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Genetically predicted sleep duration in relation to risk of breast tumor

Sleep duration and breast tumor

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Abstract

Aim: To investigate the causal relationship between genetically predicted sleep duration and breast tumors using a bidirectional two-sample Mendelian randomization (MR) analysis.

Methods: A bidirectional two-sample MR analysis was performed using genome-wide association study (GWAS) summary statistics. The inverse variance weighted (IVW) method was used as the primary analysis, with MR Egger regression, weighted median, weighted mode, and simple mode analyses conducted for validation. Sensitivity analyses, including heterogeneity, horizontal pleiotropy, and leave-one-out tests, were performed to evaluate the robustness of the findings. Reverse MR analysis was conducted to assess whether breast tumors causally influence sleep duration.

Results: Genetically predicted longer sleep duration was associated with an increased risk of breast cancer (OR=1.328, 95% CI: 1.013–1.741, p=0.04). Borderline associations were observed for ER+ (p=0.051) and ER− (p=0.077) breast cancer, whereas no significant causal association was found for HER2+/HER2− breast cancer, benign breast tumors, or carcinoma in situ. Sensitivity analyses supported the robustness of the main findings despite heterogeneity. Reverse MR analysis showed no evidence that breast cancer or its molecular subtypes causally affected sleep duration.

Conclusion: This study provides evidence that genetically predicted longer sleep duration may increase the risk of breast cancer but not benign breast tumors or carcinoma in situ. No reverse causal relationship between breast tumors and sleep duration was identified. Maintaining an appropriate sleep duration may contribute to breast cancer prevention.

Keywords

sleep duration, breast cancer, mendelian randomization, causal effect, prevention

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Introduction

Breast cancer is the most common cancer among women worldwide, accounting for a substantial number of new cancer cases and cancer-related deaths. Lifestyle factors play a critical role in the development of breast cancer. For instance, smoking has been identified as a potential risk factor, particularly in premenopausal women.[1] Conversely, regular physical activity has been linked to a decreased risk of breast cancer.[2] Understanding these modifiable factors empowers individuals to make informed choices and adopt healthy behaviors that can potentially reduce their risk of developing breast cancer.

Sleep-related characteristics, such as sleep duration, quality, habits, rhythms, and disorders, have been extensively studied in relation to health outcomes.[3] Numerous studies have explored the association between sleep traits and the incidence of breast cancer, revealing that both insufficient sleep duration and excessive sleep duration are associated with an increased risk of breast cancer.[4] For instance, a study involving 23,620 cases found that sleeping less than 6 hours per night was associated with a 43% higher risk of cancer, including breast cancer.[5] Other studies have also reported a positive correlation between long sleep duration (more than 9 hours per night) and breast cancer.[6,7] Nevertheless, a definitive causal relationship between sleep duration and breast cancer has not been firmly established. Several epidemiological studies and meta-analyses have consistently reported no significant association between sleep duration and the risk of developing cancer.[8,9]

Mendelian randomization is a statistical technique that leverages genetic variants as instrumental variables to establish causal relationships between exposures and outcomes.[10] In this approach, genetic variants are utilized as proxies or instrumental variables for the modifiable risk factors or exposures of interest in Mendelian randomization studies.

These genetic variants are randomly allocated during meiosis and inherited independently of confounding factors, mimicking the process of a randomized controlled trial. This random allocation provides a natural experiment-like setting, making Mendelian randomization a powerful tool for causal inference. By using genetic variants, which are determined at conception and not influenced by confounders, the risk of confounding bias is greatly reduced.

In this study, we employed a two-sample Mendelian randomization (MR) analysis to examine the potential causal relationship between sleep duration and various types of breast tumors, including breast cancer, ER+/ER- breast cancer, HER2+/HER2- breast cancer, benign breast tumors, and carcinoma in situ of the breast. Additionally, we conducted a reverse Mendelian randomization analysis to investigate whether breast tumors have a causal effect on sleep duration.

