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Original Research

The Role of Body Mass Index and Insulin-Treated Diabetes on the Education-Mediated Risk of Developing Stress Urinary Incontinence: A Mediation Mendelian Randomization Study

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Abstract

Background: This study aimed to investigate the causal relationship between educational attainment and stress urinary incontinence (SUI) using Mendelian randomization (MR) analysis, as well as to explore the potential mediating roles of body mass index (BMI), chronic obstructive pulmonary disease (COPD), and insulin-treated diabetes. **Methods**: Genome-wide association studies (GWAS) data for educational attainment levels were obtained from the Social Science Genetic Association Consortium (SSGAC); SUI data were sourced from the Integrative Epidemiology Unit Open GWAS Project (IEU OpenGWAS project); and data for BMI, COPD and insulin-treated diabetes were obtained from the FinnGen database. The inverse variance weighted (IVW) method was used as the primary analytical approach to investigate causal relationships. Sensitivity analyses, including heterogeneity tests, pleiotropy tests, funnel plot, and leave-one-out sensitivity analysis, were conducted to assess the robustness of the results. **Results**: A causal relationship was observed between higher educational attainment and a reduced risk of SUI (odds ratio [OR] = 0.995, 95% CI: 0.993–0.996, $p = 1.851 \times 10^{-10}$). Besides, higher educational attainment was associated with lower BMI (OR = 0.817, 95% CI: 0.792–0.843, $p = 4.825 \times 10^{-37}$) and diabetes (OR = 0.661, 95% CI: 0.615–0.709, $p = 2.266 \times 10^{-30}$). Genetically predicted higher BMI (OR = 1.002, 95% CI: 1.000–1.003, $p = 0.048$) and insulin-treated diabetes (OR = 1.001, 95% CI: 1.000–1.002, $p = 0.012$) were associated with an increased risk of SUI. Sensitivity analyses confirmed the robustness of the findings. Mediation analyses showed that BMI (8.08%, $p = 0.048$) and insulin-treated diabetes (7.47%, $p = 0.004$) partially mediated the protective effect of higher educational attainment on SUI risk. **Conclusions**: Improving educational attainment levels may reduce the risk of SUI. Furthermore, BMI and insulin-treated diabetes partially mediate the protective effect of higher educational attainment on SUI risk.

Keywords: educational attainment; stress urinary incontinence; BMI; diabetes (insulin treatment); mediation Mendelian randomization study

1. Introduction

Stress urinary incontinence (SUI) is the most common type of incontinence [1], characterized by the sudden, involuntary leakage of urine during activities that increase intra-abdominal pressure, such as coughing, laughing, sneezing, or exercising [2,3]. This condition affects millions of people globally, severely impacting the quality of life of patients and creating a huge economic burden [1,4]. It is more prevalent in middle-aged and older women, especially those with a history of multiple vaginal deliveries and pelvic surgery [5]. SUI is more prevalent among male patients who have undergone prostate surgery or radiotherapy, experienced nerve damage due to trauma, or possess congenital developmental abnormalities [6–10]. Current research suggests that the average prevalence of SUI is as high as 25% [11,12]. Accordingly, it is of great clinical significance to clarify the etiology of SUI and early intervention for preventing and delaying its onset and progression.

Factors such as the level of education, pregnancy, childbirth, pelvic surgery, and chronic diseases, including metabolic syndrome, diabetes, obesity, and overweight, have been associated with the development of SUI [12–14]. Accordingly, controlling these associated factors is essential for delaying the onset and preventing SUI [15]; however, several factors, such as level of education, pregnancy, and childbirth, are non-modifiable. Furthermore, the association between education level and SUI may be mediated by several modifiable factors related to exposure and outcome, such as height, body mass index (BMI), diabetes mellitus, smoking, and alcohol consumption. Due to economic development, lifestyle change, environmental impact, population aging and other factors, the incidence

