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Research Article

# Does androgenic alopecia aggravate the risk of prostate cancer? Evidence from Mendelian randomization

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A B S T R A C T

**Background:** Epidemiological reports indicate a potential association between androgenic alopecia (AGA) and increased prostate cancer (PC) prevalence, but conflicting reports also exist. This study aims to elucidate the causality of AGA on PC risk using Mendelian randomization (MR) analysis.
**Materials and methods:** Two-sample MR analyses utilized public genome-wide association studies summary data for single-nucleotide polymorphisms associated with AGA. Four statistical methods were used: inverse variance weighted (IVW), MR-Egger, weighted median, and weighted mode, with IVW as the preliminary estimation method. Additionally, sensitivity analyses were conducted to address pleiotropic bias.
**Results:** Genetically proxied AGA did not demonstrate a causal effect on PC risk (IVW $P > 0.05$). Consistently, complementary methods yielded results aligned with IVW.
**Conclusions:** Our MR analysis indicates no causal relationship between genetically predicted AGA and PC risk, suggesting that observed associations in epidemiological studies may not be causal.
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## 1. Introduction

Androgenetic alopecia (AGA) represents a hereditary and androgen-dependent dermatologic condition characterized by progressive scalp hair thinning following a specific pattern. As the most prevalent form of nonscarring alopecia, AGA stems from genetic predisposition and an exaggerated response of hair follicles to androgens, predominantly affecting males. This process gradually transforms terminal hair on the scalp into vellus hair.[1,2] While AGA itself is not life-threatening, it can significantly impact an individual's psychosocial well-being, particularly among females and younger males.

Androgens play a pivotal role in the pathophysiology of AGA. Testosterone, the primary and most potent androgen in males, undergoes conversion by type 2, 5-alpha reductase into dihydrotestosterone (DHT). Excessive DHT leads to hair follicle

shrinkage, replacing terminal hairs with vellus hairs and contributing to AGA progression.[3] Previous research has demonstrated that inhibiting DHT production can halt AGA progression and potentially facilitate hair regrowth.[4] Additionally, elevated levels of the androgen receptor have been associated with AGA, leading to follicle miniaturization and shortening the anagen phase of the hair cycle.[5]

As the second most common malignancy among men worldwide, prostate cancer (PC) has the highest incidence rate in Europe, with more than 100,000 patients dying from this disease every year.[6] Coincidentally, androgens have also been strongly implicated in PC carcinogenesis. Increased androgen-receptor activity and expression, along with androgen-receptor-gene mutations, promote PC growth.[7] However, attempts to establish a common pathophysiological link between AGA and PC have yielded inconsistent and inconclusive findings. Pooled meta-analyses have shown that certain degrees of alopecia do not affect PC incidence or cancer-specific mortality.[8] Similarly, Amoretti et al revealed the absence of a statistically significant association between AGA and PC utilizing a meta-analysis comprising seven case-control studies (4078 cases and 4916 controls).[9] Notably, certain studies reported similar results,[10] whereas others reported conflicting results. For instance, Jin et al conducted a meta-analysis of 17 studies, comprising 68,448

participants, and revealed that male pattern baldness was associated with an increased risk of aggressive PC.[11] Similarly, another screening trial indicated that frontal plus moderate vertex baldness at age 45 years was associated with an increased risk of aggressive PC.[12]

This controversy surrounding the relationship between AGA and PC primarily stems from observational studies, which are susceptible to residual confounding and reverse causation. Hence, in this study, a two-sample Mendelian randomization (MR) study is conducted to dissect the underlying causal effect of AGA and PC risk, which utilized germline genetic single-nucleotide polymorphisms (SNPs) as a randomized instrument of the exposure of interest to minimize the biases using summary statistics from large-scale genome-wide association studies (GWAS). The results of this study aim to provide more robust evidence elucidating the connection between AGA and PC.

## 2. Materials and methods

### 2.1. MR assumptions and study design

To ensure the validity of MR analysis, three key assumptions regarding instrumental variables (IVs) must be satisfied: (a) genetic variations must strongly associate with the exposure; (b) genetic variations should remain independent of both measured and unmeasured confounding factors in the exposure–outcome relationship; and (c) genetic variants must remain independent of the outcome given the exposure and confounders.[13] The study flowchart is illustrated in Fig. 1, and GWAS summary datasets for both exposures and outcomes are provided in Table 1.

