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Article

Common Genetic Influence on the Relationship Between Gaming Addiction and Attention Deficit Hyperactivity Disorder in Young Adults: A Twin Study

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Abstract

Although the relationship between gaming addiction (GA) and attention deficit hyperactivity disorder (ADHD) is well established, the causal mechanism of this relationship remains ambiguous. We aimed to investigate whether common genetic and/or environmental factors explain the GA-ADHD relationship. We recruited 1413 South Korean adult twins (837 monozygotic [MZ], 326 same-sex dizygotic [DZ], and 250 opposite-sex DZ twins; mean age = 23.1 ± 2.8 years) who completed an online survey on GA and related traits. Correlational analysis and bivariate model-fitting analysis were conducted. Phenotypic correlation between GA and ADHD in the present sample was 0.55 (95% CI [0.51, 0.59]). Bivariate model-fitting analysis revealed that genetic variances were 69% (95% CI [64%, 73%]) and 68% (95% CI [63%, 72%]) for ADHD and GA respectively. The remaining variances (ADHD: 31%; GA: 32%) were associated with nonshared environmental variances, including measurement error. Genetic and nonshared environmental correlations between ADHD and GA were 0.68 (95% CI [0.62, 0.74]) and 0.22 (95% CI [0.13, 0.30]) respectively, which indicates that shared genes can explain 82% of the phenotypic correlation between ADHD and GA. Our study demonstrated that the ADHD-GA association was largely due to shared genetic vulnerability.

Keywords: Gaming addiction; Attention deficit hyperactivity disorder; Twin study; Genetic correlation

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Gaming addiction (GA) is an increasing public concern for its negative effects on health. The prevalence of internet gaming disorder (IGD) ranges from 0.7% to 25.5% worldwide and is generally high in Asian countries (Mihara & Higuchi, 2017). Although debate is ongoing on the nosology of IGD (Fergusson et al., 2011), it is now included in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013) Appendix as a condition needing further research. The present study defined GA as preoccupation with gaming, tolerance, withdrawal, and inability to control participation in gaming despite problems. This definition followed the description of IGD in the DSM-5. In the literature, however, GA has been used interchangeably with internet and other technology-related disorders such as pathological video gaming, internet addiction, problematic internet use, compulsive internet use (King et al., 2013). The mixed use of these terms may be due to high correlations among them (Andreassen et al., 2016; Chiu et al., 2013), but it also reflects a lack of consistent definitions among researchers (Dong & Potenza, 2014).

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Prior research demonstrated a strong relationship between GA and attention deficit hyperactivity disorder (ADHD; Andreassen et al., 2016; Kuss et al., 2014). A meta-analysis showed that patients with internet addiction were 2.51 times more likely to be diagnosed with ADHD compared with patients without internet addiction (Wang et al., 2017). To explain the causal direction of the ADHD-GA relationship, Ko et al. (2009) conducted a 2-year longitudinal study on more than 2000 adolescents and found that ADHD was the most significant predictor of the development of internet addiction. Those with attention problem and impulsivity tend to pursue immediate reward (Barkley, 1997), which increases the risk for developing GA. Also, a lack of self-control in those with ADHD may cause them to experience difficulty in controlling gaming, which may make them progress to GA (Ko et al., 2009). However, other studies suggested that gaming preceded the diagnosis of ADHD. In a longitudinal study on adolescents without ADHD symptoms, Ra et al. (2018) found that the frequent use of digital media was associated with later development of ADHD symptoms. Additionally, ADHD children with pathological gaming behavior exhibited more severe ADHD symptoms than those without (Shuai et al., 2021). Adolescents who played games reported an increase in attention problems after playing games (Gentile et al., 2012). These studies reported that gaming may, at least, exacerbate ADHD symptoms. Digital games are fast-paced and frequently offer immediate rewards. People can adapt to the intense cognitive stimulation in games through elevated levels of arousal; thus,

gaming may lead people to perceive low-stimuli environments as deprivation, which fosters restlessness or inattention, a key feature of ADHD (Lang et al., 2000).

