Risk and protective factors for (internet) gaming disorder: A meta-analysis

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Abstract

Background: This large-scale meta-analysis aimed to provide the most comprehensive synthesis to date of the available evidence on risk and protective factors for (internet) gaming disorder (as defined in the DSM-5 or ICD-11) across all studied populations.

Methods: The risk/protective factors included demographic characteristics, psychological, psychopathological, social, and gaming-related factors. In total, we have included 1578 effects from 250 different studies, summarizing data from 206690 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.

Results: The available evidence suggests the existence of numerous moderately strong and highly heterogeneous risk factors but only a few empirically robust protective factors (all having markedly smaller effect sizes).

Conclusions: 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.

Registration number: Preregistered at PROSPERO under ID:CRD42020187776.

Keywords: gaming disorder, internet gaming disorder, risk factor, protective factor, meta-analysis

Risk and protective factors for (internet) gaming disorder: A meta-analysis

Gaming disorder is the first official mental health condition directly associated with the use of digital technology. A global pooled prevalence for gaming disorder of approx. 3% estimated by two recent meta-analyses (Stevens et al., 2020; Kim 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 (WHO, 2019) were officially introduced, a large amount of evidence in a form of associations between IGD or GD and different psychological, socio-economic, gaming or other phenomena, has been accumulated (Kuss et al., 2018; Paulus et al., 2018; Burleigh et al., 2019). Inclusion of GD into the 11th revision of the International Classification of Diseases (WHO, 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). This emphasizes the importance of synthesizing evidence on risk or protective factors of (internet) gaming disorder which could play a central role in the development of theoretical models and preventive programs. A 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), 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 BDI score of 10.31 in individuals with significant IGD symptoms. 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.

Present study

Being first recognized as a mental disorder relatively recently (WHO, 2019), gaming disorder was derived from substance use disorder or pathological gambling (see Aarseth et al., 2017) and is still considered largely atheoretical (Kardefelt-Winther et al., 2017). A better understanding of (internet) gaming disorder and the development of more effective preventive/intervention programs thus first requires rigorous mapping of evidence on causally

relevant psychological but also non-psychological factors, which the present meta-analysis aimed to deliver. 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.

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. Data, R code, and analytical outputs can be found at <https://osf.io/9mzgr/>.

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 and was limited to primary empirical studies written in English and published from 2013. 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.

Inclusion criteria

(1) Population: We included studies on all populations, including the general population, clinical sample, or groups of specific gamers. (2) Correlates/Exposures: The study had to report at least one zero-order association between GD and any specific risk or protective factor, e.g., individual, external, or gaming-related factor (see King & Delfabbro, 2019). (3) Outcome: The study

had to assess the presence and/or severity of gaming disorder. We included only studies where the definition and measurement of GD complied with the DSM-5 or ICD-11.

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 Figure 1).

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.

Analysis

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 more than 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 (Pustejovsky & Tipton, 2020). To test for equality of effect sizes across the levels of the moderators studied, we used the robust HTZ-type Wald test (Pustejovsky & Tipton, 2020). The p-values for individual contrasts were adjusted using Holm’s method.

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.

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.

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 by Hunter & Schmidt (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.

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) (McShane et al., 2016; Bartoš et al., 2021) 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 (see Ropovik, Adamkovic, & Greger, 2021). A more elaborate description and specification of the models can be seen in Appendix A.

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, decline bias (see Fanelli, Costas, & Ioannidis, 2017), 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.

Quality of evidence assessment

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

Assessment of evidential value

First, we estimated the evidential value of the available studies using a permutation-based p -curve analysis (Simonsohn, Nelson, & Simmons, 2014). This analysis aimed to provide insight into the degree of selective reporting in the literature on different risk factors. 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).

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.

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.

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.

Results

A complete breakdown of the study selection process is shown in the PRISMA flowchart, Figure 1. We were able to recompute the effect sizes for 1578 effects from 250 studies (reported in 208 papers). These effects summarized data from 206690 independent participants, with a median N across all the eligible effect sizes equal to 467. 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 esports (42%), 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 (13%), 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 (20%), 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.