### 2.2. Selection of IVs

IVs were selected based on a study by Rui L et al, which included 3891 cases and 8915 controls, yielding 645 IVs strongly associated with AGA.[14] From the AGA's GWAS summary data from FinnGen Biobank (https://www.finngen.fi), SNPs exhibiting strong correlations with AGA ($P$ value $< 5 \times 10^{-6}$) were retained as candidate IVs, ensuring a robust association between IVs and exposure factors, with an exception for F value < 10. This selection process used the formula: $F = (\beta/se)$.[2] The SNP linkage disequilibrium value ($r^2$) was set to 0.001, and a genetic distance of 10,000 kb was utilized to mitigate the impact of linkage disequilibrium and maintain the independence of the selected IVs.

### 2.3. MR analysis

Several statistical methods were employed, namely inverse-variance weighting (IVW, comprising fixed-effect and random-effects), MR-Egger, weighted median, and weighted mode. Among these, the IVW method was prioritized as the primary approach, providing reliable causal estimates even in the presence of heterogeneity.

### 2.4. Heterogeneity, pleiotropy, and sensitive analyses

Heterogeneity was assessed using the Cochran Q statistical test. The random-effects model IVW method was used when Cochran Q-derived $P$ value was <0.05; otherwise, the fixed-effects IVW method was used as the primary outcome. Directional and horizontal pleiotropy tests were conducted using the MR-Egger regression-derived intercept method and MR pleiotropy residual
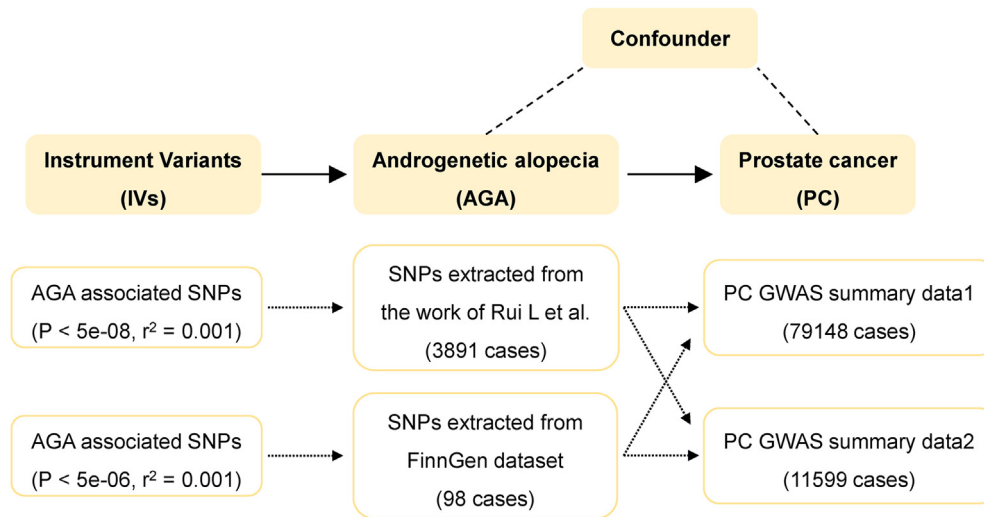


**Figure 1.** Schematic of the study design.

**Table 1**
GWAS summary datasets of exposures and outcomes used in this study.

| Trait | IEU GWAS id | Author | Consortium | Number of cases | Number of controls | Number of variants | Year | PMID | Population | Gender |
|---|---|---|---|---|---|---|---|---|---|---|
| Androgenic alopecia | NA | Rui L | MAAN | 3891 | 8915 | 2391230 | 2012 | 22693459 | European | Males |
| Androgenic alopecia | finn-b-L12_ALOPECANDRO | NA | FinnGen | 98 | 119087 | 16379661 | 2021 | NA | European | Males and females |
| Prostate cancer | ieu-b-85 | Schumacher | PRACTICAL | 79148 | 61106 | 20346368 | 2018 | 29892016 | European | Males |
| Prostate cancer | ebi-a-GCST90018905 | Sakaue S | NA | 11599 | 199628 | 24119306 | 2021 | 34594039 | European | Males |

Abbreviations: GWAS, genome-wide association studies; IEU, integrative epidemiology unit; PMID, PubMed unique identifier.

sum and outlier (MR-PRESSO) method, respectively.[13] Additionally, leave-one-out analyses were performed to evaluate the robustness of the MR analysis results by iteratively removing the SNPs one by one.

