The role of executive function deficits, delay aversion and emotion dysregulation in internet gaming disorder and social media disorder: Links to psychosocial outcomes

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ABSTRACT

Background and aims: It has been argued that it is important to consider underlying mechanisms of mental health problems. Previous studies have shown that executive deficits, delay aversion, and emotion dysregulation are related to Internet Gaming Disorder (IGD) and Social Media Disorder (SMD). However, the present study is the first to investigate whether these neuropsychological deficits show additive effects or if they interact. The present study also investigated whether these deficits mediate the association between IGD/SMD and psychosocial outcomes. *Methods:* The study involved 995 university students who completed a survey measuring IGD/SMD symptom severity, neuropsychological functions, and psychosocial outcomes. Both dimensional and categorical analyses were used to assess the associations between neuropsychological functions and IGD/SMD. Simple and multiple mediation analyses were conducted to examine if neuropsychological functioning mediates the association between IGD/SMD and psychosocial outcomes. *Results:* All neuropsychological functions were significantly associated with both IGD and SMD symptom severity. However, only inhibition and emotion regulation, as well as delay aversion for SMD, remained significant when controlling for the overlap between different functions. Associations were significantly stronger for men compared to women for IGD. In the categorical analyses, individuals with IGD/SMD were more likely to have neuropsychological deficits (odds ratios between 3.33 and 8.81). Finally, all neuropsychological functions, except inhibition, were significant mediators in the link between IGD/SMD and psychosocial outcomes. *Discussion and conclusions:* These results shed light on the neuropsychological underpinnings of IGD/SMD, which can be used to identify more homogenous subgroups and provide more individualized treatment options.

KEYWORDS

internet gaming disorder, social media disorder, neuropsychological functions, psychosocial problems, mental health

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INTRODUCTION

In the latest version of the Diagnostical and Statistical Manual of Mental disorders (DSM-5; American Psychiatric Association [APA], 2013), Internet Gaming Disorder (IGD) was introduced as a disorder in need of further research. For problematic use of social media, there is no officially recognized disorder within the DSM-5. However, similarly to previous

research, we chose to use the term Social Media Disorder (SMD) within the present study, and it is operationalized using the same symptom criteria as presented in DSM-5 (APA, 2013) to describe IGD (Bouna-Pyrrou et al., 2018; Burén, Nutley, Sandberg, Ström Wiman, & Thorell, 2021; Moretta, Buodo, Demetrovics, & Potenza, 2022; van den Eijnden, Lemmens, & Valkenburg, 2016). The terms "digital media" and "IGD/SMD symptom severity" are used as overarching constructs, which includes both gaming and social media. Although a relatively large number of previous studies have examined gaming and social media, the mechanisms underlying IGD and SMD are still poorly understood. This is important as it provides a better understanding of the factors that trigger and maintain an addiction, which in turn will aid in identifying targets for treatment. As explained further below, previous studies have linked both IGD and SMD to executive function deficits, delay aversion, and emotion dysregulation. However, as these neuropsychological functions have not been investigated within the same study, we do not know if they have additive or interaction effects in relation to IGD and SMD. Furthermore, we currently do not know if they can explain the association between IGD/SMD symptom severity and psychosocial outcomes. The overall aim of the present study was therefore to examine the role of neuropsychological deficits in IGD and SMD, and to what extent these deficits mediate the association between IGD/SMD symptom severity and three different types of psychosocial outcomes: psychosomatic problems, self-concept, and social problems.

According to the DSM-5 (APA, 2013), there are nine proposed symptom criteria for IGD and it has been suggested (Burén, Nutley, Crisci, & Thorell, 2022; Wichstrøm, Stenseng, Belsky, von Soest, & Hygen, 2019) that they can be divided into symptoms related to excessive usage (e.g., "gaming has become the dominant activity in daily life") and negative consequences (e.g., "jeopardizing relationships or career/educational opportunities due to gaming"). At least five of nine symptoms need to be endorsed to meet the criteria for IGD. It has been argued that because there are many similarities between IGD and SMD, the same symptom criteria used to assess IGD could be adopted for SMD by replacing the word "gaming" with "social media use" (e.g., Bouna-Pyrrou et al., 2018; Burén et al., 2021; Moretta et al., 2022; van den Eijnden et al., 2016).

