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A Family Study of Executive Function in Gambling Disorder

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Abstract

Impulsivity-characterized executive function impairments have been hypothesized to represent mechanisms underlying the symptomology associated with gambling disorder (GD). Despite this, a clear profile of executive function within GD has yet to be established. Furthermore, it remains unclear whether executive function deficits represent a vulnerability marker for the disorder. This study assessed executive function performance within a GD sample compared to a sample of familial relatives and community controls. Using a family study methodology, a broad assessment of executive function was administered to analyze performance differences and their potential characterization by impulsivity between a sample of individuals meeting criteria for GD, their first-degree familial relatives, and a community control sample. Performance differences emerged regarding the capacity to delay gratification and inhibit automatic task-irrelevant responses between the GD and control samples. Results support the presence of impulsive choice and impulsive cognitive bias as components of the GD executive functioning profile. Similar difficulties inhibiting automatic attentional shifting were observed within the first-degree relative sample. Executive functioning within GD appears to be characterized by an impulsive pattern of behaviours/decisions but impacts processes differently. Evidence suggests that individuals diagnosed with GD demonstrate a statistically different capacity to delay gratification (e.g. a propensity towards smaller, more immediate rewards as opposed to larger delayed rewards) and inhibit cognitive biases (e.g. difficulty shifting attention away from task-irrelevant stimuli). This latter difference may represent a vulnerability marker of GD as preliminary evidence was provided for similar difficulties in a first-degree relative sample. Further research must replicate these findings and assess the impact of task modality, symptom severity, and comorbidity on the observation of executive functioning impairment.

Keywords Gambling disorder · Executive function · Family study · Decision-making · Response inhibition · Vulnerability markers

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Gambling disorder (GD) is characterized by persistent and habitual patterns of problematic behaviours despite their negative consequences leading to clinically significant impairment (American Psychiatric Association, 2013). The presence and severity of gambling symptoms have been shown to predict functional outcomes for the individual (Petry & Armentano, 1999) and their familial relatives (Kourgiantakis et al., 2013). Research has demonstrated that problematic gambling behaviours are associated with familial conflict (Shaw et al., 2007), as well as legal (May-Chahal et al., 2017), psychiatric (Lorains et al., 2011), and financial difficulties (Grant et al., 2010). To improve our capacity to treat GD, there is a need for further research aimed at understanding both its maintenance and etiological factors.

Broadly, deficits in executive function, a hierarchical category of high-order cognitive processes (e.g. working memory, response inhibition, mental flexibility), may underlie and maintain addictive disorders, including GD (Noël et al., 2013; Pallanti et al., 2021). Such models of GD are supported by evidence of impairments related to cognitive flexibility (Ellis et al., 2018; Leppink et al., 2016), planning (Ellis et al., 2018; Kräplin et al., 2014), response inhibition (Brevers et al., 2012a, 2012b; Mestre-Bach et al., 2020), working memory (Brevers et al., 2012a, 2012b; Leiserson & Pihl, 2007), and decision-making (Ciccarelli et al., 2017; Fauth-Bühler et al., 2017; Perandrés-Gómez et al., 2021). While such findings have not consistently been replicated (Albein-Urios et al., 2012; Boog et al., 2014; Brevers et al., 2012a, 2012b; Hur et al., 2012; Ledgerwood et al., 2012, 2012; Manning et al., 2013; Sharif-Razi et al., 2019; Yan et al., 2014), it is widely accepted that disrupted executive function reflects a core component of the GD presentation.

More recently, executive functioning impairment within GD has been recognized to reflect a pattern of impulsivity (Mestre-Bach et al., 2020; Tiego et al., 2018; Yücel et al., 2019). Impulsivity refers to risky, hastily initiated, and inappropriate behaviours frequently leading to adverse outcomes (Evenden, 1999). Importantly, impulsivity is a complex multifactorial construct. Models of impulsivity have identified several separable domains, including but not limited to impulsive cognitive bias (i.e. difficulty suppressing inappropriate attentional bias), impulsive choice (i.e. propensity towards smaller, more immediate rewards as opposed to larger delayed rewards), impulsive behaviour (i.e. difficulty inhibiting inappropriate motor responses), and impulsive decision-making (i.e. tendency to make risky choices, specifically within situations of ambiguity) (MacKillop et al., 2016; Tiego et al., 2018, 2019). Applying this model, a recent meta-analysis provided evidence of deficits within GD across all domains of impulsivity, suggesting generalized impulsivity characterizes the cognitive profile of GD (Ioannidis et al., 2019). Despite such findings, results remain inconsistent, and further research using novel and broad neuropsychological measures is necessary to clarify the manifestation of impulsive behaviours/traits within GD samples.

Of note, previous research has highlighted the presence of impulsivity in the form of personality traits, cognitive control issues, and decision-making deficits in individuals with GD and their familial relatives. This suggests that impulsivity may represent a vulnerability marker of the disorder (Black et al., 2015a, 2015b). In further support of this finding, results from the Ioannidis et al. (2019) meta-analysis suggest that evidence of impulsive decision-making is present within samples characterized by problem gambling (i.e. individuals engaging in disordered gambling behaviour that does not meet GD diagnostic criteria). Despite this, it remains unclear whether broad executive functioning impairment characterized by impulsivity represents a vulnerability marker of GD or a product of chronic gambling behaviour. In light of this, there is clinical utility in the identification of methodology which supports the assessment of vulnerability markers for developing GD. One

such method is comparisons of individuals with GD and unaffected first-degree familial relatives (i.e. parents, siblings, or children) (Ersche et al., 2010; Gottesman & Gould, 2003; Robbins et al., 2012). Research using a twin study methodology has suggested genetic factors account for approximately 50% of the risk of developing GD (Ibanez et al., 2003; Lobo, 2016; Slutske, 2000). Family studies, therefore, offer a unique opportunity to assess the manifestation of possible vulnerability markers in a sample that shares genetic and environmental factors (Hodgins et al., 2011). Despite this, research to date assessing first-degree relatives of individuals with GD has been limited, with even fewer studies assessing possible neurocognitive mechanisms. Additional research characterizing cognitive performance in GD samples and samples of their familial relatives could provide evidence supporting the identification of vulnerability markers and underlying mechanisms.

