Systematic Review and Meta-Analysis

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Male pattern baldness and incidence of prostate cancer

A systematic review and meta-analysis

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Abstract

Background: The relationship between male pattern baldness and incidence of prostate cancer remains inconclusive. Hence, we performed the present meta-analysis based on all eligible cohort and case–control studies.

Methods: A comprehensive literature search was performed in October 2017 based on PubMed and Web of Science databases. Pooled relative risk (RR) and its 95% confidence interval (95% CI) was calculated with a DerSimonian and Laird random-effects model.

Results: A total of 15 studies were included in this meta-analysis. Overall, no statistically significant association between baldness (any pattern) and prostate cancer risk was identified (RR: 1.03, 95% CI 0.96–1.11). There was obvious heterogeneity across included studies ($P < .078$ for heterogeneity, $I^2 = 36.4\%$). When subgroup analysis by types of baldness, a statistically significant association was observed for vertex baldness (RR 1.24, 95% CI 1.05–1.46) but not for other types of baldness.

Conclusion: Individuals with vertex baldness may have an increased risk of prostate cancer. Given the obvious heterogeneity and null results in overall analysis and most of subgroup analyses, further large well-designed prospective cohort studies are warranted to confirm our preliminary findings.

Abbreviations: CI = confidence interval, NOS = Newcastle–Ottawa Scale, OR = odds ratio, RR = relative risk.

Keywords: male pattern baldness, meta-analysis, prostate cancer, risk

1. Introduction

Prostate cancer is the second-most commonly diagnosed cancer among men worldwide, with a total of 1,111,700 new cases and 307,500 deaths estimated in 2012.[1] With the wide utilization of the prostate-specific antigen (PSA) testing, a growing number of prostate cancer cases at local or regional stages have been diagnosed.[2] The only well-established risk factors for prostate cancer are age, race/ethnicity, and family history of prostate cancer.[3] Hence, an additional study to improve our understanding of the etiology of prostate cancer is warranted.

Recently, several researchers have explored whether male pattern baldness (or androgenic alopecia) is a potential indicator for future prostate cancer incidence, as male pattern baldness seems to share pathologic mechanisms with prostate cancer, such as advancing age, hereditability, and endogenous hormones.[4,5] As male pattern baldness generally occurs decades earlier than prostate cancer, baldness may serve as a potential noninvasive screening indicator for prostate cancer if a clear association between male pattern baldness and prostate cancer could be established. However, findings from previous epidemiologic studies on the relationship between male pattern baldness and prostate cancer risk are inconclusive due to the different study designs, limited sample size, various baldness measurements and classification, and time window (age of baldness onset). Al Edwan et al,[6] Zeigler-Johnson et al,[7] and Giles et al[8] reported a significant positive association of baldness with prostate cancer risk, while many other epidemiological studies failed to confirm this association.

Given the conflicting findings in literature as discussed above, we performed the present systematic review and meta-analysis to summarize evidence on the association of male pattern baldness with risk of prostate cancer.

2. Materials and methods

2.1. Literature search

A comprehensive literature search was performed in October 2017 based on PubMed and Web of Science databases with the following search algorithm: ("baldness" or "alopecia" or "hair loss") and ("prostate cancer" or "prostate neoplasm" or "prostatic carcinoma"). In addition, the reference lists of

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The authors declare no conflict of interest.

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retrieved articles and related reviews were checked to identify any potential relevant studies. No language or publication date restrictions were adopted. The present systematic review and meta-analysis was designed, performed, and reported in accordance with the standards of quality for reporting a systematic review and meta-analysis.[9]

2.2. Selection criteria

Articles included in this study had to meet all the following criteria: the exposure of interest was male pattern baldness; the outcome of interest was risk of prostate cancer; study design was cohort, nested case–control, or case-control; and the risk estimates with their corresponding 95% CIs were reported or sufficient data were provided to calculate them. If multiple articles used the same study population, the article with the largest sample size was included in the present meta-analysis.

2.3. Quality assessment

Newcastle–Ottawa Scale (NOS) was introduced to evaluate the quality of included studied by two independent authors (HH and BX). NOS is a 9-star system, which includes 3 dimensions: selection (4 items), comparability (1 item), and exposure/outcome (3 items) (http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp). Each item represents 1 point, except for comparability (2 points). A study with ≥ 7 points was considered as a high-quality study.