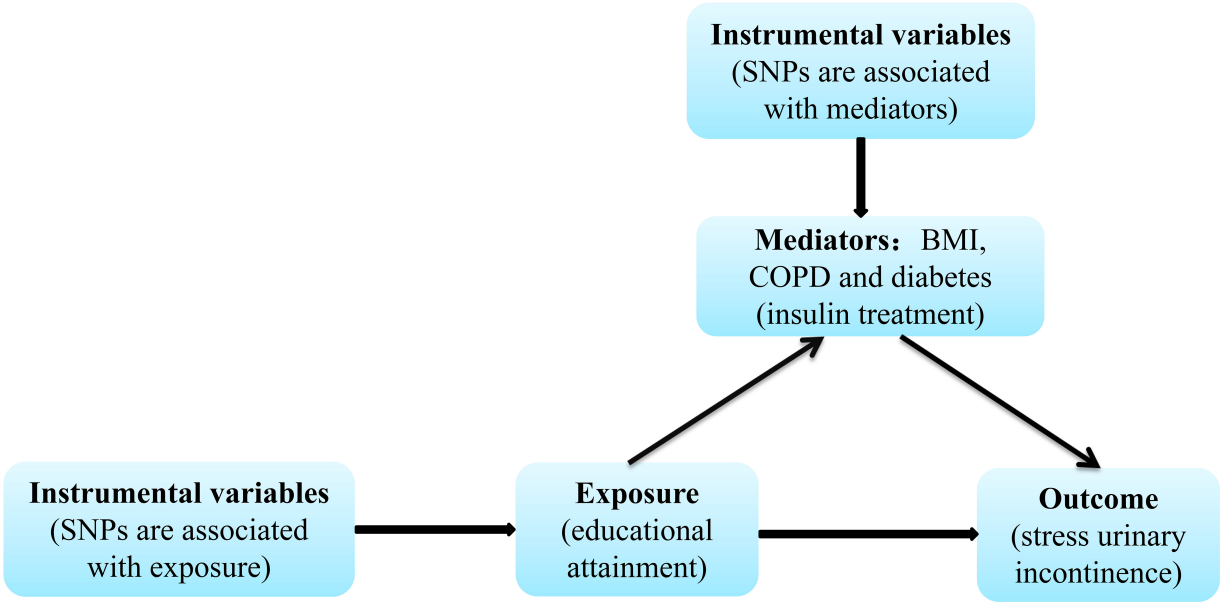

Fig. 1. Assumption and design for the MR study. SNP, single-nucleotide polymorphism; BMI, body mass index; COPD, chronic obstructive pulmonary disease; MR, Mendelian randomization.

of obesity and diabetes is increasing year by year around the world, which poses a serious threat to public health [16,17]. In 2023, a Mendelian study conducted by Zhang *et al*. [13] revealed that higher educational attainment exerted a causal protective effect against SUI. Nevertheless, this study did not investigate the mediating factors, whether risk or protective, that are associated with the onset of SUI. This limitation poses challenges for early-stage clinical interventions and prevention strategies for SUI, an issue that merits further investigation. Therefore, the present study explored the causal relationship between educational attainment level and SUI risk using Mendelian randomization (MR) and examined the moderating role of BMI, chronic obstructive pulmonary disease (COPD), and insulin-treated diabetes in this relationship using mediated MR analysis.

2. Materials and Methods

2.1 Research Design and Data Sources

A two-sample MR analysis was used to explore the causal relationship between educational attainment level and SUI, as well as to determine the mediating roles of BMI, COPD and insulin-treated diabetes in this relationship. This study follows three assumptions of MR research: (1) The association assumption, which states that there is a close association between genetic variants (instrumental variables) and exposure factors; (2) The exclusivity assumption: Instrumental variables can only influence the outcome by affecting exposure factors and, consequently, the outcome. (3) The exclusion restriction assumption: There is no association between instrumental variables, confounders, and outcome variables. These assumptions are illustrated in Fig. 1.

In our study, genome-wide association studies (GWAS) summary statistics for educational attainment level were obtained from the Social Science and Human Behavioral Genetics Data [18] (Social Science Genetic Association Consortium, SSGAC, https://www.thessgac.org/). In the analysis, educational attainment was measured by the number of years of schooling completed (n = 765,283 European participants). GWAS data for SUI were provided by the Integrative Epidemiology Unit Open GWAS Project (IEU OpenGWAS project) (https://gwas.mrcieu.ac.uk/). GWAS data for BMI, COPD and diabetes (insulin treatment) were obtained from the FinnGen database [19] (version 11, https://r11.finngen.fi/), with a public release date of June 24, 2024. A summary of all data sources and sample characteristics is provided in Table 1.

