

# The co-occurrence between symptoms of internet gaming disorder and psychiatric disorders in childhood and adolescence: prospective relations or common causes?

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**Background:** Internet gaming disorder (IGD) is highlighted as a condition for further study in the Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5). Some studies indicate that IGD appears comorbid with other psychiatric disorders. We examine concurrent and prospective links between symptoms of IGD and symptoms of common psychiatric disorders in childhood and adolescence to determine whether observed comorbidity is a result of (a) reciprocal relations or (b) common underlying causes. **Methods:** A community sample ( $n = 702$ ) of Norwegian children completed the Internet Gaming Disorder Interview (IGDI) to assess DSM-5 defined IGD symptoms at ages 10, 12 and 14 years. The Child and Adolescent Psychiatric Assessment (CAPA) assessed symptoms of depression, anxiety, attention-deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD) and conduct disorder (CD) at the same time points. **Results:** A Random Intercept Cross-lagged Panel Model (RI-CLPM), which captures pure within-person changes and adjusts for all unmeasured time-invariant factors (e.g., genetics, parent education) revealed no associations between IGD symptoms and psychopathology, except that increased IGD symptoms at ages 10 and 12 predicted decreased symptoms of anxiety two years later. **Conclusions:** No support emerged for concurrent or prospective relations between IGD and psychiatric symptoms, except in one case: increased IGD symptoms forecasted reduction in anxiety symptoms. Observed co-occurrence between IGD symptoms and mental health problems can mainly be attributed to common underlying factors. **Keywords:** Child development; adolescence; longitudinal studies.

## Introduction

Digital games have become widely popular, with more than two billion people playing at different levels of involvement (Statista, 2018). For most players, gaming is a positive recreational activity. However, a few children and adolescents manifest addiction-like engagement in gaming with associated difficulties in everyday functioning (Kuss & Griffiths, 2012). Some studies find that children and adolescents scoring high on symptoms of Internet gaming disorder (IGD) have more symptoms of common psychiatric disorders than other children, Cohen's  $d$  typically somewhat above 1 (e.g., Kim et al., 2016; Percy, McEvoy, & Roberts, 2017), whereas other work chronicles very small associations with mental health problems (e.g.,  $r = .07-.15$ ; Wichstrøm, Stenseng, Belsky, von Soest, & Hygen, 2019).

Given the increase in gaming and continuing concern about its mental health consequences, as well as limits to existing study designs (see below), further investigations of relations between gaming and well-being are called for. Notably, the evidence

already cited led to IGD being included as a condition for further study in the Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5; American Psychiatric Association, 2013). Nevertheless, there is ongoing scientific debate as to whether there is a convincing scientific basis for formally establishing gaming disorder in psychiatric nosology (Van Rooij et al., 2018). The fact that excessive gaming appears transient (Rothmund, Klimmt, & Gollwitzer, 2018) might seem to support those arguing against the establishment of a new psychiatric disorder.

Three explanations exist for the comorbidity of psychiatric symptoms (Angold, Costello, & Erkanli, 1999). The first is that psychiatric disorders are not sharply delineated, with identical symptoms sometimes, indeed often, characterizing supposedly different disorders. Because there appears to be little overlap between symptoms of IGD and the most prevalent psychiatric disorders, we focus on the remaining two explanations.

The second explanation for psychiatric comorbidity suggests that there may be a causal relation between IGD and other mental health problems, with IGD operating as an etiological factor – and/or the

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other way around. The third explanation is that there may be common causes to IGD and other disorders spuriously linking the other two (e.g., genetics, personality, parent-child relationships). Here, we address these latter two accounts of symptom co-occurrence by examining (a) prospective and potentially reciprocal relations between symptoms of IGD and common psychiatric disorders in childhood and adolescence (explanation two), while evaluating (b) whether such associations might be due to stable common causes (e.g., genes, persistent personality traits, (explanation three)).

