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Risk and protective factors for (internet) gaming disorder: A meta-analysis of pre-COVID studies

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ABSTRACT

This large-scale meta-analysis aimed to provide the most comprehensive synthesis to date of the available evidence from the pre-COVID period on risk and protective factors for (internet) gaming disorder (as defined in the DSM-5 or ICD-11) across all studied populations. The risk/protective factors included demographic characteristics, psychological, psychopathological, social, and gaming-related factors. In total, we have included 1,586 effects from 253 different studies, summarizing data from 210,557 participants. Apart from estimating these predictive associations and relevant moderating effects, we implemented state-of-the-art adjustments for publication bias, psychometric artifacts, and other forms of bias arising from the publication process. Additionally, we carried out an in-depth assessment of the quality of underlying evidence by examining indications of selective reporting, statistical inconsistencies, the typical power of utilized study designs to detect theoretically relevant effects, and performed various sensitivity analyses. The available evidence suggests the existence of numerous moderately strong and highly heterogeneous risk factors (e.g., male gender, depression, impulsivity, anxiety, stress, gaming time, escape motivation, or excessive use of social networks) but only a few empirically robust protective factors (self-esteem, intelligence, life satisfaction, and education; all having markedly smaller effect sizes). We discuss the theoretical implications of our results for prominent theoretical models of gaming disorder and for the existing and future prevention strategies. The impact of various examined biasing factors on the available evidence seemed to be modest, yet we identified shortcomings in the measurement and reporting practices.

1. Introduction

Gaming disorder is the second official mental health condition (just after gambling disorder) directly associated with the use of digital technology. A global pooled prevalence for gaming disorder of approx. 3 % estimated by two recent meta-analyses (Kim et al., 2022; Stevens, Dorstyn, et al., 2021) potentially constitute 200 million of people playing pathologically. Over just a few years during which both internet gaming disorder, IGD (APA, 2013) and gaming disorder, GD (World Health Organization, 2019) were officially introduced, a large amount of evidence in a form of associations between IGD or GD and different psychological, physical, socio-economic, gaming or other phenomena, has been accumulated (Burleigh et al., 2019; Kuss et al., 2018; Paulus et al., 2018). Inclusion of GD into the eleventh revision of the International Classification of Diseases (World Health Organization, 2019) is therefore justified not only from the clinical perspective and public health needs (Rumpf et al., 2018) but also based on extensive longitudinal evidence on the negative consequences (e.g., increased anxiety, depression, loneliness, or emotional distress, and decreased life satisfaction, school performance, poor relationship with parents, or social competence) (Richard et al., 2020). Several countries have reported growing needs for treatment facilities specialized on gaming disorder

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Rumpf et al., 2018). This is closely related to the requirement for proper training of health professionals for providing not only treatment but also prevention and early intervention strategies. Since prevention should be preferred over treatment, development of preventive approaches still represents "a pressing need" (Saunders et al., 2017, p. 275). Most of the existing preventive approaches are selective - applied only to gamers who have a greater risk of developing the disorder (King et al., 2018). The current early stage of the development of preventive programs (Rumpf et al., 2018) would benefit from a synthesis of evidence on risk or protective factors of (internet) gaming disorder. Such a synthesis would be beneficial also for the development of theoretical models that hypothesize the existence of various predisposing factors (e.g., reward-seeking behavior or diminished cognitive control in the cognitive-behavioral model by Dong and Potenza (2014); high impulsivity or low self-esteem in the I-PACE model by Brand et al. (2016). The need for robust evidence is also underscored by the actions of the gaming industry (Association, 2018; Foundation, 2018, p. 1; Twist, 2018) that either overlook or downplay the negative consequences of gaming disorder and also cast doubt on the existing evidence („The evidence for its [GD into the ICD 11] inclusion remains highly contested and inconclusive").

The few meta-analyses published so far summarized only a portion of this evidence, reporting moderate associations of IGD with the male gender (Su et al., 2020, Kim et al., 2022; Stevens, Dorstyn, et al., 2021), impaired response inhibition (Argyriou et al., 2017), or a lower probability discounting rate (Chung et al., 2021). Ostinelli et al. (2021) reported pooled prevalence of 32 % for comorbidity of depression and pooled Beck's Depression Inventory score of 10.31 in individuals with significant IGD symptoms. meta-analyses studying the prevalence of gaming disorder (Fam, 2018; Gao et al., 2022; Kim et al., 2022; Liao et al., 2022; Stevens, Dorstyn, et al., 2021) have shown that factors such as type of measure, sample size, and sample type, participant age, or gender significantly moderate the pooled prevalence. A recently published meta-analysis by Ji et al. (2021) summarized the risk and protective factors of IGD in a Chinese population. They identified maladaptive cognitions, achievement motivation, and rule-breaking behavior as the strongest risk factors, while self-control, school climate, and school engagement seemed to act protectively. The existing body of literature uses the term risk or protective factor to denote any variable that is meaningfully related to IGD/GD. Thus, what is considered a risk or protective factor is not determined based on the method (e.g., experiment, longitudinal study) but rather a theoretical assumption associated with the existence of some form of monotonic relationship (e.g., younger age associated with more severe IGD/GD is considered as a predisposing or a risk factor).

1.1. Present study

Being first recognized as a mental disorder relatively recently (World Health Organization, 2019), gaming disorder was derived from substance use disorder or pathological gambling (Aarseth et al., 2017) and is still considered largely atheoretical (Kardefelt-Winther et al., 2017). So far, only a few theoretical models explain this phenomenon - the I-PACE model (Brand et al., 2016), cognitive-behavioral model (Dong & Potenza, 2014), and model of compensatory use (Kardefelt-Winther, 2014). Any theory building process should, at the same time, be preceded by strong evidence for the existence of such a phenomenon (for more detail see the TCM framework, Borsboom et al. (2021)). A better theoretical understanding of (internet) gaming disorder and the development of more effective preventive/intervention programs thus first requires rigorous mapping of evidence on potentially causally relevant psychological but also non-psychological factors, which the present meta-analysis aimed to deliver. As opposed to evidence from individual studies or systematic reviews, meta-analyses, in general, do provide more precise estimates of the relations among variables that are not so much laden with idiosyncrasies and study-specific biases of individual studies. This meta-analysis aimed to provide the most comprehensive synthesis to date of risk and protective factors of both gaming disorder and internet gaming disorder (henceforth referred to as GD). Apart from estimating these predictive associations and relevant moderating effects, we implemented state-of-the-art adjustments for publication bias, psychometric artifacts (measurement error and different types of selection effects), and other forms of bias arising from the publication process. Additionally, we carried out an in-depth assessment of the quality of underlying evidence by examining indications of selective reporting, statistical inconsistencies, the typical power of utilized study designs to detect theoretically relevant effects, and performed various sensitivity analyses.