2.4. Data extraction

Data were extracted independently by the same 2 authors (HH and BX) and any discrepancies were resolved by discussion. The following information from each included study were recorded: first author's surname, the country where the study was conducted, study design, publication year, age of study population, sample size and number of cases, types of baldness, fully adjusted risk estimates with their corresponding 95% confidence intervals (95% CIs), and matched or adjusted variables in the study design or statistical analysis.

2.5. Statistical methods

Considering that prostate cancer is a rare disease, the odds ratio (OR) was assumed approximately the same as relative risk (RR), and the RR was adopted as the study outcome. Pooled RR and its 95% CI was calculated with a DerSimonian and Laird random-effects model,[10] which takes account for both within- and between-study variability. For studies that reported separate effect size estimates for different types of baldness, we combined these risk estimates within each study, weighted by inverse of the variance.[11] Subgroup analyses were performed based on geographical region, types of baldness, age of baldness onset, study design, and stage of prostate cancer.

Heterogeneity across included studies was assessed with the Cochran Q test (the level of significance was set at 0.1).[12] The I^2 score was also used to determine the degree of heterogeneity ($I^2 <$ 25%, no obvious heterogeneity; $I^2 = 25$–50%, moderate heterogeneity; $I^2 > 50\%$, large or extreme heterogeneity).[12] Finally, Galbraith plot [13] was introduced to identify the studies that contributed to the heterogeneity and meta-analysis was performed again after removing these studies.

In sensitivity analysis, meta-analysis was reconducted after omitting each study in turn. Potential publication bias was evaluated with Begg test [14] and Egger test.[15] Statistical analyses were completed with STATA 11.0 (StataCorp, College Station, TX), using 2-sided P values (set at .05). All analyses of this meta-analysis were based on previous published studies, and this meta-analysis did not have original data. Thus, no ethical approval and patient consent are required.

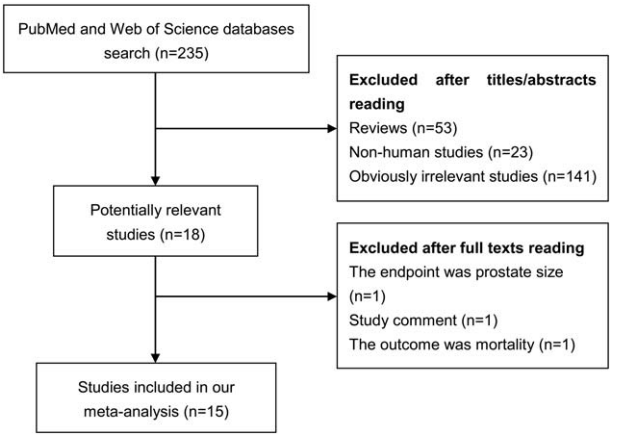

Figure 1. Literature search and study selection.

3. Results

3.1. Study search and characteristics

The detailed process of literature search is shown in Fig. 1. A total of 15 eligible studies [6–8,16–27] were finally included in the present meta-analysis. These studies were carried out in the following geographical regions: Europe (n = 5), North America (n = 8), and Oceania (n = 2). There were 4 cohort studies and 11 case–control studies, which were published between 1997 and 2016. Information on exposure (male pattern baldness) was collected by face-to-face interview or self-reported questionnaire. Study outcome (prostate cancer) was histopathologically proved in most of the included studies. Quality scores evaluated by the Newcastle–Ottawa Scale (NOS) ranged from 5 to 8. The main characteristics of all included studies have been summarized in Table 1.

3.2. Overall analysis and heterogeneity assessment

Multivariable adjusted RRs with their 95% CIs extracted from each included study that evaluated baldness (any pattern) and prostate cancer risk are present in Fig. 2. Overall, in a random effects model combining these studies, no statistically significant association between baldness (any pattern) and prostate cancer risk was identified (RR: 1.03, 95% CI 0.96–1.11). There was statistically significant heterogeneity across included studies ($P < .078$ for heterogeneity, $I^2 = 36.4\%$). Thus, we adopted the Galbraith plot to explore the studies that led to the heterogeneity. Consequently, studies by Al Edwan et al,[6] Zeigler-Johnson et al,[7] and Giles et al[8] were the major sources of heterogeneity (Fig. 3). Overall analysis was reconducted after omitting these 3 studies. The heterogeneity was disappeared ($P = .908$, $I^2 = 0.0\%$), while the trend direction of combined risk estimate was not changed (RR: 0.99, 95% CI 0.94–1.04).