### *Prospective and reciprocal links between symptoms of IGD and mental health*

Currently, associations between IGD in adolescents and mental health are largely unexplained due to disproportionate reliance on cross-sectional designs. To our knowledge, only one prior study has investigated whether IGD actually forecasts mental health problems (or the reverse; Wartberg, Kriston, Ziegler, Lincoln, & Kammerl, 2019). Nevertheless, there is some prospective work that documents links between extensive gaming and poor mental health, though this was before the release of the DSM-5 criteria for IGD (e.g., Brunborg, Mentzoni, & Frøyland, 2014; Lemmens, Valkenburg, & Peter, 2011). Also notable perhaps is that some research documents beneficial effects of internet gaming (Granic, Lobel, & Engels, 2014). We thus consider both possibilities.

### *Detrimental effects of IGD symptoms*

A core symptom of IGD is preoccupation with gaming, implying that much time is spent on gaming, leaving fewer opportunities for exposure to anxiety-provoking situations (e.g., unstructured social interactions with peers – social anxiety) or stimuli (e.g., unfamiliar dogs – specific phobia). Such avoidance is a hypothesized maintaining factor for anxiety – whereas exposure to threatening situations and objects is perhaps the most important feature of its treatment. Hence, symptoms of IGD could maintain *anxieties* once established by preventing children from learning that what he/she fears is not so catastrophic after all. Consistent with this claim, some, prior research finds that pathological gaming predicts anxiety (Wartberg et al., 2019).

Extensive gaming may also jeopardize important relationships, including friendships, which may explain why pathological gaming forecasts loneliness (Lemmens et al., 2011) as well as physical inactivity (Melkevik, Torsheim, Iannotti, & Wold, 2010). Importantly, both loneliness (Cacioppo, Hughes, Waite, Hawley, & Thisted, 2006) and limited physical activity (Zahl, Steinsbekk, & Wichstrøm, 2017) are risk factors for *depression*.

Such observations may explain why some investigations have discerned links between gaming and depression (Brunborg et al., 2014; Wartberg et al., 2019).

Sustained attention may also be undermined by extensive gaming. After all, gaming provides stimuli at a fast pace. The scan-and-shift hypothesis (Jensen et al., 1997) postulates that gaming with images flashing across the screen at a fast pace may lead children to adopt a high-speed mode of attentional focus, thereby undermining attentional capabilities. To the extent that children adapt to the intense cognitive stimulation in games through elevated levels of baseline arousal, such activity may also lead children to perceive low-stimuli environments as deprivation, thereby fostering restlessness or hyperactivity (Lang, Zhou, Schwartz, Bolls, & Potter, 2000). Despite such possibilities, several studies have failed to document links between pathological gaming and symptoms of ADHD (Ferguson & Cera-noglu, 2014; Wartberg et al., 2019).

The three most popular games in the United States in 2019 (Call of Duty, Grand Theft Auto and Fortnite) contain violence, and there has been speculation that the *in vitro* aggressiveness in digital games may foster *in vivo* aggressive behavior – common to both *conduct disorder* (CD) and *oppositional defiant disorder* (ODD)). Some, (Greitemeyer & Mugge, 2014; Prescott, Sargent, & Hull, 2018) even if not all (Ferguson, 2015), meta-analyses indicate that violent games do increase aggressive behavior. Recent preregistered studies find no evidence; however, linking violent video games to aggressive behavior (e.g., McCarthy, Coley, Wagner, Zengel & Basham, 2016; Przybylski & Weinstein, 2019).

Despite the aforementioned arguments and (limited) evidence linking gaming with symptoms of psychiatric disorders, the possibility that heavy involvement in gaming may actually enhance mental health, perhaps by reducing psychiatric symptoms, should not be overlooked. Novel games often involve children playing with other children (Olson, 2010). Participation in the virtual world may provide players with a sense of (online) community, which may fulfill the need to establish and maintain positive relationships (i.e., the-need-to-belong hypothesis; Baumeister & Leary, 1995); if so, this could reduce loneliness which, as already noted, is itself a risk factor for depression (Cacioppo et al., 2006). Moreover, online relationships may convert to offline relationships, perhaps further fulfilling one's need-to-belong. In addition to offering a training ground for improving social skills, gaming may also protect against depression. Offline friendships may increase exposure to anxiety-provoking social situations, thereby reducing social anxiety over time. Notable in this regard is a randomized controlled trial by Russoniello et al. (2013) which found that increased gaming reduced symptoms of depression.

