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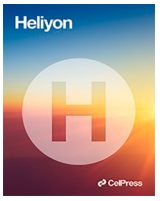

Research article

Associations of education attainment with gestational diabetes mellitus and the mediating effects of obesity: A Mendelian randomization study

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ABSTRACT

We aim to assess the causal association between educational attainment and gestational diabetes mellitus, and the mediating effect of obesity on this association. We estimated the causal effects of educational attainment on gestational diabetes mellitus using European ancestry genome-wide association study summary data with two-sample univariate Mendelian randomization (UVMR) approach. Two-stage Mendelian randomization analysis was performed to assess the potential mediating role of obesity traits in this association and to calculate the mediating proportion. UVMR analysis demonstrated that higher educational attainment was associated with a reduced risk of GDM (OR 0.76, 95% CI 0.67–0.86; $p < 0.01$). EA has also been associated with decreased obesity in women. Mediation Mendelian randomization results indicated that body mass index (BMI) was the most significant mediating factor in the relationship between educational attainment and GDM, accounting for 42.52% (95% CI 37.75–55.44%) of the effect, followed by waist-to-hip ratio (WHR) at 34.35% (95% CI 29.82–46.41%), body fat percentage at 28.95% (95% CI 35.99–46.81%), and WHR adjusted for BMI (WHRadjBMI) at 12.51% (95% CI 36.2–58.5%). educational attainment exerts a potential causal protective effect against gestational diabetes mellitus, and obesity-related risk factors play a mediating role. Attention should be paid to the educational attainment of women, and obese women with lower educational attainment may represent a higher risk group for GDM than those with higher educational attainment.

1. Introduction

Gestational diabetes mellitus (GDM) is characterized by glucose intolerance, which manifests for the first time during pregnancy [1, 2] since its initial description in 1964. GDM is a common complication during pregnancy, with a global incidence rate ranging from

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7.1% to 27.6% in different areas [3,4], and affects approximately 17 million pregnancies worldwide [5]. GDM increases the risk of adverse pregnancy outcomes [6,7] and long-term maternal and offspring complications [1,5,8–10]. GDM is a growing public health concern that has garnered widespread attention worldwide.

Education attainment (EA) is a vital socioeconomic characteristic and a predictor of other factors, such as occupation and income. It substantially impacts the adoption of healthy lifestyles and the accessibility of healthcare services [11]. Moreover, EA may serve as a causal factor in the development and mortality of various diseases. Mendelian randomization (MR) is a substantially more robust approach than conventional clinical research methods for evaluating causal relationships based on observational data. Recent MR studies have shown that higher EA is causally associated with a decreased risk of obesity traits [12–14] and identified a causal association between lower EA and a higher risk of type 2 diabetes [15,16]. However, little is known about the causal relationship between genetically predicted EA and GDM risk as estimated using recent genome-wide association study (GWAS) data.

Hence, our objective was to comprehensively examine the causal effects of EA on the risk of GDM and ascertain the mediating influence of obesity using two-sample univariate MR (UVMR) and MR-based mediation analyses.

2. Materials and methods

2.1. Study design

The study design is illustrated in Fig. 1. The MR approach was based on three key assumptions [17]. First, the instrumental variables used were associated with the risk factors. Second, these factors were not associated with confounding factors. Finally, the effect of these genetic variants on the outcome should be solely through risk factors and no other pathways.

2.2. Data sources for genetic instruments

This MR study utilized summary-level data from GWASs to obtain data sources for exposure, mediators, and outcomes. Most GWASs have focused on individuals of European ancestry (Table 1).

2.3. Genetic instruments for EA and obesity traits

EA is the number of years of formal education a person has completed, starting with kindergarten or its equivalents [18]. Genetic instruments for education were selected from a GWAS of years of schooling in individuals of European ancestry. The most recent GWAS was conducted by the Social Science Genetic Association Consortium (SSGAC), with summary data available for 765,283 participants after excluding data from individuals in 23andMe owing to limitations in reporting more than 300,000 SNPs [18].