From a theoretical perspective, it has been emphasized that behavioral addictions should be seen as heterogeneous conditions related to multiple underlying mechanisms, including cognitive, emotional, as well as motivational factors (e.g., Brand et al., 2019; Dong & Potenza, 2014). This is also supported by previous empirical studies, which have linked IGD and SMD (i.e., operationalized as IGD/SMD diagnosis or SMD/IGD symptom severity) to neuropsychological deficits. The neuropsychological functions that have been most strongly linked to IGD/SMD are executive functioning such as working memory (Ioannidis et al., 2019) and inhibition (Argyriou, Davison, & Lee, 2017), as well as to emotion regulation (Kuss, Pontes, & Griffiths, 2018; Yang, Wang, Elhai, & Montag, 2022). A relatively recent meta-analysis

(Cheng, Ko, Sun, & Yeh, 2021) also showed that motivational factors such as delay aversion (i.e., the tendency to prefer smaller sooner rewards over larger later rewards) were more common among individuals with internet addiction compared to a comparison group. Thus, although the number of empirical studies is relatively limited, there appears to be both theoretical and empirical support for a link between problematic use of digital media and executive functioning deficits, emotion dysregulation, and delay aversion. The present study therefore chose to focus on these three neuropsychological deficits, and whether they have additive or interaction effects in relation to IGD and SMD symptom severity. This should be considered an important addition to previous research as it is well-known from previous empirical studies that neuropsychological functions are interrelated (e.g., Castellanos & Tannock, 2002), and they most likely interact in a complex way in relation to addictive behaviors (Brand et al., 2019; Dong & Potenza, 2014). In addition, IGD and SMD have seldom been examined within the same study, which limits our understanding of possible differences and similarities with regard to the neuropsychological underpinnings of these two digital media addictions (Wegmann & Brand, 2020).

Another limitation of previous research is that most studies have only investigated group differences between individuals with either IGD or SMD and a comparison group. Although this information is valuable, it has been argued that it is important to identify subtypes based on the underlying mechanism of mental health problems (e.g., Lee, Lee, & Choo, 2017). Investigations of mean group differences should therefore be complemented with person-oriented analyses (i.e., investigating the proportion of individuals with clear neuropsychological deficits based on a predefined cut-off).

When investigating associations between problematic digital media and psychosocial outcomes, a distinction can be made between "direct effects" (i.e., effects that are a result of the content of the digital media) and "indirect/displacement effects" (effects that result from the fact that digital media crowds out health promoting activities such as sleep and exercise). Thus, problematic use of digital media can have negative effects with regard to a range of different outcomes. Previous studies have for example found that both gaming (e.g., Cheng, Cheung, & Wang, 2018; Männikkö, Ruotsalainen, Miettunen, Pontes, & Kääriäinen, 2020; Müller et al., 2015; Paulus, Ohmann, von Gontard, & Popow, 2018; Teng, Pontes, Nie, Griffiths, & Guo, 2021) and social media use (Boer et al., 2022; Pontes, Taylor, & Stavropoulos, 2018; Stiglic, Masterson Creber, & Cilar Budler, 2022; van den Eijnden, Koning, Doornwaard, van Gurp, & Ter Bogt, 2018) are related to negative outcomes such as depression, anxiety, low self-esteem, psychosomatic symptoms, and poor social relations.

Previous research has also shown that the negative outcomes mentioned above are associated with neuropsychological deficits, with several reviews linking executive function deficits, emotion dysregulation, and delay aversion to a range of social and mental health problems (e.g., Amlung et al., 2019; Baune, Fuhr, Air, & Hering, 2014; Lopes, Salovey, Coté,

& Beers, 2005; Sheppes, Suri, & Gross, 2015; Weyandt et al., 2014). It can therefore be hypothesized that neuropsychological deficits mediate the association between IGD/SMD symptom severity and negative psychosocial outcomes. However, to our knowledge, this has not been examined in previous studies. Understanding the links between IGD/SMD symptom severity, neuropsychological deficits and negative psychosocial outcomes would potentially provide better insights into the underlying mechanisms of problematic use of digital media, which could in turn lead to prevention of problems with earlier and better risk management and treatment.

Aims of the present study

To address the limitations of the previous research described above, the overall aim of the present study was to investigate problematic use of gaming and social media in university students and to what extent this is related to neuropsychological functions and psychosocial outcomes using both dimensional and categorical analyses. More specifically, we examined the following three research questions:

1. Can different neuropsychological functions (i.e., inhibition, working memory, emotion dysregulation, and delay aversion) be described as having additive or interaction effects on IGD and SMD symptom severity?
2. Do individuals meeting the symptom criteria for IGD/ SMD, or those being in an at-risk group for IGD/SMD, more often have neuropsychological deficits than a comparison group?
3. Do neuropsychological functions mediate the association between IGD/SMD symptom severity and psychosocial outcomes (i.e., psychosomatic problems, self-concept, and social problems)?

METHODS

Participants and procedure

The present study included data from 995 (68.7% females) university students from Italy and Sweden. The mean age was 25.00 years ($SD = 6.31$). The participants were recruited via social media, flyers, face-to-face interaction on university campuses, and through contact with university professors. The survey was available both via pen-and-paper and online. Responses were anonymous. Background data is provided in Table 1.