### *Reciprocal effects: do symptoms of psychiatric disorders affect gaming?*

Digital games capture the gamer's attention and can foster a sense of mastery and joy. Youth suffering from emotional and behavioral disorders might use gaming as an escape from negative emotions and thoughts. Such escapism is an important motivation for gaming (Kuss & Griffiths, 2012) and fairly prevalent (15%; Wichstrøm et al., 2019). Indeed, loneliness in adolescents predicts later pathological gaming (Lemmens et al., 2011), thereby underscoring selection effects into IGD-like behavior.

Digital games are fast-paced and offer frequent rewards. Inability to sustain attention is a key feature of ADHD and children with ADHD display strong preferences for immediate rewards over delayed ones (Barkley, 1997). All this makes gaming an attractive activity for children with symptoms of ADHD (Stenseng, Hygen, & Wichstrøm, 2019), perhaps increasing risk of developing IGD. Children with ADHD also show a preference for stimulus-rich environments and may in fact perform better than others when tracking rapidly moving objects in shifting environments (Grossman, Hoffman, Berger, & Zivotofsky, 2015) – a situation characteristic of many games. It is perhaps for these and related reasons that attention problems (Peeters, Koning, & van den Eijnden, 2018), poor regulation skills (Wichstrøm et al., 2019) and higher levels of hyperactivity/inattention each predict symptoms of pathological gaming (Wartberg et al., 2019).

In sum, while there are theoretical and empirical grounds for concern about the adverse effects of pathological gaming on children's mental health and that children experiencing mental health issues may develop gaming problems, the possibility also exists that gaming can exert positive effects on children's well-being. It is for these reasons that we consider all possibilities herein, including reciprocal effects. Critical to recognize, however, is that even if adverse or beneficial 'effects' emerge in longitudinal inquiry of gaming disorder and mental health, this by itself does not imply causation. After all, the possibility of confounding effects needs to be entertained.

### *Common causes?*

Preliminary evidence from family studies suggests that problematic Internet use is partly under genetic influence (Deryakulu & Ursavaş, 2014). Given that factor analytic work reveals a strong common component to most psychopathology in childhood and adolescence (McElroy, Belsky, Carragher, Fearon, & Patalay, 2018) and that shared genetic influence partly accounts for this comorbidity (Caspi & Moffitt, 2018), one may expect that genetics also accounts for some of the co-occurrence between IGD and other disorders (or

symptoms), to the extent they emerge in empirical inquiries. Moreover, although work on identifying risk factors for IGD is still in its early stages, a range of personal factors not fully explained by genetics have found to predict IGD and psychiatric disorders (Kotov, Gamez, Schmidt, & Watson, 2010), including personality (Müller, Beutel, Egloff, & Wölfling, 2014) and social competence (Wichstrøm et al., 2019). The list of potential common causes of IGD and other psychiatric disorders is certainly longer, including factors of which we remain unaware. Indeed, it is for these reasons that we adopt a particular methodological approach to the investigation of prospective relations between gaming and mental health, one designed to address this possibility of common causes.

If IGD symptoms and other mental health problems are contributing to each other, these effects take place at the within-person level. Notably, though estimates derived from traditional covariate approaches – which have been used in all observational research on IGD cited above (except for one study, Brunborg et al., 2014) – might be biased, they do not separate within- from between-person effects (Hamaker, Kuiper, & Grasman, 2015).

To overcome this methodological concern, we implement a Random Intercept Cross-Lagged Panel Model (RI-CLPM) (Hamaker et al., 2015). This statistical method distinguishes within- and between-person effects to investigate the prospective interrelation of presumed causes and consequences, and thus in the case of the current inquiry mental health problems and gaming. Potential evidence of etiological effects would emerge if prospective relations between symptoms of IGD and other disorders emerge *once stable between-person effects are discounted*. Detection of such time-invariant between-person effects, however, would still be informative, chronicling common origins of IGD and other disorders as a source of comorbidity. In sum, the application of this analytical approach brings us one step closer to determining whether there is a true prospective effect of symptoms of IGD on psychiatric disorders and/or the reverse, or whether statistical associations linking the two constructs and thus evidence of co-occurrence is a result of common underlying factors. While this does not mean that we are positioned to document indisputable causal effects, it does mean that in the case of any identified prospective relations that these would not be the result of time-invariant effects, whether reflecting personal (e.g., personality, genetics) or contextual factors (e.g., socioeconomic status (SES)).