Although the direction of causation between ADHD and GA remains unclear, Hygen et al. (2020) showed that a common underlying factor caused the correlations between IGD and mental disorders (e.g., ADHD, depression, and anxiety). However, the authors failed to address the common factor because they employed nontwins in their longitudinal cross-lagged panel analysis. Interestingly, pharmacological studies found that medicine to treat ADHD symptoms was effective in mitigating GA (Han et al., 2009; Park et al., 2016), which indicates that common biological mechanisms may underlie the GA-ADHD association. Using summary statistics from genomewide association studies (GWAS), Vink et al. (2021) found a significant genetic correlation (r = .53) between ADHD and nicotine dependence. Similarly, Koller et al. (2024) found several common genetic variants between ADHD and substance use disorders (SUDs), confirming genetic contribution to the comorbidity. Given the evidence of the strong relationship between SUDs and GA (Ko et al., 2009), common genetic factors may mediate the relationship between GA and ADHD.

Twin studies are useful for determining the common genetic and environmental etiology of comorbid disorders such as ADHD and GA. Twin studies of GA are scanty. Nonetheless, extant twin studies consistently demonstrated that internet-related disorders were strongly heritable in adolescents and young adults, with heritability estimates ranging from 48% to 66% (Deryakulu & Ursavas, 2014; Li et al., 2014; Vink et al., 2016). These studies found that shared environmental influences were not significant, and that environmental influences were those not shared by family members (hereafter, nonshared environmental influences). In support of twin studies, GWAS identified 72 single nucleotide polymorphisms associated with internet addiction disorder (Haghighatfard et al., 2023).

In contrast to GA, numerous twin studies and GWAS have been conducted to reveal genetic influences on ADHD. A review of twin studies of ADHD suggested a mean heritability of 74% (Faraone & Larsson, 2019). In addition, recent GWAS meta-analyses discovered multiple gene variants associated with ADHD (Demontis et al., 2023). Given the evidence of the strong genetic influences on GA and ADHD, the present study sought to examine common genetic factors that contribute to the GA-ADHD relationship in a sample of South Korean adult twins. To the best of our knowledge, this is the first twin study to report the genetic relationship between ADHD and GA.

Material and Methods

Sample

Twins were invited through a survey link posted on online communities in universities and the websites of the Kookmin Twin Research Institute and twin clubs throughout South Korea in 2022−2023. The survey included informed consent form, ADHD, GA and zygosity questionnaires, and gaming-related questions. A mobile gift coupon was sent to participants who completed the survey. The survey was originally developed for adolescents and young adults. However, only adult twin participants (age: 20 to 35 years old) were included for the present study. The zygosity questionnaire, which was adopted from Ooki et al. (1993), included questions on the physical similarity of twins, frequency of confusion about twins, and the self-perception of zygosity. Eight twin pairs were removed from data analysis because their zygosity was ambiguous. The final sample included 1413 twins (mean age: 23.1 ± 2.8 years), consisting of 837 monozygotic (MZ) twins (416 complete pairs and 5 cotwin missing twins), 326 same-sex dizygotic (DZ) twins (162 complete pairs and 2 cotwin missing twins), and 250 opposite-sex DZ twins (125 complete pairs). Females exceeded males (64% vs. 36%), partially because military service is compulsory for South Korean young adult men. Moreover, females tend to participate in online surveys more frequently than males do (Wu et al., 2022). MZ twins outnumbered DZ twins (59% vs. 41%), which likely reflected the twin birth rates in South Korea in the 1990s and early 2000s (Hur, 2021).

Measures

Gaming addiction (GA). The Korean Game Addiction scale (Choi et al., 2013) was used to assess GA. The scale contains 20 items measuring tolerance, withdrawal, compulsive use of game, impairment of self-control, impairment of daily activities, excessive time consumption for gaming, and continued gaming despite problems in the past year. A sample item includes, 'I tried to reduce or stop playing the game several times but failed'. The items were rated using a 4-point Likert-type scale (0: *not at all true*; 3: *almost always true*). Scores were calculated by summing the responses, with high scores indicating severe GA. The Cronbach's alpha reliability of the scale in the present sample was .96.

Attention Deficit Hyperactivity Disorder (ADHD). The Korean version of the Conners' Adult ADHD Rating Scale (K-CAARS; E. J. Kim, 2003) was used to assess ADHD. The K-CAARS includes 26 items measuring inattention, hyperactivity, impulsivity, and self-concept problems. The items were rated using a 4-point Likert-type scale (0: *not at all true*; 3: *almost always true*). Scores were calculated by summing the responses, with high scores indicating severe symptoms. The Cronbach's alpha reliability in the present sample was .94.