Measures

Digital media addiction. Problematic digital media use was measured by the Gaming and Social Media Questionnaire (GSMQ-9), an instrument that was created and validated in a previous study using the same sample as in the present study (Burén et al., 2022). The GSMQ-9 includes nine items, one for each symptom criteria of IGD (APA, 2013). For each

Table 1. Demographics data for the sample

	Full sample
Number of participants	995
Age (years, [SD])	25.00 (6.31)
Sex (% women)	72.8
Country of origin (%)	
Sweden	68.5
Italy	31.1
Area of study (%)	
Medicine	16.2
Natural/Technical sciences	13.9
Social sciences	20.0
Psychology	30.3
Education	10.2
Economy	4.6
Law	4.9

item, two separate scores were provided, one for gaming and one for social media, using a five-point Likert scale ranging from 0 ("Doesn't correspond at all") to 4 ("Corresponds very well"). In the dimensional analyses, we used the mean of the nine items to assess IGD and SMD symptom severity. In the categorical analyses, participants were considered to meet a specific symptom criterion if their score was ≥ 3 and they were then classified into the IGD/SMD groups (≥ 5 symptoms), "at-risk" groups (3 or 4 symptoms), and comparison groups (≤ 2 symptoms). This instrument has good psychometric properties, including excellent test-retest reliability (ICC > 0.90) in a subsample of adults ($n = 115$) tested 1–2 weeks apart, and good internal consistency ($\alpha \geq 0.83$; $\omega = \geq 0.83$).

Executive function. The Teenage Executive Functioning Inventory (TEXI; Thorell, Lazarević, Milovanović, & Bugarski Ignjatović, 2020) was used to investigate working memory and inhibition. For this study, we used an abbreviated version consisting of eight items of which four assessed working memory (e.g., "When someone asks me to do several things, I sometimes cannot remember all of them") and four assessed inhibitory control (e.g., "sometimes cannot stop myself from laughing or smiling even though I know that it is inappropriate at that time"). Items were rated on a scale ranging from 1 ("definitely not true") to 5 ("definitely true"). We selected the items that best represented the different aspects captured by each scale. Using data from a previous study (Thorell, Lazarević, et al., 2020), the selected items were shown to correlate very highly with the original scales with regard to both working memory ($r = 0.96$) and inhibition ($r = 0.91$). In addition, confirmatory factor analysis was used to show that the two-factor structure (i.e., working memory or inhibition), was appropriate also for the abbreviated version used in the present study (CFI = 0.963; RMSEA = 0.064; SRMR = 0.032). We used the mean score for working memory and inhibition, with higher values indicating more severe deficits. The test-retest reliability has been shown to be adequate in a previous study (Thorell, Lazarević, et al., 2020), as well as within the present study (ICC = 0.71 for working memory and ICC = 0.61 for inhibition).

Emotion regulation. The "Negative Impact" subscale (5 items, e.g. "I have problems with my studies/work because I cannot control my negative feelings") from the Comprehensive Emotion Regulation Inventory (CERI; Thorell, Tilling, & Sjöwall, 2020) was used to investigate emotion regulation. We selected this subscale because we were not primarily interested in investigating different strategies for regulating emotions but rather to what extent the participants had problems with emotion regulation in a way that impacted on their daily life functioning. Ratings were made on a scale ranging from 1 ("definitely not true") to 5 ("definitely true") and we used the mean score, with higher values indicating more severe problems with emotion regulation. The test-retest reliability and the internal consistency have been shown to be adequate in previous research (Thorell, Tilling, & Sjöwall, 2020) and both the internal consistency ($\alpha = 0.81$; $\omega = 0.82$) and the test-retest reliability (ICC $= 0.79$) was adequate also within the present study.

Delay aversion. We measured delay-related behaviors using an abbreviated 4-item version of the Quick Delay Questionnaire (QDQ; Clare, Helps, & Sonuga-Barke, 2010). The four selected items captured both "delay aversion" and "delay discounting" and they were shown to be very highly correlated ($r = 0.93$) with the full 10-item scale using data from a previous study (Sjöwall & Thorell, 2022). Ratings were made on a five-point Likert scale ranging from 1 ("definitely not true") to 5 ("definitely true"), and we used the mean score, with higher values indicating more severe delay aversion. The test-retest reliability has been shown to be adequate in a previous study (Thorell, Sjöwall, Mies, & Scheres, 2017), as well as within the present study (ICC $= 0.55$).

Psychosocial problems. We included three aspects of psychosocial functioning: self-concept, social problems, and psychosomatic problems. First, we used the subscale "Self-concept" (six items, e.g., "I feel frustrated with myself") and the subscale "Social problems" (five items, e.g., "I have problems keeping friends") from the self-report version of the Weiss Functional Impairment Rating Scale (WFIRS-S; Weiss, 2000). Ratings were made on a scale ranging from 1 ("definitely not true") to 5 ("definitely true") and we used the mean score of the items, with higher values indicating more severe psychosocial problem. The internal consistency was good for both self-concept ($\alpha = 0.91$; $\omega = 0.91$), and social problems ($\alpha = 0.87$; $\omega = 0.88$). A previous review has also showed good psychometric properties, including high test-retest reliability, for the WFIRS-S (Canu, Hartung, Stevens, & Lefler, 2020; Weiss, McBride, Craig, & Jensen, 2018).