Body mass index (BMI), body fat percentage (BF%), waist-to-hip ratio (WHR) and WHR adjusted for BMI (WHRadjBMI) were selected as obesity traits. BMI, calculated as total body weight (kg) divided by standing height squared (m^2), is a widely used indicator of overall obesity. WHR and WHRadjBMI are considered measures of central obesity. Genetic instruments for BMI, WHR, and WHRadjBMI were identified through a meta-analysis of GWASs, combining data from the Genetic Investigation of Anthropometric Traits and the UK Biobank. These studies included 806,834 individuals with BMI (434,794 women); 697,734 individuals with WHR (381,152 women); and 694,649 individuals with WHRadj BMI (379,501 women) [19]. The summary statistics of the female European ancestry used in this study were obtained from the following website: https://zenodo.org/record/1251813#.Y6lY_BVBzQw. We also obtained BF% from the Neale Lab, summary data available for 331,117 participants of European ancestry, and summary statistics obtained from the following website: https://gwas.mrcieu.ac.uk/datasets/ukb-b-8909/

Linkage disequilibrium analyses were performed using $r^2 < 0.001$, a distance threshold of 10,000 kb, and SNPs with a significance level of $P < 5 \times 10^{-8}$ as primary genetic instruments for EA, BMI, BF%, WHR, and WHRadjBMI. We also calculated the F-value to identify weak instrumental variables, which were excluded if the F-value was below 10.

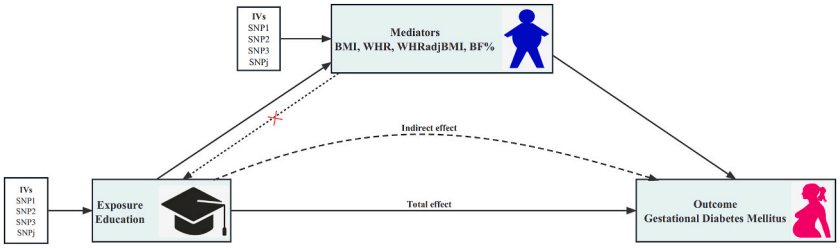

Fig. 1. Overview of the study design. This study consisted of two stages of analysis. In stage 1, we assessed the causal associations of EA with GDM using univariate Mendelian randomization, which was the total effect in stage 2. We assessed the causal associations of EA with obesity trait mediators (BMI, WHR, WHRadjBMI, and BF%) and obesity trait mediators with GDM. We then performed bidirectional Mendelian randomization to ensure that obesity trait mediators had no causal association with EA. Finally, indirect effects of individual mediators (BMI, WHR, WHRadjBMI, BF%) using 2-step MR. EA, education attainment; GDM, gestational diabetes mellitus; BMI, body mass index; WHR, waist-to-hip ratio; WHRadjBMI, WHR adjusted for BMI; BF%, body fat percentage.

Table 1
Overview of GWAS data used in the MR Analyses.

Phenotype	No of participants	Ancestry	Consortium/cohort	Author	Year of publication	PubMed ID
EA	765,283	European	SSGAC	Okbay et al.	2022	35361970
BMI	262,817	European	GIANT + UKB	Pulit, SL et al.	2018	30239722
WHR	263,148	European	GIANT + UKB	Pulit, SL et al.	2018	30239722
WHRadj BMI	262,759	European	GIANT + UKB	Pulit, SL et al.	2018	30239722
BF%	331,117	European	Neale Lab	Neale et al.	2017	NA
GDM	210,870	European	FinnGen	Kurki M.I. et al.	2023	NA

Abbreviations: EA: education attainment, GDM: gestational diabetes mellitus, BMI: body mass index (female), WHR: waist-to-hip ratio, WHRadjBMI: WHR adjusted for BMI, BF%: body fat percentage.

2.4. Genetic instruments for GDM

GDM is characterized by the development of diabetes mellitus during pregnancy [20] GDM summary statistics data of GDM were obtained from a GWAS of European ancestry by the FinnGen Consortium [21]. FinnGen, an ongoing project in Finland, merges genomic information with digital healthcare data. Version 9 of the FinnGen data was used, comprising 210,870 females (13,039 cases and 19,7831 controls) with a prevalence of 6.18%. Data were downloaded from https://storage.googleapis.com/finngen-public-data-r9/summary_stats/finngen_R9_GEST_DIABETES.gz.

2.5. Statistical analysis

2.5.1. UVMR analyses and sensitive analyses

We performed a two-sample UVMR analysis to assess the causal relationship between EA and GDM, EA, and obesity trait mediators (BMI, BF%, WHR, and WHRadjBMI), and obesity trait mediators and GDM development separately. We evaluated the causal association between EA and the mediators using bidirectional MR.

