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# Associations between psoriasis and risk of 33 cancers: a Mendelian randomization study

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## Abstract

**Background** Several observational studies have reported epidemiologic associations between psoriasis and risk of some cancers, but systematic evidence is lacking. Our aim was to comprehensively estimate the association between psoriasis and the risk of 33 common cancers using systematical Mendelian randomization based on genetic data.

**Method** Forty-nine independent single-nucleotide polymorphisms (SNPs) significantly associated with psoriasis were extracted as instrumental variables from a large-scale meta-analysis study of genome-wide association study (GWAS) for psoriasis. Outcome GWAS data were obtained from the FinnGen consortium ($n=500{,}348$), UK Biobank ($n=420{,}531$), and other large-scale cancer datasets. The inverse-variance weighted (IVW) was used as the primary method to infer the association between psoriasis and risk of cancer, and finally the results from multiple databases were pooled by meta-analysis.

**Results** In the UK Biobank, genetically predicted psoriasis had a suggestive association with colon (OR = 1.055, 95%CI: 1.001–1.113, $P=0.046$) and uterine corpus cancer (OR = 0.922, 95%CI: 0.852–0.997, $P=0.042$). In the FinnGen consortium, psoriasis had a suggestive association with vulvar cancer (OR = 1.182, 95%CI: 1.023–1.366, P = 0.024), uterine corpus cancer (OR = 0.937, 95%CI: 0.883–0.993, $P=0.028$), and prostate cancer (OR = 0.973, 95%CI: 0.948–0.999, $P=0.045$). In an additional large-scale cancer dataset, psoriasis also showed a suggestive association with prostate cancer (OR = 0.968, 95%CI:0.942–0.995, $P=0.020$). The meta-analysis confirmed the suggestive association of psoriasis with uterine corpus (OR = 0.931, 95% CI: 0.889–0.976, $P=0.003$) and prostate cancer (OR = 0.976, 95% CI: 0.955–0.997, $P=0.023$). Whereas the effect of psoriasis on colon and vulvar cancer was not in the same direction across different populations. Furthermore, no association between genetically predicted psoriasis and other cancers were observed.

**Conclusions** This comprehensive MR study suggests that psoriasis may be a potential protective factor for uterine corpus cancer in women and prostate cancer in men.

**Keywords** Cancer, Psoriasis, Association, Mendelian randomization, UK biobank, FinnGen consortium

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## Introduction

Psoriasis is an immune-mediated, systemic, chronic, inflammatory skin disease characterized by the appearance of clearly demarcated red papular scaly plaques on the skin [1]. As a common and incurable chronic disease, psoriasis affects more than 60 million people worldwide. Its prevalence ranges from 0.1% to 1.99% in different regions. However, the prevalence in high-income and European countries commonly exceeds 2% and can even be as high as 10% [2, 3]. Worryingly, the prevalence of psoriasis is steadily increasing [3, 4]. Psoriasis has a clear genetic predisposition and is a polygenic genetic disease with interactions between multiple factors, including genetic and environmental factors, and is associated with many diseases, including cardiovascular disease (CVD), central nervous system, and cancer [5]. The most recent genome-wide association studies (GWAS) have identified 63 susceptibility loci for psoriasis and explain approximately 28% of the heritability [6].

Some studies showed that patients with psoriasis appear to have a slightly increased risk of cancer, with an overall prevalence of 4.78% and an incidence of 11.75 per 1000 people per year among patients with psoriasis [7]. In addition, several observational studies have shown that psoriasis is associated with an increased risk of some cancers. For example, Lee et al. found that psoriasis increased the risk of testicular cancer in Koreans [8]; He et al. found that psoriasis increased the risk of lung cancer in Europeans by approximately 60% [9]. However, the inherent limitations of observational studies, such as confounding factors, information bias, and reverse causality, may make the observed association between psoriasis and cancer subject to incidental findings and thus require further validation.

Mendelian randomization (MR) analysis is a methodological approach that uses genetic variants as instrumental variables to examine evidence for potential causal links between exposures and outcomes. The random allocation of genetic alleles during meiosis reduces susceptibility to certain biases inherent in conventional observational studies, such as environmental confounding. However, it should be noted that MR inferences may still be influenced by residual biases including pleiotropy and population stratification, particularly when utilizing GWAS data from diverse populations [10–14]. Genome-wide association studies (GWAS) in psoriasis have identified 63 significant loci and provided complete summary statistics [6]. We obtained GWAS data for 33 cancers from UK Biobank and FinnGen. Additionally, some other large-scale cancer databases have also been included. All these provided robust data for conducting MR analyses.