Psychosomatic problems were measured using nine items. Five items (i.e., feeling irritated/bad mood, feeling nervous/anxious, feeling down, having headaches, and having stomach aches) were taken from the HSBC symptom checklist used by WHO in well-being surveys. We also included two items measuring sleep ("difficulty falling asleep" and "daytime tiredness"), one item measuring loneliness ("feeling lonely"), and one item measuring stress ("feeling stressed"). Each item was measured on a six-point

scale (0 = never, 1 = sometimes, 2 = once a week, 3 = twice a week, 4 = at least three times a week, 5 = every day) and we used the mean score as a measure of psychosomatic problems. The internal consistency for the nine items was good ($\alpha = 0.89$; $\omega = 0.87$), and the test-retest reliability was excellent (ICC $= 0.94$).

Statistical analyses

In the dimensional analyses, we first used Pearson correlation analyses to investigate associations between symptoms of IGD/SMD and neuropsychological functions for men and women separately. Due to the relatively large sample size of the present study, we interpreted coefficients of at least medium effects sizes (i.e., $r \geq 0.30$) as being meaningful rather than focusing on statistical significance (i.e., $p < 0.05$). Second, hierarchical linear regression analyses were used to explore additive and interaction effects. The value for the Variance Inflation Factor (VIF) was < 1.75 for all four neuropsychological functions, which means that multicollinearity was not a problem in these analyses. In the regression analyses, sex was entered in the first step and the four neuropsychological functions in the second step. In the third step, we entered interaction effects, with each interaction being entered separately. Both interactions between the four neuropsychological functions and interactions between sex and neuropsychological functioning were analyzed. All p-values for the interaction effects were adjusted with the Holm-Bonferroni method (Holm, 1979). In the categorical analyses, we used chi-square tests and odds ratios to investigate if the IGD or SMD group (i.e., ≥ 5 symptoms) or the at-risk groups (i.e., 3 or 4 symptoms) had neuropsychological deficits more often than the comparison group (≤ 2 symptoms). In line with several previous studies (e.g., Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Sjöwall, Roth, Lindqvist, & Thorell, 2013), the cut-off for having a neuropsychological deficit was set to scoring above the 90th percentile. As complementary analyses, we also used Analyses of Variances (ANOVAs) to compare mean group differences for the four neuropsychological deficits. Finally, we tested for simple and multiple mediation using Preacher and Hayes (2008) method. We used 5,000 bootstrap resamples and the percentage of the total effect explained by each mediator was calculated as a measure of effect size.

Ethics

The study procedures were carried out in accordance with the Declaration of Helsinki. The national ethical review authority approved the study. All subjects were informed about the study, and all provided informed consent.

RESULTS

Associations between IGD/SMD and neuropsychological functions

All four neuropsychological functions were positively and significantly associated with both IGD and SMD symptom

severity (see Table 2). However, effect sizes were small (i.e., $r < 0.30$) for associations between all neuropsychological functions and IGD symptom severity for females, as well as for associations between delay aversion and both IGD and SMD symptom severity for males. In the regression analyses (Table 3), the four neuropsychological functions explained 9% of the variance in IGD, with inhibition and emotion regulation contributing independently. For SMD, the neuropsychological functions explained 22% of the variance, with inhibition, delay aversion and emotion regulation contributing independently. Significant interaction effects between sex and each one of the four neuropsychological functions were found for IGD symptom severity ($\beta s = 0.17$ to 0.20, $p < 0.001$), indicating that the neuropsychological functions were more strongly associated with IGD symptom severity for men compared to women (Table 2). The neuropsychological functions explained 17% of the variance in IGD for men and 6% for women. No significant interaction effects for sex were found for SMD (all $\beta s < -0.03$ to 0.05, $p > 0.29$). None of the interaction effects between the four neuropsychological functions were significant for either IGD or SMD.

In the categorical analyses (see Table 4), we found that the participants in the IGD and SMD groups were significantly more likely to have neuropsychological deficits than the comparison group, with odds ratios ranging from 3.33 (delay aversion for SMD) to 8.81 (working memory for IGD). Similar results were found when using ANOVAs to compare mean group differences (see supplementary material). In total, 57.1% of the participants who met the criteria for IGD had multiple neuropsychological deficits, 21.4% had a single deficit, and 21.4% had no neuropsychological deficits at all. The corresponding numbers for SMD were 50% with multiple deficits, 22.5% with a single deficit, and 27.5% with no deficits.

When conducting similar analyses for the at-risk groups (see Table 4), individuals at risk of IGD were not significantly more likely to have any type of neuropsychological deficits than the comparison group. However, individuals in the at-risk group for SMD were significantly more likely than the comparison group to have deficits in inhibition and emotion regulation. They were also more likely to have single or multiple deficits, and less likely to have no deficits than the comparison group. In total, 18.2% of those in the at-risk group for IGD had multiple deficits, 36.4% had single deficits and 45.5% had no deficits. The corresponding numbers for the at-risk group for SMD were 22.5% with multiple deficits, 29.6% with single deficits, and 49.4% with no deficits.