Materials and Methods

Study Design

We conducted a bidirectional two-sample Mendelian randomization (MR) analysis to examine the causal effect of sleep duration on breast tumors. Both sleep duration and breast tumors were considered as exposure factors to assess their respective causal effects on each other. In the bidirectional analysis, we used specific genetic variants, known as single-nucleotide polymorphisms (SNPs), that are associated with sleep duration or breast tumors as instrumental variables (IVs) to infer causal relationships between the exposures and outcomes. To obtain the necessary GWAS datasets, we used the IEU OpenGWAS database (https://gwas.mrcieu.ac.uk/), which contains over 40,000 GWAS datasets with SNPs associated with various traits.

Determination of IVs

All SNPs that were correlated with the exposure trait at genome-wide significance ($p<5 \times 10-8$) were extracted as potential IVs. SNPs in high linkage disequilibrium ($r2 > 0.001$ or clump windows < 10,000 kb) were excluded to eliminate bias caused by linkage disequilibrium (LD). Harmonization was performed to eliminate ambiguous SNPs with non-concordant alleles. When a SNP was not presented in the outcome summary statistics, a proxy SNP highly correlated with the variant of interest (LD, $r2 > 0.8$) was selected for substitution. However, if a substitute could not be identified, the SNP was excluded. Palindromic SNPs were aligned when minor allelic frequencies were less than 0.3. IVs with an F-statistic of < 10 were excluded due to their weak correlations.

MR Statistics

We used the inverse-variance weighted (IVW) method to obtain the main results of the two-sample MR analysis.[11] MR-Egger regression, weighted median, weighted mode, and simple mode analyses were used as complementary methods to assess the consistency of the findings. A clear causal relationship between exposure and outcome was considered for IVW results with p-values < 0.05, and the p-values from the other four methods were consistent with the IVW p-value. Sensitivity analyses were performed, including the heterogeneity test, the horizontal pleiotropy test, and a leave-one-out analysis, to further validate the causal relationships obtained and assess the reliability of the results. All MR statistical analyses were conducted using the TwoSampleMR package (version 0.5.7) in R software (version 4.2.2).

Ethical Approval

This study was approved by the Ethics Committee of the Affiliated Hospital of Shaanxi University of Traditional Chinese Medicine (Date: 09.05.2024, Decision No: SZFYIEC-KYBC-2024-03).

Statistical Analysis

Statistical analyses were performed using the TwoSampleMR package (version 0.5.7) in R software (version 4.2.2). A two-sided p-value <0.05 was considered statistically significant.

Reporting Guidelines

The study was reported in accordance with relevant recommendations for Mendelian randomization studies, where applicable.

Results

Data for MR Analysis

Figure 1 displays the workflow of the study. The sleep duration-related dataset (GWAS ID: ukb-b-4424) that was selected comprised 460,099 samples and 9,851,867 SNPs from European populations. GWAS breast tumor datasets included breast cancer datasets (malignant, HER2, and ER status undefined), ER+/ER- breast cancer, HER2+/HER2- breast cancer, benign breast tumors, and carcinoma in situ of the breast (Table 1).

Causal Effect of Sleep Duration on Breast Cancer

Figure 2 and Supplementary Table 1 display the results of the MR analysis of the causal effect estimate of sleep duration on breast cancer. A total of 66 SNPs were included in the MR analysis. We identified a significant causal relationship between sleep duration and the risk of breast cancer, with a p-value of 0.04 (odds ratio (OR) = 1.328, 95% confidence interval (CI): 1.013 – 1.741) in the IVW analysis (Figures 2A and 2B). A positive result was also detected using the weighted median method (OR = 1.320, 95% CI: 1.030–1.693, p=0.028). While the secondary indicators (MR-Egger, simple mode, and weighted mode methods) did not indicate statistically significant results, b values consistently pointed in the same direction. This consistency indicates a positive association between sleep duration and breast cancer, despite the lack of significant findings.