Current Study

This study characterizes the manifestation of a broad range of executive functioning processes within a sample of GD, their first-degree biological relatives, and a community control sample. The objective is to offer further evidence characterizing GD's executive functioning impairment as impulsive and providing characterizations of the cognitive profile of first-degree biological relatives of individuals with GD. To facilitate this, this study administered tasks validated in their assessment of domains of executive functioning, which have been previously identified as measures of impulsive attentional bias (i.e. the Colour-Word Interference Task; Hierarchy Paper), impulsive choice (i.e. the Delayed Discounting Task), impulsive decision-making (i.e. the Balloon-Analogue Risk Task, the Tower of London Task), impulsive behaviours (i.e. Stop-Signal Anticipation Task (Bonini et al., 2018, p. 201; Ioannidis et al., 2019; MacKillop et al., 2016; Tiego et al., 2018), and working memory (i.e. the Spatial Working Memory Task).

Based on the previously discussed literature, several hypotheses were proposed. We hypothesized that the GD sample would demonstrate broad executive functioning impairments reflective of an impulsive response pattern. Specifically, we anticipated that the GD sample would demonstrate a reduced capacity for response inhibition and delayed gratification compared to the control sample. Results reflecting decision-making, attention, and working memory have been more inconsistent within the literature, yet impulsive performance patterns have been observed. Therefore, we hypothesized that performances on measures of these domains would demonstrate a pattern of impulsivity within the GD sample compared to the control sample. Finally, given previous research, which has suggested that impulsivity may reflect a vulnerability marker of GD, we hypothesized that performances observed within the first-degree relative sample would demonstrate a similar pattern of impulsivity to that observed within the GD sample.

Methods

Participants

The study sample consisted of 40 participants meeting lifetime criteria for GD (DSM-5; American Psychiatric Association, 2013), 19 of their first-degree biological relatives, and 50 community controls (see Table 1). Using a convenience sampling methodology, the

Table 1 Demographic characteristics for gambling disorder, control, and relative samples

	Gambling Disorder (GD) (n = 40)	*Relatives (n = 19)*	*Controls (n = 50)*	*F, χ2 or Fisher's Exact (df)*	*p*	*Contrasts*
Age ($\bar{x}$, SD)	44.00 (15.32)	45.26 (16.45)	41.76 (13.83)	.476, (2, 106)	$p = .623$	N.S.
Education ($\bar{x}$, SD; years)	14.10 (1.85)	*14.39 (1.85)*	*15.82 (1.67)*	*11.29, (2, 103)*	$p < .001$	GD/R < C**
Female (%)	30.00%	63.20%	62.00%	10.59, (2)	$p = .005$	GD < R/C****
Handedness (% right)	80.00%	78.9%	93.9%	8.72, (4)	$p = .068$	N.S.
Marital status (%)				15.24, (2)	$p = .362$	N.S.
Single, never married	46.2%	31.6%	36.1%			
Common-law	7.7%	5.3%	4.6%			
Married, never divorced	12.8%	47.4%	30.6%			
Divorced, not remarried	20.5%	10.5%	17.6%			
Divorced, remarried	2.6%	5.3%	4.6%			
Separated	2.6%	0.0%	2.8%			
Widowed	5.1%	0.0%	2.8%			
# of Times Married ($\bar{x}$)	0.77	0.79	0.83	0.069, (2)	$p = .933$	N.S.
Employment status (%)				12.99, (12)	$p = .373$	N.S.
Full time (40 h + /week)	37.5%	57.9%	53.1%			
Halt-time (20 h/week)	22.5%	0.0%	12.2%			
Quarter-time (10/week)	2.5%	5.3%	4.1%			
Unemployed	12.5%	0.0%	10.2%			
Retired	12.5%	21.1%	8.2%			
Student	10.0%	15.8%	12.2%			
Disabled	2.5%	0.0%	0.0%			
Annual income (%)				14.72, (10)	$p = .143$	N.S.
Under $10,000	2.6%	0.0%	0.0%			
$10,000–$20,000	5.3%	0.0%	6.0%			
$20,000–$30,000	2.6%	11.1%	6.0%			
$30,000–$50,000	23.7%	27.8%	8.0%			
$50,000–$95,000	39.5%	11.1%	36.0%			

Table 1 (continued)

	Gambling Disorder (GD) (n = 40)	*Relatives (n = 19)*	*Controls (n = 50)*	*F, χ2 or Fisher's Exact (df)*	*p*	*Contrasts*
$95,000 & <	26.3%	50.0%	44.0%			
HAMD ($\bar{x}$, SD)	3.70 (5.15)	1.63 (2.27)	1.30 (2.05)	5.43, (2, 106)	$p = .006$	GD < R*/C**
YMRS ($\bar{x}$, SD)	1.63 (1.78)	1.63 (2.27)	1.04 (1.41)	1.57, (2, 106)	$p = .212$	N.S.
WTAR ($\bar{x}$, SD)	103.00 (11.35)	103.84 (13.01)	107.16 (10.73)	1.61, (2, 106)	$p = .203$	N.S.
PGSI ($\bar{x}$, SD)	9.50(6.03)	1.16 (2.24)	0.20 (0.73)	71.75, (2, 106)	$p < .001$	GD < R/C***
SOFAS ($\bar{x}$, SD)	66.00 (12.06)	79.32 (9.13)	80.00 (8.08)	22.42, (2, 106)	$p < .001$	GD < R/C***

Notes. HAMD: Hamilton Depression Rating Scale (index of depression symptoms in the last week); YMRS: Young Mania Rating Scale (index of mania symptoms in the last week); WTAR: Wechsler Test of Adult Reading PGSI: Problem Gambling Severity Index; SOFAS: Social & Occupational Functional Assessment Scale. N.S: Non-significant; * $p < .05$, ** $p < .005$, *** $p < .001$, **** $p <$ Bonferroni Adjusted Alpha

GD and the control samples were recruited using community announcements (e.g. online ads, posters), advertisements at local treatment facilities, and an existing research registry. Control participants were excluded if they met lifetime criteria for GD or had a family history of GD. Relatives were contacted using information provided by the GD sample and included parents ($n=7$), children ($n=5$), and siblings ($n=7$). The following exclusion criteria were applied to all participants and subgroups: (1) age less than 18 years; (2) IQ less than 80; (3) diagnosis with a neurological condition (e.g. multiple sclerosis, epilepsy, stroke, AIDS, traumatic brain injury); or (4) diagnosis with a psychotic disorder that could confound measurement of cognitive function. Participants were also excluded if they reported any condition(s), medical or otherwise, that would make participation in the study difficult or confound the study's analysis.