2. Method

The protocol of this meta-analysis has been preregistered at PROSPERO (ID:CRD42020187776), with a list of deviations from the protocol disclosed in Appendix B. An extended description of our methodology is available in Appendix A. Appendices, data, R code, and analytical outputs can be found at https://osf.io/9mzgr/.

2.1. Search strategy

Two different strategies were used to identify relevant studies. First, after piloting the search string, we searched the following databases: Scopus, Web Of Science Core Collection, PsycInfo, PubMed, OSF Preprints, ThesisCommons, ProQuest Dissertations and Theses. The search was carried out on 09/06/2020[2] and was limited to primary empirical studies written in English and published from 2013 (year in which IGD was first defined in the DSM). Second, we carried out a forward citation search in Google Scholar by screening studies that cited the 13 most widely used measures of GD adhering to the DSM-5 or ICD-11 definition of GD (referenced in Appendix A). The exact search string and its translations for all databases can be found in Appendix C.

2.2. Inclusion criteria

(1) Population: As the diagnostic criteria for gaming disorder apply equally to all gender and age groups (Petry et al., 2014 and empirically supported by e.g., Sigerson et al., 2017), we applied no exclusion criteria regarding the target population. The population of interest thus included general population, clinical sample, as well as groups of specific gamers. (2) Correlates/Exposures: Given that any variable can act as a risk or protective factor, the study had to report at least one zero-order association between GD and any correlate, e.g., individual, external, or gaming-related factor (King et al., 2019). (3) Outcome: The study had to assess the presence and/or severity of gaming disorder, i.e., a binary format typical for diagnostic manuals (either the polythetic DSM 5 classifying format where some of the criteria needs to be met or the ICD

[2] The fact that our literature search was limited to 2020, may be viewed as a benefit when considering the effects of COVID and behavioral restrictions. Several studies (e.g., Deng et al., 2021; Ettman et al., 2020; Oka et al., 2021) reported either an increase in GD symptoms from the beginning of the COVID period or increased prevalence of other psychopathological factors (e.g., depression or anxiety) that were included in our *meta*-analysis as risk factors. We believe that synthesizing effects from time periods with significantly different prevalence and comorbidity rates of both the dependent variable and its predictors may induce bias in meta-analytic effects.

11 monothetic classifying format where all of the criteria needs to be met) or a continuous variable format typical for research settings, respectively. We included only studies where the definition and measurement of GD complied with the DSM-5 or ICD-11.[3] Lastly, studies for which we were unable to obtain a full text, or studies not written in English language were excluded.

2.3. Study selection

The set of records identified in the search phase was checked for duplicates and retractions. Screening of abstracts was carried out in Rayyan (Ouzzani et al., 2016) by two coders blinded to each other's decisions. Full texts of non-excluded records were further screened for eligibility. None of the studies were excluded unless agreed upon by both coders (an additional coder was consulted in case of disagreement). Studies for which we were unable to obtain a full text were excluded. The detailed flow of study selection is outlined in the Prisma Flowchart (see Fig. 1). The studies included in and excluded from the meta-analysis are referenced in the separate Supplementary document that can be found at https://osf.io/f3xvs/.

2.4. Data extraction and effect size recomputation

We included all the relevant zero-order effects from each study. Following the screening and selection of the studies, the data were extracted by two coders. We checked for the inter-rater agreement and, if needed, adjusted the coding scheme in a two-stage procedure (see Appendix A for details). We coded the bibliographic and scientometric information, methodological characteristics, outcome and risk factor-related variables, gaming-related variables, sample characteristics, and effect size data. In case of missing data required to recompute the effect size, we contacted the corresponding author. If ultimately, we were unable to recover the missing data, the given effect was excluded.

In case the effect size was not reported in the zero-order Pearson's *r* metric, we converted from the reported test statistics, different effect sizes, or the reported descriptives. The computation and conversion of all effect sizes were carried out in code, using formulas laid out in Borenstein et al. (2009). Full coding scheme, a list of general principles applied to coding, and additional variable-specific coding rules can be found in Appendix A.

3. Analysis

3.1. Strategy for data synthesis

The available evidence was synthesized using multilevel random-effects models. For any identified risk factor, we chose to refrain from doing any quantitative inference unless the given analytic model was based on >10 studies. We modeled both types of dependencies among the effects – nesting of effects within papers and clustering due to the estimation of effects based on the same sample – using the robust sandwich-type variance estimation (RVE) with the "Correlated and hierarchical effects" working model (Borenstein et al., 2009). To test for equality of effect sizes across the levels of the moderators studied, we used the robust HTZ-type Wald test (Pustejovsky & Tipton, 2021). The *p*-values for individual contrasts were adjusted using Holm's method. All *p*-values were two-tailed. All models were fitted using restricted maximum-likelihood estimation (REML) using R packages metafor, version 2.5 (Viechtbauer, 2010) and clubSandwich, version 0.4.2. (Pustejovsky, 2020).

Prior to our analyses, we carried out an in-depth diagnosis of the random-effects meta-analytic model for each separate correlate. Specifically, we screened for influential outliers using the Baujat plot and influence diagnostics indices. Outliers exerting an excessive influence on the meta-analytic model (standardized residual > 2.58) were then excluded in a sensitivity analysis.

3.2. Adjustment for bias

In the present meta-analysis, we attempted to adjust the included effects for (1) psychometric artifacts like measurement error, range restriction/enhancement, and collider bias, (2) publication bias, as well as (3) some other more specific forms of biases in the publication process.

3.2.1. Psychometric artifacts

First, we carried out a correction for various psychometric artifacts that tend to bias individual effect sizes. We employed the psychometric meta-analysis (Schmidt & Hunter, 2014), adjusting for the attenuation of the studied associations due to measurement error in IGD (unreliability, group misclassification). We also accounted for selection effects of range restriction/enhancement and collider bias using the artifact-distribution method (Wiernik & Dahlke, 2020). For a more detailed description of these corrections and related sensitivity analyses, see Appendix A.

3.2.2. Publication bias

More importantly, we also tried to adjust the meta-analytic estimates for publication bias. We used (1) permutation-based implementation of multiple-parameter selection models (in frequentist and Bayesian model-averaging framework) (Bartoš et al., 2021; McShane et al., 2016) and multi-level regression-based models (Stanley & Doucouliagos, 2014). These were supplemented by several exploratory analyses assessing the sensitivity of adjusted effect size estimates to different assumptions about the biasing process. If the adjusted estimates from selection models markedly diverged from the crude meta-analytic estimates, we mainly relied on bias-corrected estimates to guide our substantive inferences (Ropovik et al., 2021). A more elaborate description and specification of the models can be seen in Appendix A.