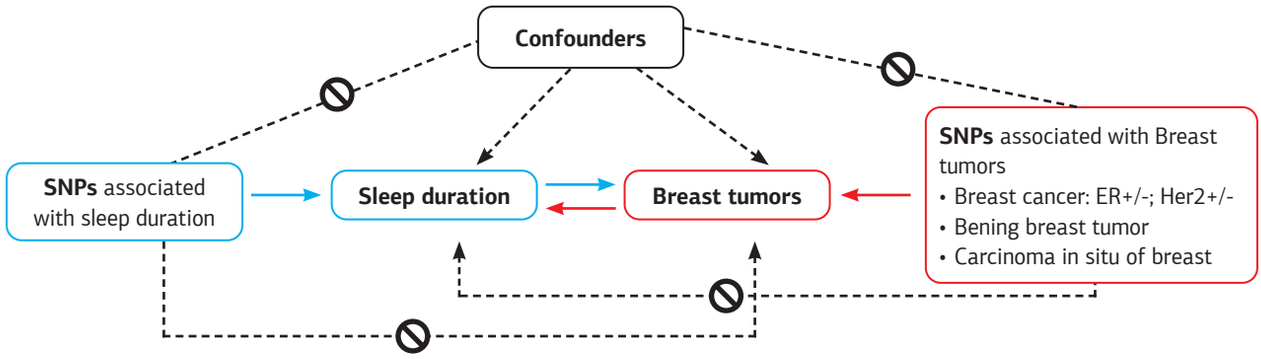

Figure 1. *Workflow of this bidirectional Mendelian randomization study*

Table 1. *Data source of different types of breast tumors*

Trait	*GWAS ID*	*publication year*	*Number of samples*	*Number of SNPs*	*Population*	*Source*
Breast cancera	ieu-a-1126	2017	228951	10680257	European	BCACb
ER+ breast cancer	ieu-a-1133	2017	69970	10680257	European	BCAC
ER- breast cancer	ieu-a-1166	2015	21695	13011123	European	BCAC
Her2+ breast cancer	finn-b-C3_BREAST_HERPLUS	2021	123302	16,379,780	European	BCAC
Her2- breast cancer	finn-b-C3_BREAST_HERNEG	2021	119039	16,379,675	European	MRC-IEU
Benign breast tumor	ukb-b-8549	2018	463,010	9,851,867	European	MRC-IEU
Carcinoma in situ of breast	finn-b-CD2_INSITU_BREAST	2021	123,579	16,379,784	European	MRC-IEU

a HER2 and ER status undefined. b BCAC: Breast Cancer Association Consortium.

The MR-Egger regression results confirmed that our results were not significantly affected by horizontal pleiotropy (p=0.995). The funnel plot and Cochran's Q test indicated significant heterogeneity among the SNPs involved in the IVW analysis (p=1.828e-19) (Figure 3A). A random-effects model was used in the analysis to avoid statistical bias due to heterogeneity. The leave-one-out analysis showed that removing a single SNP did not significantly affect the overall estimates (Figure 3B), suggesting that the results were highly reliable.

Causal Effect of Sleep Duration on ER+/ER- or HER2+/HER2- Breast Cancer

Additional MR analyses were performed in ER+/ER- and HER2+/HER2- breast cancers to further verify the causal effect between sleep duration and different molecular subtypes of breast cancer. A total of 65 SNPs were finally selected for the causal effect analysis of sleep duration on ER+ breast cancer. Although the IVW method did not detect statistical significance, the p-value was close to 0.05 (p=0.051). Analyses using the weighted median and weighted mode methods revealed a significant causal relationship between sleep duration and ER+ breast cancer (weighted median, p=0.003; weighted mode, p=0.036) (Supplementary Figure 1, Supplementary Table 1). Similarly, a positive causal effect was identified between sleep duration and ER- breast cancer. A p-value with borderline statistical significance (p=0.077) was obtained using the IVW method. However, a significant statistical difference was obtained by the weighted median method (p=0.028). The results obtained by the other three methods showed similar trends in the causal effect of sleep duration on ER-breast cancer. Still, they were not statistically significant (MR-Egger p=0.569, simple mode p=0.090, weighted mode p=0.097) (Supplementary Figure 2, Supplementary Table 1).