## **Method**

### *Procedure and participants*

Parents of two birth cohorts (2003/2004) in Trondheim, Norway ( $N = 3,456$ ) were invited to participate in a study of

child development (Steinsbekk & Wichstrøm, 2018). An invitation letter was mailed to their homes prior to the routine health checkup for 4-year-olds. In total, 3,358 families (97%) attended the health checkup. Parents ( $n = 176$ ) with insufficient proficiency in Norwegian were excluded. A total of 2,475 parents consented (82.1%). Upon receiving invitation to participate, parents completed the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997), a screening tool for mental health problems. To ensure oversampling of children with emotional and behavioral problems, SDQ total problem scores were divided into four strata. The likelihood of being included in the study increased with an increasing SDQ score. 1,250 of the consenting families were drawn to participate, and we succeeded in interviewing 1,007 at T1. Data have been collected biannually, and the current inquiry is based on measurements from ages 10 ( $n = 703$ ;  $M_{\text{age}} = 10.51$  years,  $SD = 0.17$ ), 12 ( $n = 666$ ;  $M_{\text{age}} = 12.49$  years,  $SD = 0.15$ ) and 14 years ( $n = 636$ ;  $M_{\text{age}} = 14.33$  years,  $SD = 0.59$ ). Children's SDQ at age 4 predicted dropout at age 10 (odds ratio (OR) = 1.06, 95% confidence interval (CI) = 1.02–1.09), age 12 (OR = 1.05, 95% CI = 1.02–1.08) and age 14 (OR = 1.06, 95% CI = 1.03–1.09). See Table S1 for further sample descriptives. The sample is comparable to the Norwegian population of 4-year-olds regarding parental education and SES (Steinsbekk & Wichstrøm, 2018). The project was approved by the Regional Committee for Research Ethics, Mid-Norway.

## Measures

*Symptoms of Internet Gaming Disorder* were assessed by the Internet Gaming Disorder Interview (IGDI; Wichstrøm et al., 2019) assessing the nine DSM-5 defined symptoms of IGD. The IGDI is an interviewer-based psychiatric interview of youth, which implies that the interviewer poses mandatory and optional follow-up questions and probes until enough information is obtained to decide whether a symptom is present during the previous 12 months. Interviews were completed face-to-face by interviewers who had a college or university degree in health or education sciences. The inter-rater reliability of blindly recoded interviews was ICC = 0.90. A symptom count score was calculated as the sum of symptoms (0 = not present, 1 = present). See Table S2 for descriptives of IGD symptoms from age 10–14 years.

*Symptoms of psychiatric disorders* were assessed by the Child and Adolescent Psychiatric Assessment (CAPA; Angold and Costello, 2000) and included symptoms counts of depressive disorders (i.e., major depression and dysthymia), ADHD, ODD, CD, and anxiety disorders (i.e., social phobia, specific phobias, separation anxiety disorder, and general anxiety disorder). One parent and the child were interviewed separately, and a symptom was considered present if reported by either parent or child. Inter-rater reliabilities derived from recoding's by blinded coders of 15% of filmed interviews ranged from ICC .86–.90 across disorders. Due to low prevalence of CD, symptoms of CD and ODD were combined to form a behavioral disorder (ODD/CD) score.

## Statistical analyses

We fitted four separate random intercept cross-lagged panel models (RI-CLPM) (Hamaker et al., 2015) for symptoms of IGD and (a) depression, (b) anxiety, (c) ADHD and (d) ODD/CD, respectively, from age 10 through age 12 and to age 14 (Figure 1), using Mplus 7.4. Separate testing of individual disorders was necessary because of the complexity of RI-CLPM, in that inclusion of all symptoms in one model would result in an inadmissible solution. RI-CLPM decomposes observed symptoms into a stable between-person part (a latent random intercept) and a latent within-person part, which assesses changes from one's own mean IGD

symptoms level as a function of changes in one's own mean symptom level of the respective disorder (e.g., depression) at a previous time point (lagged effects), and *vice versa*. Each RI-CLPM model fit was compared to a corresponding CLPM, using the Satorra–Bentler scaled chi-square test. Because the sample was stratified at screening, analyses were weighted, to arrive at correct population estimates, with a factor corresponding to the number of youths in the population divided by the number of youths in the stratum. A robust maximum likelihood estimator was used, and missing data were handled with full information maximum likelihood estimation.