The data in this paper were obtained from open-source public databases and with permission from these databases, without the need for additional ethical approval.

2.2 Selection of Instrumental Variables

To ensure the independence of single-nucleotide polymorphisms (SNPs) and to reduce the influence of genetic pleiotropy on the results, we implemented specific analytical methods. First, SNPs were selected based on a significance threshold of $p < 5 \times 10^{-8}$, a linkage disequilibrium r^2 value < 0.001, and a clumping distance of 10,000 kb. However, in the reverse MR analysis of SUI on education level, the $p < 5 \times 10^{-8}$ threshold yielded only one SNP. Since this result does not meet the minimum criterion of having at least 10 eligible instrumental variables, a less stringent significance threshold of $p < 5 \times 10^{-6}$ was applied, yielding 12 SNPs. Besides, to avoid bias from weak

Table 1. Information on GWAS data for exposure, mediating factors, and outcome datasets.

Parameter	Data sources	GWAS ID	Population	Sex	Sample size	Cases/controls	SNP
Exposure: educational attainment	SSGAC	/	European	Males and Females	765283	765283/NA	10985947
Mediating factors							
BMI	FinnGen database	finngen_R11_BMI_IRN	European	Males and Females	321672	321672/NA	21304205
Diabetes (insulin treatment)	FinnGen database	finngen_R11_KELA_DIAB_INSUL_EXMORE	European	Males and Females	437353	68356/368997	21306517
COPD	FinnGen database	finngen_R11_J10_COPD	European	Males and Females	394244	21617/372627	21305758
Outcome: SUI	IEU OpenGWAS project	ukb-b-9873	European	Males and Females	463010	4340/458670	9851867

GWAS, genome-wide association studies; SSGAC, Social Science Genetic Association Consortium; SUI, stress urinary incontinence; IEU OpenGWAS project, Integrative Epidemiology Unit Open GWAS Project; NA, not applicable.

Table 2. Tests of heterogeneity between SNPs and pleiotropy of selected SNPs.

	Number of SNPs	Heterogeneity test		Pleiotropy test	
		Cochran's Q	*p* value	β	*p* value
Education level and SUI	381	412.166	0.123	-3.842×10^{-6}	0.936
Education level and BMI	356	908.072	3.835×10^{-50}	1.248×10^{-4}	0.885
BMI and SUI	270	350.583	0.001	2.957×10^{-5}	0.474
Education level and diabetes (insulin treatment)	385	761.002	4.285×10^{-27}	–0.002	0.374
Diabetes (insulin treatment) and SUI	140	166.811	0.054	3.426×10^{-5}	0.480
Education level and COPD	395	615.578	5.676×10^{-12}	–0.005	0.052
COPD and SUI	16	18.433	0.241	-1.821×10^{-4}	0.225

Table 3. The mediation effect of education attainment on SUI via BMI and diabetes.

Mediators	Total effect β (95% CI)	Direct effect A β (95% CI)	Direct effect B β (95% CI)	Mediation effect β	Mediated proportion (%)	*p* value
BMI	–0.005 (–0.007, –0.004)	–0.202 (–0.233, –0.171)	0.002 (1.218×10^{-5}, 0.003)	-4.040×10^{-4}	8.08	0.048
Diabetes	–0.005 (–0.007, –0.004)	–0.415 (–0.486, –0.344)	8.609×10^{-4} (1.895×10^{-4}, 0.002)	-3.735×10^{-4}	7.47	0.004

instrumental variables, we excluded those with an F-statistic of less than 10. Finally, before performing the final MR analysis, we conducted an MR-Pleiotropy RESidual Sum and Outlier (PRESSO) analysis to exclude potential pleiotropic variables and ensure the reliability of the results, ultimately obtaining the final instrumental variables.

2.3 MR Analysis

Inverse variance weighted (IVW) was the primary analytical method used in this study. Besides, four additional MR analytical methods, MR-Egger regression, weighted median estimator (WME), simple modeling, and weighted modeling, were employed to examine the relationship between exposure and outcome in supplementary causal analyses. The odds ratio (OR) and 95% CI were used to estimate the causal effect of exposure on the outcome. Scatter plots were used to visualize the causal relationship between exposure and outcome.