3.3. Subgroup analysis

Next, we performed stratified analyses according to various study characteristics (Table 2). When stratified analysis by geographi-

Table 1

Summary characteristics of the studies included in this meta-analysis.

Author	Year	Country	Design	Cohort/Control	Case	Age	Variables	NOS
Al Edwan et al[6]	2016	Canada	Case–control	200	194	62.7	Age, PSA, and DRE abnormalities	5
Sarre et al[26]	2016	Finland	Cohort	11,795	757	66	Age	7
Zhou et al[25]	2015	USA	Cohort	32,583	2306	50–76	Age, ethnicity, marital status, Charlson comorbidity index, BMI, alcohol, smoking, and aspirin us	7
Zhou et al[25]	2015	USA	Cohort	39,070	1138	55–74	Age, screening arm, center, education, marital status, diabetes, BMI, smoking, aspirin us, and myocardial infarction	6
Zeigler-Johnson et al[7]	2013	USA	Case–control	219	318	33–93	Age	5
Thomas et al[23]	2013	USA	Case–control	312	167	62.4	Age, race, family history, and BMI	5
Muller et al[22]	2013	Australia	Cohort	9448	476	27–81	Age and country of birth	8
Yassa et al[21]	2011	France	Case–control	281	388	67.2	Age	5
Wright et al[20]	2010	USA	Case–control	942	999	35–74	Age, race, PSA screening, family history, BMI, and finasteride use	7
Cremers et al[19]	2010	Netherlands	Case–control	2160	938	NA	Age and family history	6
Faydaci et al[27]	2008	Turkey	Case–control	108	44	50–75	Age	5
Giles et al[8]	2002	Australia	Case–control	1390	1446	$\leq$ 69	Age, study center, calendar year, family history, and country of birth	7
Demark-Wahnefried et al	2000	USA	Case–control	145	134	45–70	Age	6
Hsieh et al[17]	1999	Greece	Case–control	246	320	NA	Age, height, BMI, and years of schooling	5
Demark-Wahnefried et al[16]	1997	USA	Case–control	156	159	50–70	Age and race	5

BMI = body mass index, DRE = digital rectal examination, NA = not available, PSA = prostate-specific antigen.

cal regions, the RRs with 95% CIs were 0.96 (0.86–1.06), 1.07 (0.95–1.20), and 1.07 (0.89–1.29) for Europe, North America, and Oceania, respectively. When further subgroup analysis was done by types of baldness, statistically significant association was observed for vertex baldness (RR 1.24, 95% CI 1.05–1.46) but not for other types of baldness. We then conducted stratified analysis by study design, the analysis limited to cohort studies yielded an RR of 0.99 (95% CI 0.93–1.04), and the analysis for case–control studies yielded an RR of 1.11 (95% CI 0.98–1.25). In the subgroup analysis by stage of prostate cancer, the RRs with