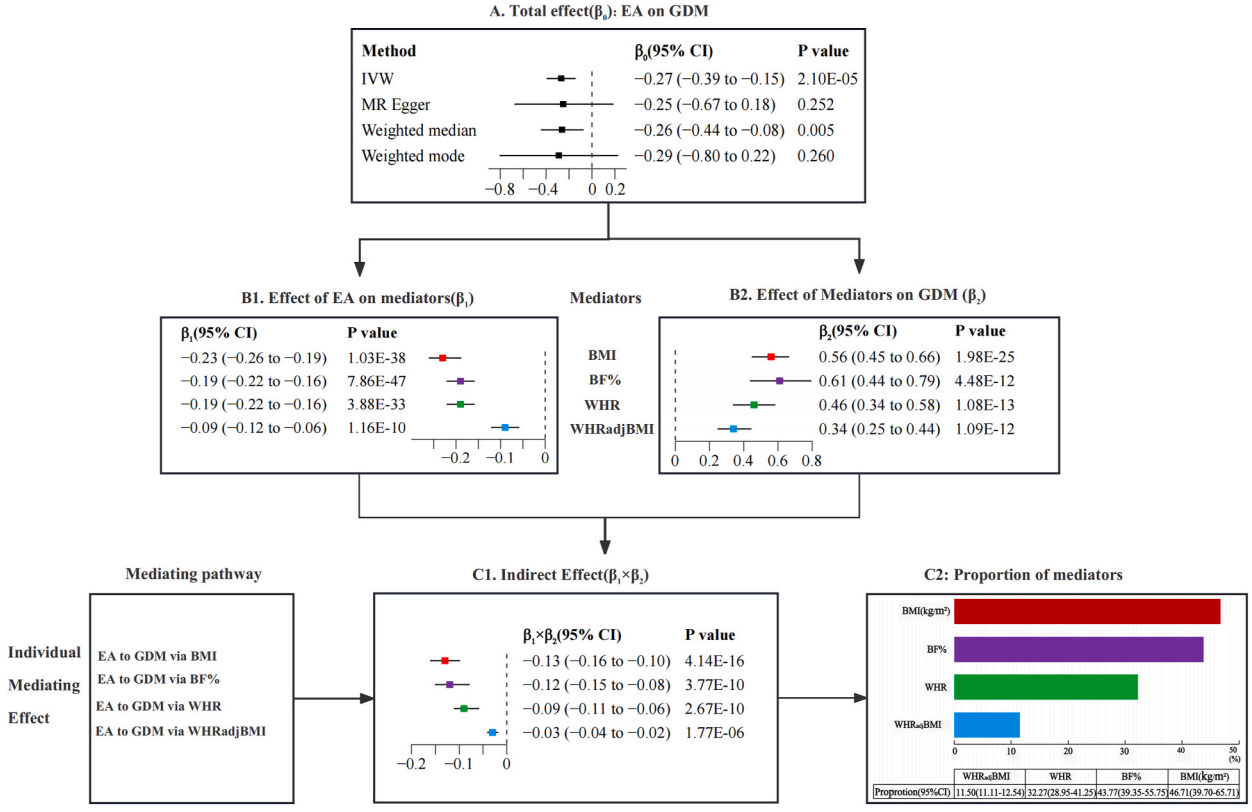

Fig. 2. (A) MR-estimated total effect of EA on GDM using different methods(β_0). (B1) MR-estimated effects of EA on each mediator (β_1). (B2) MR-estimated effects of mediators on GDM (β_2). (C1) MR-estimated effects of indirect effects of each mediator separately($\beta_1 \times \beta_2$) by the product of coefficients method with delta method-estimated 95% CIs. (C2) Calculated proportions mediated (%) are presented with 95% CIs. EA, education attainment; GDM, gestational diabetes mellitus.

The primary analysis employed an inverse-variance-weighted (IVW) approach, utilizing different genetic variants under the assumption of instrument validity. We deleted confounding SNPs associated with triglyceride, high-density lipoprotein, and APOA1 (see Supplementary Data 1) based on the literature [13] using PhenoScanner (cam.ac.uk). We incorporated three alternative methods into our MR analysis to address potential pleiotropy: MR-Egger regression, weighted median, and weighted mode. Heterogeneity across genetic instruments was evaluated using Cochran's Q test. We employed the MR-Egger intercept test to identify horizontal pleiotropy. In cases where the MR-Egger intercept indicated horizontal pleiotropy, we identified and excluded outliers using the pleiotropy residual sum and the outlier (PRESSO) method (see Supplementary Data 2). We conducted an MR-Steiger directionality test to confirm the causal direction of UVMR. The reliability of the findings was validated using a leave-one-out approach. Finally, the results were presented visually using scatter, funnel, and leave-one-out plots.

