sensation seeking	−0.04	0.11	−0.30	0.14	0.451	−0.03	0.10	−0.26	0.15	0.552	−0.05	0.10	−0.28	0.10	0.345	0.05	0.14	−0.16	0.38	0.447
CHI-T: Trait compulsivity	0.12	0.06	−0.01	0.25	0.071	0.01	0.06	−0.11	0.12	0.915	0.03	0.06	−0.09	0.15	0.603	−0.02	0.09	−0.20	0.14	0.744

Note. β: Standardized coefficient; SE: Standard error; $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

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

	Baseline (N = 294)					Baseline predicting 6-months (N = 198)					Baseline predicting 3-months (N = 206)					3-months predicting 6-months (N = 138)				
			95% CI					95% CI					95% CI					95% CI		
Variable	β	*SE*	LL	UL	*p*	β	*SE*	LL	UL	*p*	β	*SE*	LL	UL	*p*	β	*SE*	LL	UL	*p*
Demographics																				
Age	−0.12	0.08	−0.31	−0.02	0.027*	0.01	0.07	−0.12	0.16	0.796	−0.00	0.08	−0.15	0.15	0.997	0.09	0.08	−0.04	0.27	0.144
Sex (F)	−0.20	0.72	−4.05	−1.27	<0.001***	−0.07	0.65	−2.14	0.40	0.193	−0.06	0.70	−2.21	0.50	0.237	−0.05	0.71	−1.87	0.84	0.456
Neurocognition																				
SST: SSRT	0.07	5.03	−3.34	16.87	0.192	0.04	4.73	−5.37	13.52	0.392	−0.01	4.68	−10.20	7.82	0.803	−0.01	5.37	−11.35	8.83	0.804
VMAC: VMAC score	−0.04	8.29	−23.04	10.08	0.437	−0.05	7.53	−22.28	7.79	0.345	−0.14	8.75	−43.89	−9.16	0.003**	−0.07	8.16	−26.60	5.95	0.223
BART: *M* pre-committed pumps	0.07	0.02	−0.01	0.07	0.180	−0.09	0.02	−0.08	0.00	0.075	−0.11	0.02	−0.10	−0.01	0.029*	0.07	0.02	−0.01	0.06	0.236
CST: Switch cost latency	0.05	0.00	−0.00	0.01	0.365	0.01	0.00	−0.00	0.00	0.810	−0.00	0.00	−0.00	0.00	0.913	−0.01	0.00	−0.00	0.00	0.845
EAT: Error awareness	−0.10	0.10	−0.04	0.00	0.052	−0.04	0.01	−0.03	0.01	0.432	−0.09	0.01	−0.04	0.00	0.068	0.03	0.01	−0.02	0.03	0.643
SDT: *w*	−0.08	0.96	−3.31	0.48	0.143	−0.04	0.86	−2.42	0.99	0.433	−0.04	0.92	−2.51	1.13	0.465	−0.07	0.85	−2.73	0.69	0.238
N-Back: 3-back d'	−0.04	0.29	−0.83	0.34	0.390	0.03	0.27	−0.38	0.65	0.603	−0.00	0.29	−0.59	0.55	0.909	0.04	0.25	−0.31	0.68	0.484
DDT: Log *k*	−0.07	0.21	−0.72	0.11	0.158	−0.03	0.19	−0.48	0.28	0.555	−0.04	0.21	−0.60	0.21	0.365	0.00	0.20	−0.38	0.40	0.936
Covariates																				
IAT	–	–	–	–	–	0.65	0.05	0.46	0.67	<0.001***	0.72	0.06	0.65	0.89	<0.001***	0.77	0.06	0.54	0.75	<0.001***
DASS: Total score	0.31	0.04	0.11	0.25	<0.001***	0.02	0.03	−0.06	0.08	0.791	−0.05	0.04	−0.10	0.05	0.477	0.04	0.03	−0.04	0.08	0.520
SUPPS-P: Lack of perseverance and premeditation	0.09	0.22	−0.07	0.80	0.107	0.01	0.20	−0.36	0.42	0.837	0.10	0.22	−0.02	0.84	0.063	−0.05	0.22	−0.57	0.27	0.492
SUPPS-P: Urgency	0.11	0.18	−0.03	0.68	0.076	0.11	0.16	−0.05	0.59	0.100	−0.01	0.18	−0.38	0.32	0.850	0.06	0.17	−0.21	0.48	0.439
SUPPS-P: Sensation seeking	−0.14	0.13	−0.57	−0.08	0.015*	−0.02	0.12	−0.27	0.19	0.739	0.01	0.13	−0.22	0.28	0.784	−0.06	0.12	−0.35	0.11	0.305
CHI-T: Trait compulsivity	0.22	0.07	0.12	0.40	<0.001***	0.07	0.07	−0.06	0.20	0.287	0.09	0.08	−0.04	0.27	0.143	0.05	0.08	−0.10	0.21	0.460