Procedure

All procedures were approved by the University of Calgary Conjoint Facilities Research Ethics Board and aligned with the 1964 Helsinki Declaration (World Medical Association, 2013). All participants were screened during a phone interview. Diagnosis was verified in person using the Structured Clinical Interview for the DSM-5 (First et al., 2016). The presence of mood, psychotic, substance use, anxiety, eating, adult attention-deficit hyperactivity, obsessive–compulsive, and trauma/stress-related disorders were assessed. Diagnosis of GD was further verified using the Composite International Diagnostic Interview (CIDI; Kessler et al., 2008). All diagnoses were verified using case discussions attended by at least one of the principal investigators (MASKED). Before any study procedures were completed, informed written consent was obtained. The measures analyzed were administered over 2 days as part of a larger test battery. Gift cards were provided as a reimbursement.

Clinical Measures

Clinical constructs were assessed using validated clinical measures to assist in characterizing the participants. Gambling symptomology and severity were indexed using the Problem Gambling Severity Index (PGSI). This is a nine-item self-report questionnaire ($\alpha=0.943$) demonstrating satisfactory psychometrics (Currie et al., 2013; Holtgraves, 2009). The PGSI queries potential problems resulting from gambling, coded from 0 ("never") to 4 ("almost always"), with responses summed for a total score. Recent psychological symptomology was similarly assessed. Depressive mood experiences and symptoms were assessed using the widely used and psychometrically acceptable (Aben et al., 2002) 17-item version ($\alpha=0.848$) of the Hamilton Depression Rating Scale (HAMD; Hamilton, 1960). Manic symptomology was assessed using the valid and reliable Young Mania Rating Scale (YMRS; Young et al., 1978). The YMRS is an 11-item measure ($\alpha=0.462$, Inter-Item Correlation $=0.074$) which assesses manic experiences over the past 48 h. Premorbid intelligence was estimated using the validated (Green et al., 2008) Wechsler Test of Adult Reading measure (WTAR; Wechsler, 2001). Based on case discussion, psychosocial and occupational functioning was measured using the validated and reliable Social and Occupational Functioning Assessment Scale (SOFAS; Morosini et al., 2000).

Cognitive Tasks

Color-Word Interference Test (CWIT) The CWIT is a valid and reliable (Shunk et al., 2006) paper and pencil-based assessment of inhibitory control from the Delis-Kaplan Executive Function System (Delis et al., 2004). This measure has moderate reliability, high internal consistency, and satisfactory validity (Delis et al., 2004). It consists of four distinct conditions (i.e. colour naming, word reading, inhibition, and inhibition/switching). Performances within each condition are scaled based on normative age-appropriate data. This assessment allows for examining performance on a factor-by-factor basis through contrasting performance within each of the four conditions, comparing speed scores, and scoring accuracy.

Tower of London Task (ToLT) The ToLT is an executive function assessment with demonstrated construct validity (Debelak et al., 2016), criterion validity, and reliability (Köstering et al., 2015) which provides a measurement of planning, decision-making, and problem-solving (Shallice, 1982). Participants are asked to recreate a presented tower with a similar tower consisting of coloured rings. Two rules are applied while completing this task (1: The number of rings staked cannot exceed the bar's capacity; 2: Only one ring can be moved at a time). Participants complete this task over ten trials.

Spatial Working Memory Task (SWMT) The SWMT is a computer-based assessment with demonstrated reliability and validity previously used to assess visuospatial working memory (Almeida et al., 2015; Glahn et al., 2002). Participants are exposed to two conditions, maintenance and manipulation. During the maintenance condition, participants must remember the spatial location of a set of three circles presented for 1500 ms. After a 6000 ms delay, the second set of three circles is presented. Participants are asked to verify whether the new set of circles is presented in the exact spatial location and orientation as the initial set. Participants are presented with similar stimuli for the manipulation condition and asked if they represent a mirror flip of the first set of circles.

Stop-Signal Anticipation Task (SSAT) The SSAT is a widely used (Clark et al., 2020) measure of response inhibition and, more specifically, the processes of reactive and proactive inhibition (Zandbelt & Vink, 2010). Three horizontal lines are displayed in this task, one above the other. On every trial, a bar is presented, which rises towards the middle-presented line. The time between the line beginning to move and reaching the middle line is set at a constant duration of 800 ms. Trials are presented within three blocks, which consisted of stop trials pseudo-randomly interspersed between go trials. In go trials, the participant is tasked with stopping the bar as close as possible to the middle line by pressing the spacebar. On stop trials, representing the minority of trials, the bar would stop automatically before reaching the second line. The sudden stopping of the line represents a stop signal and signals to the participant that a button depress response should be inhibited. The likelihood of a stop signal being presented is manipulated across each trial. It is cued based upon the colour of the lines presented (i.e. green: 0%, yellow: 17%, amber: 20%, orange: 25%, and red: 33%). The presentation of the stop signal is adjusted throughout the task based on the participants responding accuracy. The task's difficulty is increased and decreased as necessary, manipulated by stop-signal presentation, so that accuracy was maintained at 50%.

Balloon Analogue Risk Task (BART) Openness to risky behaviours was measured using the demonstrated reliable and valid (Lejuez et al., 2007; Weafer et al., 2013; White et al., 2008) computerized BART (Lejuez et al., 2002). With this measure, participants inflate a balloon, earning a single point for each pump. If the balloon pops, the participant loses all the collected points. This procedure is repeated over 30 trials. Unfortunately, participants are not provided with an opportunity to test the propensity for popping (i.e. evaluate risk) as there are no practice trials in this measure.

Delayed Discounting Task (DDT) The capacity to delay gratification was assessed using the DDT, a valid and reliable computer-based assessment of the subjective value of a delayed reward (Bailey et al., 2021; Reynolds & Schiffbauer, 2004; Weafer et al., 2013). The DDT comprises 100 questions, which query whether the participant would prefer a reward now or a larger award later. Questions are presented using an adjusted-amount procedure (Richards et al., 1999). The amount of immediate reward is adjusted across successive trials until an amount is reached that the participant judges to be equivalent to the delayed reward. This amount represents the participant's subjective valuation of the delayed reward. This point of subjective equality is referred to as the indifference point. Indifference points were assessed at seven different delays: 1 week, 2 weeks, 1 month, 6 months, 1 year, 5 years, and 25 years. After each question, the amount of the immediate reward is adjusted. If the smaller-sooner reward was selected, the amount of that reward is decreased by $25 in the subsequent choice trial. Alternatively, if the larger-latter reward was selected, the amount of the sooner reward was increased by $25. Subsequent adjustments to the immediate reward were 50% of the preceding adjustment. The amount of immediate reward following the tenth-choice trial was used as the indifference point for that delay. At each subsequent delay, the amounts of the smaller-immediate reward and the larger-delayed reward were returned to $50 and $100, respectively, and the titration procedure was repeated.