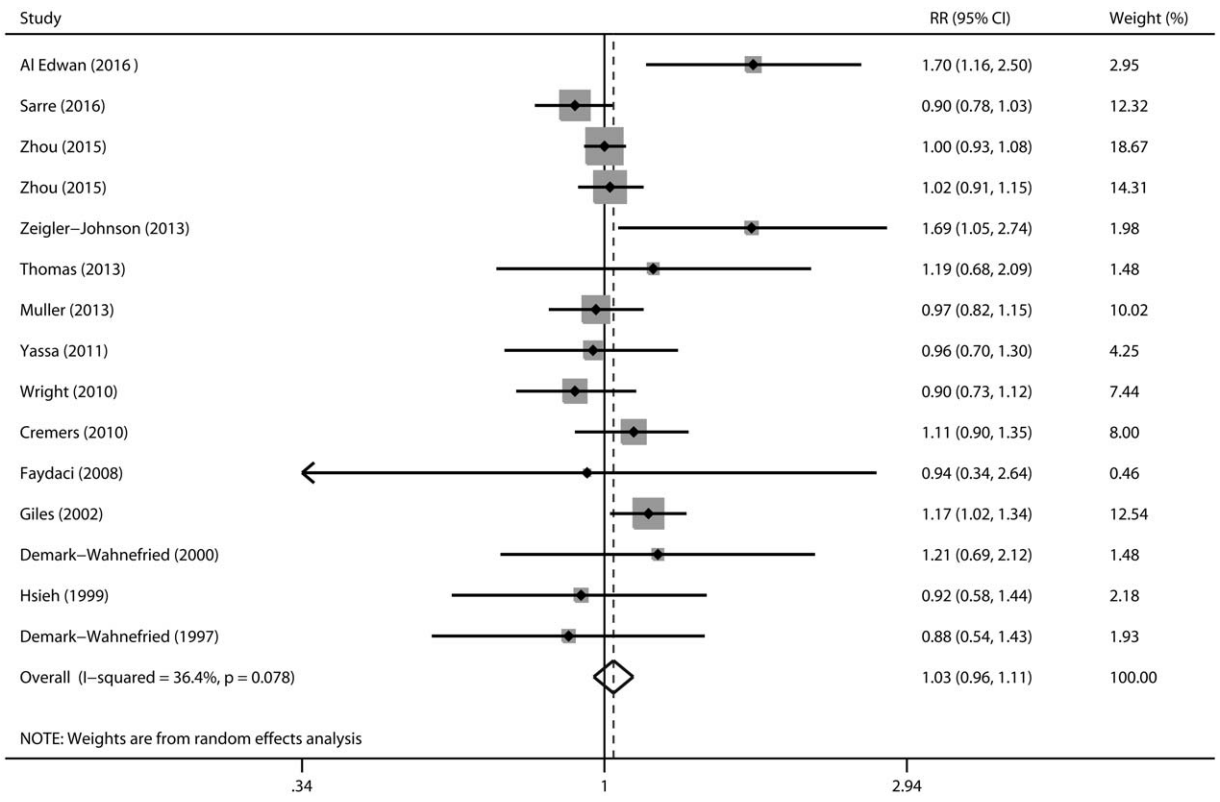

Figure 2. A forest plot showing effect size estimates from case–control and cohort studies evaluating the association between male pattern baldness and prostate cancer risk.

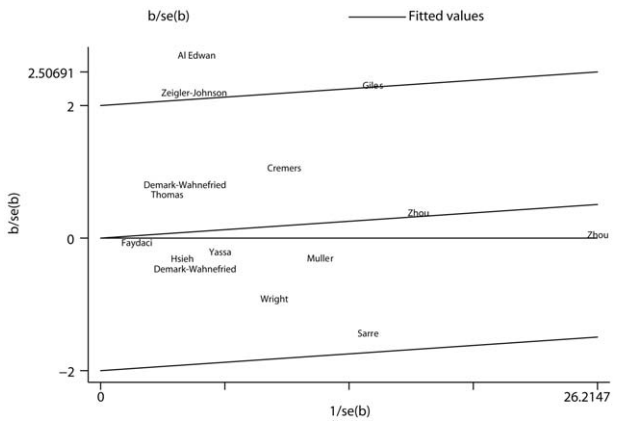

Figure 3. Galbraith plot analysis was performed to detect potential sources of heterogeneity.

95% CIs were 1.04 (0.92–1.17) and 1.14 (0.96–1.35) for nonaggressive and aggressive prostate cancer, respectively. Finally, in the stratified analysis by the age of baldness onset, male pattern baldness at age 20 years, age 30 years, and age 40 years were not associated with overall prostate cancer risk.

3.4. Sensitivity analysis and publication bias

Sensitivity analysis was performed to determine the influence of each included study on the pooled RR by repeating the meta-analysis after removing each study in turn. As shown in Fig. 4, no individual study obviously dominated the pooled RR. The 15 study-specific RRs ranged from a low of 1.01 (95% CI 0.94–1.08) to a high of 1.05 (95% CI 0.98–1.13) after omitting the study by Giles et al [8] and the study by Sarre et al,[26] respectively. Finally, no significant publication bias was detected by Begg test (Fig. 5, $P = .428$) or Egger test ($P = .627$).

Table 2

Subgroup analysis of the association between male pattern baldness and risk of prostate cancer.