The IVW approach did not show a significant causal connection in the MR analysis of sleep duration on HER2+/HER2- breast cancer (HER2+ breast cancer: OR = 0.963, 95% CI: 0.564 – 1.642, p=0.889; HER2- breast cancer: OR = 1.825, 95% CI: 0.958 – 3.477, p=0.067) (Supplementary Figure 3, Supplementary Figure 4, Supplementary Table 1). Although the p-value for the causal effect of sleep on HER2- breast cancer was close to 0.05, we did not believe that there was a clear causal relationship between sleep duration and HER2- breast cancer because MR-Egger's method detected a causal effect in the opposite direction (b = -0.278).

Causal Effect of Sleep Duration on Benign Breast Tumor

A total of 44 SNPs were used for the MR analysis of sleep duration on benign breast tumors. The IVW approach did not show a causal effect of sleep duration on benign breast tumors (OR = 1.002, 95% CI: 0.999–1.005, p=0.242). Secondary analysis methods, including MR-Egger regression (OR = 0.996; 95% CI: 0.970 – 1.022, p=0.757), weighted median (OR = 1.001; 95% CI: 0.997 – 1.005, p=0.606), simple mode (OR = 0.999; 95% CI: 0.991 – 1.008, p=0.826), and weighted mode (OR = 1.000; 95% CI: 0.991 – 1.008, p=0.949) approaches, exhibited the same results (Supplementary Figure 5, Supplementary Table 1).

Causal Effect of Sleep Duration on Carcinoma in Situ of the Breast

The causal relationship between sleep duration and breast carcinoma in situ was also examined. Heterogeneity was not found for changes in SNPs (p=0.811). The MR analysis found no statistically significant causal effect using the IVW method (OR = 1.090, 95% CI: 0.403–2.947, p=0.086) and the other four methods. Finally, the MR-Egger regression intercept provided no evidence for directional pleiotropy (p=0.743) (Supplementary Figure 6, Supplementary Table 1).

Reverse MR Results

We performed a reverse MR analysis to verify the possibility of reverse causality, in which breast tumors were used as the exposure and sleep duration as the outcome. A total of 126 SNPs related to breast cancer were finally included in the reverse MR analysis. The IVW analysis results did not indicate that having breast cancer could affect sleep duration (OR = 0.998, 95% CI: 0.989 – 1.006, p=0.670) (Supplementary Table 2). Further analysis based on breast cancer molecular typing generated similar results, that ER+/ER- and HER2+/HER2- breast cancer did not have a causal effect on sleep duration. The analysis investigating the causal relationship