### 2.5. Statistical analysis

All two-sample MR analyses were conducted using R (version 4.2.2, https://www.rproject.org) and R package 'TwoSampleMR' (version 0.5.6, https://github.com/MRCIEU/TwoSampleMR). A Bonferroni-corrected two-side $P$ threshold of 0.0125 was considered statistically significant (0.05/4, 2 exposures and 2 outcomes). For sensitivity analysis, a $P$-value of <0.05 indicated a significant heterogeneity and horizontal pleiotropy.

## 3. Results

### 3.1. Genetic instruments

Following the specified parameters, seven variants and four variants were used as IVs for exposure 1(AGA_Rui L) and exposure 2 (AGA_FinnGen), respectively (Fig. 2). The Wald ratio method was used to assess the individual causal effects of each SNP on outcome 1 (Fig. 2A, PC, ieu-b-85) and outcome 2 (Fig. 2B, PC, ebi-a-GCST90018905).

### 3.2. Effects of genetically proxied AGA on PC risks

Fig. 3 illustrates the causal estimate of AGA on PC risk. All statistical methods indicated no significant causal relationship between AGA and PC (Fig. 3 and Supplemental Figure 1). Furthermore, no evidence of horizontal pleiotropy was observed, as evaluated by the interception of Egger regression (Table 2, all $P > 0.05$), suggesting that the outcomes remained unaffected by potential confounding pathways. These findings further support the validity and reliability of our results. Lastly, leave-one-out tests did not suggest a potential causality after excluding any one SNP (Supplemental Figure 2).

## 4. Discussion

Although AGA does not primarily affect physical health, it exerts a significant impact on patient's mental health and quality of life. The widely accepted role of androgens in AGA pathogenesis has led to the clinical use of finasteride, a specific inhibitor that targets type 2, 5-alpha reductase and blocks androgen conversion to DHT. Given the close association between androgens and PC progression, androgen deprivation therapy, such as abiraterone, which competitively binds to androgen receptors, is crucial to PC treatment,[15,16] despite its impact on cognitive function.[17]
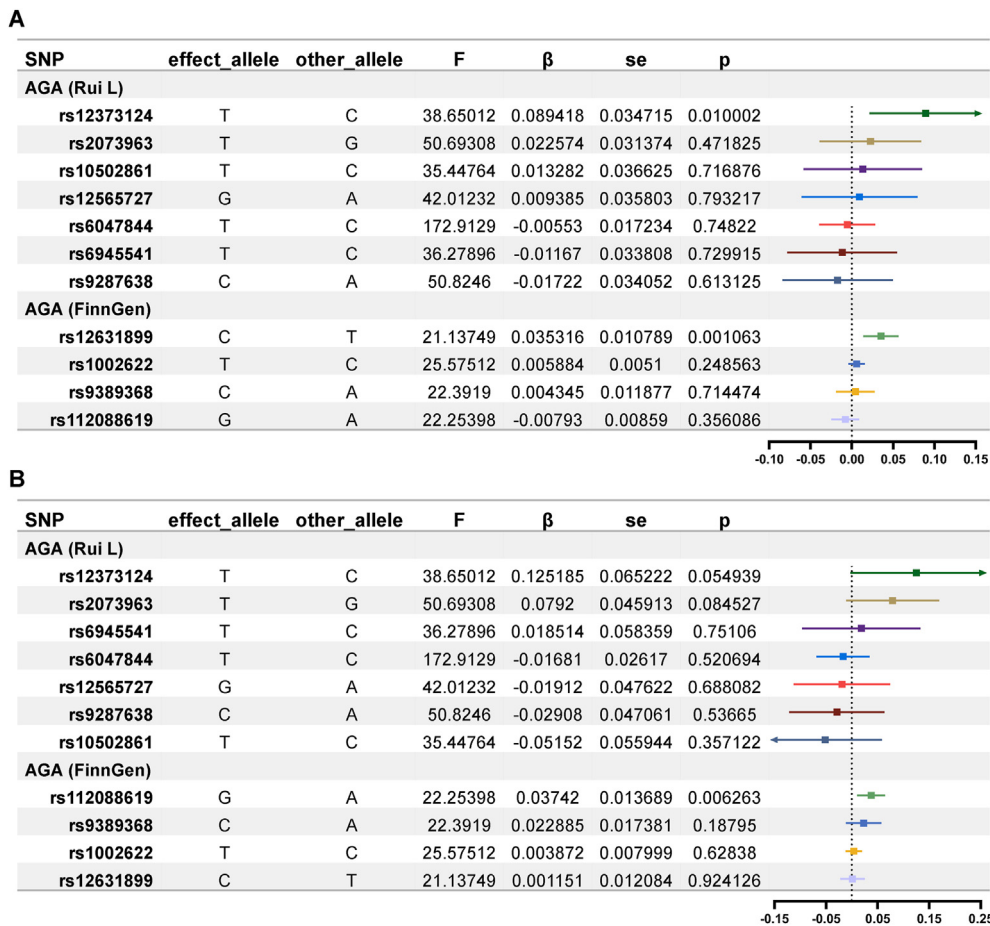
**A**