2.5.2. Mediation MR analyses

As there was no sample overlap, we employed GWAS data from FinnGen as the primary data source for GDM to perform mediator screening. A two-step MR analysis was conducted to evaluate the individual mediating effect of an intermediate factor between EA and obesity traits (BMI, BF%, WHR, and WHRadjBMI; Fig. 2).

In the first step, UVMR analysis was used to calculate the total direct effect of EA on GDM risk (β_0; Fig. 2(A)). In the second step, we estimated the effect of EA on obesity traits (β1; Fig. 2 (B1)) and the effects of different obesity traits (BMI, WHR, WHRadjBMI, and BF %) on GDM (β2; Fig. 2 (B2)), and then calculated the indirect effect ($\beta_1 \times \beta_2$) (Fig. 2(C1)). $\beta_1 \times \beta_2$ was divided by the total effect (β_0) to determine the proportion of the mediated effect (Fig. 2(C2)).

Effect sizes were reported as odds ratios (OR), β-coefficients, proportions, and their respective 95% confidence intervals (CI). All analyses were performed using the TwoSampleMR (version 0.5.7) and MRPRESSO packages (version 1.0.0) in R Software 4.3.1 (https://www.R-project.org).

3. Results

3.1. Genetic instruments

Data on SNPs and their associations with EA, mediators, and GDM are provided in the Supplementary Information (see Supplementary Data 3–16).

3.2. Effects of EA on GDM (β_0)

Univariate MR analyses demonstrated that an elevated genetic predisposition to EA was associated with an increased risk of GDM. Moreover, for each SD increase in genetically predicted EA, there was a corresponding 24% reduction in the risk of developing GDM (OR = 0.76; 95% CI:0.67–0.86) (see Fig. 2(A), sTable 1, sFig. 1a.). We conducted a reverse MR analysis and the MR-Steiger directionality test to evaluate potential directional horizontal pleiotropy. There was no causal association between GDM and EA (IVW, $p > 0.05$) (see Supplementary Data 12, sTable 3).

3.3. Effect of EA on mediators (β_1)

The effects of genetically predicted EA on each mediator are illustrated in Fig. 2 (B1) and summarized in sTable 2. It was observed that for every SD increase in genetically predicted EA, there was a significant association with lower BMI (β = −0.23; 95% CI: −0.24 to −0.17), reduced WHR (β = −0.19; 95% CI: −0.22 to −0.16), decreased WHRadjBMI (β = −0.09; 95% CI: −0.12 to −0.06), and diminished FA% (β = −0.19; 95% CI: −0.22 to −0.16). These findings highlight the inverse relationship between genetically predicted EA and obesity.

We further employed bidirectional MR to validate the causal relationship between EA and different obesity-related traits. The results indicated no evidence of a reverse causal association between obesity-related traits and education (see Supplementary sTable 3, sFigure 1b).

3.4. Effect of mediators on GDM (β_2)

Fig. 2 (B2) and sTable 4 show the significant associations between each mediator and GDM. According to these findings, an increase of one SD in genetically predicted BMI was linked to a 1.74-fold higher likelihood of GDM (OR = 1.74, 95% CI: 1.57 to 1.94). Similarly, an increase of one SD in the genetically predicted WHRadjBMI was associated with an increased risk of GDM (OR = 1.41, 95% CI: 1.28 to 1.55). Moreover, a one SD increase in genetically predicted WHR resulted in 1.58-fold higher odds of developing GDM (OR = 1.58, 95% CI: 1.40 to 1.78). Finally, a one SD increase in genetically predicted FA% was associated with a 1.85-fold higher likelihood of GDM (OR = 1.85, 95% CI: 1.55 to 2.20). These findings confirm the significant impact of these mediators on the risk of developing GDM (see Supplementary sFig. 1c).

3.5. Indirect effect and individual mediating effect ($\beta_1 \times \beta_2$)

Fig. 2 (C1) and Fig. 2 (C2) illustrate the individual contributions of each mediator in explaining the indirect effects of EA on GDM.