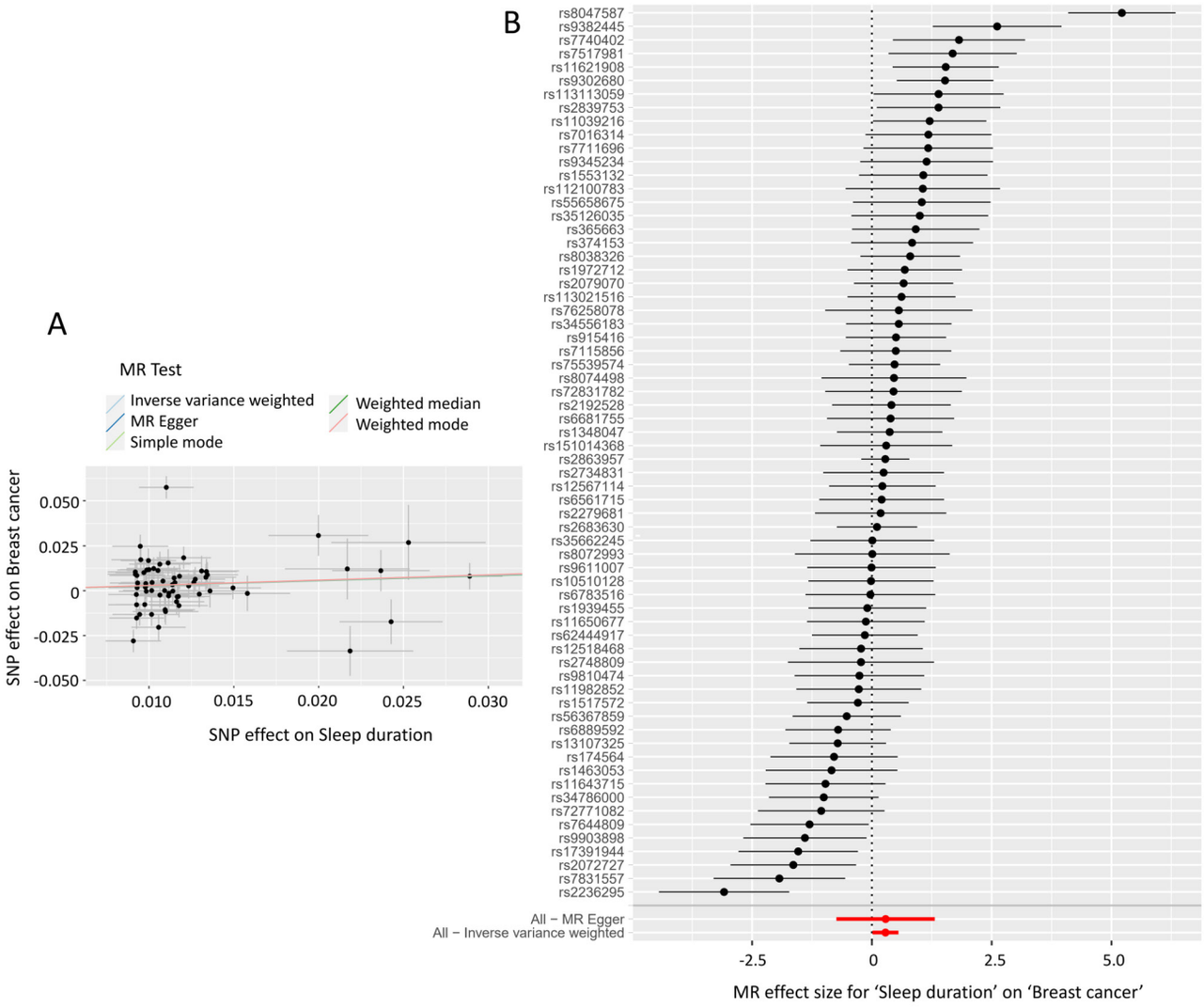

Figure 2. *The causal estimates of sleep duration on breast cancer via two-sample MR analysis. (A) Scatter plot, (B) forest plot*

between sleep duration and benign breast tumors did not yield statistically significant results (odds ratio = 2.011, 95% confidence interval: 0.26-153.896, p=0.752). It's important to note that this analysis was based on only one SNP and had low confidence. As for the analysis of the causal relationship between sleep duration and carcinoma in situ of the breast, no relevant SNP was identified, yielding no results for this aspect of the reverse analysis.

Discussion

Our results suggested evidence for causal effects between sleep duration and the risk of breast cancer. Women with longer sleep duration might have a significantly increased risk of breast cancer. However, we did not detect a clear causal effect of sleep duration on HER2+/HER2- breast cancer, benign breast tumors, or carcinoma in situ of the breast, nor did we find that having breast tumors increased or decreased sleep duration.

There is a traditional belief that shorter sleep duration might be a risk factor for breast cancer, and long sleep duration might reduce the risk of cancer.[12,13] However, our study generated inconsistent conclusions. Sleep duration had a causal effect on breast cancer, and longer rather than shorter sleep duration might increase the risk of breast cancer. The positive association between long sleep duration and breast cancer has attracted considerable attention in recent years.[2] A study by Wang et al. reported a positive association between long sleep duration (> 9 hours a night) and breast cancer incidence compared to a reference sleep duration (6.1 – 8.9 hours a night).[14] Another case-control study revealed that the risk of breast cancer increased with increasing sleep duration for every additional sleeping hour (OR = 1.06, 95% CI: 1.01 – 1.11).[15] In addition, a meta-analysis including 10 studies showed that women with longer sleep durations had a significantly increased risk of breast cancer, and the effect was dose-dependent.[16] Our study provides genetic evidence suggesting a possible association between longer sleep duration and increased breast cancer risk. Unlike prospective or retrospective cohort studies, this analysis eliminated biases that could arise from confounding factors, making the conclusions more reliable.