Subgroup	Included studies	Combined RR (95% CI)	Heterogeneity I^2 (%)	Heterogeneity *P*
Total	15	1.03 (0.96–1.11)	36.4	.078
Geographical region				
Europe	5	0.96 (0.86–1.06)	0.0	.586
North America	8	1.07 (0.95–1.20)	48.7	.058
Oceania	2	1.07 (0.89–1.29)	65.0	.091
Type of baldness				
Frontal	14	0.98 (0.91–1.06)	0.0	.757
Vertex	11	1.24 (1.05–1.46)	47.1	.041
Both	7	1.01 (0.95–1.08)	0.0	.818
Study design				
Cohort	4	0.99 (0.93–1.04)	0.0	.542
Case–control	11	1.11 (0.98–1.25)	31.4	.148
Stage of prostate cancer				
Nonaggressive	5	1.04 (0.92–1.17)	47.1	.109
Aggressive	7	1.14 (0.96–1.35)	78.4	< .001
Age of baldness onset, y				
20	2	1.28 (0.59–2.75)	81.3	.021
30	6	1.15 (0.89–1.48)	74.3	.002
40	5	0.97 (0.82–1.13)	40.9	.148

CI = confidence interval, RR = relative risk.

4. Discussion

The present systematic review and meta-analysis summarized the relationship between male pattern baldness and incidence of prostate cancer based on 4 cohort studies and 11 case–control studies. Overall, there was no statistically significant association between baldness (any pattern) and prostate cancer risk (RR: 1.03, 95% CI 0.96–1.11). However, in the subgroup analysis by types of baldness, a statistically significant association was observed for vertex baldness (RR 1.24, 95% CI 1.05–1.46).

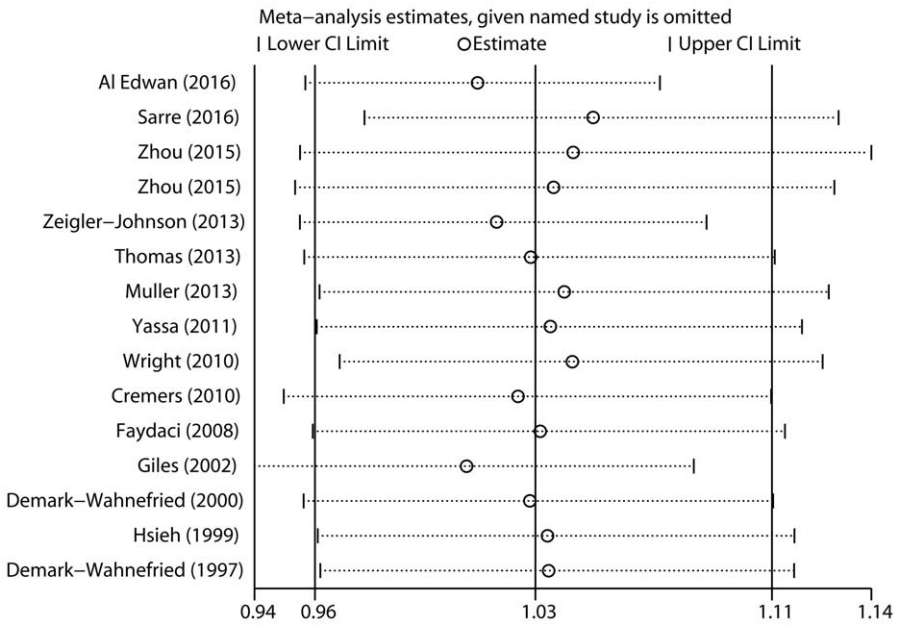

Figure 4. Sensitivity analysis was carried out by omitting each study in turn and recalculating the pooled risk estimates.

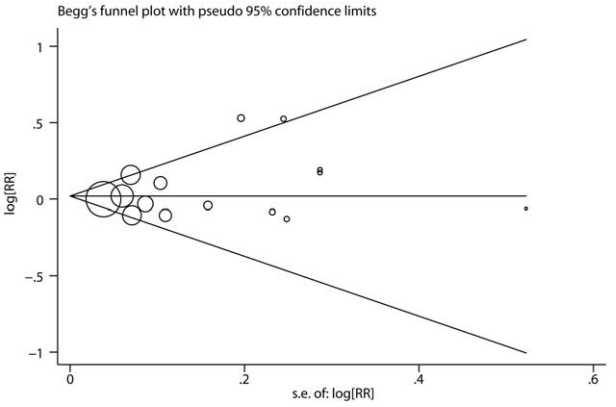

Figure 5. Publication bias was evaluated by Begg test.