Two previous Mendelian randomization analysis studies explored the causal relationship between psoriasis and lung cancer, but the findings were inconsistent [15, 16]. To obtain a more comprehensive perspective, we used Mendelian randomization analysis to systematically assess the association of psoriasis with the risk of 33 common cancers and synthesized the results by meta-analysis. The results of this study could provide a valuable reference for cancer screening and surveillance of patients with psoriasis.

## Methods

### Study design

The design of this study is shown in Fig. 1. We used two-sample Mendelian randomization approach to study whether there was an association between psoriasis and 33 cancers risk. SNPs that were significantly associated with psoriasis were used as instrumental variables to proxy for psoriasis. Cancer data were obtained from multiple independent GWAS databases, UK-Biobank, FinnGen Consortium, and other large-scale cancer databases, and the results were finally combined using meta-analysis. This study adhered to the STROBE-MR statement (Supplementary file 1) [17]. This study was not pre-registered.

### Psoriasis GWAS and selection of instrumental variables

We obtained genetic instruments from the hitherto largest genome-wide association meta-analysis of psoriasis which included 13,229 psoriasis cases and 21,543 controls, with cases diagnosed by experienced physicians. All cases were from European ancestry, and no significant heterogeneity was reported. This study identified 63 psoriasis susceptibility loci, which explained about 28% of genetic heritability [6]. For the selection of instrumental variables, we extracted single nucleotide polymorphisms (SNPs) that were significantly associated with exposure at the genome-wide level ($P < 5e^{-08}$). To ensure the independence of instrumental variables, an aggregation algorithm with $r^2 < 0.001$ and kb = 10,000 was used to avoid linkage disequilibrium (LD). We identified SNPs significantly associated with cancer or some confounders by searching the PhenoScanner V2 database [18]. If a candidate instrumental variable for psoriasis has been found in previous studies to be associated with cancer, smoking, alcohol consumption, or obesity at a genome-wide level ($P < 5 \times 10^{-8}$), it will be excluded. Only one SNPs (rs34517439) was excluded because it was directly associated with lung cancer. To avoid bias introduced by weak instrumental variables, the F statistic $(F = beta/se)^2$ was used to assess the strength of association of each SNP with exposure. We considered a strong association with exposure when the F value >10 [19]. The final 49 SNPs were obtained, as shown in *Supplementary file 2.*