3.2.3. Other forms of bias

Apart from examining the impact of psychometric artifacts and publication bias, we also explored other more specific biasing effects of the publication process, that is, the citation bias (testing whether more highly-cited studies report larger effect sizes), decline bias (Fanelli et al., 2017; whether studies showing more extreme (possibly opposite) results appear early in the research line rather than late, as data accumulate), and bias arising from the presence of a conflict of interests. The specification, results, and the interpretations of these analyses can be found in Appendix E.

3.3. Quality of evidence assessment

Apart from answering substantive questions, we also assessed the quality and integrity of the synthesized evidence.

3.3.1. Assessment of evidential value

First, we estimated the evidential value of the available studies using a permutation-based *p*-curve analysis (Simonsohn et al., 2014). This analysis aimed to provide insight into the degree of selective reporting in the literature on different risk factors. In case there is evidential value in the given literature, then regardless of power, a right-skewed distribution of *p*-values can be expected. On the other hand, a left-skewed distribution of *p*-values may indicate a substantial prevalence of

[3] Information about all included measures is available in Appendix E, section Descriptives/GD measures used. Excluded studies in most cases used the Chen Internet Addiction Scale (Chen et al., 2003), the Internet Addiction Test (Young, 1998), the Game Addiction Scale (Liao et al., 2009), the Scale for the Assessment of Internet and Computer game Addiction (World Health Organization, 2019), the Video Game Addiction Test (van Rooij et al., 2012), or the Problematic Online Gaming Questionnaire Short-Form (Pápay et al., 2013).

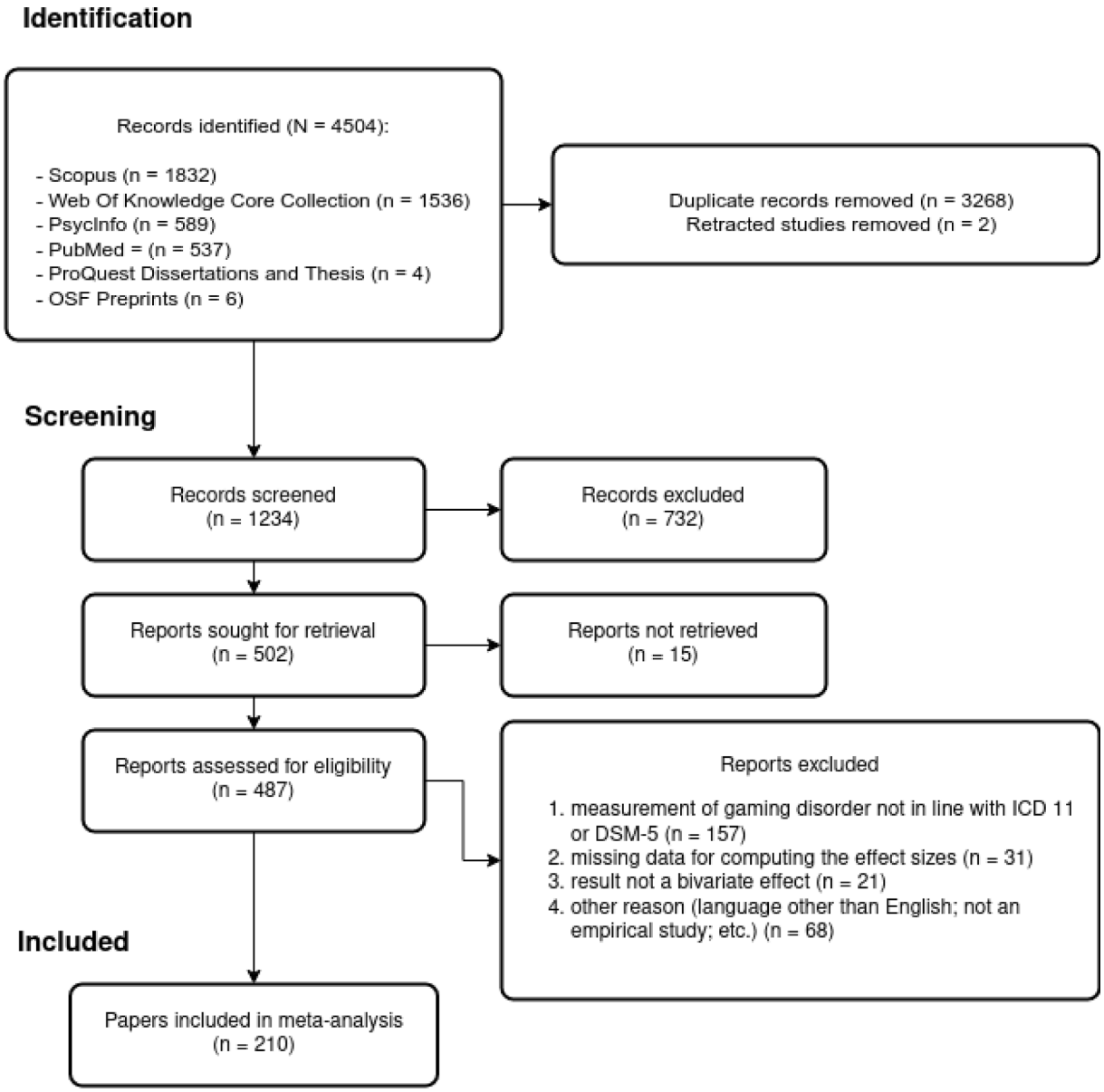

Fig. 1. PRISMA Flowchart.

questionable research practices in the literature. We employed *p*-values recomputed from reported test statistics. The within-study clustering of *p*-values was addressed employing a permutation-based procedure, iteratively sampling only a single focal *p*-value per study, estimating the *p*-curve, and averaging over the set (by selecting the model with the median *z*-score for the right-skew of the full *p*-distribution). We considered the synthesized set of effects to have adequate evidential value if the *z*-test for the right-skew of the half *p*-curve was significant at $p < .05$.

3.3.2. Numerical inconsistencies in reported p-values

Second, all included papers were screened using a machine-based procedure for the presence of inconsistencies in reported *p*-values, employing the statcheck package (Epskamp & Nuijten, 2018). The method works as follows. First, pdf files are converted to plain text files. These are scanned in full for statistical results reported in APA style. Next, test statistics and degrees of freedom are extracted to recompute the *p*-values. Lastly, the recomputed *p*-values are compared to the reported *p*-values. Based on these extracted data, we computed how often were the reported *p*-values inconsistent with the reported test statistics and how many of those cases led to an error in inference (*p*-values actually > 0.05 regarded as significant).

3.3.3. Median statistical power in the literature

Third, we computed the median statistical power to detect various smallest effect sizes of interest ($r = 0.10$, 0.30, and 0.50), as well as median power to detect the bias-corrected (4-parameter selection model and PET-PEESE) estimates. This analysis served to assess the empirical robustness of the evidence underlying the set of synthesized effects.