Mediation analyses

The simple mediation analyses (Table 5) showed that all four neuropsychological functions were significant mediators for all three psychosocial outcomes. However, in the multiple mediation analyses for IGD symptom severity (see Fig. 1A), the mediating effect of inhibition was not significant for self-concept and social problems. Together, the mediators explained 54.9% of the total effect for social problems, 51.4% for self-concept and 62.8% for psychosomatic problems. For SMD symptom severity (see Fig. 1B), all mediators except inhibition were significant for all three psychosocial outcomes. Together, the mediators explained as much as 79.8% of the total effect for social problems, 69.6% for self-concept, and 46.0% for psychosomatic problems.

In all mediation analyses, emotion regulation had the strongest independent effect. Although the mediators investigated in this study explained a large proportion of the variance, the direct effect of symptoms of IGD or SMD also remained significant, except for the association between symptoms of SMD and social problems (see Fig. 1B).

Table 3. Results of the regression analyses

	Step 1 Main effects		Step 2 Main effects	
	β	ΔR^2	β	ΔR^2
IGD symptom severity		0.08		0.09
Sex	0.29***			
Working memory			0.07	
Inhibition			0.14***	
Delay aversion			0.00	
Emotion regulation			0.15***	
SMD symptom severity		0.00		0.22
Sex	0.02			
Working memory			0.06	
Inhibition			0.21***	
Delay aversion			0.08*	
Emotion regulation			0.24***	

*$p < 0.05$, ***$p < 0.001$.

Table 2. Correlations between neuropsychological functions and IGD/SMD symptom severity for men and women

	Men		Women	
Neuropsychological functions	IGD symptom severity	SMD symptom severity	IGD symptom severity	SMD symptom severity
Working memory	0.32***	0.36***	0.16***	0.31***
Inhibition	0.36***	0.40***	0.19***	0.39***
Delay aversion	0.29***	0.25***	0.09*	0.32***
Emotion regulation	0.33***	0.39***	0.20***	0.39***

*$p < 0.05$, ***$p < 0.001$.

Table 4. Results from the categorical analyses indicating differences in neuropsychological deficits between IGD/SMD groups and the at-risk groups with the comparison group

	Group			χ^{2a} ($df = 1$)		Odds ratio (95% CI)	
	IGD/SMD group (%)	At-risk Group (%)	Comparison Group (%)	IGD/SMD group	At-risk group	IGD/SMD group	At-risk group
IGD	(*n* = 14)	(*n* = 22)	(*n* = 959)				
Working memory deficits	7 (50.0)	2 (9.1)	98 (10.2)	22.79***	0.07	8.81 (3.03, 25.63)	0.83 (0.19, 3.59)
Inhibition deficits	7 (50.0)	6 (27.3)	121 (12.6)	16.26**	3.68	6.72 (2.32, 19.49)	2.48 (0.95, 6.44)
Delay aversion	5 (35.7)	3 (13.6)	89 (9.3)	10.89**	0.39	5.36 (1.76, 16.34)	1.48 (0.43, 5.08)
Emotion dysregulation	7 (50.0)	5 (22.7)	96 (10.0)	22.49***	3.28	8.71 (3.00, 25.34)	2.48 (0.90, 6.87)
Multiple deficits	8 (57.1)	4 (18.2)	99 (10.3)	31.06***	1.21	11.62 (3.95,34.15)	1.84 (0.61, 5.53)
Single deficits	3 (21.4)	8 (36.4)	161 (16.8)	0.20	5.99*	1.34 (0.37, 4.85)	2.88 (1.19, 6.98)
No deficits	3 (21.4)	10 (45.5)	711 (72.5)	18.08***	7.91**	0.10 (0.03, 0.37)	0.31 (0.13, 0.73)
SMD	(*n* = 40)	(*n* = 81)	(*n* = 874)				
Working memory deficits	18 (45.0)	13 (16.0)	76 (8.7)	50.93***	2.58	7.96 (4.41, 16.72)	1.67 (0.89, 3.13)
Inhibition deficits	18 (45.0)	22 (27.2)	94 (10.8)	35.56***	14.19**	5.92 (3.08, 11.36)	2.67 (1.57, 4.53)
Delay aversion	10 (25.0)	12 (14.8)	75 (8.6)	11.02**	2.57	3.33 (1.57, 7.03)	1.70 (0.88, 3.26)
Emotion dysregulation	16 (40.0)	17 (21.0)	75 (8.6)	36.59***	9.36**	6.25 (3.21, 12.20)	2.40 (1.35, 4.28)
Multiple deficits	20 (50.0)	17 (21.0)	72 (8.2)	65.13***	9.10**	9.73 (5.04, 18.77)	2.37 (1.33, 4.22)
Single deficits	9 (22.5)	24 (29.6)	142 (16.2)	0.89	10.00**	1.44 (0.67, 3.09)	2.23 (1.34, 3.71)
No deficits	11 (27.5)	40 (49.4)	660 (75.5)	40.28***	22.52***	0.13 (0.07, 0.27)	0.34 (0.22, 0.54)

Note: χ^2 = chi-square value; df = degrees of freedom.
[a]Fisher's exact test was used when expected cell count fell below 5 in at least one cell.
**$p < 0.01$, ***$p < 0.001$.