Specifically, BMI accounted for 46.71% (95% CI: 39.70%–65.71%) of the total effect of EA on GDM, with a coefficient of −0.13 (95% CI: −0.16 to −0.10). BF% explained 43.77% (95% CI: 39.35%–55.75%) of the total effect, with a coefficient of −0.12 (95% CI: −0.15 to −0.08). WHR accounted for 32.27% (95% CI: 28.95%–41.25%) of the total effect, with a coefficient of −0.09 (95% CI: −0.11 to −0.06). Finally, WHRadjBMI explained 11.50% (95% CI: 11.11%–12.54%) of the total effect, with a coefficient of −0.03 (95% CI: −0.04 to −0.02). These findings shed light on the individual contribution of each mediator to the relationship between EA and GDM.

3.6. MR sensitivity analyses

Heterogeneity was assessed using Cochran's Q test. We observed substantial heterogeneity from the genetic instruments for EA to GDM and mediators, as well as from mediators to GDM ($p < 0.05$) (see Supplementary sTable 5).

To evaluate potential directional horizontal pleiotropy, we conducted a reverse MR analysis and the MR-Steiger directionality test (see Supplementary Data 12–16 and sTable 6). Additionally, we performed an MR-Egger regression to assess whether the mean value of the Egger intercept was nonzero, indicating possible directional pleiotropy. Furthermore, we employed the MR-PRESSO method to identify and remove outlier SNPs (see Supplementary Data 2). Using a funnel chart to visualize SNP outliers can aid in a better understanding of the distribution of data, identify anomalies, and facilitate appropriate data processing and analysis (see Supplementary sFig. 2a, sFig. 2b and sFig. 2c). These approaches enhance the robustness and validity of the results by eliminating the potential influence of outliers.

Our study found no significant evidence of horizon pleiotropy as a mediator of EA ($p > 0.05$) (see Supplementary sTable 6). Moreover, the MR-weighted median and weighted mode methods were generally consistent with the MR-IVW method in terms of effect size and direction (see Supplementary sTable 1 and sTable 2), suggesting that horizontal pleiotropy did not significantly bias the results.

Finally, we conducted a leave-one-out cross-validation to validate the reliability of our findings. These consistent validation results provide additional support for the robustness and reliability of our results (see Supplementary sFigure 3a, sFigure 3b and sFigure 3c).

4. Discussion

In this updated MR study of EA to GDM, we found evidence of a causal protective relationship between EA and GDM, where each SD increase in schooling was associated with about a 24% decrease in GDM risk (OR = 0.76; 95% CI:0.67 to 0.86; $P = 2.10 \times 10^{-5}$). Our MR-based mediation analysis indicates that obesity traits may mediate the relationship between EA and GDM. Specifically, BMI accounted for 46.71% of the mediation, BF% 43.77%, WHR 32.37%, and WHR-adjusted BMI 11.50%. These findings emphasize the importance of addressing obesity traits to understand the relationship between complex EA and GDM.

In a previous study [22], an evaluation was used to determine the causal relationship between EA and GDM using MR analysis. This study used EA-related GWAS data from Okbay [23] and GDM-related GWAS data from the UKbiobank [24]. However, owing to the limited records in the GDM database, only 240 cases were identified using questionnaire-based criteria. Consequently, MR analysis found no causal association between EA and GDM (OR = 1.00; 95%CI:1.00–1.00; P = 0.171). Thus, updating the MR results for the EA-GDM relationship using the latest databases is crucial.

To address this issue, we acquired data from a genome-wide association study (GWAS) of European ancestry conducted by the FinnGen Consortium. This database comprises 210,870 females, with 13,039 cases and 197,831 controls in 2023. Additionally, we obtained the latest GWAS data on European ancestry (EA) from the SSGAC in 2022 [18] which incorporates a whole-genome polygenic risk score or polygenic index explaining 12–16% of the EA variance and aids in the risk prediction of 10 diseases.

EA is a well-established determinant of social health [25]. In our study, we found a compelling causal association between higher EA and a reduced incidence of GDM and a decrease in obesity-related indicators such as BMI, BF%, WHR, and WHRadjBMI. Notably, existing reports suggest that education has more pronounced health effects on women than that on men [26]. Therefore, increasing the EA of women may have a more significant potential in positively addressing female obesity.