The detailed mechanism by which sleep duration affects the occurrence of breast cancer is not clear. Several studies suggested that reduced melatonin secretion due to sleep deprivation might be a major mechanism promoting cancer development, and melatonin is known to have an effect on protecting the body from cancer.[7,17] However, such an explanation does not fit our results. Long-duration sleepers have higher cortisol levels than short-duration sleepers.[18,19] Cortisol is involved in multiple processes involved in the genesis and development of breast cancer, such as 1) the regulation of mammary epithelium growth, 2) the impairment of immune activity, and 3) the suppression of natural killer cell activity.[20,21] These effects of cortisol may be the underlying mechanisms by which long-term sleep duration increases the risk of breast cancer.[22]

Patients with cancer were reported to be at a high risk of sleep

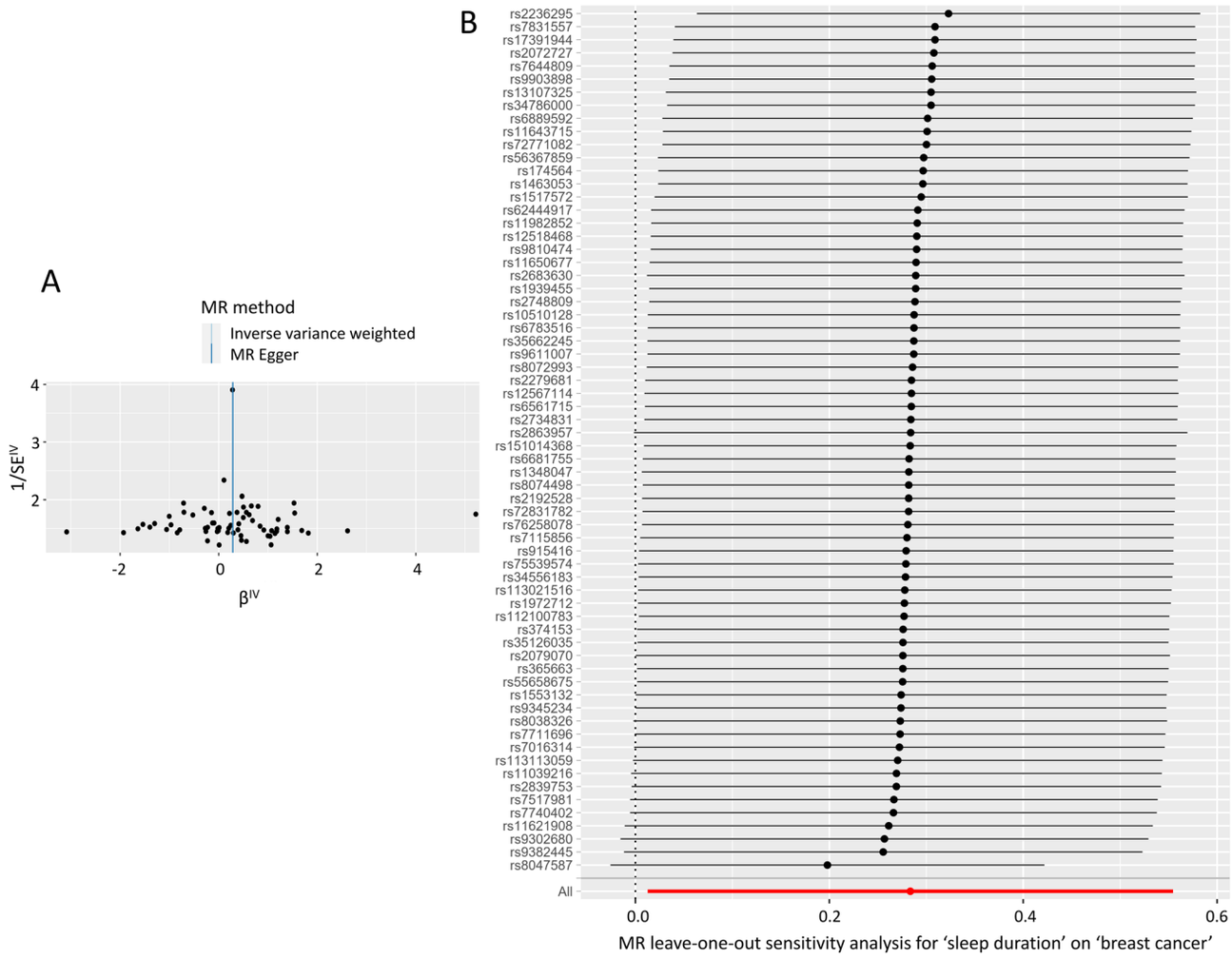

Figure 3. *The sensitivity analysis of causal estimates of sleep duration on breast cancer. (A) funnel plot ;(B) leave-one-out sensitivity analysis*

disorders, the most prominent of which was insomnia. No causal effect of breast tumors on sleep duration was detected in our reverse MR analysis. However, we could not draw a strong conclusion that breast tumors do not affect patients' sleep duration because there were too few SNPs as IVs in the evaluation of ER- breast cancer (SNPs = 7), HER2+/HER2- breast cancer (SNPs = 8/4), benign neoplasm of the breast (SNPs = 1), and carcinoma in situ of the breast (SNPs = 0). In fact, the causal effect of breast tumors on sleep duration is complex. Although some patients may experience sleep problems during cancer or treatment, not all patients are affected. The prognosis of patients with breast cancer is generally good, and endocrine therapy or targeted therapy can replace traditional chemotherapy and reduce toxic side effects.[23] Therefore, the psychological stress caused by the disease and treatment of breast cancer patients is less than that of other malignant tumors, such as liver cancer and lung cancer. Psychological stress-related anxiety, depression, etc., are causes of sleep problems. In contrast, sleep disorders in cancer patients are closely related to treatment factors, individual differences, economic factors, and the sleeping environment.[24]