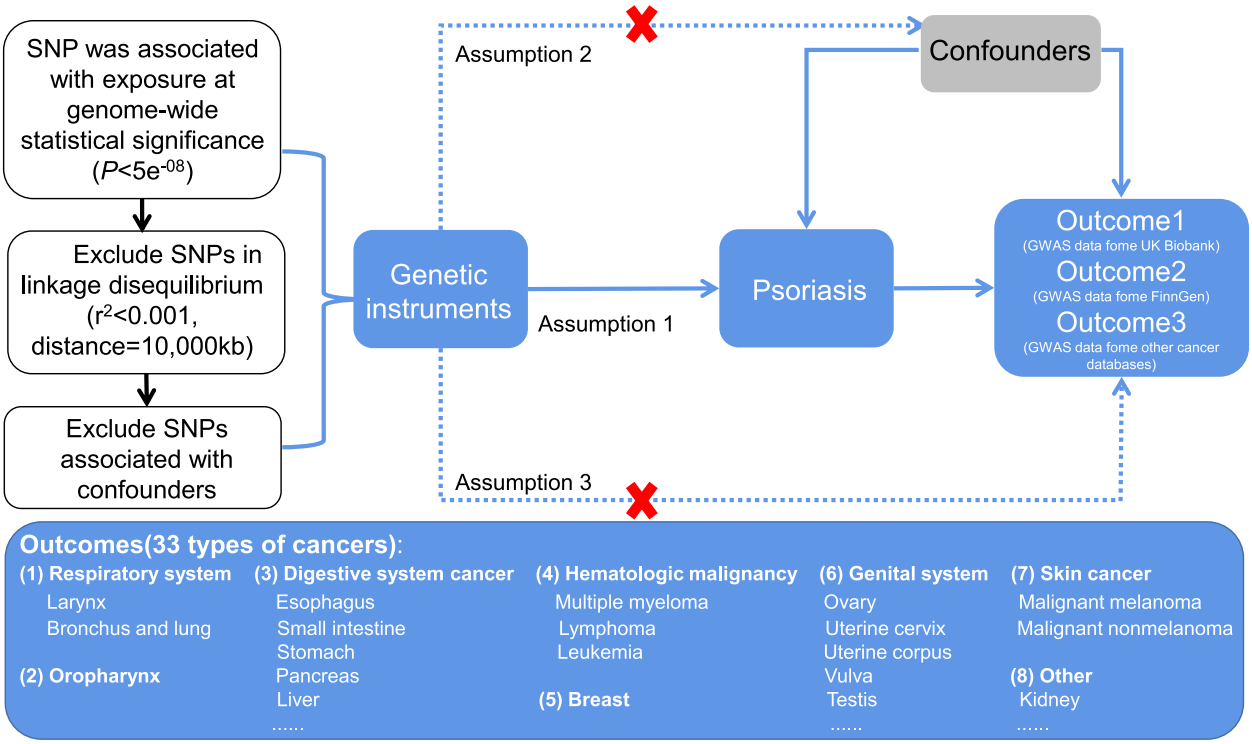


**Fig. 1** Research Design of Mendelian randomization. The outcome data for this Mendelian randomization study were obtained from UK Biobank, FinnGen Consortium, and other large-scale cancer databases. Assumption 1 (relevance assumption): There is a strong association between genetic variants and exposure factors. Assumption 2 (independence assumption): Genetic variants are independent of confounders affecting "exposure and outcome". Assumption 3 (exclusion restriction assumption): Genetic variation can only contribute to outcome through exposure, but not through other pathways

### Data sources for cancer

We obtained effect size estimates associated with cancer from the FinnGen consortium, UK Biobank and other large-scale cancer databases (Supplementary file 3). For FinnGen, we used the latest publicly released data FinnGen R12 version (https://r12.finngen.fi/) with a total sample size of 500,348 (282,064 women and 218,284 men), which include 2,502 available disease endpoints (phenotypes). The UK Biobank collects biological and medical data from 500,000 people aged between 40 and 69 living in the UK, including large-scale gene-wide association data for a wide range of cancer categories (https://pan.ukbb.broadinstitute.org/). Other large-scale cancer datasets include oropharynx cancer, bronchus and lung cancer, liver and intrahepatic bile ducts cancer, colorectum cancer, bladder cancer, prostate cancer, brain cancer, thyroid cancer and breast cancer (https://gwas.mrcieu.ac.uk/). Cancer diagnosis relies on International Classification of Diseases (ICD codes). The cancer classifications in UK Biobank, FinnGen Consortium and other databases are shown in the Supplementary file 3. UK Biobank, FinnGen Consortium and other databases were approved by the relevant ethical review boards and participants provided informed consent.

Our study evaluated the potential association between psoriasis and the risk of 33 common cancers, including respiratory system cancers: oropharynx, larynx, bronchi and lungs; digestive system cancers: esophagus, stomach, small intestine, pancreas, liver and intrahepatic bile ducts, extrahepatic bile ducts (including gallbladder), colon, rectum; urinary system cancers: kidney and bladder; reproductive system cancers: ovary, uterine cervix, uterine corpus, vulva, testes, prostate; malignant hematologic diseases: multiple myeloma, Hodgkin's lymphoma, non-Hodgkin's lymphoma, acute myeloid leukemia and acute lymphocytic leukemia, chronic myeloid leukemia and chronic lymphocytic leukemia; skin cancers: melanoma and malignant non-melanoma; other: brain, eye and adnexa, thyroid, breast, bone. Both psoriasis and cancers were dichotomized variables.

### MR analysis

We used the inverse variance weighted (IVW) model as the primary analysis to assess the association between psoriasis and cancer risk, and we also used the weighted

median (WM) and MR-Egger methods as additional analyses to assess the robustness of the results [20]. The result is shown in the Supplementary file 4. In addition, we performed a leave-one-out sensitivity analysis to check whether the results were dominated by any single SNP. These results were presented as odds ratios (ORs) and 95% confidence intervals (CIs). This study used Cochran's Q-test to assess heterogeneity between individual instrumental variables, which was considered significant when the p-value was < 0.05. To account for potential heterogeneity, we employed a random effects IVW model as the primary analysis to provide more conservative estimates. We used the MR Egger intercept method to test the horizontal pleiotropy of MR analysis [21]. The Mendelian randomization pleiotropy residual sum and outlier (MR-PRESSO) test was used to detect the potential heterogeneity and horizontal pleiotropy [22]. When outliers were present, we repeated the main analysis and the heterogeneity and pleiotropy analysis after removing the outliers. In addition, we conducted post hoc power calculations for the primary IVW analyses using an online web-based calculator (https://sb452.shinyapps.io/power/), with the significance level set to 0.05 [23]. Afterwards, we conducted a meta-analysis of the multiple results using a random effects model to draw final conclusions.