| SNP | effect_allele | other_allele | F | β | se | p |
|---|---|---|---|---|---|---|
| **AGA (Rui L)** | | | | | | |
| **rs12373124** | T | C | 38.65012 | 0.089418 | 0.034715 | 0.010002 |
| **rs2073963** | T | G | 50.69308 | 0.022574 | 0.031374 | 0.471825 |
| **rs10502861** | T | C | 35.44764 | 0.013282 | 0.036625 | 0.716876 |
| **rs12565727** | G | A | 42.01232 | 0.009385 | 0.035803 | 0.793217 |
| **rs6047844** | T | C | 172.9129 | -0.00553 | 0.017234 | 0.74822 |
| **rs6945541** | T | C | 36.27896 | -0.01167 | 0.033808 | 0.729915 |
| **rs9287638** | C | A | 50.8246 | -0.01722 | 0.034052 | 0.613125 |
| **AGA (FinnGen)** | | | | | | |
| **rs12631899** | C | T | 21.13749 | 0.035316 | 0.010789 | 0.001063 |
| **rs1002622** | T | C | 25.57512 | 0.005884 | 0.0051 | 0.248563 |
| **rs9389368** | C | A | 22.3919 | 0.004345 | 0.011877 | 0.714474 |
| **rs112088619** | G | A | 22.25398 | -0.00793 | 0.00859 | 0.356086 |

-0.10   -0.05   0.00   0.05   0.10   0.15

**B**

| SNP | effect_allele | other_allele | F | β | se | p |
|---|---|---|---|---|---|---|
| **AGA (Rui L)** | | | | | | |
| **rs12373124** | T | C | 38.65012 | 0.125185 | 0.065222 | 0.054939 |
| **rs2073963** | T | G | 50.69308 | 0.0792 | 0.045913 | 0.084527 |
| **rs6945541** | T | C | 36.27896 | 0.018514 | 0.058359 | 0.75106 |
| **rs6047844** | T | C | 172.9129 | -0.01681 | 0.02617 | 0.520694 |
| **rs12565727** | G | A | 42.01232 | -0.01912 | 0.047622 | 0.688082 |
| **rs9287638** | C | A | 50.8246 | -0.02908 | 0.047061 | 0.53665 |
| **rs10502861** | T | C | 35.44764 | -0.05152 | 0.055944 | 0.357122 |
| **AGA (FinnGen)** | | | | | | |
| **rs112088619** | G | A | 22.25398 | 0.03742 | 0.013689 | 0.006263 |
| **rs9389368** | C | A | 22.3919 | 0.022885 | 0.017381 | 0.18795 |
| **rs1002622** | T | C | 25.57512 | 0.003872 | 0.007999 | 0.62838 |
| **rs12631899** | C | T | 21.13749 | 0.001151 | 0.012084 | 0.924126 |