Statistical Analysis

All tests were performed using SPSS for Windows, version 26.0 (IBM, 2019) and were two-tailed. Outliers, normality, and test assumptions for each analysis were assessed and corrected if necessary. Demographic information for the three groups was compared using univariate ANOVA, Chi-Square test, and Fisher's exact test when appropriate analyses of variance (ANOVA) models were conducted for each administered tasks to observe for statistical between-group performance differences. Effect sizes, p values, and 95% confidence intervals are reported and interpreted based on accepted guidelines. To test the proposed hypotheses, planned comparisons were conducted regardless of the presence of a significant main effect. Preliminary analyses included family ID as a nested variable to assess for variance related to familial relationships. Familial relationships failed to demonstrate a significant interaction with the group condition in any of the analyses conducted and, therefore, was excluded from the analysis to preserve power.

Cognitive Task Analysis

Separate multivariate ANOVAs (MANOVA) were conducted to test for performance differences on the CWIT. Performance on each primary condition (DV: 4 levels: colour naming, word reading, inhibition, and inhibition/switching) was compared using a

MANOVA with group (IV: 3 levels; GD, relatives, controls) as the between-subjects factor. Contrast differences were assessed using a MANOVA with inhibition vs. colour naming, inhibition/switching vs. colour reading + word reading, inhibition/switching vs. inhibition, inhibition/switching vs. colour naming, and inhibition/switching vs. word reading as the dependent variables and group (3 levels: GD, relative, controls) as the independent variable. Finally, the frequency of errors was compared between the groups using a MANOVA with group (3 levels: GD, relative, control) as the independent variable and colour naming errors, word reading errors, inhibition errors, and inhibition/switching errors as the dependent variables.

A univariate ANOVA (group: 3 levels: GD, relative, control) was used to assess differences in relation to overall achievement scores (DV) on the ToLT. An additional MANOVA was conducted to further query between-group performance differences with group as the between-subject factor and total rule violations percentile, scaled mean time until the first move, scaled time-per-move ratio, move accuracy ratio, and rule violations per item ratio as the dependent variables.

To assess for between-group differences regarding accuracy (IV: total score) and reaction time (IV: mean reaction time) on the SWMT, separate ANOVAs were conducted. Both ANOVAs followed a 2 (group: GD, relative, control) × 2 (condition: maintenance, manipulation) mixed model design.

Performance on the SSAT was assessed for differences in the respective processes of reactive and proactive inhibition. Reactive inhibition was assessed using average stop-signal reaction time (SSRT), calculated using the integration method (Verbruggen & Logan, 2008). This method facilitates the calculation of stop-signal reaction time by subtracting the *n*th reaction time from the mean stop-signal delay. The *n*th reaction time is identified by multiplying the number of trials by the probability of the participant responding on the stop-signal trial. Outliers for the analysis were identified as reaction times that exceeded 1.5 × the inter-quartile range away from the 25th and 75th percentiles of the reaction time distribution. The remaining reaction times were averaged per task condition (4 stop-signal probability levels) and compared between groups. A mixed model ANOVA was used to assess for differences regarding proactive inhibition with group (3 levels: GD, relatives, controls) as the between-subject variable and stop-signal probability (4 levels: 17%, 20%, 25%, 30%) as the within-subject variable.

Performance on the BART was assessed using a MANOVA, with group (3 levels: GD, relative, controls) as the independent variable and each of the outcome scores of interests (3 levels: adjusted total score, total number of pumps, average reaction time) as the dependent variables. Cutoff was applied to the reaction time data to reduce the confounding impact of poor effort. Per group cutoffs were specified as average reaction times greater than the mean + 2 SDs.

Differences regarding performance on the DDT were assessed using model fit estimates and a follow-up univariate ANOVA. Median indifference points, reflecting the point where the amount of the smaller-sooner outcome is regarded as equal to the present value of the larger-later outcome, for each group were fit to the Mazur hyperbola (Mazur, 1987) and the Myerson and Green (Myerson & Green, 1995) hyperboloid models. Indifference points were calculated in alignment with the process outlined above in the measures section. Akaike information criterion (AIC), a weighted index of variance accounted for by the model as a factor of its number of parameters, was utilized to select the most appropriate analysis model. Based upon the model of best fit, between-group comparisons were carried out using a one-way (group; 3 levels; GD, relative, control) ANOVA using the average area under the curve (AUC), the sum of the trapezoidal area between each set of adjacent

indifference points for each group as the dependent variable. Since it was demonstrated that the Myerson and Green hyperboloid ((Myerson & Green, 1995) provided the best fit for the data, the free-parameter k, indicating a measure of the degree of discounting, was not used. In the Myerson and Green model, k interacts with the value of s, an exponential scaling parameter, and therefore is not an independent index of delayed discounting. Finally, non-systematic delayed discounting data were excluded from the analysis based on the criteria and algorithm developed by Johnson and Bickel (2008).

Results

Demographics

The samples were shown to be not statistically different regarding age, handedness, marital status, level of employment, annual income, mania symptom ratings (i.e. YMRS), and estimated intelligence (i.e. word reading; WTAR). However, samples differed on gender, the number of years of education, gambling symptoms, depression symptom ratings, and psychosocial/occupational functioning. See Table 1 for a list of statistics and significance values. Comorbid mental health disorders for each sample are displayed in Table 2.

Neuropsychological Measures

The results for each impulsivity domain assessed are outlined below. See Table 3 for means, standard deviations, and contrast statistics.