Figure 1*PRISMA Flowchart***Identification** **Results****Individual risk/protective factors**

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 & Figure 2). 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 ($r \sim .40$), 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 more than 2/3), compared to the strongest identified risk factors. Almost all synthesized sets of effects showed high relative heterogeneity ($I^2 > 90\%$).

Figure 2

Forest plot for individual risk/protective factors and aggregate correlate types

Aggregate correlate types

When comparing aggregated correlate types (see Table 2 & Figure 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 $F(4, 86.5) = 41.0, p < .001$. 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.

Figure 3

Heatplot for moderation effects

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). As notable from the heatmap (Figure 3), 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, while 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.

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 $>.1$ 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 seven correlates (by $>.2$ for four correlates).

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, $z_{half} = -67.9$, $p_{half} < .001$. The median p -curve for the full literature can be seen in Figure 4. 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 .58 median power to detect a hypothetical small effect size ($r = .10$) and more than adequate power $>.99$ to detect an effect size of medium magnitude ($r = .30$). The values of median power to detect bias-adjusted estimates for individual correlates are reported in Table 1.

Figure 4

p-curve for the full literature

Note: The observed p -curve includes 156 statistically significant ($p < .05$) results, of which 146 are $p < .025$. There were 22 additional results entered but excluded from p -curve because they were $p > .05$.

Table 1
Results for individual risk/protective factors

Risk/protective factor	<i>k</i>	Effect size <i>r</i> [95% CI]	<i>Tau</i> [<i>I</i> ²]	4-PSM estimate	PET-PEESE estimate	Measurement artifacts- corrected <i>r</i>	Median power to detect <i>r</i> = .10
Age	118	-0.01 [-0.04, 0.01]	0.12 [92%]	-0.01	0.00	0.03	0.58
Depression	93	0.40 [0.36, 0.44]	0.18 [97%]	0.37	0.32	0.34	0.39
Gaming time	84	0.38 [0.33, 0.43]	0.20 [99%]	0.35	0.32	0.32	0.62
Being male	78	0.18 [0.14, 0.21]	0.15 [97%]	0.20	0.18	0.14	0.82
Anxiety (state)	63	0.31 [0.27, 0.35]	0.13 [94%]	0.29	0.30	0.34	0.41
Internet addiction	53	0.59 [0.52, 0.65]	0.21 [99%]	0.55	0.50	0.29	0.22
Impulsivity	29	0.38 [0.31, 0.45]	0.17 [95%]	0.31	0.27	0.34	0.26
Education	24	-0.13 [-0.21, -0.04]	0.17 [92%]	-0.12	-0.05	0.04	0.12
Social motive	24	0.19 [0.11, 0.26]	0.15 [97%]	0.18	0.19	0.20	0.64
Stress	23	0.39 [0.32, 0.45]	0.14 [93%]	0.41	0.43	0.38	0.41
Time for internet use	21	0.41 [0.26, 0.57]	0.29 [99%]	0.20	0.31	0.45	0.57
Competition/achievement motive	20	0.24 [0.19, 0.29]	0.10 [94%]	0.24	0.22	0.28	0.75
Hostility/aggressivity (trait)	19	0.37 [0.28, 0.45]	0.16 [94%]		0.18	0.22	0.52
Social support	18	-0.05 [-0.16, 0.07]	0.20 [97%]	0.04	-0.04	0.11	0.21
Gaming time weekdays	16	0.34 [0.21, 0.46]	0.23 [97%]	0.18	0.06	0.21	0.56
Gaming time weekends	16	0.35 [0.22, 0.49]	0.25 [98%]	0.34	0.06	0.21	0.56
Loneliness	16	0.27 [0.21, 0.33]	0.11 [94%]	0.26	0.26	0.29	0.77
Escape motive	15	0.42 [0.34, 0.51]	0.15 [98%]	0.44	0.39	0.46	0.64
Life satisfaction	15	-0.13 [-0.19, -0.07]	0.10 [91%]	-0.15	-0.08	0.09	0.75
Skill development motive	15	0.20 [0.14, 0.29]	0.13 [96%]	0.23	0.16	0.20	0.64
Social networking addiction	15	0.39 [0.25, 0.52]	0.24 [99%]		0.62	0.51	0.72
Fantasy motive	14	0.32 [0.24, 0.4]	0.13 [96%]	0.35	0.30	0.32	0.71
Recreation motive	14	0.08 [0.02, 0.15]	0.11 [95%]	0.07	0.05	0.07	0.80
Self-esteem	14	-0.24 [-0.32, -0.15]	0.13 [90%]	-0.25	-0.24	0.21	0.35
Gaming frequency	13	0.29 [0.19, 0.38]	0.15 [97%]	0.29	0.31	0.21	0.70
ADHD	12	0.26 [0.15, 0.38]	0.16 [95%]		0.33	0.36	0.35
Coping motive	12	0.30 [0.20, 0.40]	0.15 [98%]	0.31	0.23	0.30	0.89
Online gaming time	12	0.22 [0.15, 0.29]	0.08 [91%]	0.22	0.20	0.19	0.74
Intelligence	11	-0.24 [-0.41, -0.07]	0.21 [72%]		-0.12	0.24	0.10
Aggregated correlate type	<i>k</i>	Effect size <i>r</i> [95% CI]	<i>Tau</i> [<i>I</i> ²]	4-PSM estimate	PET-PEESE estimate	RoBMA estimate	Median power to detect <i>r</i> = .10
Psychological	510	0.23 [0.2, 0.26]	0.19 [97%]	0.21	0.22	0.11	0.59
Psychopathological	416	0.41 [0.38, 0.44]	0.21 [98%]	0.39	0.36	0.43	0.41
Demographic	253	0.08 [0.06, 0.10]	0.15 [96%]	0.15	0.07	0.08	0.61
Gaming-related	215	0.32 [0.29, 0.36]	0.21 [99%]	0.30	0.28	0.07	0.65
Maladaptive personality traits	93	0.33 [0.29, 0.38]	0.16 [95%]	0.33	0.26	0.4	0.39
Other	76	0.23 [0.15, 0.31]	0.26 [99%]	0.28	0.11	0.39	0.72
Social	43	0.15 [0.08, 0.23]	0.18 [96%]	0.22	0.19	0.26	0.59