3.4. Additional sensitivity analyses

In Appendix E, we also report the results of the following, more general sensitivity analyses. (1) We used Fisher's *z*-transformed effect sizes instead of Pearson's *r*. (2) We excluded all effect sizes coming from studies employing a selection inference approach, i.e., disregarding non-significant effects using, e.g., stepwise selection procedures or regularization techniques. Namely, these effect sizes are inflated, on average, leading to overestimation of the mean effect sizes.

Table 1
Results for individual risk/protective factors.

Risk/protective factor	*k*	Effect size *r* [95 % CI]	Odds ratio [95 % CI]	*Tau* [I^2]	*r* 95 % PI [LB, UB]	4-PSM estimate	PET-PEESE estimate	Measurement artifacts-corrected *r*	Median power to detect $r = 0.10$
Age	118	−0.01 [−0.04, 0.01]	0.98 [0.93, 1.02]	0.12 [92 %]	[−0.25, 0.22]	−0.01	0.00	0.03	0.58
Depression	93	0.40 [0.36, 0.44]	2.07 [1.93, 2.22]	0.18 [97 %]	[0.05, 0.75]	0.37	0.32	0.34	0.39
Gaming time	87	0.38 [0.33, 0.42]	1.98 [1.82, 2.16]	0.20 [99 %]	[−0.02, 0.77]	0.36	0.32	0.32	0.61
Being male	78	0.18 [0.14, 0.21]	1.38 [1.29, 1.47]	0.15 [97 %]	[−0.12, 0.48]	0.20	0.18	0.14	0.82
Anxiety (state)	63	0.31 [0.27, 0.35]	1.75 [1.63, 1.88]	0.13 [94 %]	[0.05, 0.57]	0.29	0.30	0.34	0.41
Internet addiction	53	0.59 [0.52, 0.65]	2.89 [2.57, 3.26]	0.21 [99 %]	[0.15, 1.02]	0.55	0.50	0.29	0.22
Impulsivity	29	0.38 [0.31, 0.45]	2.00 [1.76, 2.26]	0.17 [95 %]	[0.03, 0.73]	0.31	0.27	0.34	0.26
Education	24	−0.13 [−0.21, −0.04]	0.79 [0.68, 0.93]	0.17 [92 %]	[−0.48, 0.23]	−0.12	−0.05	0.04	0.12
Social motive	24	0.19 [0.11, 0.26]	1.40 [1.22, 1.61]	0.15 [97 %]	[−0.15, 0.52]	0.18	0.19	0.20	0.64
Stress	23	0.39 [0.32, 0.45]	2.01 [1.79, 2.27]	0.14 [93 %]	[0.10, 0.68]	0.41	0.43	0.38	0.41
Time for internet use	21	0.41 [0.26, 0.57]	2.12 [1.60, 2.81]	0.29 [99 %]	[−0.22, 1.05]	0.20	0.31	0.45	0.57
Competition/achievement motive	20	0.24 [0.19, 0.29]	1.54 [1.40, 1.70]	0.10 [94 %]	[0.02, 0.46]	0.24	0.22	0.28	0.75
Hostility/aggressivity (trait)	19	0.37 [0.28, 0.45]	1.95 [1.67, 2.28]	0.16 [94 %]	[0.02, 0.71]		0.18	0.22	0.52
Social support	19	−0.04 [−0.15, 0.07]	0.94 [0.77, 1.14]	0.19 [97 %]	[−0.48, 0.41]	0.04	−0.01	0.06	0.21
Loneliness	17	0.28 [0.22, 0.34]	1.67 [1.49, 1.87]	0.11 [95 %]	[0.03, 0.53]	0.28	0.28	0.31	0.80
Gaming time weekdays	16	0.34 [0.21, 0.46]	1.84 [1.47, 2.30]	0.23 [97 %]	[−0.16, 0.83]	0.18	0.06	0.21	0.56
Gaming time weekends	16	0.35 [0.22, 0.49]	1.90 [1.49, 2.43]	0.25 [98 %]	[−0.19, 0.90]	0.34	0.06	0.21	0.56
Escape motive	15	0.42 [0.34, 0.51]	2.15 [1.84, 2.51]	0.15 [98 %]	[0.09, 0.75]	0.44	0.39	0.46	0.64
Life satisfaction	15	−0.13 [−0.19, −0.07]	0.79 [0.71, 0.88]	0.10 [91 %]	[−0.36, 0.09]	−0.15	−0.08	0.09	0.75
Skill development motive	15	0.20 [0.14, 0.29]	1.47 [1.28, 1.69]	0.13 [96 %]	[−0.07, 0.50]	0.23	0.16	0.20	0.64
Social networking addiction	15	0.39 [0.25, 0.52]	2.01 [1.57, 2.57]	0.24 [99 %]	[−0.15, 0.92]		0.62	0.51	0.72
Fantasy motive	14	0.32 [0.24, 0.4]	1.79 [1.55, 2.07]	0.13 [96 %]	[0.03, 0.62]	0.35	0.30	0.32	0.71
Recreation motive	14	0.08 [0.02, 0.15]	1.17 [1.03, 1.32]	0.11 [95 %]	[−0.16, 0.32]	0.07	0.05	0.07	0.80
Self-esteem	14	−0.24 [−0.32, −0.15]	0.65 [0.56, 0.76]	0.13 [90 %]	[−0.54, 0.07]	−0.25	−0.24	0.21	0.35
Gaming frequency	13	0.29 [0.19, 0.38]	1.69 [1.41, 2.01]	0.15 [97 %]	[−0.05, 0.63]	0.29	0.31	0.21	0.70
ADHD	12	0.26 [0.15, 0.38]	1.62 [1.31, 2.00]	0.16 [95 %]	[−0.10, 0.63]		0.33	0.36	0.35
Coping motive	12	0.30 [0.20, 0.40]	1.72 [1.43, 2.06]	0.15 [98 %]	[−0.05, 0.65]	0.31	0.23	0.30	0.89
Online gaming time	12	0.22 [0.15, 0.29]	1.49 [1.31, 1.68]	0.08 [91 %]	[0.02, 0.42]	0.22	0.20	0.19	0.74
Intelligence	11	−0.24 [−0.41, −0.07]	0.65 [0.48, 0.89]	0.21 [72 %]	[−0.74, 0.26]		−0.12	0.24	0.10

Note. *k* = number of analyzed effects; *r* = Pearson's bivariate correlation; CI = confidence interval; PI = prediction interval; *Tau* = standard deviation of the true effect sizes; I^2 = relative heterogeneity; 4-PSM = 4-parameter selection model; PET-PEESE = regression-based publication bias-adjustment model. RoBMA = model-averaging Robust Bayesian meta-analysis.