Statistical Analyses

We first computed MZ and DZ twin correlations for GA and ADHD, phenotypic correlation between GA and ADHD, and cross-twin cross-trait correlations between GA and ADHD for MZ and DZ twins. Then, we conducted bivariate Cholesky model-fitting analysis to estimate genetic and environmental influences on GA and ADHD, genetic and environmental correlations between GA and ADHD, and bivariate heritability and environmentality.

The twin method involves the decomposition of phenotypic variances and covariances into additive genetic (A), shared environmental (C), and nonshared environmental variances, including measurement error (E). MZ twins share 100% of their genes, while DZ twins share, on average, 50%. Thus, if MZ twin correlation is greater than DZ twin correlation, genetic effects are presumed existing. If DZ twin correlation is greater than half the MZ twin correlation, shared-environmental effects are likely present. MZ twin correlation less than 1 indicates non-shared environmental influences. Cross-twin cross-trait correlation involves correlating the score of twin 1 for one trait (e.g., ADHD) with the score of twin 2 for another trait (e.g., GA). Greater MZ than DZ cross-twin cross-trait correlation indicates genetic contribution to the covariance between two variables. DZ cross-twin cross-trait correlation greater than half the corresponding MZ correlation indicates shared environmental contribution to covariance.

The Cholesky model partitions the covariance of ADHD and GA into additive genetic (A1 and A2), and shared (C1 and C2), and

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Table 1. Means and standard deviations of raw scores of attention deficit hyperactivity disorder (ADHD) and gaming addiction (GA) by sex and zygosity

Trait	Men	Women	MZ	DZ
ADHD[a]	18.5 (± 13.1)	21.7 (± 14.7)	21.4 (± 14.9)	19.2 (± 13.1)
GA[b,c]	8.3 (± 9.7)	6.5 (± 10.8)	8.5 (± 11.5)	5.2 (± 8.3)

Note: *SD*s are enclosed in parenthesis. MZ, monozygotic twins; DZ, dizygotic twins. DZ twins include 250 opposite-sex twins.
[a]Significant mean and variance differences between sexes and between the two zygosity groups at $p < .01$.
[b]Significant mean difference between sexes and between the two zygosity groups at $p < .01$.
[c]Significant variance difference between the two zygosity groups at $p < .01$.

nonshared environmental (E1 and E2) variance components. This model provides genetic, shared environmental, and nonshared environmental influences on each phenotype as well as genetic (r_a), shared environmental (r_c), and nonshared environmental (r_e) correlations between two variables. These correlations indicate the extent to which the same set of genes or shared and non-shared environments influence two variables. Using these correlations, bivariate heritability (the proportion of phenotypic correlation due to additive genetic factors (i.e., $[\sqrt{a1} \times r_a \times \sqrt{a2}]$/phenotypic correlation), and bivariate environmentality (the proportion of phenotypic correlation due to environmental factors (i.e., $[\sqrt{e1} \times r_e \times \sqrt{e2}]$/phenotypic correlation) can be calculated.

Mx (Neale et al., 2003) was used to conduct correlation and model-fitting analysis. Mx produces −2 log likelihood (−2LL). The difference between −2LL of two nested models is distributed as a chi-square (χ^2) with degrees of freedom (*df*) equivalent to the difference in the number of parameters between the two models. The relative goodness-of-fit for nested models was compared with that of the full model, including additive genetic, shared environmental, and nonshared environmental variances and their covariances, to determine the best-fitting model for the data. Parameter estimates were then calculated with 95% confidence intervals using the maximum likelihood method. The best-fitting model was selected based on the log-likelihood ratio test and the Akaike's information criteria (AIC). Models exhibiting lower AIC were considered more parsimonious and were thus preferred (Akaike, 1987).

Results

Descriptive Statistics

Table 1 presents means and standard deviations of raw scores of ADHD and GA by sex and zygosity. Age was not significantly correlated with ADHD or GA ($r < .05$). Women had significantly higher mean and variance of ADHD than men. This was consistent with other South Korean university samples (e.g., E. J. Kim, 2003; H. Y. Kim et al., 2005) but not with other ethnic groups where no significant sex difference was found (e.g., Stibbe et al., 2020). Although sex difference in variance of GA was not significant, men had significantly higher mean than women, consistent with the literature of GA (Mihara & Higuchi, 2017).