We incorporated data sources from GWAS pertaining to potential mediators, including BMI, diabetes, and COPD, to examine possible intermediate factors that may lie on the causal pathway from educational attainment to SUI (Table 1). First, the effects of exposure (educational attainment) on the mediators (BMI, diabetes, and COPD) were determined using a univariable model by performing a regression analysis of the mediators with respect to the exposure. Next, the effects of the three mediators on the outcome (SUI) were estimated using a univariable model by regressing the outcome on each mediator. The indirect effect of the exposure on the outcome was determined by calculating the product of the two regression estimates obtained from mediation analysis. The total effect represents the overall causal impact of the exposure on a specified outcome. The proportion of mediation can be determined by calculating the ratio of the indirect effect to the total effect, employing the product of coefficients method. Finally, a Sobel test was performed to explore whether the mediating effects were significant (http://www.quantpsy.org/sobel/sobel.htm).

To assess the robustness of the findings, sensitivity analyses were conducted, including heterogeneity tests, pleiotropy tests, funnel plot, and leave-one-out sensitivity analysis. Cochran's Q test was used to determine whether heterogeneity was present among the instrumental variables, considering a p-value < 0.05 to be statistically significant. MR-Egger was used to explore any potential bias introduced by pleiotropy ($p < 0.05$). The funnel plot was applied to visualize the symmetry of the distribution of the variant-specific effect estimates. Besides, to evaluate the sensitivity of individual SNPs, a leave-one-out sensitivity analysis was performed. All statistical analyses were conducted using R statistical software (version 4.2.3, R Foundation for Statistical Computing, Vienna, Austria).

3. Results

3.1 Screening of Instrumental Variables

After a rigorous quality control pipeline to identify and harmonize instrumental variables, the following numbers of SNPs were used for each analysis: education and SUI (n = 381), education and diabetes (n = 385), and diabetes and SUI (n = 140), as detailed in Table 2. The number of SNPs associated with education level and BMI was 356, and the number of SNPs associated with BMI and SUI was 270. The study identified 395 SNPs associated with education level and COPD, while 16 SNPs were associated with both COPD and SUI. Refer to Table 2 for further details.

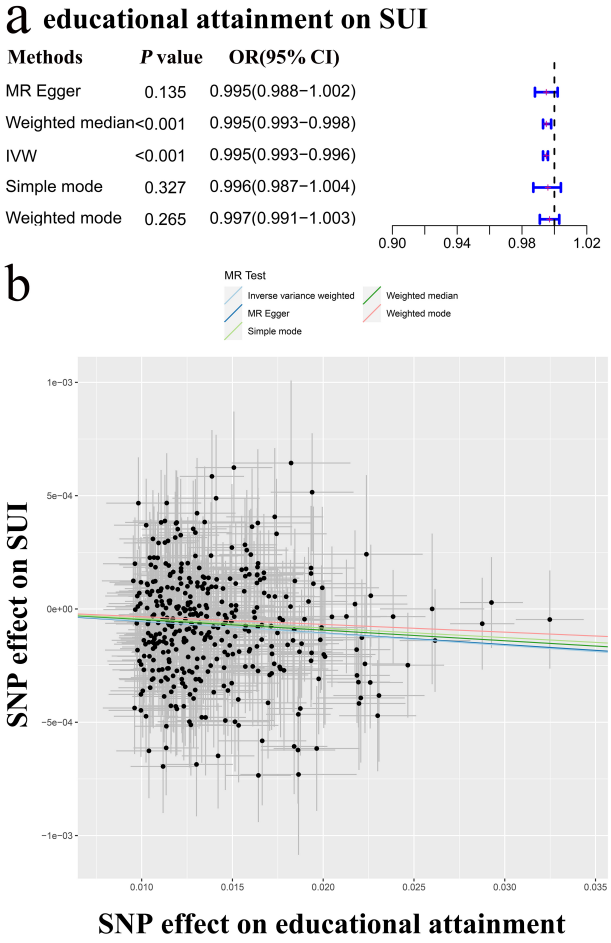

Fig. 2. Results of MR between educational attainment and SUI. (a) Forest plot. (b) Scatter plot. OR, odds ratio; IVW, inverse variance weighted.