Table 5. Results of the simple mediation analyses

|	IGD symptom severity				SMD symptom severity			
	Est.	SE	95% CI	Indirect effect %	Est.	SE	95% CI	Indirect effect %
Psychosomatic problems								
Working memory	0.51*	0.04	0.43–0.58	36	0.40*	0.04	0.32–0.47	23
Inhibition	0.47*	0.04	0.39–0.55	41	0.34*	0.04	0.27–0.42	24
Delay aversion	0.50*	0.04	0.42–0.58	23	0.39*	0.04	0.31–0.47	20
Emotion regulation	0.63*	0.04	0.54–0.71	46	0.50*	0.04	0.42–0.58	32
Low self-concept								
Working memory	0.56*	0.04	0.48–0.63	31	0.51*	0.04	0.44–0.58	36
Inhibition	0.46*	0.04	0.38–0.53	31	0.40*	0.04	0.32–0.48	34
Delay aversion	0.50*	0.04	0.43–0.58	18	0.44*	0.04	0.36–0.51	28
Emotion regulation	0.69*	0.04	0.62–0.77	40	0.65*	0.04	0.58–0.73	51
Social problems								
Working memory	0.35*	0.03	0.29–0.41	30	0.33*	0.03	0.27–0.39	38
Inhibition	0.28*	0.03	0.22–0.34	30	0.25*	0.03	0.19–0.32	35
Delay aversion	0.31*	0.03	0.25–0.37	17	0.27*	0.03	0.21–0.33	29
Emotion regulation	0.52*	0.03	0.45–0.58	54	0.50*	0.03	0.44–0.57	65

Note: SE = standard error; CI = confidence interval.
*Significant mediator (i.e., zero is not contained within the confidence intervals).

DISCUSSION

The aim of the present study was to examine associations between neuropsychological functions and both IGD and SMD symptom severity and to what extent neuropsychological functions mediate the association between IGD/SMD symptom severity and psychosocial outcomes. The results showed that all four neuropsychological functions were significantly related to both IGD and SMD symptom severity. However, some associations were small, and it was primarily inhibition and emotion regulation that contributed to explaining the variance in IGD and SMD symptom severity when controlling for the overlap between different neuropsychological functions. Significant interactions for sex indicated that the associations between neuropsychological functions and IGD symptom severity were stronger for men
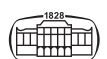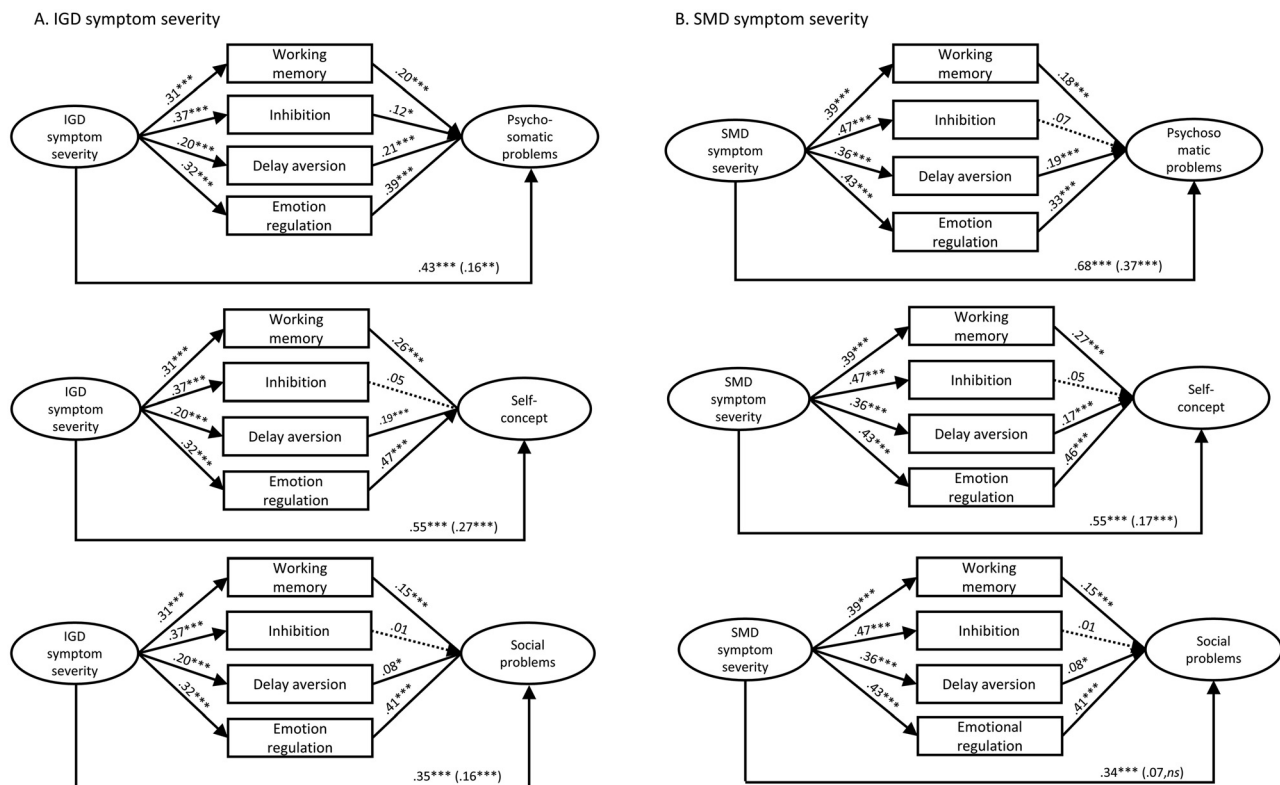
Fig. 1. Results of the multiple mediation analyses for IGD (left-hand side) and SMD (right-hand side) with the numbers representing unstandardized coefficients. The numbers within parentheses are unstandardized coefficients after taking the effect of the mediators into account (i.e., the direct effect)