Skewness indices were 0.6 for ADHD and 1.7 for GA. Thus, the scores of GA were log transformed, which resulted in a skewness of 0.4. To increase sample size and, thus, statistical power, we performed correlation and model-fitting analyses using data combined across males and females. Because twins are the same age and gender (except opposite-sex pairs), failing to correct for the effects of age and sex when they exist will lead to biased estimation of twin correlation and model parameters (McGue & Bouchard, 1984). Prior to correlation and model-fitting analyses, raw scores of ADHD and log-transformed scores of GA were corrected for sex, age, age^2, and age × sex effects using multiple regression analysis. The standardized residuals were used in subsequent analysis. Variances of standardized residual scores of ADHD and GA were not significantly different between two zygosity groups.

Correlational Analysis

Figure 1 depicts the results of correlational analysis. MZ and DZ twin correlations were 0.70 (95% CI [0.65, 0.75]) and 0.35 (95% CI [0.24, 0.44]), respectively, for GA and 0.71 (95% CI [0.66, 0.76]) and 0.39 (95% CI [0.29, 0.49]), respectively, for ADHD. For both traits, MZ twin correlations were significantly higher than DZ twin correlations, which indicated that genetic influences were large, whereas shared environmental influences were negligible. The phenotypic correlation between GA and ADHD was substantial (*r*: 0.55, 95% CI [0.51, 0.59]), which confirmed the significant relationship between the two traits. Cross-twin cross-trait correlations were 0.50 (95% CI [0.42, 0.57]) for MZ and 0.27 (95% CI [0.16, 0.37]) for DZ twins, which suggested that common genetic factors mediated the relationship between GA and ADHD.

Bivariate Cholesky Model-Fitting Analysis

As means of ADHD and GA were significantly different between MZ and DZ twins, zygosity-specific means were implemented in the Cholesky model. Table 2 presents the results of model-fitting analysis. Although omitting all genetic variances/covariances yielded a significant change in chi-square (model 1: $\Delta\chi^2$: 60.1, Δdf 3, $p < .00$), omitting all shared environmental variances/covariances did not (model 2: $\Delta\chi^2$: 5.8, Δdf 3, $p = .12$). These results indicated that shared environmental variances/covariances for ADHD and GA were negligible, consistent with the results of correlational analysis. We individually omitted genetic covariance and nonshared environmental covariance from model 2 (models 3 and 4). The resulting chi-square differences were significant for both models, which indicates that common genetic and nonshared environmental influences contribute to the relationship between ADHD and GA.

Figure 2 shows the path coefficients in the best-fitting bivariate model, which should be squared to estimate additive genetic and nonshared environmental influences. Additive genetic variances were 69% (95% CI [64%, 73%]) for ADHD and 68% (95% CI [63%, 72%]) for GA. The remaining variances (ADHD = 31%; GA = 32%) were attributable to nonshared environmental variances, including measurement error. Genetic and nonshared environmental correlations between ADHD and GA reached .68 (95% CI [.62, .74]) and 0.22 (95% CI [.13, .30]), respectively. Bivariate heritability for ADHD and GA was 82% $[(\sqrt{.68} \times \sqrt{.69} \times .68)/.55]$, which indicates that shared genes can explain 82% of the correlation between ADHD and GA. Bivariate environmentality for ADHD and GA was 18% $[(\sqrt{.31} \times \sqrt{.32} \times .22)/.55]$, which suggests that 18% of the correlation was due to common nonshared environmental factors.

We also found genetic and nonshared environmental variances unique to GA. Of 68% of genetic variance for GA, 31% were variance shared with ADHD, and the remaining 37% were the genetic variance unique to GA. Of 32% of the nonshared environmental variance for GA, only 1% were variance shared with ADHD, and the remaining 31% were the nonshared environmental variance unique to GA.

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Table 2. Results of bivariate Cholesky decomposition model-fitting analysis for gaming addiction (GA) and attention deficit hyperactivity disorder (ADHD)

		Goodness-of-fit indices					
Model	Description	−2LL	*df*	AIC	$\Delta\chi^2$	Δdf	*p*
Full	Full model	6947.3	2815	1317.3			
1	Drop all genetic variances/covariances	7007.4	2818	1371.4	60.1	3	.00
2	**Drop all shared environmental variances/covariances**	**6953.1**	**2818**	**1317.1**	**5.8**	**3**	**.12**
3	Drop all shared environmental variances/covariances; genetic covariance	7209.0	2819	1571.0	261.7	4	.00
4	Drop all shared environmental variances/covariances; non-shared environmental covariance	6976.9	2819	1338.9	29.6	4	.00

Note: -2LL, −2 log likelihood; AIC, Akaike information criterion; *df*, degrees of freedom. The best-fitting model is indicated in bold.