4. Results

We start by characterizing the set of synthesized studies/effects, followed by the presentation of the results on individual risk/protective factors and aggregate correlate types. Furthermore, we test for the effects of several theoretically relevant moderators, examine the meta-analytic estimates after applying multiple adjustments for publication bias and psychometric artifacts, and assess the quality and integrity of the evidence underlying the synthesized effects.

4.1. Descriptives

A complete breakdown of the study selection process is shown in the PRISMA flowchart, Fig. 1. We were able to recompute the effect sizes for 1586 effects from 253 studies (reported in 210 papers). Large majority, 95 % of effects came from studies utilizing different forms of cross-sectional designs, 1 % from randomized experiments, and 4 % from longitudinal studies. Altogether, these effects summarized data from 210,557 independent participants, with a median *N* across all the eligible effect sizes equal to 468. One third of the sampled participants (*SD* = 21 %) were female, on average, with a weighted mean age of included samples at 22.5 years. The included effects pertained to different types of populations: gamers other than esport (43 %), general population (26 %), university students (12 %), clinical population (11 %), and other (8 %).

The included studies most frequently examined relationships with psychological correlates (32 %), followed by psychopathological (26 %), demographic (16 %), gaming-related correlates (14 %), maladaptive personality traits (6 %), and social correlates (3 %), with other types of correlates accounting for 5 %. GD, on the other hand, was most frequently assessed using the IGDS9-SF (21 %), DSM-5 criteria (16 %), clinical interview (12 %), IGDS (12 %), IGDT-10 (11 %), and IGDC (8 %). The complete frequency table of GD measures used in the literature can be seen in the full analysis outputs in Appendix E.

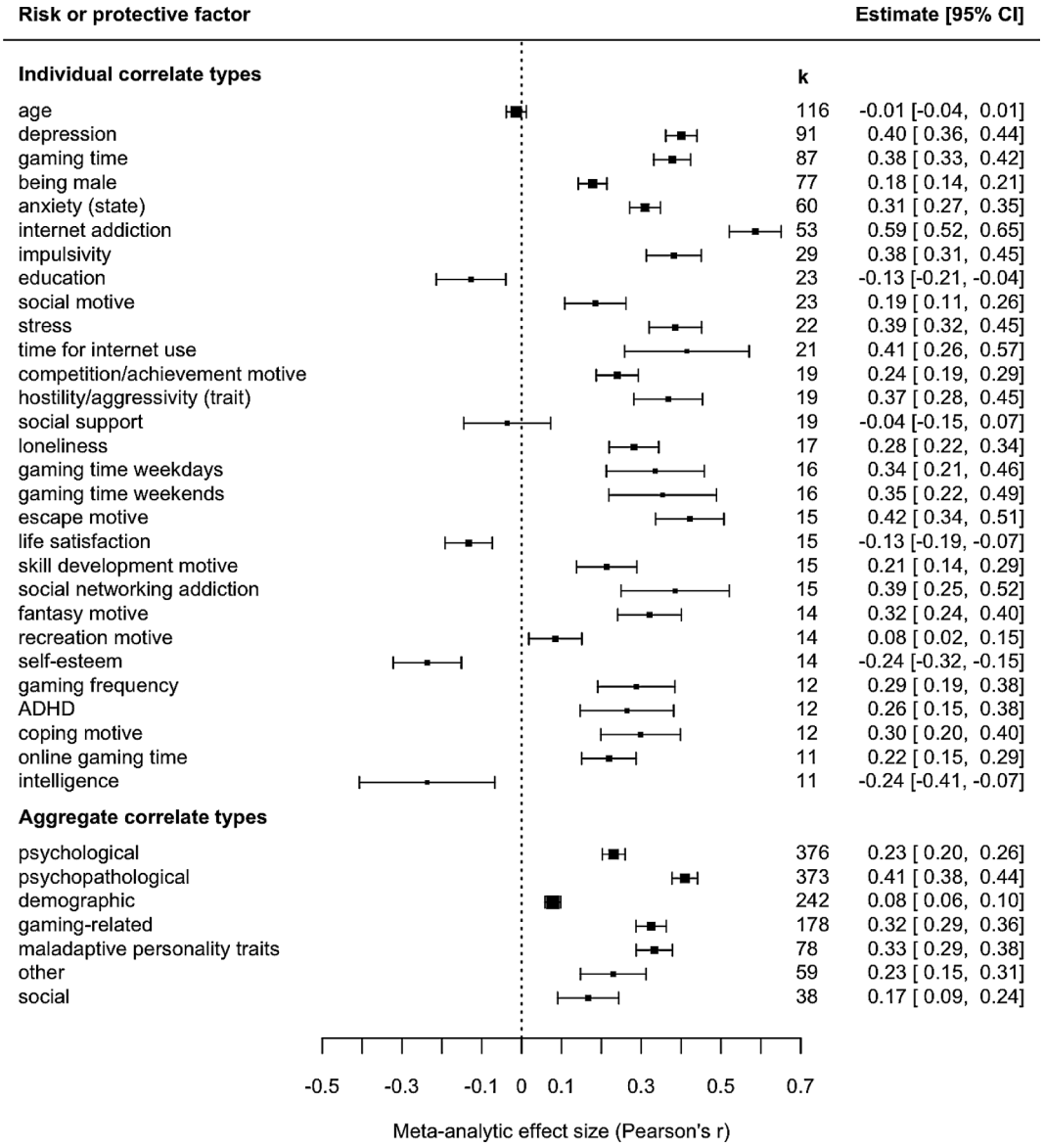

Fig. 2. Forest plot for individual risk/protective factors and aggregate correlate types *Note*. It is possible to visually judge the significance of the difference between any two risk/protective factors by the overlap of the 95 % confidence intervals. The point of marginal overlap corresponds to a difference test at the $\alpha = 0.005$ level. If the confidence intervals overlap by no more than half the length of one side of an interval, the meta-analytic estimates can be considered statistically significant at the $\alpha = 0.05$ level.

4.2. Individual risk/protective factors meta-analysis

Overall, the included studies reported effect size data on 414 distinct constructs. For 29 of those constructs, there were at least 10 effects, allowing for quantitative synthesis (see Table 1 & Fig. 2). We have not used any pre-existing classification of risk or protective factors in the search stage of the research allowing for the inclusion of any potential variable that could act as a risk or protective factor. What is considered a risk or protective factor in this study was objectively determined based on the nature of the relationship between the specific correlate and GD: positive relationship defining the risk factor and negative relationship defining the protective factor (e.g. correlation between age and GD was negative, thus age was considered a protective factor – the higher the age, the lower the GD). GD, as could be expected, was most closely associated with internet addiction and time for internet use. Apart from that, there were several other moderately strong risk factors of essentially the same magnitude ($r \sim 0.40$), namely depression, gaming time, impulsivity, stress, hostility/aggressivity as a trait, escape motive, or social networking addiction. On the other hand, we were able to identify only 4 empirically robust protective factors: self-esteem, intelligence, life satisfaction, and education, all having a markedly smaller effect size (by >2/3), compared to the strongest identified risk factors. Almost all synthesized sets of effects showed high relative heterogeneity ($I^2 > 90$ %), which should be considered when assessing the informativeness of the synthesized effect sizes. As a consequence, the 95 % prediction intervals for most risk factor estimates were relatively wide, with an interval width exceeding $r = 0.50$ (and a median width of $r = 0.68$). This suggests that the meta-analytic estimates are only modestly informative with regards to the expected range of true effects in similar studies.