than women. Together, the four neuropsychological functions explained 9% (6% for women and 17% for men) of the variance in IGD and 22% of the variance in SMD in the dimensional analyses. In the categorical analyses, participants meeting the symptom criteria for IGD/SMD were at significantly higher risk of having deficits in all four neuropsychological functions, with 50% or more having multiple deficits compared to only 8–10% in the comparison group. In the multiple mediation models, all the neuropsychological functions, except inhibition, mediated the association between IGD or SMD and psychosocial outcomes.

Associations between neuropsychological deficits and IGD/SMD symptom severity

The results of the dimensional analyses showed that both IGD and SMD symptom severity were significantly related to the four neuropsychological functions. The categorical analysis also confirmed that the IGD/SMD groups were more likely to have neuropsychological deficits. This was expected as previous studies have shown links between IGD/SMD and executive functioning (e.g., Argyriou et al., 2017; Ioannidis, et al., 2019; Kuss et al., 2018), emotion dysregulation (Kuss et al., 2018; Yang et al., 2022) and delay aversion (Cheng et al., 2021). However, due to the relatively large sample size of the present study, even negligible associations were sometimes significant. This was primarily the case for associations between IGD symptom severity and

neuropsychological deficits for females and for some associations for delay aversion for both sexes.

The present study also adds new information by investigating several different neuropsychological functions within the same study. It is interesting to note that, even though many previous studies have emphasized the importance of neuropsychological deficits for behavioral addictions, the total explained variance for all four functions was 9% for IGD and 22% for SMD. Thus, a substantial proportion of the variance in IGD/SMD could not be explained by neuropsychological functions. Other variables therefore need to be considered. This is also supported by other researchers who have argued that digital addiction results from an interaction between multiple biological, psychological, and social factors (e.g., Brand, Young, & Laier, 2014; Dong & Potenza, 2014; Wei, Zhang, Turel, Bechara, & He, 2017).

In addition to examining associations using dimensional analyses, we also used person-oriented analyses to determine how many of those meeting the full symptom criteria for IGD or SMD reported more severe deficits in neuropsychological functioning. In these analyses, odds ratios were around 8 or above for emotion dysregulation and working memory deficits for IGD and for working memory deficits for SMD. For multiple deficits, odds ratios were around 10 or above, which should be considered very high. Thus, even though only a small proportion of the variance could be explained in the regression analyses, individuals meeting the symptom criteria were much more likely than

the comparison group to have neuropsychological deficits. First, this emphasizes the need to conduct both dimensional and categorical analyses as they complement each other. Second, this could be taken to mean that the association between neuropsychological deficits and IGD/SMD symptom severity is not linear. Perhaps it is primarily more severe neuropsychological deficits (in this study defined as reporting problems above the 90th percentile) that are associated with increased risk of developing IGD and SMD.

When investigating the role of specific neuropsychological functions, the present study showed that all four neuropsychological functions were related to both IGD and SMD symptom severity for both men and women, except for delay aversion for women. However, when we jointly entering them in the regression model, it was primarily inhibition and emotion regulation that remained significant predictors for both IGD and SMD. Several reviews (Dong & Potenza, 2014; Lee, Hoppenbrouwers, & Franken, 2019; Yang et al., 2022) have emphasized that emotion regulation and inhibition should be regarded as two central components for digital media addiction. However, the interplay between these two neuropsychological deficits have not been examined in previous research. Thus, the results of the present study add valuable new information by showing that they have additive, but not interaction effects, in relation to both IGD and SMD symptom severity. As it has been previously shown that digital media is commonly used as a coping mechanism to escape negative emotions (Blasi et al., 2019), our finding linking emotion regulation to IGD and SMD is not surprising. Maladaptive emotion regulation strategies have also been shown to maintain addictive behaviors (Wartberg & Lindenberg, 2020). In line with the present study, which showed that delay aversion had a significant independent effect in relation to SMD, it has also been emphasized that the ability to prioritize long term goals is of importance for internet addiction (Cheng et al., 2021). However, it is important to emphasize that the effect of delay aversion was generally small, and the results of the present study therefore indicate that although previous research has demonstrated that delay aversion is related to addictive use of digital media (e.g., Cheng et al., 2021), this effect appears to be of less importance. Conclusively, the results of the present study show that it is primarily inhibition and emotion regulation that contribute to the explained variance in both IGD and SMD symptom severity when controlling for the overlap between different neuropsychological deficits.