For *P* values, Bonferroni correction was applied. Statistical significance was considered to exist when $P < 0.0015$ (0.05/33 [1 exposure × 33 outcomes]), and suggestive association was considered to exist when *P* values ranged between 0.0015 and 0.05. We used R software to analyze the data and construct graphs.

## Results

We extracted instrumental variables significantly associated with psoriasis ($P < 5 \times 10^{-8}$) and removed linkage disequilibrium (LD) ($r^2 < 0.001$, 10,000 kb) from the hitherto largest genome-wide association meta-analysis of psoriasis. Subsequently, in the PhenoScanner database, we found a psoriasis instrumental variable SNP (rs34517439) directly associated with lung cancer and therefore was excluded. Also, palindromic SNPs (SNPs whose alleles consisted of bases and complementary bases) were ruled out. We did not replace missing variants with proxies as a way to ensure the consistency of SNPs used as instrumental variables in the analysis. Finally, the 49 SNPs screened were included in the study for further analysis (Supplementary file 2). GWAS data for both psoriasis and cancer were derived from European ancestry. There was no overlap between exposure and outcome data.

For the UK Biobank, at Bonferroni-corrected significance thresholds, a suggestive association between genetically predicted psoriasis and high-risk colon cancer (OR = 1.055, 95%CI: 1.001–1.113, *P* = 0.046) and low-risk uterine corpus cancer (OR = 0.922, 95%CI: 0.852–0.997, *P* = 0.042) was found, but not statistically significant (Fig. 2). The effect of psoriasis on colon cancer was inconsistent in direction in the FinnGen consortium database (OR = 0.990, 95% CI: 0.947–1.034, *P* = 0.638), while the effect on uterine corpus cancer was consistent (OR = 0.937, 95% CI: 0.883–0.993, *P* = 0.028). The post hoc statistical power for the IVW results of colon cancer and uterine corpus cancer were 33.5% and 30.8%, respectively. Power analyses for other cancer types were also low, with most being below 10% (Supplementary file 5). In addition, heterogeneity analysis and horizontal pleiotropy analysis showed that the association between psoriasis and colon and uterine corpus cancers was robust and there was no potential heterogeneity and horizontal pleiotropy. The leave-one-out sensitivity analysis showed that these results were robust (Supplementary file 6). Although there was significant heterogeneity in laryngeal cancer, liver and intrahepatic cholangiocarcinoma and melanoma, the results did not change significantly after removal of outliers using the MR-PRESSO test. Although there was significant heterogeneity in oropharyngeal, bronchial and lung cancers, multiple myeloma and non-Hodgkin's lymphoma, no outliers were found after MR-PRESSO test.

For the FinnGen, after Bonferroni correction, we observed the suggestive associations of genetically predicted psoriasis with vulvar (OR = 1.182, 95%CI: 1.023–1.366, *P* = 0.024), uterine corpus (OR = 0.937, 95%CI: 0.883–0.993, *P* = 0.028), and prostate (OR = 0.973, 95%CI: 0.948–0.999, *P* = 0.045) cancers (Fig. 2). However, in the UK Biobank data, the direction of effect of psoriasis and vulvar cancer was opposite (OR = 0.960, 95%CI: 0.739–1.246, *P* = 0.757), while the effect on uterine corpus cancer (OR = 0.922, 95%CI: 0.852–0.997, P = 0.042) and prostate cancer (OR = 0.980, 95% CI: 0.945–1.016, P = 0.268) were consistent. Besides, there was no significant heterogeneity and horizontal pleiotropy between psoriasis and vulvar cancer. In the statistical power analysis, the power of IVW results for vulvar, uterine corpus, and prostate cancers was 38.5%, 43.2%, and 40.7%, respectively. The power of MR results for most other cancer types was around 10% (Supplementary file 5). There was horizontal pleiotropy in uterine corpus cancer, but no outliers were found after MR-PRESSO test. The leave-one-out sensitivity analysis showed robust results and the results did not change significantly after removing outliers by MR-PRESSO test (Supplementary file 7), although there was significant heterogeneity in bronchial and lung cancer, rectum cancer, malignant nonmelanoma skin and breast cancer. In addition, for stomach cancer, colon cancer, kidney cancer, chronic lymphocytic leukaemia and brain cancer with significant heterogeneity, no outliers