## Results

The bivariate correlations between symptoms of mental disorders are presented in Table S3. At all ages (10, 12 and 14) symptoms of IGD were positively, significantly, though modestly correlated with symptoms of depression, anxiety, ADHD, and ODD/CD at ages 10, 12, and 14 (range of  $r = .09$ –.19).

In order to arrive at correct estimates of prospective within-individual associations between symptoms of IGD and mental disorders, while controlling for stable between-person variance, we fitted four separate RI-CLPMs (Figures 2, Figures S1–S3). All models showed good model fits (Table S4) and had significantly better model fits than the traditional CLPMs, based on the Satorra–Bentler scaled chi-square test.

At the between-person (or group) level, more IGD symptoms were associated with more symptoms of anxiety ( $r = .53$ ,  $p = .028$ ), ODD/CD ( $r = .36$ ,  $p = .048$ ), and ADHD ( $r = .23$ ,  $p = .028$ ), but not depression ( $r = .26$ ,  $p = .14$ ). In contrast to the concurrent correlations of IGD symptoms with symptoms of psychopathology (in Table S3), no significant concurrent correlations between IGD symptoms and those of disorders emerged at the within-person level (Figure 2, Figures S1–S3). Moreover, with respect to prospective associations at the within-person level, only a single set of relations proved statistically significant, such that an increase in symptoms of IGD predicted a decrease in symptoms of anxiety two years later, in the 10–12 years ( $\beta = -.17$ ,  $p = .015$ ) as well as the 12–14 years ( $\beta = -.16$ ,  $p = .010$ ) age spans (Figure 2).

## Discussion

Although IGD and symptoms of IGD co-occur with symptoms of psychopathology (e.g., Wichstrøm et al., 2019), it remains unclear why this is the case. Using a novel analytical technique that disentangles between- and within-person effects, we were, for the first time, positioned to evaluate two viable explanations of this covariance: (a) prospective within-person relations between symptoms of IGD and other disorders and (b) common etiology. By controlling for stable associations at the between-person level and hence for common causes, we evaluated