Inhibitory Control A non-significant main group effect was observed regarding performance on the primary four conditions of the CWIT (Pillai's Trace = 0.11, $F(8, 96) = 1.34$, $p = 0.227$, $n_p^2 = 0.056$). Planned comparisons in alignment with the study's goals were carried out. Non-significant differences were observed between the samples regarding colour naming, word reading, and inhibition/switching performance. In contrast, a significant effect was observed regarding inhibition performance ($F(2, 93) = 4.28$, $p = 0.017$,

Table 2 Comorbid mental health disorders for GD, control, and relative samples

	GD (n = 40)	*Relatives (n = 19)*	*Controls (n = 50)*
Bipolar disorder, % total (*n*)	5%, (2)	0%, (0)	0%, (0)
Depressive disorder, % total (*n*)	47.5% (19)	*21.1%, (4)*	*28%. (14)*
Psychotic disorder, % total (*n*)	0%, (0)	0%, (0)	0%, (0)
Substance use disorder, % total (*n*)	55%, (22)	21.1%, (4)	22%, (11)
Anxiety disorder, % total (*n*)	20% (8)	5.3%, (1)	8%, (4)
Obsessive–compulsive disorder, % total (*n*)	2.5% (1)	0%, (0)	0%, (0)
Eating disorder, % total (*n*)	7.5% (3)	0%, (0)	0%, (0)
Trauma-related disorder, % total (*n*)	5% (2)	10.5%, (2)	0%, (0)
ADHD, % total (*n*)	5% (2)	0%, (0)	0%, (0)
Other disorder, % total (*n*)	7.5% (3)	5.3%, (1)	0%, (0)

Notes. *ADHD*, attention deficit hyperactivity disorder

Table 3 Means (standard deviations) of primary outcome measures for executive function neuropsychological assessment organized by study group

	GD	Relatives	Controls	Contrasts
CWIT ($\bar{x}$, SD)	($n=38$)	($n=17$)	($n=41$)	
Condition 1. Colour naming	9.39 (2.63)	9.47 (1.33)	10.05 (2.17)	N.S.
Condition 2. Reading score	10.13 (2.42)	9.82 (1.94)	10.88 (1.57)	N.S.
Condition 3. Inhibition	10.50 (2.42)	9.76 (2.14)	11.56 (2.25)	GD*/R** > C
Condition 4. Inhibition/ switching	10.63 (2.42)	10.35 (1.90)	11.07 (2.39)	N.S.
Inhibit vs. colour naming	11.00 (2.63)	10.60 (0.19)	11.68 (1.93)	N.S.
Inhib/Swit vs. Cond 1 + 2	10.63 (2.79)	10.67 (1.59)	10.78 (1.73)	N.S.
Inhib/Swit vs. inhibition	10.26 (1.64)	10.60 (2.17)	9.77 (1.83)	N.S.
Inhib/Swit vs. colour naming	11.26 (3.10)	11.20 (1.74)	11.45 (2.01)	N.S.
Inhib/Swit vs. word reading	10.42 (2.86)	10.60 (2.10)	10.53 (1.68)	N.S.
Colour naming scaled errors	77. 32 (37.95)	74.83 (41.94)	95.62 (18.57)	N.S.
Reading scaled errors	78.16 (39.05)	89.50 (30.60)	95.19 (20.04)	N.S.
Inhibition scaled errors	10.08 (2.05)	10.39 (2.09)	10.89 (1.37)	N.S.
Inhib/Swit scaled errors	10.78 (1.65)	10.67 (1.53)	10.89 (1.51)	N.S.
ToLT ($\bar{x}$, SD)	($n=37$)	($n=17$)	($n=47$)	
Total achievement	10.73 (1.64)	11.18 (3.37)	10.45 (2.29)	N.S.
Total rule violations (%Rank)	63.58 (35.50)	90.47 (21.23)	74.16 (34.81)	R > GD **
Mean 1st move time (scaled)	9.17 (3.68)	8.35 (3.12)	8.53 (3.69)	N.S.
Time-per-move-ratio (scaled)	9.83 (2.75)	8.71 (2.87)	10.11 (1.96)	N.S.
Move accuracy (ratio)	9.22 (3.05)	9.76 (2.77)	8.69 (2.63)	N.S.
Rule violation per item (ratio)	10.11 (1.70)	10.71 (0.47)	10.04 (2.15)	N.S.
SWMT ($\bar{x}$, SD)	($n=36$)	($n=15$)	($n=47$)	
Maintenance score	17.83 (1.40)	16.87 (1.80)	17.76 (1.59)	N.S.
Maint reaction time (s)	31.84 (11.97)	30.74 (6.12)	31.16 (14.28)	N.S.
Manipulation score	15.77 (2.33)	15.80 (1.82)	16.15 (2.23)	N.S.
Manip reaction time (s)	42.89 (14.97)	37.41 (5.91)	37.84 (16.62)	N.S.
SSAT ($\bar{x}$, SD)	($n=24$)	($n=12$)	($n=30$)	
SSP 17%	820.86 (28.36)	823.07 (31.63)	818 (23.00)	N.S.
SSP 20%	821.52 (27.52)	827.45 (34.55)	825.15 (24.47)	N.S.
SSP 25%	821.07 (28.56)	827.08 (31.19)	827.43 (23.58)	N.S.
SSP 33%	825.75 (28.29)	826.84 (37.22)	828.44 (23.69)	N.S.
SSP Overall	820.71 (25.88)	823.71 (31.91)	822.42 (20.53)	N.S.
SSRT	259.75 (28.72)	242.54 (9.23)	246.59 (29.80)	N.S.
BART ($\bar{x}$, SD)	($n=36$)	($n=16$)	($n=43$)	
Total adjusted score	5738.33 (1896.69)	5962.35 (2225.67)	5764.77 (2156.36)	N.S.
Total number of pumps	803.88 (342.80)	737.29 (314.77)	750.54 (369.21)	N.S.
Average reaction time (ms)	309.58 (133.20)	348.22 (164.80)	261.48 (85.44)	R > C*

Notes. *CWIT*, Colour-Word Interference Task; *ToLT*, Tower of London Task; *Spatial Working Memory Task*, Maintenance & Manipulation Task; *SSAT*, Stop-Signal Anticipation Test; *SSP*, Stop-Signal Probability; *SSRT*, Stop-Signal Reaction Time; *BART*, Balloon Analogue Risk Task, * $p < .05$, ** $p < .005$

$n_p^2 = 0.084$). The relative (Mean $\Delta = 1.80$, $p = 0.008$, *95% CI* [0.48, 3.11]) and the GD samples (Mean $\Delta = 1.06$, $p = 0.043$, *95% CI* [0.03, 2.09]) both demonstrated cognitive inhibitory performances significantly below that demonstrated by the control sample. Performance analysis regarding the primary contrasts returned a similar non-significant main effect of group (Pillai's Trace = 0.053, $F(8, 176) = 0.60$, $p = 0.779$, $n_p^2 = 0.026$). Non-significant between-group differences were supported based on planned comparisons. Similar non-significant differences were observed from the MANOVA assessing between-group differences regarding error rates on each condition (Pillai's Trace = 0.072, $F(8, 172) = 0.799$, $p = 0.604$, $n_p^2 = 0.036$) and the conducted planned comparisons.