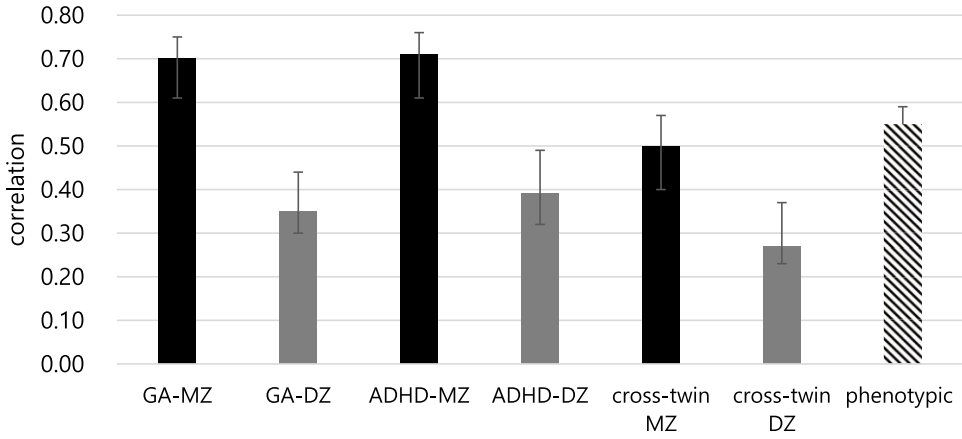

Figure 1. Results of correlational analysis. Note: GA, gaming addiction; ADHD, attention deficit hyperactivity disorder; MZ, monozygotic twins; DZ, dizygotic twins. Error bars represent 95% confidence intervals.

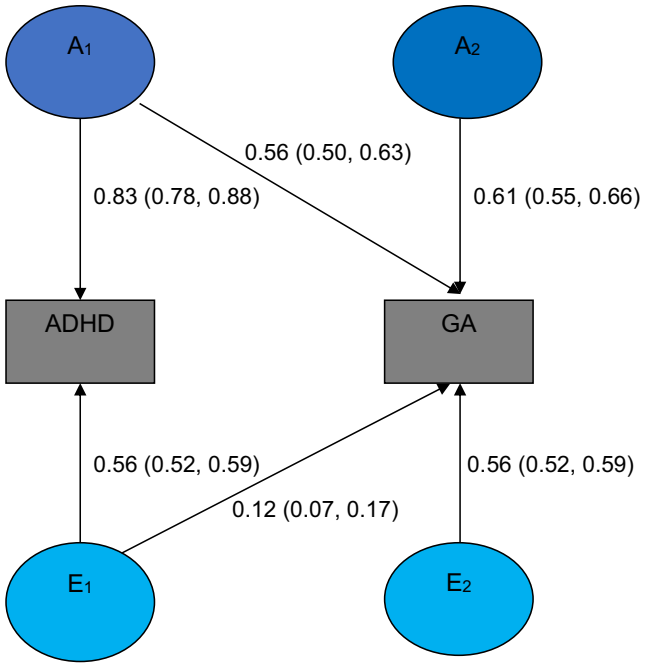

Figure 2. Parameter estimates in the best-fitting bivariate Cholesky model. 95% confidence intervals are in parenthesis. A: additive genetic influences, E: nonshared environmental influences. Path coefficients should be squared to estimate additive genetic and nonshared environmental influences.
Note: GA, gaming addiction; ADHD, attention deficit hyperactivity disorder.

Discussion

Using South Korean adult twins, the present study demonstrated that the common genetic factors mainly influenced the ADHD-GA association. Although common nonshared environmental influence attained statistical significance, it was much smaller than common genetic influence (.68 vs. .22).