4.3. Aggregate correlate types meta-analysis

In contrast to the preregistration, where we planned to aggregate the effects into only 3 types based on the King and Delfabbro (2019) - individual, external, and gaming-related factors, after the data extraction process we expanded the number of aggregated types into 7. The number of types or their meaning is not supported by any theory and is the result of our reasoning based on our understanding of the coded data. This taxonomy should only be viewed as a guidance to researchers and practitioners (e.g. differentiating non-pathological psychological factors from pathological ones; or psychological states from personality traits). The following seven types accomodated all 1586 effects: 1) psychological, 2) psychopathological, 3) demographic, 4) maladaptive personality traits, 5) social, 6) gaming-related factors, and 7) other. For further detail see our data (https://osf.io/aeym7), column Correlate type.

When comparing aggregated correlate types (see Table 2 & Fig. 2), these were found to significantly differ. This held even when controlling for design-related factors that are likely prognostic with respect to the effect sizes, namely mean age, gender, gaming style, and sample type, Wald's-type $F(4, 87) = 41.2$, $p < .001$. More specifically, the relatively strongest (in absolute values) appear to be the psychopathological, gaming-related factors, and maladaptive personality traits. The relatively weakest were the demographic and social factors.

4.4. Moderation effects

The effect of several synthesized risk/protective factors on GD was shown to be moderated by gender (% of females in the sample), mean age, sample type (0 = general population; 1 = gamers, including esports gamers), and criteria by which the presence of GD was assessed (0 = GD as a binary variable based on a symptom score cut-off or clinical assessment; 1 = GD as a continuous variable). Although - as preregistered - we coded also gaming style, game genre, and platform, underreporting of these study aspects (see Appendix E) precluded any reasonable, empirically sound moderation analysis. The results of the moderation analyses can be seen in the heatplot (Fig. 3). For each individual protective or risk factor (rows), we figure shows the effect of four moderators (columns). For a more natural interpretation, we report standardized coefficients β for continuous moderators (% female and Mean age) and unstandardized coefficients *B* for categorical moderators (Sample type, GD criteria). Apart from the estimates, each cell also reports the results of the *Q* test, the *Q*-statistic and *p*-value denoting the statistical significance of the given moderation effect.

As notable from the heatplot, the mean age and sample type tend to moderate the effect of different risk/protective factors in a rather idiosyncratic way. On the other hand, the female gender generally seems to attenuate the effect of risk factors and the assessment of GD using metric measures (vs binary assessment) yielded more detectable effects overall. We also aimed to examine moderation effects of gaming style, game genre, and platform, but underreporting of these study aspects (see Appendix E) precluded any reasonably sound moderation analysis.

4.5. Adjustment for publication bias and psychometric artifacts

The publication bias-corrected estimates (see Tables 1 & 2) proved to be remarkably similar to the unadjusted estimates for a large majority of correlates. Moreover, the estimates were mostly very stable across both different publication bias correction methods in particular, and varying assumptions about forms and severity of bias in general. Although 76 % of included effects were considered by original authors to be sufficiently focal to warrant a mention in the abstract, it can be concluded that the available evidence does not appear to be subject to selection by statistical significance and thus not impacted by publication bias in a meaningful way. Full results for these analyses are available in Appendix E.

Adjustment of the meta-analytic estimates for psychometric artifacts showed that the observed effects tended to be rather slightly overestimated due to unreliability in the measurement of the correlate and range restriction/enhancement. The estimates of only two correlates differed by a delta of > 0.1 in Pearson's *r* metric (Tables 1 & 2). When adjusting for indirect selection (i.e., bias due to conditioning on a collider), the adjusted estimates diverged by at least the same delta for

Table 2
Results for aggregated risk/protective factors.

Aggregated correlate type	*k*	Effect size *r* [95 % CI]	Odds ratio [95 % CI]	*Tau* [I^2]	*r* 95 % PI [LB, UB]	4-PSM estimate	PET-PEESE estimate	RoBMA estimate	Median power to detect $r = 0.10$
Psychological	512	0.23 [0.2, 0.26]	1.52 [1.44, 1.60]	0.19 [97 %]	[−0.15, 0.61]	0.21	0.22	0.11	0.59
Psychopathological	418	0.41 [0.38, 0.44]	2.10 [1.98, 2.22]	0.21 [98 %]	[0.00, 0.82]	0.39	0.36	0.44	0.39
Demographic	253	0.08 [0.06, 0.10]	1.15 [1.11, 1.19]	0.15 [96 %]	[−0.23, 0.38]	0.15	0.07	0.08	0.61
Gaming-related	218	0.32 [0.29, 0.36]	1.80 [1.68, 1.93]	0.21 [99 %]	[−0.1, 0.75]	0.30	0.28	0.09	0.65
Maladaptive personality traits	93	0.33 [0.29, 0.38]	1.83 [1.68, 1.99]	0.16 [95 %]	[0, 0.66]	0.33	0.26	0.40	0.39
Other	76	0.23 [0.15, 0.31]	1.52 [1.31, 1.76]	0.26 [99 %]	[−0.3, 0.76]	0.28	0.11	0.39	0.72
Social	44	0.17 [0.09, 0.24]	1.35 [1.18, 1.56]	0.18 [97 %]	[−0.22, 0.56]	0.24	0.21	0.29	0.59

Note. *k* = number of analyzed effects; *r* = Pearson's bivariate correlation; CI = confidence interval; PI = prediction interval; *Tau* = standard deviation of the true effect sizes; I^2 = relative heterogeneity; 4-PSM = 4-parameter selection model; PET-PEESE = regression-based publication bias-adjustment model. RoBMA = model-averaging Robust Bayesian meta-analysis.