Note. *k* = number of analyzed effects; *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.

Discussion

The synthesis of 1578 effects provided several implications for both theory and intervention.

Theoretical implications

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. Our results add support to the proposed role of stress as a driving force in the cognitive-behavioral model or to the role of specific motivation, especially the escape motivation, being central to the model of compensatory use and a component in the I-PACE model. The I-PACE model assumes several predisposing factors, many of which we identified to be moderately-to-strongly tied to gaming disorder (impulsivity, low self-esteem, loneliness, perceived stress).

Implications for prevention strategies

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 (King et al., 2017) or directly intervene at the level of these factors in population at-large (Bender et al., 2020). 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 (King et al., 2017; Stevens 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 (Bender et al., 2020), such as getting more satisfaction from life or developing healthy self-esteem.

Quality of the evidence

The impact of various examined biasing factors on the available evidence seems to be relatively modest. Advancement in the understanding of GD, however, requires a clearer empirical demarcation from related yet distinct constructs like internet addiction or excessive gaming time,

and improved reporting of psychometric details, aspects like gaming style, game genre, or gaming platform.

Conclusions

Synthesizing empirical evidence published from 2013 to 2020 for (internet) gaming disorder, we have found robust evidence for moderate to strong positive relationships with internet addiction, depression, anxiety, and gaming time, weak relationship with being male, and almost zero relationship with age. Only education, life satisfaction, self-esteem, and intelligence were identified as protective factors.

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Data accessibility

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

Authors' contributions

Ivan Ropovik: Conceptualization, Project administration, Methodology, Investigation, Formal analysis, Data curation, Writing - Original Draft, Writing - Review & Editing. **Marcel Martončík:** 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. IR and MM are joint first authors of this paper.

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