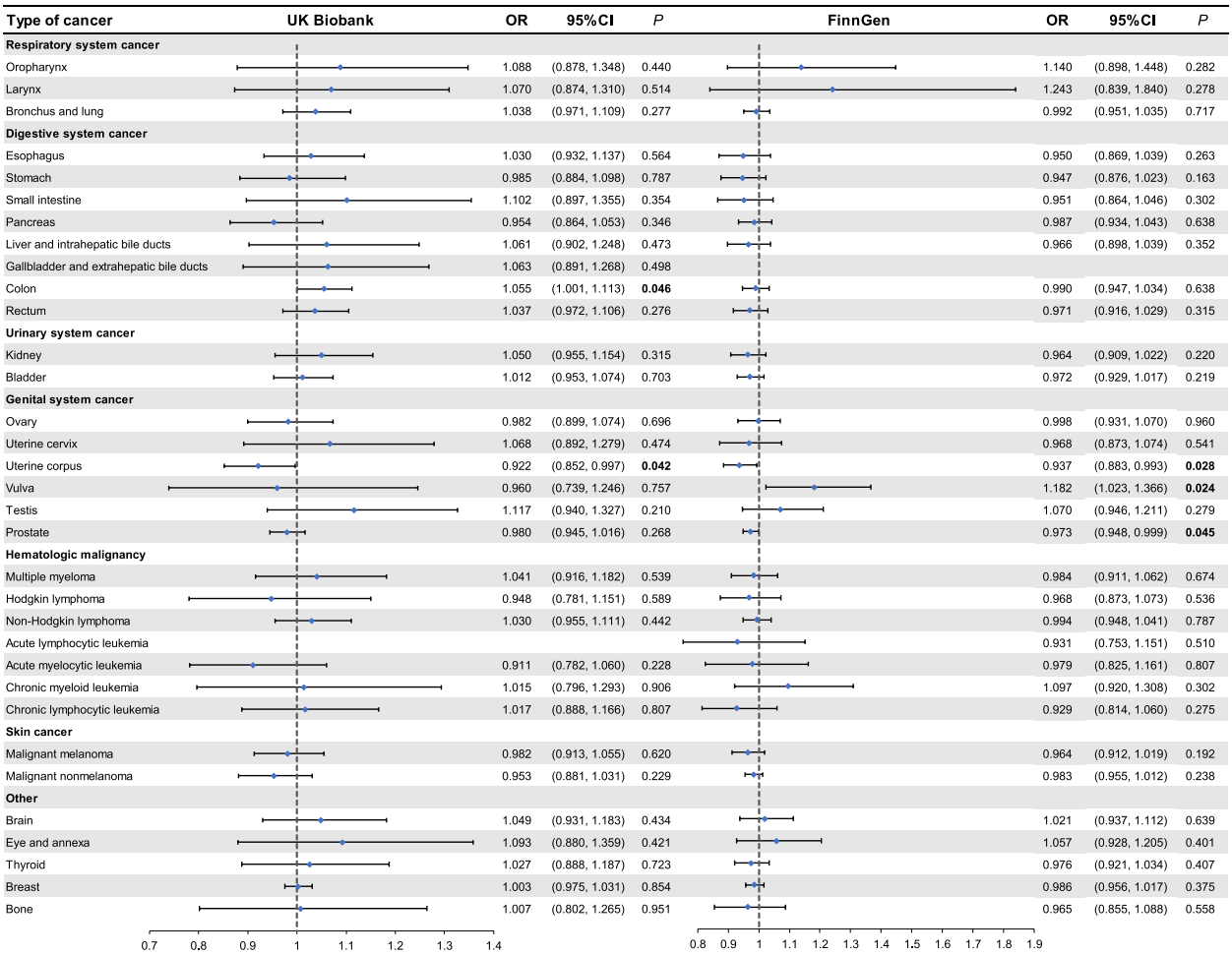


**Fig. 2** Forest plot of MR analysis results in the UK Biobank and the FinnGen. Abbreviations: OR: odds ratio; CI: confidence interval

were found after MR-PRESSO test. Initial IVW analysis showed no significant association between genetically predicted psoriasis and melanoma risk (OR = 0.964, 95% CI: 0.912–1.019, $P$ = 0.192) but a high degree of heterogeneity ($P$ = 5.8 $\times 10^{-5}$). MR-PRESSO identified one aberrant SNP whose heterogeneity remained significant after exclusion ($P$ = 0.011), but the IVW result changed to a potential protective effect (OR = 0.946, 95% CI: 0.901–0.994, $P$ = 0.026). However, the weighted median method (OR = 0.949, 95% CI: 0.893–1.009, $P$ = 0.093) and MR-Egger (OR = 0.984, 95% CI: 0.895–1.082, $P$ = 0.740) results did not support robust associations. Interestingly, the cancer-promoting effects of psoriasis therapeutic agents (such as tofacitinib) in observational studies contradicted the protective direction of genetic tools [24]. In summary, current evidence is insufficient to support a potential association between psoriasis and melanoma.