Decision-Making/Planning Total achievement scores derived from the TolT were shown to be statistically similar between the samples ($F(2, 98) = 0.642$, $p = 0.528$, $n_p^2 = 0.013$). The main effect of group derived from a MANOVA analyzing the optional performance measures was non-significant (Pillai's Trace = 0.156, $F(10, 188) = 11.59$, $p = 0.113$, $n_p^2 = 0.078$). Planned comparisons generally provided further evidence of similar performances between the study samples regarding mean first move time, mean time-per-move ratio, move accuracy ratio, and rule-violations per item ratio. Total rule violations, represented as a scaled percentile rank, reflected a significant between-group difference ($F(2, 81) = 4.70$, $p = 0.012$, $n_p^2 = 0.104$) reflective of the relative sample performing significantly worse than the GD sample (Mean $\Delta = -31.097$, $p = 0.004$, *95% CI* [−51.84, −10.36]).

Working Memory The repeated measures ANOVA analyzing working memory performance returned an expected significant effect of condition ($F(1, 95) = 28.28$, $p < 0.001$, $n_p^2 = 0.229$). In contrast, the between-group effect ($F(2, 95) = 1.13$, $p = 0.327$, $n_p^2 = 0.023$) and interaction ($F(2, 95) = 0.793$, $p = 0.455$, $n_p^2 = 0.016$) were non-significant. A similar pattern of effects was observed with the repeated measures ANOVA assessing reaction time with an expected significant effect of condition ($F(1, 95) = 54.01$, $p < 0.001$, $n_p^2 = 0.046$), a non-significant between-group effect ($F(2, 95) = 0.61$, $p = 0.544$, $n_p^2 = 0.013$), and non-significant interaction ($F(2, 95) = 2.29$, $p = 0.107$, $n_p^2 = 0.362$). All planned comparisons between the study samples regarding differences in working memory performance and reaction times on the separate conditions were non-significant.

Reactive and Proactive Inhibition Despite slower average performance within our proband sample (see Table 3) compared to both the relative and control sample, measured by SSRT and reflective of poorer reactive inhibition, the overall effect of group failed to reach significance ($F(2, 52) = 1.93$, $p = 0.153$, $n_p^2 = 0.074$). Planned comparisons between the study samples corroborated this performance pattern with no significant differences. Regarding proactive inhibition, a significant main effect of trial probability was demonstrated (Wilks Lambda = 0.81, $F(3, 57) = 4.35$, $p = 0.008$, $n_p^2 = 0.19$), while neither the between-group comparison ($F(1, 59) = 0.093$, $p = 0.911$, $n_p^2 = 0.003$) nor the interaction (Wilks Lambda = 0.87, $F(6, 114) = 1.39$, $p = 0.226$, $n_p^2 = 0.068$) reached significance. Planned comparisons were carried out, which supported the lack of significant between-group differences.

Risky Behaviour Propensity The main effect of group was observed to be significant (Pillai's Trace = 0.140, $F(6, 186) = 0.2.33$, $p = 0.034$ $n_p^2 = 0.070$). Univariate comparisons revealed non-significant differences regarding adjusted total score (i.e. risky behaviour propensity) ($F(2, 97) = 0.07$, $p = 0.936$, $n_p^2 = 0.001$) and total number pumps ($F(2, 94) = 0.31$,

$p = 0.736$, $n_p^2 = 0.007$), but indicated a significant between-group difference associated with average reaction time ($F(2, 94) = 3.61$, $p = 0.031$, $n_p^2 = 0.071$). Follow-up planned comparisons revealed significantly slower reaction times within the relative sample compared to the control sample (Mean $\Delta = 86.73$, $p = 0.014$, *95% CI* [18.29, 155.18]). No other significant differences were observed.

Delayed Discounting Model fit analysis results, utilizing proportional median indifference points derived from the DDT, are presented in Table 4 below. Figure 1 below displays median indifference points, expressed as a proportion of the delayed reward amount, organized by study group. Model parameters (e.g. k, s, R^2) for each model are displayed in Table 4. This analysis returned a significant main effect of group ($F(2, 60) = 3.22$, $p = 0.047$, $n_p^2 = 0.097$). Planned comparisons revealed significant differences between the GD and control sample, with the GD sample having a significantly steeper discounting curve than the control sample (Mean $\Delta = -0.17$, $p = 0.015$, *95% CI* [−0.30, −0.04]).

Discussion

The first objective of this study was to provide further evidence characterizing GD's executive functioning profile. The results offer mixed support for the hypotheses posed related to this aim. In alignment with the hypotheses, performances observed with our GD sample reflecting cognitive control and capacity to delay gratification were impulsive compared to those observed with the community control sample. In contrast, motor response inhibition, planning, visuospatial working memory, and propensity for risky behaviours were statistically similar between the GD and control samples. This overall pattern of results suggests that while patterns of impulsivity can characterize the executive functioning profile of GD, these impairments impact specific domains of executive functioning and are not broadly observed.

In comparison to the control sample, the GD sample was characterized by a reduced capacity to delay gratification. This performance pattern has been observed in previous studies revealing similarly elevated tendencies of choice impulsivity in GD samples compared to controls (Albein-Urios et al., 2014; Amlung et al., 2017; Dixon et al., 2003; Ioannidis et al., 2019; Petry, 2001). It has been hypothesized that GD is associated with a hypoactive reward system, biasing reward representation and valuation and, as a result, biasing motivations leading to dysfunctional behaviours (Brevers et al., 2012a, 2012b; Lorains et al., 2014; Madden & Bickel, 2010). In other words, individuals diagnosed with GD appear to pay greater attention to potential gains as opposed to potential losses during tasks tapping decision-making processes. This attentional or motivational bias has been proposed to explain this consistently observed performance pattern, reflecting impulsive choice, associated with delay discounting tasks (Ioannidis et al., 2019; O'Connor et al., 2014).