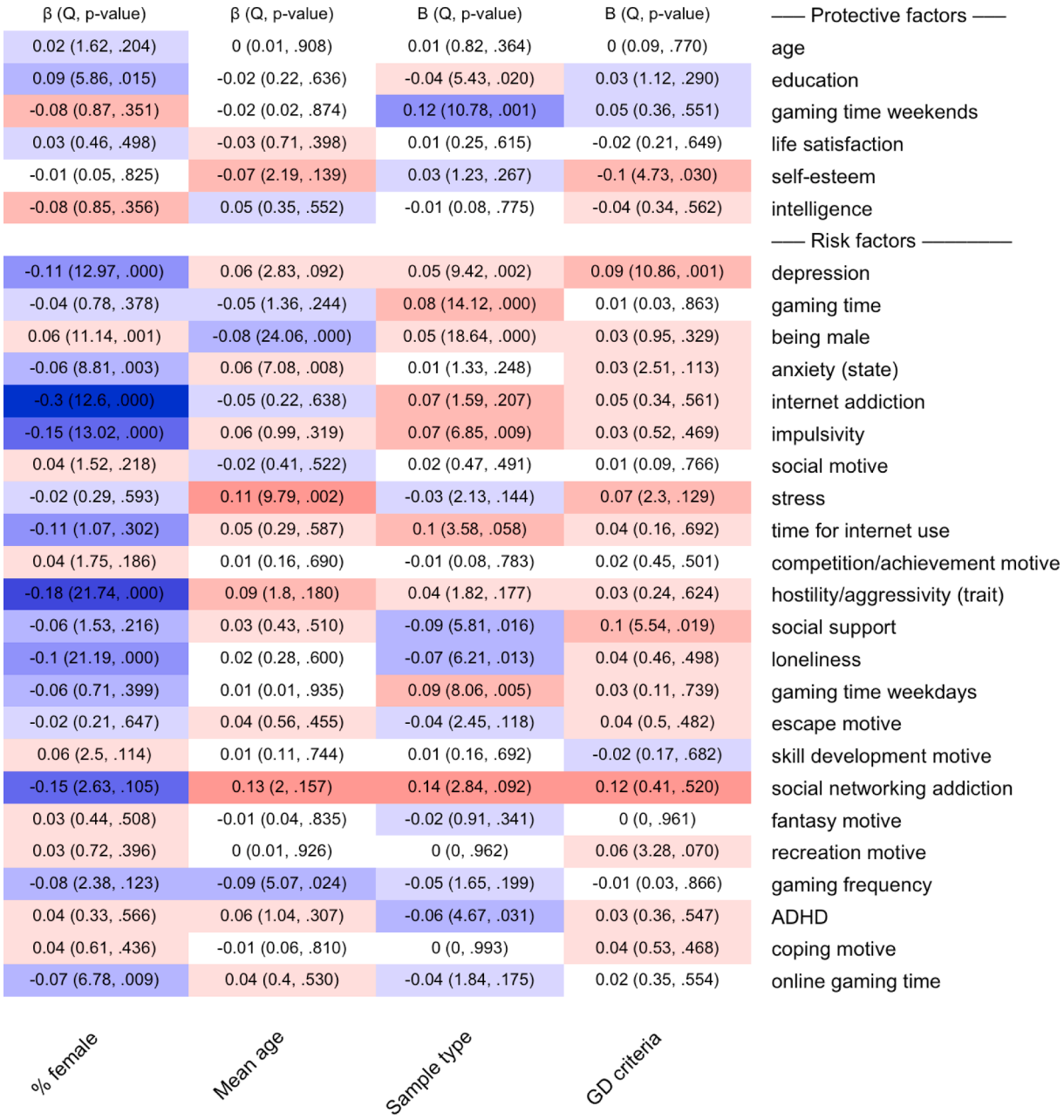

Fig. 3. Heatplot for moderation effects *Note*. Q = Q_M statistic with $df_Q = 1$ for all moderation analyses.

seven correlates (by > 0.2 for four correlates).

4.6. Quality of evidence assessment

Selective reporting did not appear to be prevalent in the set of included effects. The permutation-based *p*-curve analysis showed a right-skewed distribution of *p*-values, $z_{half} = -68.4$, $p_{half} < 0.001$. The median *p*-curve for the full literature can be seen in Fig. 4. As the *p*-curve had a steep right skew, we did not identify patterns consistent with a large prevalence of selective reporting in none of the risk/protective factors either.

We were able to identify 264 effects which could be checked for statistical inconsistencies (only 14 % of the papers reported results in APA format). Fourteen percent were associated with an inconsistent *p*-value. Out of those errors, 11 % had an effect on the resulting inference (significant/non-significant). Overall, 28 % of screened papers contained at least one reported inconsistency.

Regarding power, the research designs used in this literature provided 0.58 median power to detect a hypothetical small effect size ($r = 0.10$) and more than adequate power > 0.99 to detect an effect size of medium magnitude ($r = 0.30$). The values of median power to detect bias-adjusted estimates for individual correlates are reported in Table 1.

5. Discussion

The synthesis of 1586 effects provided several implications for both theory and intervention. Three prominent theoretical models were proposed for the conceptualization of GD, the cognitive-behavioral model (Dong & Potenza, 2014), the model of compensatory use (Kardefelt-Winther, 2014), and the I-PACE model (Brand et al., 2016). This meta-analysis aimed to synthesize and assess the robustness of evidence on which these theories build on. The cognitive-behavioral model (Dong & Potenza, 2014) hypothesizes the central role of reward-seeking behavior, reduced response-inhibition (cognitive-control), and stress

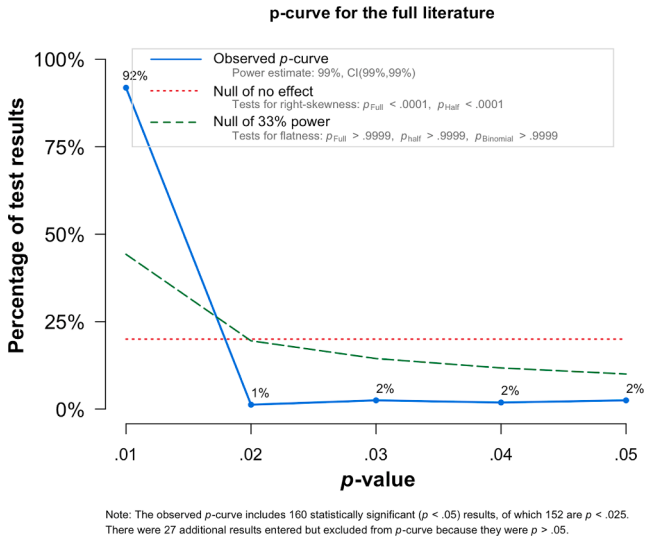

Fig. 4. P-curve for the full literature.

reduction tendencies to further drive the seeking motivation that leads to excessive gaming. We have found relatively robust evidence for the moderately strong association of GD with stress and impulsivity but there was an insufficient number of studies for reward sensitivity. Among all of the included types of motivation, GD was most strongly associated with the escape motive and coping that further supports the proposed theory. Motivational drives and stress reduction are central also to the model of compensatory use (Kardefelt-Winther, 2014). In the model, the internet or gaming act as a compensatory medium for solving real-life problems such as unsatisfactory social interactions, physical disabilities, or school- or work-related stress. Excessive gaming may be the result of different motivations for play, triggered by different real-life situations or problems. Loneliness may trigger social motives, anger may trigger achievement motives, or real life stress may trigger escape motives. Thus motivation originated in psychosocial problems should be more strongly connected to gaming disorder than healthy motivation. Our results support this assumption, where escape and coping motivation were the most strongly connected to GD, followed by fantasy, achievement/competition, and social motive, with recreation motivation being barely connected to GD.