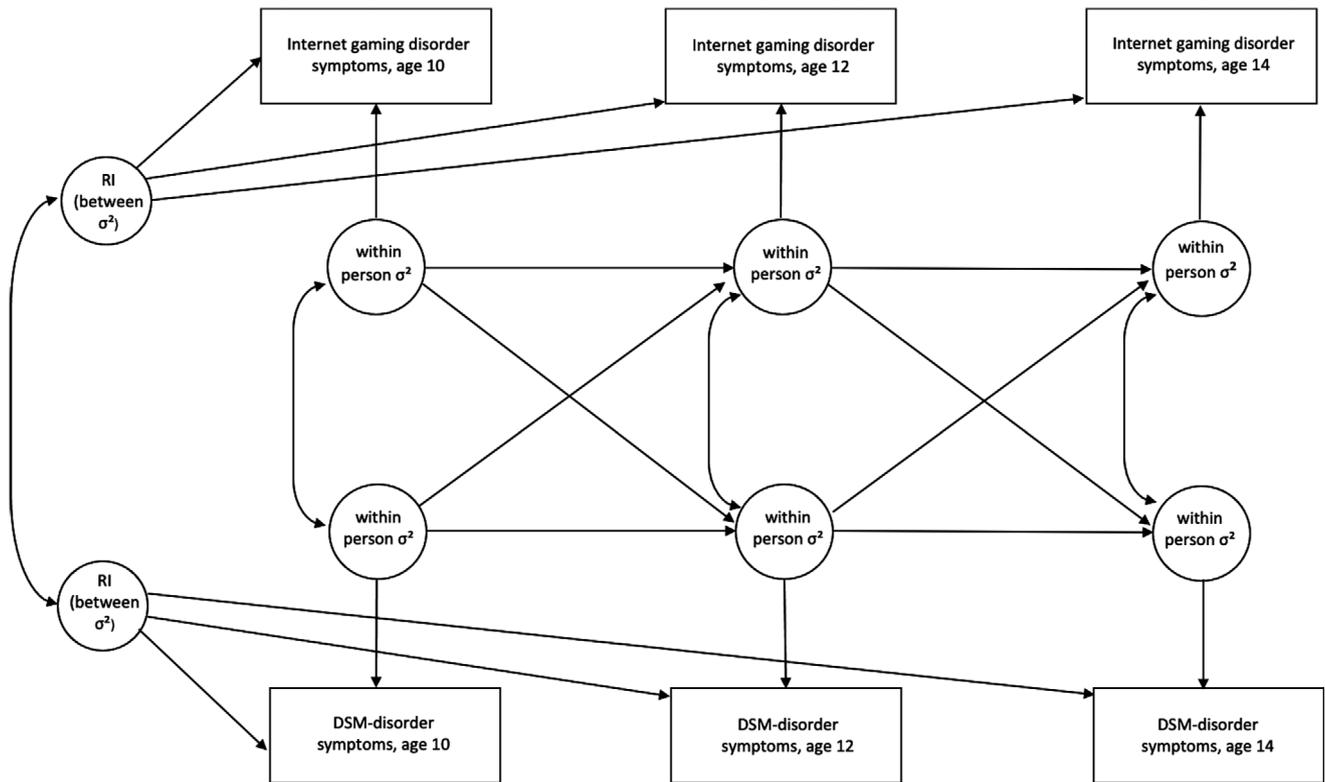


Figure 1 Illustrative model for random intercept cross-lagged modeling. Note:  $\sigma^2$  = variance