Response inhibition impairments are a relatively well-established observation associated with GD samples (Brevers & Noël, 2013; Chowdhury et al., 2017; Goudriaan et al., 2005, 2006; Smith et al., 2014). However, this effect has not consistently been replicated, with some researchers suggesting that motor response inhibition is not a central expression of GD impulsivity (Brevers et al., 2012a, 2012b; Leppink et al., 2016; Sharif-Razi et al., 2019). In support of this latter conclusion, and inconsistent with our hypotheses, this study

Table 4 Delayed discounting task outcome variables organized by group

	GD ($n=22$)		Relatives ($n=10$)		Controls ($n=31$)	
Model fit comparisons for the Mazur (1987) *hyperbola and* Myerson and Green (1995) *hyperboloid*						
	Mazur Hyperbola	Myerson & Green Hyperboloid	Mazur Hyperbola	Myerson & Green Hyperboloid	Mazur Hyperbola	Myerson & Green Hyperboloid
AIC	−24.43	**−29.48**	**−25.94**	−20.65	−20.55	**−22.13**
Median R^2	.725	**.926**	.802	**.953**	.715	**.927**
Measure parameters (k, s, R^2) for fit to median indifference points						
R^2	.830	.970	.918	.916	.715	.936
k	.201	1.15	.119	1.12	.125	.290
s		.375		.315		.575
Indifference points ($\bar{x}$, SD, 95% CI)						
$\bar{x}$	0.396		0.477		0.479	
SD	0.257		0.303		0.315	
95% CI	0.276, 0.515		0.300, 0.653		0.372, 0.586	
AUC ($\bar{x}$, SD, 95% CI)						
$\bar{x}$	0.114		0.181		0.281	
SD	0.132		0.190		0.301	
95% CI	0.012, 0.216		0.031, 0.333		0.195, 0.366	

Notes. Bolded statistics represent models with superior fit. Median R^2 values were calculated by fitting each model to each participant's data, obtaining respective R^2 values, and calculating the median

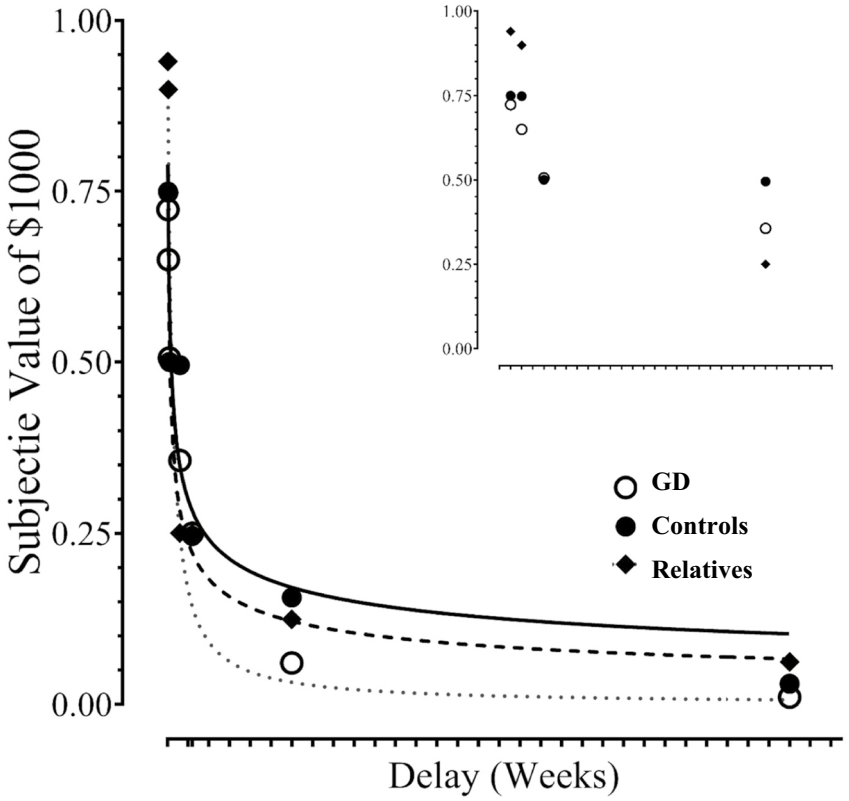

Fig. 1 Discounting models are organized by study group. Points on the figure represent median indifference points with lines indicating the best fitting discounting function (Myerson & Green, 1995). The inset figure represents the same data. The *X*-axis has been scaled to better represent the median indifference points at the shorter delays (i.e. 1 week, 2 weeks, 1 month, and 6 months)

failed to replicate previous findings suggesting individuals with GD demonstrate impaired proactive or reactive response inhibition.

In contrast, compared to the community controls, impairments were observed in the ability to inhibit highly automatic behaviours such as reading, specifically the capacity to inhibit the more salient and automatic behaviour of reading a word instead of naming the dissonant ink colours. Models of impulsivity suggest that this performance pattern reflects an impulsive cognitive bias, in which individuals are less able to inhibit attentional shifting towards task-irrelevant stimuli or highly automatic behaviours. Notably, the GD and control samples were shown to have comparable error rates across all conditions, testing various inhibitory processes on the CWIT. This suggests that impairment reflects the speed at which the inhibitory process is elicited, not explicitly a lack of capacity to inhibit overlearned responses which would manifest as elevated error rates. Therefore, the pattern of results observed here suggests that while individuals with GD can inhibit and shift their attention away from task-irrelevant responses, the speed at which this process is elicited, specifically for overlearned and highly automatic behaviours, is slower than that observed within the non-GD population.

Finally, this study failed to provide evidence supporting the existence of impulsive planning, working memory, or a propensity for risk-taking behaviours within the analyzed GD sample. This suggests that these domains of cognition are preserved within the GD cognitive profile. These results align with literature which has supported the normal functioning of verbal and spatial working memory (Albein-Urios et al., 2012; Brevers et al., 2012a, 2012b; Ledgerwood et al., 2012; Manning et al., 2013; Yan et al., 2014) and planning (Manning et al., 2013) within GD samples. On the other hand, these results contradict other efforts at characterizing executive functioning within the GD population (Kertzman et al., 2017; Ledgerwood et al., 2012; Mallorquí-Bagué et al., 2018; Zhou et al., 2016), including the recent meta-analysis that supported broad impulsivity-characterized executive functioning impairment within GD (Ioannidis et al., 2019). Our failure to detect between-group differences regarding the noted cognitive domains may reflect cognitive profile heterogeneity within the GD population. Alternatively, cognitive task variance regarding psychometrics and specificity may impact the observation of performance differences and explain our lack of between-group findings. Replicating such findings will be necessary to clarify.