3.2 MR Analysis Results

As shown in Figs. 2,3,4 and the **Supplementary Table 1**, five MR methods were applied, with the IVW method serving as the primary estimate. A causal relationship was observed between education level and the risk of SUI (OR = 0.995, 95% CI: 0.993–0.996, $p = 1.851 \times 10^{-10}$).

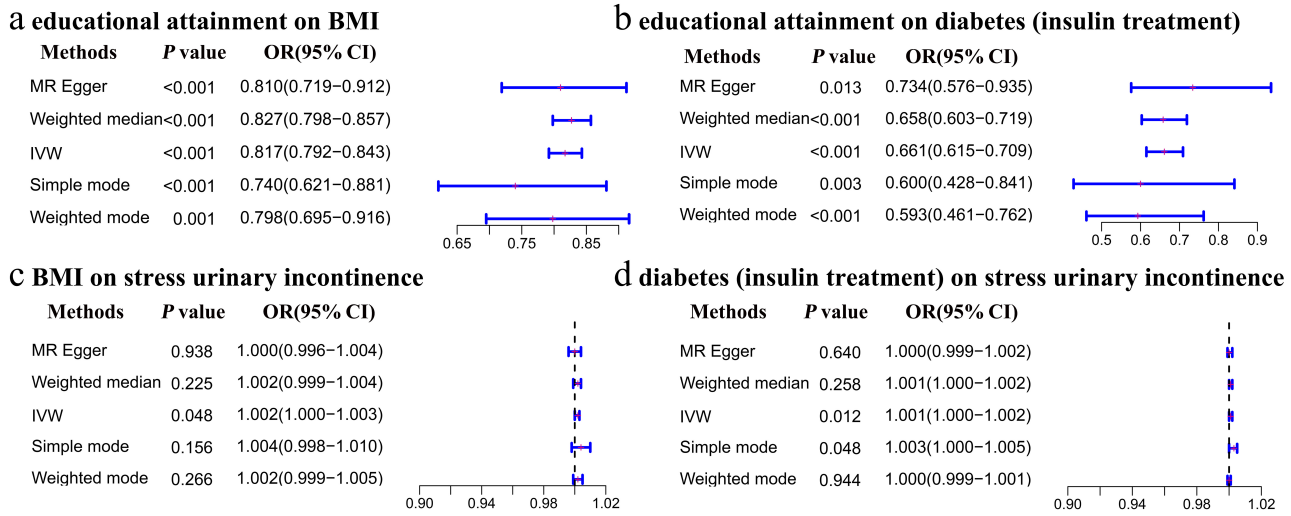

Fig. 3. Forest plots of MR analysis. (a) Educational attainment and BMI. (b) Educational attainment and diabetes (insulin treatment). (c) BMI and SUI. (d) Diabetes (insulin treatment) and SUI.

Genetically predicted higher educational attainment demonstrated a protective causal effect on BMI (OR = 0.817, 95% CI: 0.792–0.843, $p = 4.825 \times 10^{-37}$), while a causal relationship was observed between higher BMI and increased risk of SUI (OR = 1.002, 95% CI: 1.000–1.003, $p = 0.048$). Consistently, a causal relationship was found between higher educational attainment and lower risk of diabetes mellitus (OR = 0.661, 95% CI: 0.615–0.709, $p = 2.266 \times 10^{-30}$). However, the diabetes mellitus was associated with increased risk of developing SUI (OR = 1.001, 95% CI: 1.000–1.002, $p = 0.012$). According to genetically predicted causal associations, education level was inversely associated with the risk of COPD (OR = 0.513, 95% CI: 0.463–0.567, $p = 3.406 \times 10^{-38}$). However, the results of the IVW method did not show a significant association between COPD and SUI (OR = 0.999, 95% CI: 0.998–1.001, $p = 0.491$). The scatter plots are shown in Figs. 2,4 and the **Supplementary Fig. 1**. The results of the IVW analysis, both prior to and following the application of the MR-Pleiotropy RESidual Sum and Outlier (PRESSO) test, are comprehensively detailed in the **Supplementary Table 2**.