For other large-scale cancer databases, after Bonferroni correction, a suggestive association between genetically predicted psoriasis and reduced risk of prostate cancer (OR = 0.968, 95%CI: 0.942–0.995, $P$ = 0.020) was shown (Supplementary file 4, Supplementary file 8). There was no significant heterogeneity and horizontal pleiotropy in analysis of psoriasis and prostate cancer. In the statistical power analysis, the power of the association between psoriasis and prostate cancer was 35.8% (Supplementary file 5). In addition, no significant association of psoriasis with other cancers was observed.

The pooled results of the meta-analysis for the UK biobank and FinnGen are shown in Fig. 3, which suggested a suggestive association between genetically predicted psoriasis and uterine corpus cancer (OR = 0.931, 95% CI: 0.889–0.976, $P$ = 0.003) and prostate cancer (OR = 0.976, 95%CI: 0.955–0.997, $P$ = 0.023). Meta-analyses incorporating additional cancer datasets yielded similar results (Supplementary file 9). In addition, two meta-analyses consistently showed genetically predicted psoriasis to have no association with other cancers.

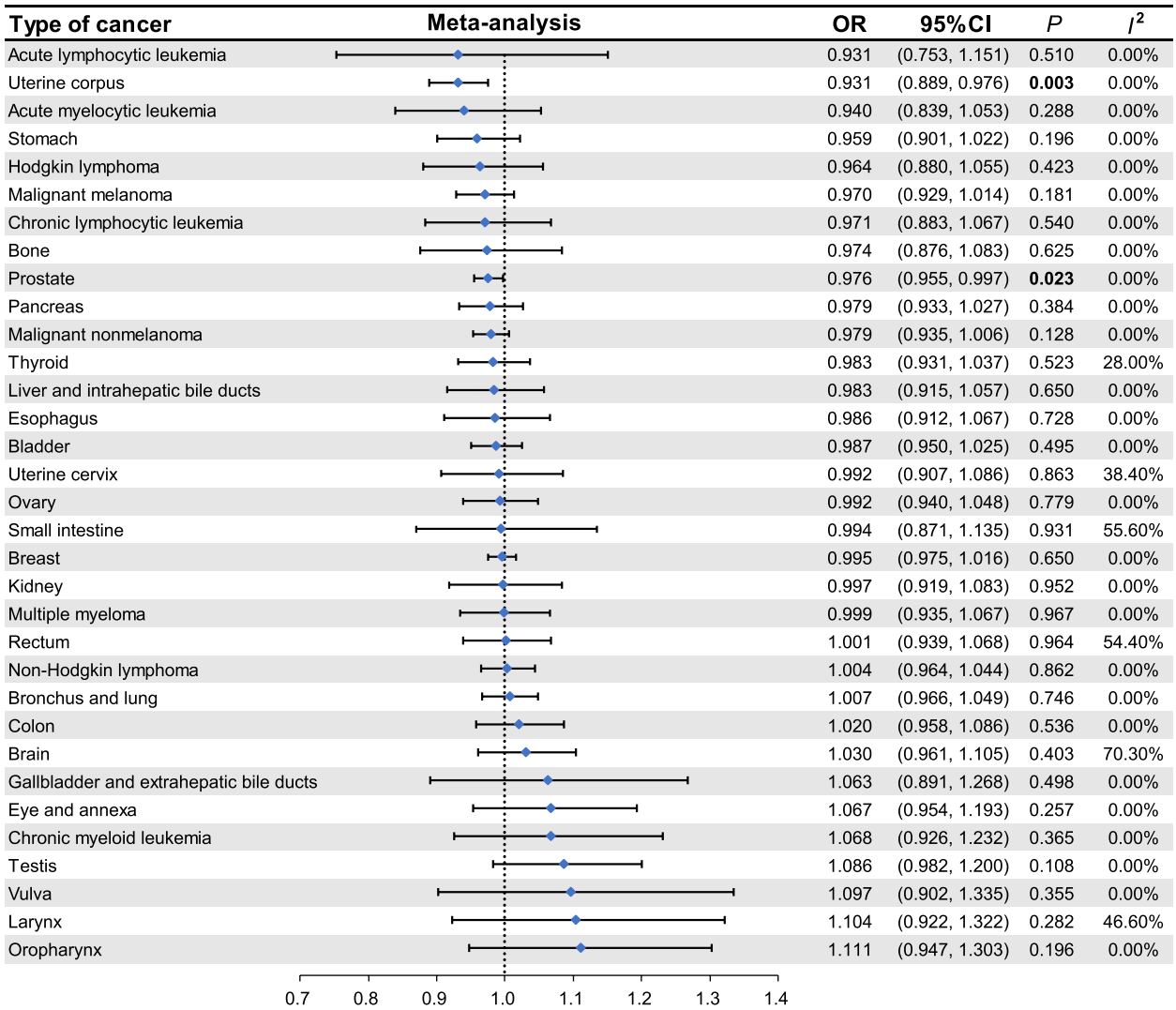


**Fig. 3** Forest plot of the pooled results of the meta-analysis from the UK biobank and the FinnGen. Cancer types are listed in ascending order of effect size (OR). Abbreviations: OR: odds ratio; CI: confidence interval