The second aim of this study was to assess whether executive functioning deficits observed within the GD sample were similarly observed with the biological relative sample, providing evidence of potential vulnerability markers of GD. The impairment that reflected impulsive attentional bias was the only domain in which similar performance patterns were observed in the relative and GD samples. This offers preliminary evidence supporting the specific inhibitory processes underlying attentional biases as a possible vulnerability marker in non-diagnosed first-degree relatives of individuals with GD. Additionally, while not significant, the overall pattern of performance on the DDT, reflecting choice impulsivity, suggested individuals with GD perform significantly more impulsively compared to controls, with the relative sample demonstrating an intermediary performance. Studies to date assessing cognitive performance patterns within familial relatives of individuals with GD have been mixed but have generally suggested that impulsivity and impaired reward-based decision-making represent candidate markers of GD vulnerability (Black et al., 2014, 2015a, 2015b; Limbrick-Oldfield et al., 2020). Although the results of this study regarding the relative sample should be reviewed cautiously, given the small sample size, they do support impulsive cognitive biases as a potential vulnerability marker in need of further assessment and replication.

Several explanations for the pattern of results observed are worth discussing. First, the contradiction between the results observed from the CWIT and the SSAT suggests that inhibitory processes may be impaired in both individuals with GD and their first-degree biological relatives but that this impairment may not be generalized and may be moderated by task-specific factors. For example, inter-task demand variation between inhibition paradigms may impact the specific inhibition process being tapped, resulting in variation in the observed results. The CWIT largely indexes verbal inhibition, while the SSAT reflects a motor inhibitory process, potentially explaining the difference in observed results. Alternatively, the subjective evaluation of the inhibited response may interact with performance, biased by underlying motivational dysfunctions and symptom severity. This explanation has been supported by evidence suggesting motor impulsivity is more consistently disrupted in severe cases of GD (Chowdhury et al., 2017; Michalczuk et al., 2011). Additionally, other studies have demonstrated that behavioural impulsivity, in the form of an increased propensity for risky behaviours, may be moderated by previous experiences. Specifically, a study by Bonini et al. (2018) reported no differences between a control, pathological, and problematic gambling sample regarding risk-taking behaviours measured by the BART. Of note, some differences did emerge within the problematic gambling group

following a predetermined series of losses, interpreted as reduced sensitivity to negative feedback and a loss-chasing tendency. These results highlight the importance of considering task-specific characteristics in observing impulsivity-characterized executive function impairment.

It has also been suggested that GD is best characterized by distinct subtypes, differentiated by course of illness (e.g. age, age of onset), symptomology (e.g. severity, comorbidity), and personality (e.g. harm avoidance, self-directedness, impulsive personality traits), which are predictive of neurocognitive functioning (Heiskanen & Toikka, 2016; Jiménez-Murcia et al., 2017; Mallorquí-Bagué et al., 2018; Moon et al., 2017, p. 201; Nower et al., 2022; Suomi et al., 2014). For example, one GD cluster has been proposed, which presents more significant executive functioning impairment in addition to higher rates of unemployment, later age of GD onset, greater endorsed negative/positive urgency, and broadly elevated comorbid psychological symptoms (Mallorquí-Bagué et al., 2018). Considering this, the results reviewed here should be interpreted with caution as, due to our modest sample size, we were unable to analyze within sample subsets. Greater than half (55%) of our GD sample met the criteria for lifetime substance-use disorder, while only 2 (5%) met the criteria for adult attention deficit hyperactivity disorder (ADHD). This is relevant as previous research has supported the importance of comorbid substance use disorder in attenuating impulsive behaviours, potentially due to the neurotoxic effects of alcohol and drugs (Chowdhury et al., 2017; Lawrence et al., 2009; Potenza, 2006). On the other hand, ADHD is consistently associated with poorer inhibition performance and greater impulsivity (Brunault et al., 2020; Lijffijt et al., 2005; Senderecka et al., 2012; Verbruggen & Logan, 2008). This highlights the need for additional studies analyzing the potential clustering of individuals with GD based on salient factors such as symptom severity and comorbidity, which may elucidate the mixed results to date.

Finally, a hypothesis has been proposed which suggests that cognitive deficits may be driven by an underlying motivational mechanism. In a study by Boog et al. (2014), mental flexibility performance was compared between a GD and a control sample. Uniquely, this study compared the capacity for mental flexibility when the rules were arbitrary versus rules that were previously reinforced with rewards. Between-group differences were non-significant when the rules were arbitrary but were significant when the rules were reward-based. This result was interpreted as evidence of a motivational impairment rather than a more generalized mental flexibility impairment. It has been hypothesized that these reward-motivation biases may explain inconsistencies within the GD neurocognitive literature (Stevens et al., 2015). While the impact of a motivational bias was not directly assessed within this study, it may offer a further explanation for the discrepancies observed and warrants further investigation.

Limitations

Several limitations of this study are acknowledged. The GD sample was recruited based on the presence or absence of GD. The presence of other psychological disorders, though assessed, did not act as an exclusion criterion. While this increases the generalizability of these observed results, it also introduces the confounding influence of comorbidity. Additionally, while the sample sizes of the GD and control samples were adequate, the relative sample was modest due to recruitment difficulties. Effect sizes are reported to reduce the impact of this issue. Future studies may wish to assess variance in neuropsychological impairment manifestation as a factor of symptom variation (i.e. remission vs.

worsening). With limitations noted, several strengths are also worth mentioning. Including a first-degree biological relative sample allowed for assessing executive functioning vulnerability markers of GD. Additionally, while differing on some characteristics (see Table 1), our samples were largely well-matched regarding demographics, except for fewer female participants within the gambling sample. Finally, this study applied a comprehensive neuropsychological assessment tapping many executive functioning domains critical to the cognitive profile of GD. This allowed for a thorough assessment of the manifestation of cognitive impairments.

Conclusions

The results of this study provide evidence of impulsive choice (i.e. a reduced capacity to delay gratification) and impulsive cognitive biases (i.e. reduced capacity for inhibiting overlearned verbal behaviours) within a sample of GD. These results contribute to the growing literature characterizing the cognitive profile of GD as impulsive while highlighting areas of interest for future studies. Specifically, the importance of the unique contributions of disorder onset, task demands, and comorbidity was reviewed and emphasized as essential areas for future research. Additionally, while preliminary and in need of replication, this study provided evidence of the characterization of impulsive cognitive biases as a potential familial vulnerability marker for GD development, as similar performance patterns were observed within the analyzed first-degree relative sample. Further research is needed to directly explore the manifestation of impulsivity as a function of task modality while considering the possible utility of symptom, cognition, and demographic-based cluster analyses.

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Declarations

Ethics Approval and Consent to Participate All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its amendments or comparable ethical standards.

Conflict of Interest The authors declare no competing interests**.**

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