3.3 Results of Heterogeneity, Pleiotropy, and Sensitivity Analysis

As shown in Table 2, there was no significant heterogeneity for the causal estimates of educational attainment on SUI, insulin-treated diabetes on SUI, or COPD on SUI (all $p > 0.05$). However, there was significant heterogeneity between education attainment and BMI, between BMI and SUI, between education attainment and diabetes (insulin treatment), and between education attainment and COPD (all $p < 0.05$). The results of the MR-Egger intercept test showed that no MR analyses exhibited significant horizontal pleiotropy (all $p > 0.05$).

The funnel plot exhibited a largely symmetrical distribution of the included SNPs, with the exception of COPD on SUI, suggesting less SNP variability in the MR analysis (**Supplementary Figs. 2–4**). The results of the leave-one-out sensitivity analysis (**Supplementary Figs. 5–11**) showed that, except for the associations of BMI with SUI and COPD with SUI, the remaining SNPs consistently clustered on the same side of the null value after iteratively removing each SNP, further suggesting the robustness of the findings.

3.4 Reverse Mendelian Analysis

Based on the results of the reverse MR analysis, there was no causal relationship between SUI and education level (OR = 0.692, 95% CI: 0.185–2.597, $p = 0.586$).

3.5 Mediation MR Analysis

We performed a two-step MR analysis to investigate the mediating roles of BMI, COPD, and insulin-treated diabetes in the pathway from educational attainment to SUI. In the first step of the analysis, it was determined that, among the three potential mediators, high educational attainment was correlated with a reduction in BMI, COPD, and diabetes. In the subsequent phase of our analysis, we assessed the causal impact of the three mediators on the risk of SUI. Our findings provided causal evidence indicating that both BMI and diabetes yielded significant effects on the risk of developing SUI. However, no significant causal association was found between COPD and SUI. Consequently, only BMI and diabetes were identified as significant mediators.

The effect value of education level on SUI ($\beta 0$) was –0.005, the effect value of education level on BMI ($\beta 1$) was –0.202, and the effect value of BMI on SUI ($\beta 2$) was 0.002. Thus, the mediating effect of BMI accounted for $\beta\% = (\beta 1 \times \beta 2) / \beta 0 = 8.08\%$ ($p = 0.048$). The effect value of edu-