The finding of genetic overlap between GA and ADHD indicates that the horizontal pleiotropic effects of genes may be partly responsible for the relationship between the two traits. Horizontal pleiotropic effects of genes occur when the same genetic factors influence multiple traits independently (Paaby & Rockman, 2013). ADHD and GA are characterized by a constant need for stimulation and an aversion to delayed rewards (Hinshaw, 2018). Neurobiological studies propose that these characteristics are related to low dopaminergic functioning (Weinstein & Lejoyeux, 2020). Thus, genes involved in dopamine regulation likely influence ADHD and GA, simultaneously. Indeed, a recent GWAS (Haghighatfard et al., 2023) illustrated that genes for dopamine pathways, such as *DRD4*, *COMT*, and *MAOB*, which were identified for GA, were shared with ADHD. Furthermore, because ADHD and GA are comorbid with depression, SUDs, and other mental disorders (Ko et al., 2009; Yen et al., 2007), genes for serotonergic activity may also influence ADHD and GA, leading to genetic correlation.

The genetic correlation found in the present study may result from vertical pleiotropic effects of genes as well. Vertical pleiotropic effects of genes occur when a trait influenced by

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genetic factors in turn influences another trait by acting as a mediator (Paaby & Rockman, 2013). Namely, genetic variants may affect ADHD through GA and vice versa. Koller et al. (2024) explored the presence of vertical pleiotropy in the relationship between ADHD and SUDs using Mendelian randomization (MR) analysis. Their results suggested bidirectional causality, but genetic effects of SUDs on ADHD were stronger than the reverse. However, in a similar study, Vink et al. (2021) failed to detect genetic effects of nicotine dependence on ADHD.

Interestingly, recent evidence shows that game-based tools that provide cognitive training are effective in decreasing ADHD symptoms (especially, inattention problems) (Peñuelas-Calvo et al., 2022). Cognitive training can reduce executive function deficits by strengthening neural networks, leading to improved attentional performance in children with ADHD (Peñuelas-Calvo et al., 2022). However, a meta-analysis suggested that long-term effects of game-based cognitive training were limited (Caselles-Pina et al., 2023). Also, because most studies of game-based interventions to date employed patients with ADHD without other psychopathology (Caselles-Pina et al., 2023), how these interventions influence patients with GA-ADHD comorbidity is largely unknown. More studies are necessary to resolve the contrasting results between the shared genetic etiology of GA-ADHD comorbidity and game-based interventions to reduce symptoms of ADHD.

Whether GA is a separate clinical entity or a manifestation of underlying psychiatric disorders is controversial (Fergusson et al., 2011). Although this study showed high genetic overlap between ADHD and GA, it identified a substantial amount of genetic variance unique to GA. This finding supports the notion that GA may be a separate clinical entity. However, given the comorbidity of GA with many forms of psychopathology (Ko et al., 2009; Yen et al., 2007), further GWAS and twin studies are necessary to elucidate the genetic architecture of GA and mental disorders.

The present findings have Implications for prevention and intervention strategies. The high genetic correlation between ADHD and GA emphasizes that family members, especially siblings of children with ADHD or GA, are also at risk, and therefore should be targeted for prevention. Common genetic mechanisms also suggest that treating ADHD symptoms may help treat GA or, conversely, treating GA may reduce the severity of ADHD in those with comorbidity.

Our findings should be interpreted with several limitations. First, due to insufficient sample size, we were not able to evaluate sex differences in the genetic and environmental correlations between ADHD and GA. Yen et al. (2009) reported that women showed higher ADHD-IGD association than men. Thus, future studies should increase sample size and explore sex differences in genetic and environmental correlations. Second, we determined twins' zygosity using a questionnaire method. A previous study indicated that the misclassification of zygosity can affect genetic and environmental influences (Odintsova et al., 2018). Notably, however, twins with ambiguous zygosity were excluded from data analysis to increase the validity of zygosity diagnosis. Third, this study employed self-reported GA and ADHD instead of clinical diagnosis or observational measures. Thus, future studies should replicate the findings with objective measures. Fourth, because GA and ADHD were measured in a single online survey, common method variance (CMV) could have occurred. In the twin modeling, if CMV had occurred, it would have increased nonshared environmental correlation. Thus, the true estimate of nonshared environmental correlation between GA and ADHD is likely lower than the estimate found in the present study. Finally, participants were South Korean adults, which primarily comprised university students. Given the difference in prevalence of GA across populations (H. S. Kim et al., 2022), whether the present results can be generalized to children, clinical samples, or other ethnic groups remains unclear.

In conclusion, the study demonstrated that the ADHD-GA association was largely due to shared genetic vulnerability. This finding could be used to support multivariate genetic approaches, like genomic structural equation modeling or multitrait analysis of GWAS, which could be used to increase power to detect genetic effects for GA.

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