The results of our meta-analysis were basically in accordance with a previous meta-analysis by Amoretti et al published in 2013,[28] which only included 7 case–control studies. They also found that any pattern baldness was not associated with a significant increase in the risk of prostate cancer, but individuals with vertex baldness had a higher incidence of prostate cancer. After their study was reported, emerging studies, especially cohort studies, on this topic have been published with conflicting results. Hence, we performed the present updated meta-analysis to summarize all available evidence. Furthermore, we also analyzed age-specific baldness in relation to prostate cancer risk and the association of male pattern baldness with different stage of prostate cancer, which were not presented in the previous meta-analysis.

Male pattern baldness is considered as a common progressive hair-loss process in a well-defined pattern and seems to share common pathophysiological mechanisms with prostate cancer (i.e., age, heritability and endogenous hormones).[4,5] Both incidences of male pattern baldness and prostate cancer increase with advancing age. Inherited genetic factors contribute to 81% of male pattern baldness [5] and 42% of prostate cancer risk.[4] A multistage genome-wide association study has identified more than 40 prostate cancer susceptibility loci, explaining ~25% of the familial risk in this disease.[29] Several independent studies indicated that some androgen receptor (AR) polyglycine repeat polymorphisms might confer susceptibility to androgenetic alopecia.[30–32] As for endogenous hormones, both hair follicles and the prostate gland respond to stimulation of androgen. Individuals with baldness seem to have a higher level of circulating androgens than those without.[33] High circulating androgens have been reported to be associated with an increased risk of prostate cancer,[16,34] although the evidence in epidemiologic studies are still conflicting.[35] Considering that male pattern baldness happens decades earlier than prostate cancer, baldness may serve as a potential noninvasive indicator for screening of prostate cancer if a clear association between male pattern baldness and prostate cancer could be confirmed by large well-designed cohort studies.

The present study had some strengths. A total of 15 epidemiologic studies were included in this systematic review and meta-analysis, which improved the statistical power and reported more reliable estimates compared with any individual study. Various subgroup analyses according to key characteristics were performed, which provided a comprehensive and in-depth evaluation of the relationship between male baldness and prostate cancer. Sensitivity analyses and Galbraith plot were carried out to confirm the robustness of pooled risk estimate. The effect size estimates were extracted from fully adjusted models in each study, which might minimize the potential confounding. No obvious publication bias was observed in Begg test or Egger test.

However, several important limitations should also be acknowledged in this study. First, statistical significantly heterogeneity was detected across included studies ($P < .078$ for heterogeneity, $I^2 = 36.4\%$), which might affect the robustness of the pooled risk estimates. We removed the studies that contributed to the heterogeneity via the Galbraith plot and recalculated the combined RR, which did not remain significant (RR: 0.99, 95% CI 0.94–1.04). This result implied that the overall analysis was not substantially affected by the heterogeneity. Second, most of the eligible studies were case–control studies, which were less conclusive than cohort studies and might introduce the select bias and recall bias. Third, although the majority of the included studies adopted the Norwood–Hamilton scale to assess the baldness, differences in exposure assessment still existed, which might distort the combined risk estimates. Finally, gray literature (e.g., conference abstracts) and studies reported in languages other than English or Chinese were less likely to be retrieved, which might lead to a certain degree of publication bias.

In conclusion, this systematic review and meta-analysis allowed us to perform overall analysis of male pattern baldness with prostate cancer risk, as well as various subgroup analyses with strong statistical power. As a result, we found that individuals with vertex baldness had an increased risk of prostate cancer. Given the obvious heterogeneity and null results in overall analysis and most of subgroup analyses, further large well-designed prospective cohort studies are warranted to confirm our preliminary findings.

Author contributions

Conceptualization: Huadong He, Bo Xie, Liping Xie.
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Visualization: Huadong He, Bo Xie, Liping Xie.
Writing – original draft: Huadong He, Bo Xie, Liping Xie.
Writing – review & editing: Huadong He, Bo Xie, Liping Xie.

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