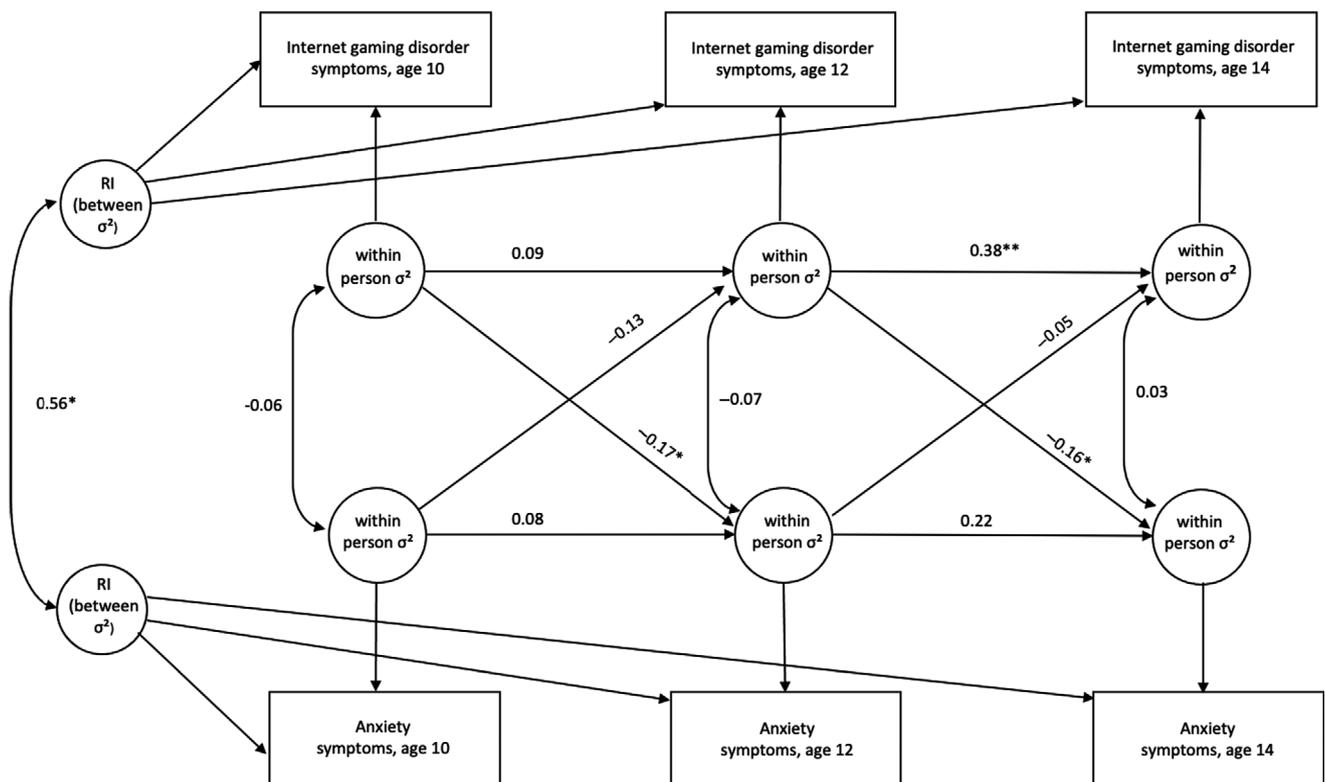


Figure 2 RI-CLPM for symptoms of IGD and anxiety. Cross-lagged paths from IGD symptoms to anxiety symptoms are hold equal over time, and all other paths are freely estimated

whether departures from an individual’s own mean level of disorder symptoms resulted in changes in other disorder symptoms. Simultaneously, we also

assessed the magnitude of symptoms’ co-occurrence before and after controlling for such underlying common factors.

Relying on repeated clinical interviews across childhood and early adolescence, the prospective influence explanation for symptom co-occurrence received little support. One opposite association emerged; *more* IGD symptoms at age 10 and 12 predicted slightly fewer symptoms of anxiety two years (at age 12 and 14, respectively). Notably, the standardized betas were small. Just as noteworthy is that the common etiology explanation of symptom covariation received empirical support, accounting for the fact that children with more IGD symptoms also evinced more symptoms of anxiety, ADHD, and ODD/CD. Moreover, our nonsignificant estimates of co-occurrence in the RI-CLPM suggest that the previously observed concurrent co-occurrence is completely due to common causes. Of course, what our work could not illuminate was which common causes might have been operative; future work will need to address this pressing issue.

### *Prospective relation between symptoms of IGD and symptoms of anxiety*

Several, though not all (Weinstein, Przybylski, & Murayama, 2017), studies chronicle longitudinal associations between IGD symptomatology or related constructs and psychopathology (e.g., Lemmens et al., 2011; Wartberg et al., 2019). What limits the just-cited prospective research is their small effect sizes (ranging from  $r=.08$  to  $r=.12$ ). Although significant in large studies, their clinical utility and validity may be difficult to discern (Ferguson & Wang, 2019). Another limitation of much available work is reliance on regression-type analytic strategies, including autoregressive cross-lagged models (Lemmens et al., 2011; Wartberg et al., 2019), in which estimates stem from a mixture of between-person and within-person information. We therefore do not know whether prospective within-person associations did exist in the cited findings. However, other research also adds to the doubt this being the case. Brunborg et al., (2014) utilized a first-difference method, which analyzes within-person changes finding, just as we did, that prior detected associations did not remain, for the most part, once participants served as their own control. In sum, the work reported herein, along with that of Brunborg et al., (2014), clearly challenges the notion that there is a causal effect of symptoms of IGD on symptoms of disorders, or the reverse, at least in the age-group studied herein. Before embracing this conclusion in its entirety, it must be noted that we did find some *potential* evidence of causal influence in that increased IGD symptoms forecasted reductions in symptoms of anxiety across two two-year time periods. Why might this have been the case? We consider two possibilities.

*Gaming reduces worry and rumination?* Spending time worrying and ruminating might increase

anxiety (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Games capture gamers' attention by combining exciting plots with sophisticated graphics and sound (Ho et al., 2017). For this reason, gaming may be an effective means to distract not only from negative moods (one of the DSM-5 symptoms), but from anxiety-maintaining thoughts. Only future research will be able to test this proposition.

*Gaming facilitating social relationships?* By middle childhood and early adolescence, generalized anxiety and social phobia are the two dominating anxieties (Copeland, Angold, Shanahan, & Costello, 2014), with the focus of worry often centering on social relationships (Dugas et al., 1998). Many of the current games involve social interaction, leading children to play with each other (Olson, 2010), resulting in immense social exchange. Online games allow anonymity, with central limits on some important nonverbal cues (e.g., of uncertainty, embarrassment). All this may make extensive gaming, a form of *tolerable* social training which may ultimately serve to reduce social anxiety.