Obesity is currently recognized as a crucial risk factor for GDM. A two-sample MR study [13] confirmed a causal association between obesity-related traits and the incidence of GDM. In another study that utilized a polygenic scoring approach, Li et al. [27] revealed that obtaining a college degree was associated with a reduced likelihood of obesity. Moreover, among individuals with college degrees, those with a higher education polygenic score exhibited a lower likelihood of obesity than those with a relatively lower education polygenic score. Howe et al. [28] conducted a within-sibship MR study that revealed a strong association between higher EA and lower BMI. Furthermore, in an observational study, Panchal et al. [29] found that individuals with lower EA (less than college graduates) had higher BMI than those with higher EA (college graduates and post-college degrees) [31.8 ± 6.4 vs. 29.6 ± 4.8 vs. 29.7 ± 5.2, p = 0.04]. These findings align with our study results, indicating a robust and significant causal association between obesity-related traits, such as BMI, BF%, WHR (indicative of central obesity), and the incidence of GDM.

The Mendelian mediation analysis further confirmed the independent mediating effects of different obesity-related indicators between EA and GDM. BMI showed the most substantial mediating effect (46.7 %), followed by BF% (43.77%), WHR (32.37%), and WHR-adjBMI (11.50%). Although BMI has been considered a shortcoming in assessing obesity in previous studies on different diseases [30] it remains the indicator with the highest independent proportion in the mediation analysis; the result was similar to that of the observational [31] and MR study [13].

The mechanisms through which EA may reduce the risk of GDM could involve the following: Higher EA could lead to lifestyle changes [32–36] such as promoting regular physical exercise and adopting a healthy diet to achieve a healthy body composition or appropriate pregnancy weight gain goals. Simultaneously, higher EA can enhance women's economic income [37] elevate their family

status, and make it easier for them to receive more family support during pregnancy. A higher EA level may make it easier to control diseases [37,38]; it is more likely that obese women of reproductive age with a higher EA level will achieve more effective preventive measures by obtaining health education before conception.

For healthcare professionals, it is essential to not only deepen their understanding of the heightened risk of GDM in overweight and obese women but also consider EA as a crucial factor in the preconception assessment for GDM. Particular attention should be paid to women of reproductive age with obesity due to lower EA. From a policymaking perspective, the EA of females is a public health concern that should be prioritized even before considering disease health status.

Our MR study first updated the association of EA with GDM and the mediators of obesity traits and identified EA as a modifiable factor preceding the disease factor (GDM). This study has some limitations that should be considered. First, the participants were solely of European origin. generalizing the findings to other ethnic populations requires further investigation in diverse cohorts. Second, the summary data used in the two-sample MR analysis did not permit stratified analyses by covariates such as age, EA status, and BMI. Third, this study only conducted a mediation analysis on some commonly used indicators of obesity and did not analyze other potential mediators such as blood lipid levels, physical activity, and socioeconomic status. Further studies are required to elucidate the potential mediating effects of EA on GDM concerning these factors. The other mediating effects of EA and GDM could not be fully explained by this study. For example, several potential mediators such as health policy, economic conditions, social background, and family support systems are not heritable, and GWASs are unavailable. Fourth, the constant heterogeneity of SNPs may have caused potential bias and affected the robustness of the MR results. Caution should be exercised when interpreting the results because of potentially unaccounted variable influences.

5. Conclusions

Our comprehensive MR study provided strong evidence supporting a causal link between higher EA and reduced GDM risk, partially with obesity traits acting as mediators. Enhancing female EA can directly affect women's health and have long-term and profound positive effects on their overall societal health.

Ethical statement

Informed consent was not required for this study because this study analyzed publicly available, de-identified data and ensured that ethical review and informed consent were obtained from all original GWAS studies.

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Data availability statement

The summary data of phenotypes can be downloaded from the website, while detailed information can be found in the methods section and Table 1 of the manuscript.

Declaration of generative AI in scientific writing

During the preparation of this work the author used AI-assisted technologies in order to improve readability and language. After using this tool, the author reviewed and edited the content as needed and take full responsibility for the content of the publication.

CRediT authorship contribution statement

Xiaoyan Wang: Writing – review & editing, Writing – original draft, Supervision, Methodology, Formal analysis, Data curation, Conceptualization. **Ying Lan:** Data curation. **Na Li:** Writing – review & editing, Conceptualization. **Jinfeng Gao:** Formal analysis, Conceptualization. **Dejiao Meng:** Formal analysis, Data curation. **Shuchuan Miao:** Writing – review & editing, Methodology, Formal analysis, Conceptualization.

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.

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Appendix A. Supplementary data

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