We found significant interactions of sex with neuropsychological functions for IGD but not SMD. These interaction effects indicated that neuropsychological functioning was more strongly associated with IGD in men compared to women. One explanation for this could be that IGD symptoms are less common among women compared to men both in the present study and in previous studies (e.g., Stevens, Dorstyn, Delfabbro, & King, 2021; Su, Han, Yu, Wu, & Potenza, 2020) limiting the variance in this group. Restriction of range in IGD symptom severity may therefore have led to lower correlations. This explanation is supported by the fact that we did not find any interaction effects of sex and neuropsychological functioning, or sex differences, for SMD.

Neuropsychological deficits as mediators between IGD/SMD symptom severity and psychosocial outcomes

Our findings of significant associations between IGD/SMD symptom severity and negative psychosocial outcomes are consistent with previous literature with regard to both IGD (e.g., Cheng et al., 2018; Müller et al., 2015) and SMD (e.g., Andreassen et al., 2016; Wong et al., 2020). More importantly, we added critical new information by showing that neuropsychological deficits mediate the association between IGD/SMD and psychosocial outcomes. Altogether, 51%–63% of the link between IGD and psychosocial outcomes and between 46% - 80% of the link between SMD and psychosocial outcomes were explained by the mediators when using multiple mediations to control for the overlap between the mediators. These percentages should be considered very high, which suggests that the neuropsychological deficits we have included in our model play an important role in explaining the link between digital media and psychosocial problems. However, it is important to note that these percentages might be somewhat overestimated because data consisted of self-ratings only.

Strengths, limitations, and future directions

One major strength was that we investigated several neuropsychological functions within the same study. This allowed us to investigate the role of each function in relation to IGD/SMD symptom severity while controlling for the overlap between them. This also made it possible to conduct multiple mediation analyses, which has not been done in previous studies. Unlike most previous research, the present study also included both IGD and SMD within the same study. The fact that the neuropsychological underpinnings of IGD and SMS appear similar supports the notion that both are part of the same overarching construct of digital addiction (e.g., van den Eijnden et al., 2016; Wegmann & Brand, 2020). Finally, we conducted both dimensional and categorical analyses. This is in line with how IGD is described in DSM-5 (APA, 2013), which presents criteria for making categorical diagnoses but also emphasizes the need to view psychiatric symptoms as varying along a dimension.

Some limitations of this study should be acknowledged. First, all data consisted of self-ratings for both neuropsychological functions and psychosocial outcomes, which might have overestimated associations. Future studies should aim to replicate these findings by measuring neuropsychological functioning with laboratory tests. Second, our study only included university students and we therefore do not know whether our findings can be generalized to adults across a broader age range, or to clinical samples. Because we included university students only, the number of individuals meeting the full symptom criteria was relatively small, especially for IGD, which limited our statistical power in the categorical analyses. Third, we included

abbreviated versions of established scales to assess some constructs. However, these abbreviated versions were shown to be highly correlated with the original versions and they were also shown to have adequate psychometric properties.

CONCLUSIONS

It has been argued (e.g., Holmes et al., 2018) that because psychiatric disorders are often heterogeneous, it is important to include measures of underlying mechanisms rather than only focusing on overt symptom levels. This allows for a better understanding of predictors, prevention and treatment targets, as well as improved precision in matching treatments to the needs of individuals. By focusing on several different neuropsychological functions, which have all been presented as potential underlying mechanisms for IGD and SMD, the present study showed that these functions are of great importance for explaining the link between IGD/SMD and psychosocial outcomes. Even though neuropsychological deficits only explained a relatively small part of the variance in IGD/SMD symptom severity, the categorical analyses showed that those meeting the full symptom criteria were much more likely to have neuropsychological deficits than the comparison group. Conclusively, neuropsychological deficits, appear to be important for both IGD and SMD and to understand the link between IGD/SMD and psychosocial outcomes. Future studies should explore whether these neuropsychological deficits, especially inhibition and emotion dysregulation, can be important when developing prevention methods, new treatments and predicting IGD and SMD symptom severity across time.

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SUPPLEMENTARY MATERIAL

Supplementary data to this article can be found online at https://doi.org/10.1556/2006.2023.00007.

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