In addition to providing online practice in social interaction, the social nature of gaming may also have offline effects. The fact that children may game with many of the same people repeatedly (e.g., same teams in games such as *Fortnite* or *Counter Strike*) may further reduce their social anxiety, as they get to know these familiar others and probably find them much less threatening than they might otherwise have presumed them to be.

### *Common cause*

By disaggregating variance into between- and within-person level, we found that the previously observed correlations between symptoms of IGD and other disorders – which stem from both between and within-person variance – were the result of between-person, not within-person variance. Together with our finding of no prospective associations at the within-person level between gaming and mental health problems (except anxiety), the results reported herein suggest that prior evidence of co-occurrence is due to time-invariant common factors affecting both the propensity toward symptoms of IGD and symptoms of these other disorders. While we suspect that genetics is an important factor here, particularly given recent evidence that many seemingly distinct psychiatric disorders share genetic etiology (Lee et al., 2013), much more work is required to delineate such specific common causes of the repeatedly documented associations between extensive gaming and mental health.

### *Limitations*

Despite apparent strengths of this work – including reliance on a representative community sample

studied longitudinally using repeated clinical interviews while applying a statistical method that discounts effects of all time-invariant factors – it was not without limitations. The identified predictions yielded effects of small size, raising questions, as already noted, regarding the meaning that can or should be breathed into them. We examined symptoms counts, not disorders. Hence, although there is no convincing evidence for most psychopathology being categorical in nature (Krueger et al., 2018), we do not know whether the present findings would generalize to diagnosed IGD or other formal diagnoses. Although we discounted all time-invariant between-person effects, we were not positioned to do so with time-varying ones (e.g., changes in friendships or relations to parents) – which could still have produced the IGD→anxiety link. It has been noted that RI-CLPM requires sample sizes above  $N = 1,000$  to obtain conventional power of  $>.80$  with typical prospective association strength and missingness (Masselink et al., 2018). Hence, prospective effects may have gone undetected in the current study. Even so, previous studies using the same sample have demonstrated expected associations when applying within-person analyses (e.g., Hygen et al., 2020). Our results indicate that there is a common, stable, vulnerability to symptoms of IGD, and common psychiatric disorders, causing an association between them. Hence, prevention or treatment of either is not likely to have an effect on the other as long as the underlying time-invariant factors are not targeted. To advance interventions, efforts should be made to identify these common factors. A final point to be made is that whether our investigation of a general population sample of Norwegian youth generalizes to clinical samples or those in other locales remains to be determined; glib generalization should not be presumed.

## Conclusion

We found, as others have, that children and adolescents with more symptoms of IGD also had more symptoms of common psychiatric disorders. Except

for increased rates of IGD symptoms predicting fewer anxiety symptoms, this co-occurrence was fully attributable to unmeasured common factors; increased IGD symptoms did not predict prospective increase in symptoms of psychiatric disorders or *vice versa*. Heavy involvement in gaming during childhood, even to the extent of acquiring proposed symptoms of Internet gaming disorder, therefore does not seem to pose a threat toward developing increased mental health problems.

## Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

**Table S1.** Sample characteristics and descriptives.

**Table S2.** Descriptives for IGD symptoms at age 10.12, and 14.

**Table S3.** Means, standard deviations, and bivariate correlations for study variables.

**Table S4.** Model fits of RI-CLPMs for IGD and each disorder symptoms.

**Figure S1.** RI-CLPM for symptoms of IGD and ODD/CD.

**Figure S2.** RI-CLPM for symptoms of IGD and depression.

**Figure S3.** RI-CLPM for symptoms of IGD and ADHD.

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### Key points

- Internet Gaming Disorder (IGD) co-occurs with common psychiatric disorders in childhood and adolescence.
- The reasons for this co-occurrence is poorly understood, examining its nature is therefore the focus of the present inquiry.
- Increased symptoms of IGD do not prospectively predict increased psychiatric ones, but a reduction in anxiety symptoms two years later was detected.
- The co-occurrence of symptoms of IGD and other psychiatric disorders is mainly due to common etiology.

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