Some limitations existed in this study. First, heterogeneity was detected in the evaluation of breast cancer, ER+ breast cancer, and ER- breast cancer. We used the random-effects IVW method to correct for heterogeneity to ensure the validity of the results. Second, we used only the IEU OpenGWAS database for analysis, which does not capture all GWAS data on sleep and breast tumors. Finally, sleep traits include sleep duration, chronotype, sleep quality, and so on. However, we only analyzed sleep duration in this study. Thus, further studies are needed.

Limitations

This study was limited by the use of a single GWAS database, the evaluation of only sleep duration, and heterogeneity in some analyses despite sensitivity analyses.

Conclusion

Our study suggested the potential causal effect of genetically predicted sleep duration on breast cancer, while a causal effect of sleep duration on benign breast tumors and carcinoma in situ of the breast was not identified. The reverse analysis did not prove the existence of a causal effect of breast tumors on sleep duration. These findings should be interpreted cautiously, and further studies are required before making clinical recommendations regarding sleep duration and breast cancer prevention.

Declarations

Animal and Human Rights Statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Declaration of Helsinki and its later amendments, or comparable ethical standards.

Informed Consent

Informed consent was not required for this study.

Data Availability

The datasets analyzed in this study are publicly available from the IEU OpenGWAS database.

Conflict of Interest

The authors declare no conflict of interest.

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Author Contributions (CRediT Taxonomy)
Conceptualization: L.S., Y.L.
Methodology: L.S., D.Z., J.J.
Investigation: L.S., D.Z., J.J.
Data Curation: L.S., D.Z.
Formal Analysis: L.S., Y.L.
Writing – Original Draft: L.S.
Writing – Review & Editing: D.Z., J.J., Y.L.
Supervision: Y.L.

AI Usage Disclosure
The authors declare that no AI-assisted technologies were used.

Abbreviations
CI: Confidence interval
ER: Estrogen receptor
GWAS: Genome-wide association study
HER2: Human epidermal growth factor receptor 2
IV: Instrumental variable
IVW: Inverse variance weighted
LD: Linkage disequilibrium
MR: Mendelian randomization
OR: Odds ratio
SNP: Single nucleotide polymorphism

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