Note. β: Standardized coefficient; SE: Standard error; $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

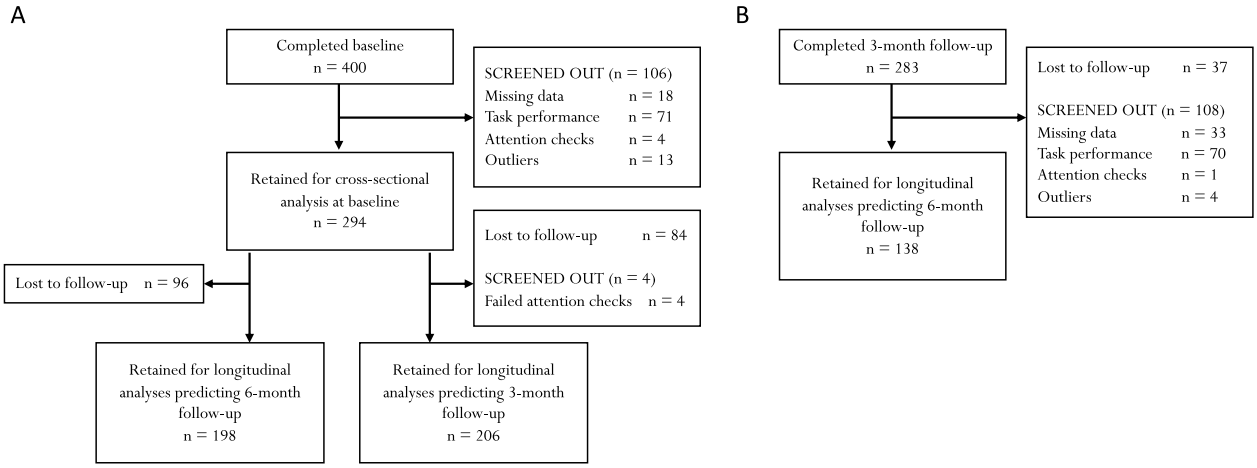

Fig. 1. Flow diagrams mapping data collection, cleaning, and reasons for exclusion. *Note*: A is the flow diagram relevant for analyses involving the baseline sample i.e. cross-sectional model, baseline predicting 3- and 6-month longitudinal models. B is the flow diagram relevant for the longitudinal model involving predictors assessed at 3-month follow-up predicting outcomes at 6-month follow-up

predicted PUI. The 3-month longitudinal models showed less reward-related attentional capture ($\beta = -0.14$, $p = 0.003$) and less risk-taking under uncertainty ($\beta = -0.11$, $p = 0.029$) significantly predicted higher PUI at 3-month follow-up, but neurocognition at 3-moth follow-up did not predict PUI at 6-months. These results held after correcting for serial correlation error (Table S8). Regressions using multiple imputation replicated these findings (Table S12), although the baseline risk-taking effect reduced to trend level (95% CI [−0.09, 0.00], $p = 0.062$).

DISCUSSION

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

Cross-sectional correlates of addictive behaviors

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 behaviors: 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, Ko, Sun, & Yeh, 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 risk use.

Longitudinal predictors of addictive behaviors

Only a minority of neurocognitive functions assessed in this study predicted addictive behaviors longitudinally, and effect sizes were generally of small magnitude. Further, neurocognition poorly predicted addictive behaviors over a 6-month time period. With the exception of delay discounting and PPU, none of the other addictive behaviors measured at 6-month follow-up were predicted by baseline neurocognition. However, when evaluating a shorter timeframe (i.e. 3-months) we found neurocognitive functions can predict addictive behaviors. This suggests that the temporal delay between the assessment of neurocognition and behavioral outcome may impact the likelihood of finding a relationship.