## Discussion

To clarify the potential associations between psoriasis and the risk of common cancers, this study used data from multiple large studies of European populations, including the UK Biobank, FinnGen consortium, and other large-scale cancer databases. The combined results of the meta-analysis suggest a potential association between genetically predicted psoriasis and lower risk of uterine corpus cancer and prostate cancer. However, the statistical significance and power level of the MR results between psoriasis and the risk of prostate and uterine corpus cancer are low, implying that the observed associations may not be robust. Moreover, no potential associations were observed between genetically predicted psoriasis and other cancers.

The relationship between psoriasis and cancer has been under scrutiny and many observational studies have explored whether they have a relevant association. For example, a study by Trafford et al. showed that patients with psoriasis may have a higher risk of cancer incidence and cancer-related mortality [25]. The study by Fu et al. showed that patients with psoriasis have an increased risk of colorectal cancer [26], and so on. Inevitably, however, these observational studies may be subject to confounders that can affect exposure and outcomes. Therefore, even if observational studies report a correlation between the two, a direct causal correlation cannot be demonstrated. Mendelian randomization, on the other hand, can avoid the influence of these confounders by using genetic instrumental variables instead of exposure, thus

obtaining a relatively accurate causal assessment and providing additional insights into the correlation between psoriasis and cancer. Interestingly, this MR study generally indicates a lack of causal evidence between psoriasis and the risk of most cancers, which is also reflected in the low post hoc power levels, with no cancer type reaching a power greater than 80%. However, for prostate cancer and uterine corpus cancer, we found some suggestive evidence that their risk may be influenced by psoriasis.

#### Psoriasis and uterine corpus cancer

This study suggests a potential inverse correlation between psoriasis and uterine corpus cancer in two independent populations, but there was horizontal pleiotropy. Notably, no aberrant SNPs were identified after MR-PRESSO test. These suggests that pleiotropy may introduce bias through a small dispersion effect of multiple SNPs. The pathogenesis of uterine corpus cancer is currently unknown, but our study may provide some evidence that psoriasis may be protective against uterine corpus cancer, which may be related to the influence of the autoimmune pathogenesis of psoriasis on the pathogenesis of uterine corpus cancer. The relationship between psoriasis and uterine corpus cancer and the mechanisms of its development needs to be further investigated to provide new targets for the prevention and treatment of uterine corpus cancer.

#### Psoriasis and prostate cancer

The conclusions of previous observational studies regarding the association between psoriasis and prostate cancer are controversial. For example, a study based on the Health Improvement Network database enrolling 198,366 patients with psoriasis found that the incidence of prostate cancer did not increase [27]. While another observational cohort study including nearly 900,000 Korean participants found that patients with psoriasis had a higher risk of prostate cancer (HR: 1.109; 95% CI: 1.057–1.165) [8]. The discrepancy between the results of the present study and these observational evidences may stem from the fact that the MR approach effectively circumvents the influence of environmental confounders through genetic instrumental variables. For example, patients with psoriasis often receive immunomodulatory therapies (TNF-α inhibitors or IL-17 antagonists), which may indirectly reduce the risk of cancer by suppressing chronic inflammation [28], whereas it is difficult for observational studies to fully correct for such treatment biases. In addition, patients with psoriasis may have an increased rate of prostate-specific antigen (PSA) testing due to frequent medical visits, leading to diagnostic bias. Future studies could further elucidate the mechanisms through cross-ethnic MR validation, resolving the tumor microenvironment in conjunction with single-cell sequencing, or using drug-targeted MR (such as anti-IL-17 therapies).

#### Strengths and limitations

Our study has some advantages. First, Mendelian randomization approach based on genetic data could reduce confounders and reverse causality to a certain extent compared to observational studies to obtain a direct association between psoriasis and 33 cancers. Second, to the knowledge of us, our MR analysis systematically explores for the first time the association between psoriasis and multiple common cancers and concludes for the first time that psoriasis may be a potential protective factor for uterine corpus cancer and prostate cancer. Third, we used cancer GWAS data from multiple databases, including UK Biobank, FinnGen consortium, and other larger cancer databases as outcome data, improving statistical validity.