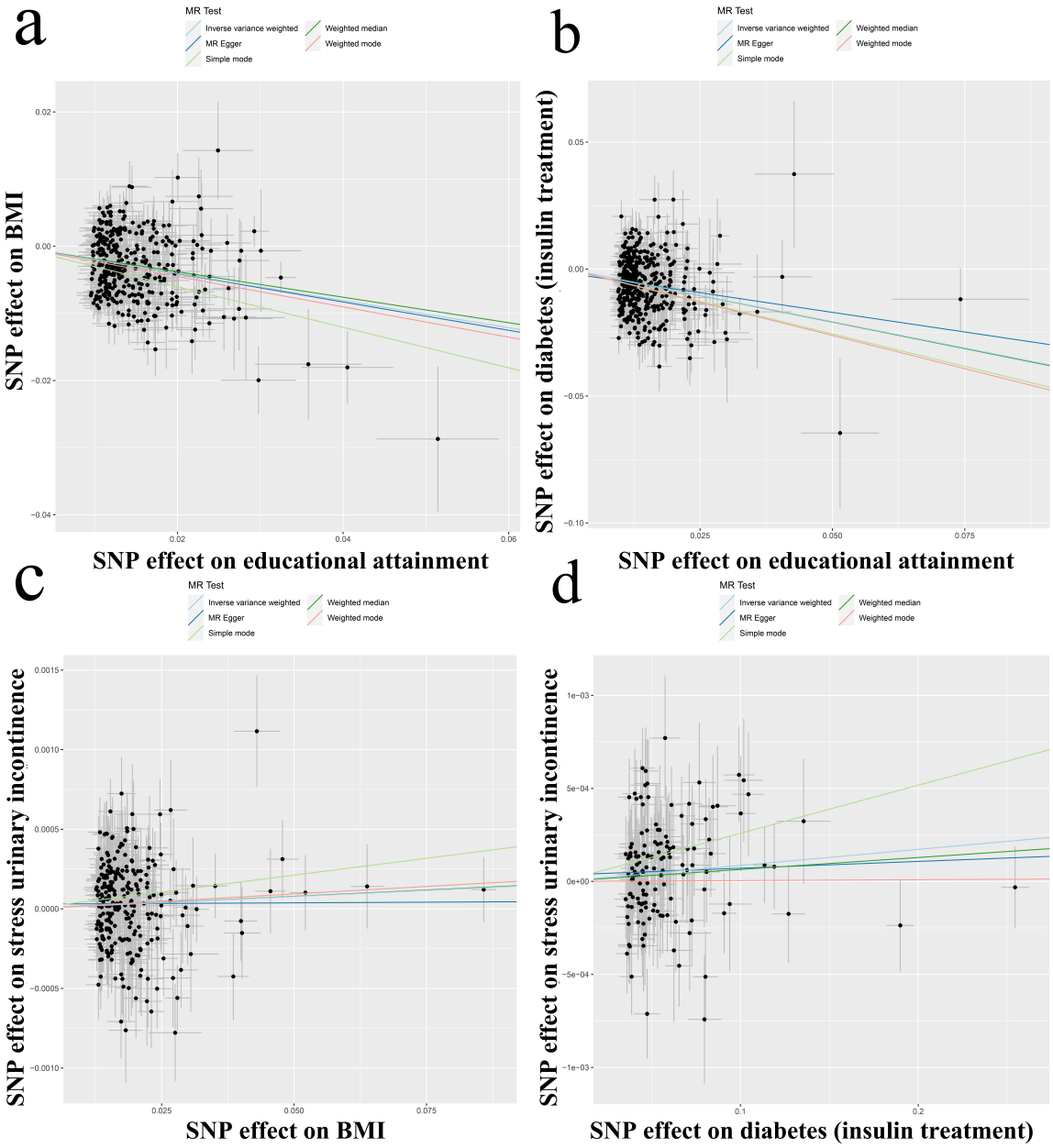

Fig. 4. Scatter plots of MR analysis. (a) Educational attainment and BMI. (b) Educational attainment and diabetes (insulin treatment). (c) BMI and SUI. (d) Diabetes (insulin treatment) and SUI.

cation level on the occurrence of diabetes ($\beta 1$) was –0.415, and the effect value of diabetes on the occurrence of SUI ($\beta 2$) was 8.609×10^{-4}. Therefore, the mediating effect of diabetes accounted for $\beta\% = (\beta 1 \times \beta 2) / \beta 0 = 7.47\%$ ($p = 0.004$) The results are comprehensively displayed in Table 3.

4. Discussion

In the present study, we investigated the causal relationship between educational attainment and the risk of developing SUI using MR analysis. Besides, the mediating roles of BMI, COPD and insulin-treated diabetes in this relationship were examined. The findings of the MR analysis indicated a significant causal relationship between educational attainment and BMI, COPD, insulin-treated diabetes and SUI. Further mediation analysis revealed that the protective effect of educational attainment on SUI was mediated by negatively modulating BMI and insulin-treated diabetes, with mediation effect ratios of 8.08% and 7.47%, respectively. Overall, higher educational attainment has a potential causal protective effect on SUI risk, and interventions targeting BMI and diabetes may be intervention targets to prevent SUI, particularly among individuals with lower education.

In recent years, several studies have demonstrated a causal relationship between educational attainment level

and the development of various diseases, including adverse pregnancy outcomes [20], chronic liver disease [21], gastroesophageal reflux disease [22], and Alzheimer's disease [23]. The results of this study substantiated that higher educational attainment could reduce the risk of SUI. However, few studies have focused on the relationship between educational attainment and SUI. A previous MR study [13] showed that a high level of education has a potential causal protective effect on SUI; however, the study did not explore the relevant mediating (risk or protective) factors for the occurrence of SUI. This gap limits the potential for early clinical intervention. For this reason, our study aimed to identify and quantify these mediators to inform more targeted prevention strategies.