Poorer performance monitoring at baseline predicted greater AE at 3-month follow-up. While 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. The present study is the first to identify that

reward-related attentional capture can explain (albeit with a small effect) significant variance in future AE behavior. The VMAC task evaluates how cues indicating high-value rewards detrimentally 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 task goals. Individuals prone to developing more severe AE are perhaps more susceptible to learning of 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 behaviors can in certain contexts 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, Bovens, & Wiers, 2021). We also found less risky decision-making in the face of uncertainty (BART) was shown to predict more PUI at 3-month follow-up. While 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 uncertainty 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 behaviors, suggesting PUI could be more characteristic of compulsive disorders. A further consideration is that PUI has been described as a quasi-trait in which meaningful variance of symptom severity occurs only at the more severe end of the spectrum (Tiego et al., 2019). Consequently, samples that contain individuals with both non-problem and problem use (such as that presented herein) may impact the psychometric stability of the IAT-10 (Tiego et al., 2019).

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 et al. (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). Further, it is pertinent to note that individuals who failed to return for 6-month follow-up had significantly higher PPU scores at baseline and were steeper discounters than those participants retained in analyses. Our models were likely biased by participant attrition which may explain the counterintuitive discounting findings. As such, the relationship between PPU and neurocognition is still unclear, 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 findings from a systematic review by the authors (Christensen, Albertella, et al., 2023) that showed longitudinal studies conducted in community settings have failed to consistently demonstrate neurocognition predicting future problem use (Goudriaan, Grekin, & Sher, 2011; Jones et al., 2021; Whelan et al., 2014). Instead, neurocognition is seemingly more relevant at riskier 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), whereas general population samples without any risk-indictors (i.e. family history, current regular use etc.) may never go on to have problems. Finally, neurocognition may be more relevant when individuals are attempting to change or control their drinking behavior (Albertella et al., 2021). In the current study, participants were not required to change their drinking. Without the motivation to change, some have argued that it is unlikely an individual's alcohol use would be predicted by cognitive disposition (Albertella et al., 2021).

A strength of this study is our broad, longitudinal evaluation of neurocognitive functions. Each neurocognitive domain was selected based on theoretical frameworks and expert-endorsement, attesting to their relevance in addiction. This approach allowed for both a comprehensive and targeted evaluation of neurocognitive functions and their role in predicting addictive behaviors. Another strength is that we investigated multiple addictive behavior types in the same sample. Given addictive behaviors often co-occur (Christensen, Albertella, et al., 2023; Ford & Håkansson, 2020), investigating influence of neurocognitive predictors on various addictive behaviors within the same group, helps us understand whether there are common factors that predict addictive behaviors across different types or if these predictors are specific to each behavior.

Limitations and future directions

While our choice to focus on a community sample contributes to the dimensional evaluation of addictive behavior, it is important to recognise that the selected sample exhibited relatively low levels of engagement in addictive behaviors, specifically in the areas of AE, alcohol use, and PPU. This limitation may have impeded our ability to

identify more 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 behavior 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.

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, was underpowered to detect small effects due to participant drop-out across the course of the study. 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, statistically mitigating the significance of this concern to a degree. Further, the effect size differences comparing individuals who were retained versus lost to follow-up were only small.

It is also important to acknowledge that our inability to identify neurocognitive predictors may have been a result of single task-based measurement error, which is inherent in investigations that opt for individual paradigms to measure what are considered to be latent mechanisms (Enkavi et al., 2019; Hedge et al., 2018). 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 concurrence among measures (Goschke, 2014; Verdejo-Garcia & Albein-Urios, 2021) may provide a more robust and comprehensive way to measure and understand neurocognitive processes. Finally, once-off assessments of neurocognitive functions at each time-point 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, Sumida, & Cook, 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 behaviors across multiple behavior types, which is in itself a significant finding from a theoretical standpoint. However, we showed that reward-related neurocognitive processes were implicated across each non-substance addictive behavior, but in different ways. Our findings suggest that there may be partially distinct neurocognitive mechanisms contributing to addiction, depending on the specific addictive behavior under consideration. However, more work should be done to interrogate these differences, particularly focusing on reward-related functions. Our findings would also benefit from being replicated in studies that specifically recruit individuals with more severe levels of addictive behavior. This could lead to a better 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 clinical and public health strategies for specific addictive behaviors.

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SUPPLEMENTARY DATA

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