-0.15   -0.05   0.05   0.15   0.25

**Figure 2.** Instrumental variants of exposures utilized in this study.
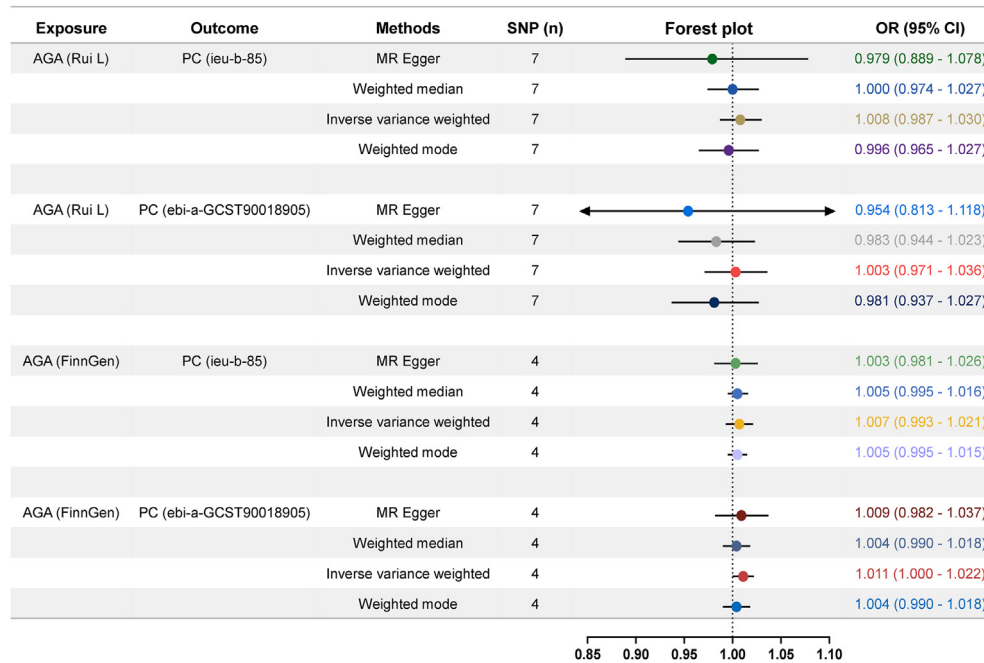
**Figure 3.** MR analysis illustrates the association between AGA and PC risk.
Abbreviations: AGA, androgenic alopecia; MR, Mendelian randomization; PC, prostate cancer.

**Table 2**
Sensitivity analyses of MR analysis of androgenic alopecia on prostate cancer.

| Exposure trait | Outcome trait | Methods | OR (95% CI) | P value | Heterogeneity/Cochran's Q | | Pleiotropy/Egger intercept | | MR-PRESSO | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Q | P value | Intercept | P value | Global test, P value | Outliers |
| AGA (Rui L) | PC (ieu-b-85) | IVW-FE | 1.008 (0.987−1.030) | 0.443 | 7.24 | 0.299 | 0.009 | 0.562 | 0.3457 | NA |
| | | IVW-RE | 1.008 (0.985−1.032) | 0.485 | | | | | | |
| AGA (Rui L) | PC (ebi-a-GCST90018905) | IVW-FE | 1.003 (0.971−1.036) | 0.871 | 8.54 | 0.201 | 0.016 | 0.552 | 0.2659 | NA |
| | | IVW-RE | 1.003 (0.965−1.042) | 0.891 | | | | | | |
| AGA (FinnGen) | PC (ieu-b-85) | IVW-FE | 1.007 (0.999−1.014) | 0.082 | 10 | **0.019*** | 0.009 | 0.707 | 0.0969 | NA |
| | | IVW-RE | 1.007 (0.993−1.021) | 0.34 | | | | | | |
| AGA (FinnGen) | PC (ebi-a-GCST90018905) | IVW-FE | 1.011 (1.000−1.022) | 0.051 | 5.65 | 0.13 | 0.005 | 0.856 | 0.2527 | NA |
| | | IVW-RE | 1.011 (0.996−1.027) | 0.156 | | | | | | |

Abbreviations: AGA, androgenic alopecia; CI, confidence interval; IVW-FE, fixed-effect inverse-variance weighting; IVW-RE, random-effects inverse-variance weighting; MR, Mendelian randomization; MR-PRESSO, MR pleiotropy residual sum and outlier; PC, prostate cancer.

Notably, the administration of finasteride has been reported to reduce the prevalence of PC by 25%,[18] whereas dutasteride, another approved drug for the treatment of male pattern hair loss, decreased the risk of PC by 23% over four years.[19] However, controversy persists regarding the association between AGA and PC risk. In this study, we used an MR design to elucidate the causal association between AGA and PC. Our results suggest that genetically determined AGA is not causally linked to PC, proving genetic evidence challenging the role of AGA in PC tumorigenesis. This underscored the need for high-quality, well-designed, randomized controlled trials to further investigate this matter.