The present study revealed that a higher educational attainment has a protective causal effect on the risk of insulin-treated diabetes and lower BMI, consistent with the literature [24,25]. Diabetes mellitus and BMI have potential causal harmful effects on the onset of SUI, and some previous studies have pointed out that diabetes mellitus and BMI are risk factors for SUI [14,26,27]. It is now understood that diabetes may affect pelvic floor microcirculation and bladder-detrusor muscle function [28] through several potential factors, such as insulin resistance [29,30] and peripheral neuropathy [31], thereby increasing the risk of SUI development. Elevated BMI has been associated with low-grade systemic inflammation and pro-inflammatory cytokine release, which generates reactive oxygen species and oxidative stress. These factors can alter collagen metabolism and, together with increased intra-abdominal pressure, lead to the development of SUI [32]. In the present study, mediation analysis revealed that insulin-treated diabetes and BMI may play a moderating role in the relationship between educational attainment and SUI. Patients with high educational attainment focused more on controlling their blood glucose levels, especially those with type 2 diabetes, by strengthening dietary control, increasing physical activity, and adhering to medication. Besides, this patient population tended to focus more on controlling their BMI at lower levels, which in turn helped manage abdominal pressure. In clinical practice, clinicians should pay closer attention to patients with diabetes mellitus who have a higher BMI and lower education level, especially female patients, to reduce their risk of developing SUI. The influence of educational attainment on SUI is complex and multidimensional. It encompasses variations in health literacy, healthcare accessibility, occupational demands, and lifestyle behaviors, among other factors [33,34]. For example, individuals with limited health awareness may perceive SUI as an inevitable consequence of aging, resulting in delayed treatment and exacerbation of symptoms [35]. Consequently, it is imperative in clinical practice to allocate additional effort and time to enhance awareness of SUI among those patients with lower educational levels.

COPD is a prevalent, preventable, and treatable condition characterized by persistent airflow limitation that progressively worsens. The primary symptoms of COPD encompass cough, expectoration, wheezing, and dyspnea [36]. In 2004, Jackson *et al*. [37] reported that the incidence of SUI in women with COPD was more than five times higher than in those without COPD. During episodes of coughing, the contraction of the abdominal muscles induces a rapid increase in intra-abdominal pressure. This elevation in pressure subsequently raises the diaphragm, augments thoracic cavity pressure, generates a high expiratory flow rate, and compels the pelvic floor muscles to contract with considerable force. SUI might occur if this compensatory mechanism is inadequate or fails to counterbalance the increased pressure from coughing [38]. Nevertheless, the findings of our study suggest the absence of a definitive causal relationship between COPD and SUI, indicating the potential for a correlation rather than direct causation.

Limitations

The present study has several strengths and limitations. To our knowledge, this study is the first to explore the causal relationship between insulin-treated diabetes, BMI, and education level, mediated by genetic factors, in relation to the occurrence of SUI. A major limitation of our study is that the genetic associations for the exposure, mediators, and outcome were predominantly sourced from large-scale GWAS conducted in individuals of European ancestry. As a result, the causal estimates presented in this study may not be applicable to populations of other ancestries, such as African, Asian, or Hispanic. Second, the analysis was constrained by the lack of sex-disaggregated data for the outcome, preventing an assessment of causality specifically in women, who are disproportionately affected by SUI. From a biological perspective, metabolic risk factors such as obesity (high BMI) and diabetes may have a more significant impact on women. Besides, the leave-one-out analysis indicated that the associations between SUI and both BMI and COPD were unstable. Consequently, this result should be interpreted with caution. Accordingly, future validation of our findings in terms of other regions, ethnic groups, and gender subgroups is warranted.

5. Conclusions

In summary, higher educational attainment is associated with a lower risk of BMI, insulin-treated diabetes, and SUI, while BMI and diabetes are risk factors for the development of SUI. Besides, BMI and diabetes serve as important mediators between education level and the occurrence of SUI. This finding reminds clinicians to pay attention to patients with lower education levels, especially those with comorbid diabetes mellitus and higher BMI, in order to reduce the risk of developing SUI.

Availability of Data and Materials

All the GWAS data involved in our manuscript is publicly available (SSGAC, https://www.thessgac.org/; IEU OpenGWAS project, https://gwas.mrcieu.ac.uk/; FinnGen database, https://r11.finngen.fi/). The main relevant data is included in the manuscript and supplemental file, further inquiries can be directed to the corresponding author.

Author Contributions

The study conception and design were contributed by HW and SY. Material preparation, data collection, and analysis were carried out by HW, SD, and LZ. The initial draft of the manuscript was written by HW, SY, and SD. All authors contributed to critical revision of the manuscript for important intellectual content. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

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Conflict of Interest

The authors declare no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.31083/CEOG44660.

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