Our study has several limitations and therefore the results need to be interpreted with caution. First, despite MR-Egger and MR-PRESSO sensitivity analyses showing weak evidence of horizontal pleiotropy ($P_{\text{pleiotropy}} > 0.05$), the use of binary exposure variables may amplify pleiotropic bias. Genetic variants associated with psoriasis could influence cancer risk through pathways independent of psoriasis itself, such as systemic inflammation, immune cell regulation, or metabolic dysfunction [29]. Although we excluded SNPs with known pleiotropic effects from the GWAS catalog, residual pleiotropy from undiscovered biological mechanisms cannot be fully ruled out. Second, survival bias may distort the observed associations. The cancer GWAS datasets primarily include individuals who survived long enough to be diagnosed, whereas severe psoriasis is linked to increased mortality from cardiovascular and metabolic comorbidities [30]. If psoriasis-associated genetic variants simultaneously elevate early mortality risk, this could lead to underrepresentation of high-risk individuals in cancer registries, potentially biasing effect estimates toward the null [31]. Third, the interpretation of Mendelian randomization estimates for binary exposures requires caution. The genetic instrument represents lifelong exposure to psoriasis susceptibility, which may not align with clinical definitions based on disease onset age or severity. Additionally, MR assumes a linear relationship between genetic liability to exposure and outcome, but the protective effect of psoriasis on certain cancers might be nonlinear or restricted to specific disease stages [31]. Fourth, our analysis relied exclusively on European ancestry populations, limiting generalizability to other ethnic groups where genetic architecture of psoriasis and cancer incidence rates may differ substantially. Fifth, although both

the psoriasis and cancer data are derived from European-ancestry populations, inter-population heterogeneity, such as environmental exposures, phenotype definitions, and participant selection, may still influence the reliability of the results. The random effects IVW model helps to reduce biases arising from statistical heterogeneity among instrumental variables, but it cannot eliminate biases introduced by differences in population structure. Finally, unmeasured confounding from "competing risks", for example psoriasis patients receiving immunosuppressive therapies may have altered risks for infection-related cancers, could distort causal estimates. While our analysis focused on genetic liability to psoriasis itself rather than its treatment effects, future studies should integrate drug-target MR approaches to disentangle disease-specific and therapy-mediated pathways.

## Conclusions

This comprehensive MR study based on multiple large-scale data sources suggests that psoriasis may be a potential protective factor for uterine corpus cancer in women and prostate cancer in man. Further validation by prospective studies and mechanistic investigations is warranted.

## Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12885-025-14243-4.

Supplementary Material 1. STROBE-MR checklist for reports of Mendelian randomization studies.

Supplementary Material 2. Single-nucleotide polymorphisms used as instrumental variable for psoriasis.

Supplementary Material 3. Cancer GWAS data information in UK Biobank, FinnGen, and other large-scale cancer databases.

Supplementary Material 4. Three main results of MR analysis in UK Biobank, FinnGen, and other large-scale cancer databases.

Supplementary Material 5. Sensitivity analyses for heterogeneity and horizontal pleiotropy.

Supplementary Material 6. Scatter plot and leave-one-out forest plot for 33 types of cancer in UK-Biobank.

Supplementary Material 7. Scatter plot and leave-one-out forest plot for 33 types of cancer in FinnGen.

Supplementary Material 8. Scatter plot and leave-one-out forest plot for 9 types of cancer in other large-scale cancer databases.

Supplementary Material 9. Forest plot of the pooled results of the meta-analysis from the UK biobank, FinnGen, and other large-scale cancer datasets.

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**Authors' contributions**
Conception and design: SD, CR, ML. Development of methodology: ML and ZS. Acquisition of data: PT, ZS, DX. Formal analysis: ML, PT, WF. Drafting of manuscript: ML, ZS, YL. Critical revision of the manuscript: ZS and SD. Study supervision: SD, CR. The final version of the manuscript was approved by all authors.

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**Data availability**
The study was conducted using anonymized summary-level data that were open to the public. The GWAS data of psoriasis were retrieved from GWAS Catalog (https://www.ebi.ac.uk/gwas/studies/GCST004346); Cancer data were obtained from the UK Biobank (https://pan.ukbb.broadinstitute.org/), FinnGen consortium (https://r12.finngen.fi/), and ieu open gwas project (https://gwas.mrcieu.ac.uk/).

## Declarations

**Ethics approval and consent to participate**
Not applicable.

**Consent for publication**
Not applicable.

**Competing interests**
The authors declare no competing interests.

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