Several studies indicating negative associations have highlighted a potential link between vertex baldness and PC risk. While frontal plus vertex baldness at age 45 years has not been demonstrated to be significantly associated with overall or nonaggressive PC risk compared to no baldness, however, it has been linked to an increased risk of aggressive PC.[12] Amoretti et al reported the absence of a statistically significant association between AGA and PC, except for vertex baldness.[9] Another study by He H et al demonstrated a statistically significant association between vertex baldness and PC using subgroup analysis; however, such an association was not observed in other types of baldness.[20] Hence, pattern-specific correlations between AGA and PC risk warrant further investigation.

As early as 1991, some scholars pointed out that the dermal papilla cells of bald hair follicles contain higher levels of androgen receptors than nonbald scalps, and the receptor levels of vertex were significantly higher than those on the occiput, which explained why different types of baldness occurred under the same stimulation of plasma free androgen.[21,22] Based on this premise, our team members speculated that the risk of PC would indeed increase under the same stimulation of plasma free androgens. However, on the other hand, the vertex area was more sensitive to androgens due to higher levels of androgen receptors. Therefore, it was theoretically easy to establish a positive correlation between PC risk and vertex baldness. Considering that the observational studies are susceptible to residual confounding factors and reverse causation, we use a two-sample MR study, which utilized germline genetic SNPs of the exposure of interest to minimize the biases. However, our study's reliance on GWAS summary data makes conducting additional baldness pattern subgroup analyses challenging. In addition, the prevalence of AGA

also varies by age and race, with vertex AGA at age 40 potentially serving as a marker for early-onset PC risk.[23] Francesca Lolli et al also suggested that up to 30% of white men are affected with AGA by the age of 30, increasing to 50% by the age of 50 and 80% by the age of 70. Furthermore, Caucasians tended to be more affected by AGA than other racial groups.[24] Thus, the baldness pattern-, age- and race-specific correlations between AGA and PC cannot be stratified, representing a limitation of this study, suggesting that caution is warranted when interpreting this finding.

Over the past two decades, GWAS studies have identified numerous susceptibility loci associated with PC.[25–30] Notably, these loci do not coincide with the two predominant susceptibility loci, which are located in the androgen-receptor region at Xq11-12 and in the region between PAX1 and FOXA2 on chromosome 20p11.22, discovered in a GWAS of AGA.[31] This genetic disparity supports our study's conclusions. However, given that the differences in androgen-receptor levels and androgen activation mechanisms between vertex baldness and other types of baldness, it is particularly crucial to identify unique vertex baldness-specific susceptibility loci and attempt to re-establish a link to PC risk, which warrants further exploration.

We note several limitations in our research. First, we only included European populations to mitigate heterogeneity issues, limiting generalizability. The result remains to be further verified in the whole population. Second, the selection of exposure 2 from the FinnGen Biobank, comprising a sex-combined population, could introduce a bias. Third, while diverse pleiotropy robust approaches were used, residual horizontal pleiotropy bias cannot be entirely ruled out. Nevertheless, MR-Egger analysis found no evidence of pleiotropic effects, and sensitivity analyses produced consistent results. Despite these limitations, it is important to acknowledge the strengths of this study. A significant strength lies in the utilization of the MR design to establish the causal relationship between AGA and PC risk, which used randomly allocated genetic variants as IVs to address the limitations of previous observational studies, such as confounders and reverse causation. Furthermore, we screened the IVs of exposure from two separate GWAS datasets to minimize bias.

## 5. Conclusion

This MR study reveals the lack of a causal relationship between genetically predicted AGA and PC risk. This suggests that observed associations between AGA and PC in certain epidemiological studies may not reflect a causal link.

## Conflicts of interest

The authors declare no conflict of interest.

## CRediT authorship contribution statement

XS designed this work and drafted the initial manuscript. XS and YP acquired and analyzed the data. JL, FL, and BL helped analyze the data. YH made contributions to data visualization. KC made contributions to the conception of work and revised the manuscript. All authors read and approved the final manuscript.

## Ethics Statement

The summary-level data were sourced from published, publicly available datasets, which were approved by the institutional review committees in their respective studies. Secondary analysis of summary data does not necessitate an ethical permit.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.prnil.2024.04.001.

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