The I-PACE model (Brand et al., 2016) assumes several groups of predisposing or vulnerability factors in the development of GD. The three main comorbid disorders, depression, (social) anxiety, and ADHD referred to in the psychopathology group were moderately-to-strongly related to GD. We also identified several of the personality group factors to be moderately-to-strongly tied to gaming disorder (impulsivity, low self-esteem, loneliness, perceived stress). On the other hand, the evidence for the lack of social support is weak at best. In addition to predisposing factors, the I-PACE model emphasizes also the role of affective and cognitive responses to different stimuli, which often take place under the influence of subjectively experienced stress. In our synthesis, stress was moderately related to GD. For the purpose of psychological intervention, the authors of the I-PACE theory describe moderator and mediator variables (e.g., coping styles and internet-related cognitive biases) for which the present meta-analytical synthesis, however, did not have sufficient data.

Given that people who play pathologically seek professional help only rarely (Konkolÿ Thege et al., 2015), more emphasis should be put on prevention rather than treatment. The identification of risk or protective factors is needed to selectively target the at-risk population or directly intervene at the level of these factors in the population at-large. Almost all of the previously reported selective prevention programs (King et al., 2018) used age for defining the target at- risk populations (elementary school students, adolescents, university students). However, our results do not support the view that younger populations and especially adolescents should be more vulnerable to gaming disorder. We concur with the statement of Bender et al. (2020, p. 557) that „when it comes to preventing GD, we believe that one of the most important preventive approaches is to inform parents, educators, and pediatricians about problematic gaming—specifically, about its risk factors and negative consequences.“ Bender et al. (2020) already highlighted the usefulness of several attention problems as potential risk factors – ADHD, emotion dysregulation, poor family functioning, and in-game needs satisfaction). The debate on risk factors can now be expanded by > 15 risk factors for which the present meta-analysis provides robust evidence. Our results (1) support the focus of prevention on people who play at high frequency and long duration, with comorbidities (ADHD, depression, anxiety), (2) question the predominant focus on males and persons with low social support (Konkolÿ Thege et al., 2015; Stevens, Delfabbro, et al., 2021), and (3) pinpoint several other factors that should be considered when developing tailored interventions, such as escape motivation, impulsivity or loneliness (and identify ones that appear irrelevant, like age). Prevention programs should also aim to increase protective factors (King et al., 2018), such as getting more satisfaction from life or developing healthy self-esteem.

We also examined the effects of several moderators. To highlight a few, the associations between all three gaming time risk factors and GD were stronger for the group of gamers than for the general population. However, the findings of King et al. (2017) question the role of the “need for increasing gaming time” within the Tolerance criterion. According to these authors, increasing gaming time should only be an indicator of increasing the difficulty in attaining goals that are just more time-consuming. There is also a substantially tighter link between GD and risk factors such as impulsivity, aggressivity, and internet addiction in males, compared to females. These results suggest that the proposed impulsive/aggressive subtype of GD players (Lee et al., 2017) may be more typical in males. Playstyle attributed to this subtype, i.e., playing to release aggressive impulses with a preference for sensation seeking, may be explained by reported gender differences in temperament (Else-Quest et al., 2006). Lastly, a stronger association of stress with GD in older players may be related to age differences in preferences for coping styles or escape motivation – an indication that needs further research.

5.1. Limitations

This meta-analysis has several inherent limitations. First, the identified links between risk/protective factors and GD are (though necessary) not sufficient to back a causal interpretation. They should purely serve as a vehicle aiding in the calibration of clinical prediction models, narrowing down the targeting of large-scale prevention programs, and tentatively informing GD theory building. Second, we were able to identify results on 414 distinct risk factors in the literature, yet for only 7 % the evidence was robust enough to warrant an informative synthesis. Interested reader can lower/increase the threshold for the minimum number of effects for carrying out a synthesis. We suggest though that the strength of resulting inferences should be proportional to the strength and breadth of underlying evidence. Third, as inherent in synthesizing bivariate predictive associations, our synthesis could not accommodate results from multivariate analyses (covariates vary widely) or latent variable models (sum score-based and latent associations would not be comparable). Fourth, we applied a relatively conservative approach and excluded all evidence that was based on an operationalization of GD that was not compliant with DSM-5 or ICD-11. That anchors the psychological validity of GD across studies and risk factor effects. On the other hand, this is at the cost of missing evidence on risk factors of constructs related to GD, like problematic internet use, pre-DSM-5 concepts of gaming addiction, etc. This evidence can potentially be informative for the understanding of GD too. Fourth, we synthesized only evidence from available English papers. We did not contact the authors or groups asking for file-drawered evidence.

6. Conclusion

The present synthesis provides comprehensive evidence on predictive associations. Methodologically-wise, the present literature-wide examination of the quality of underlying evidence suggests that the impact of various examined biasing factors on the reported results seems to be relatively modest. Researchers should also pay attention to the congruence between the conceptual definition of the construct and its operationalization. As was often the case in our systematic literature search, using the Internet Addiction Test (Young, 1998) for the measurement of GD (as defined by the DSM 5) should be viewed as psychometrically problematic. Future research should also improve in terms of transparency (e.g., sharing data and materials) and reporting practices (e.g., reporting psychometric details, a more detailed operationalization of GD when no standard measure was used, demographic characteristics of the sample, and substantive aspects like gaming style, game genre, or gaming platform). In general, the advancement in the understanding of GD, requires a clearer empirical demarcation from related yet distinct constructs like internet addiction or excessive gaming time, a more coherent operationalization across studies, and tighter link with progressively more formal theory.

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CRediT authorship contribution statement

Ivan Ropovik: Conceptualization, Project administration, Methodology, Investigation, Formal analysis, Data curation, Writing – original draft, Writing – review & editing. **Marcel Martončik:** Conceptualization, Project administration, Investigation, Data curation, Methodology, Writing – original draft, Writing – review & editing. **Peter Babinčák:** Investigation, Resources, Writing – review & editing. **Gabriel Baník:** Investigation, Methodology, Writing – review & editing. **Lenka Vargová:** Investigation, Resources, Writing – review & editing. **Matúš Adamkovič:** Conceptualization, Methodology, Investigation, Writing – original draft, Writing – review & editing.

Declaration of Competing Interest

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

Data availability

Appendices, data, R code, and full analytic outputs are freely available at https://osf.io/9mzgr/.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.addbeh.2022.107590.

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