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Review

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Association of body mass index and smoking at onset with glenohumeral osteoarthritis: A systematic review and meta-analysis

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Abstract

Objectives: In this review, we synthesize quantitative and qualitative evidence on the relationship between body mass index (BMI), smoking and the development of glenohumeral (GH) osteoarthritis (OA).

Materials and methods: We conducted a comprehensive literature search in electronic databases such as MEDLINE/PubMed, EMBASE and Web of Science for studies published between 2000 to 2023. Studies examining the association between BMI, smoking, and GH OA were selected. Among the included studies, two studies were cross-sectional, two were case control, one was retrospective, and one prospective. We used Newcastle-Ottawa Quality Assessment Scale (NOS) to assess the quality of the studies and used RStudio to perform the meta-analysis.

Results: A total of 24 articles reporting on various risk factors for GH OA were initially retrieved. Of these, five studies specifically investigated BMI and three examined smoking as potential risk factors. The odds ratio (OR) for BMI (OR=1.35; 95% confidence interval [CI] 1.04-2.22) and smoking (OR=3.14; 95% CI 1.23-9.38) were significantly associated with GH OA.

Conclusion: The present systematic review and meta-analysis suggests that higher BMI and smoking are significantly associated with an increased risk for GH OA. Future studies should focus on larger, more diverse populations and evaluate the potential impact of structured lifestyle interventions in reducing the incidence or delaying the progression of GH OA.

Keywords: Body mass index, glenohumeral osteoarthritis, meta-analysis, smoking, systematic review.

Osteoarthritis (OA) is the leading cause of joint pain and disability. By 2020, an estimated 595 million individuals (7.6% of the global population), were affected by OA, with numbers expected to rise significantly by 2050.[1,2] Osteoarthritis is characterized by degeneration of articular

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cartilage, subchondral bone, periarticular bone, and soft tissue such as muscles and ligaments.[3] Glenohumeral (GH) OA is the third largest joint affected by OA following the hip and knee OA, with a prevalence of 17 to 19% among individuals aged above 40 years and 60 years, respectively.[4-6] Typically, GH OA causes pain and limits everyday activities. The etiology of GH OA is not clear and involves multiple factors such as age, sex, obesity, smoking, genetics, and occupations involving overuse of the shoulder.[6] It has a complex pathogenesis and is categorized into two groups: primary (age related degeneration) and secondary (due to trauma, inflammation, and dislocation).[7] The role of risk factors remains ambiguous, of these risk factors body mass index (BMI) and smoking have been reported to play an important role in the pathogenesis of knee or hip OA.[7]

Body mass index is a well-established modifiable risk factor associated with increased incidence and progression of OA in weight bearing joints such as hip and knee.[8-10] While a few studies have reported an association between BMI and GH OA,[11] the relationship still remains unclear. There is growing evidence that genetic architecture of OA is joint-specific, driven by localized gene expression patterns, such as *HOX gene* signatures, that shape the tissue microenvironment in each joint.[12] Of note, BMI is known to influence key biological processes including regulating cell phenotypes, cell fate, extracellular matrix metabolism and inflammation, all of which are involved in the pathophysiology of degenerative joint diseases. Given these systemic effects, it is plausible that BMI may also play a role in GH OA, despite it being a non-weight-bearing joint.[13] Similarly, while smoking is a well-established risk factor associated with the prevalence of OA,[14] its role in GH OA remains unclear. To date, only three studies have reported a potential association between smoking and GH OA.[7,10] Additionally, preoperative smoking has been linked to increased complications following total shoulder arthroplasty.[15] Given the limited and inconsistent evidence, a systematic understanding of the relationship between BMI, smoking and GH OA is lacking.

To the best of our knowledge, a limited number of studies, spanning cross-sectional, case-control, prospective and retrospective designs, have examined the role of BMI and smoking in relation to GH OA. However, precise estimates of the association between these risk factors and GH AO remain lacking. In the present review, we hypothesized that higher BMI and smoking were significantly associated with an increased risk of GH OA. We, therefore, synthesize quantitative and qualitative evidence on the relationship between BMI, smoking and the development of GH OA.

MATERIALS AND METHODS

Search strategy

We conducted the systematic review following guidelines on Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA). A protocol was developed before literature search and registered in the PROSPERO (CRD: CRD42022371283). A systematic literature search was conducted using a combination of search words on the following electronic databases: MEDLINE (PubMed), EMBASE and Web of Science up to March 15th, 2023. Appropriate grouping of keywords such as GH OA, BMI, obesity, smoking, and former smokers, were used to perform the literature search (Supplementary Table 1).

Study selection

A total of 7,073 articles were identified from all the databases and exported into EndNote 20 bibliographic software (Thomson Reuters, NY, USA). After removing duplicates, all articles were transferred into Rayyan, a free platform (http://rayyan.qcri.org) to screen the titles and abstracts easy for reviewers. Two independent investigators conducted an initial screening of all the relevant research articles and in case of disagreement between two investigators, a decision was taken by a third investigator. Studies were excluded, if they did not meet the inclusion criteria or those were not original articles. Eligibility criteria for inclusion of studies were study design (cross-sectional, case control, retrospective, prospective or retrospectively prospective) and publication in English literature. Studies with an age group ≥ 18 years assessing the relationship

between GH OA with BMI and smoking were included in the review and meta-analysis.

Quality assessment

According to the guidelines of Newcastle-Ottawa Quality Assessment Scale (NOS), two independent researchers conducted the methodological quality assessment, which is the functioning of assessing inter-rater reliability. In case of any discrepancies, a consensus was achieved through consultation with a third assessor. The NOS is a quality assessment instrument used for non-randomized studies including case-control and cohort studies in a systematic review. The NOS produces a comprehensive measure by addressing study design, content, and the quality of outcome reporting.[16] Studies were categorized into three groups and a score of good (7–8 stars), fair (5–6 stars) and poor (4 stars) allocated based on NOS criteria.[16]

Assessment of risk factors

Smoking and BMI were examined as risk factors associated with GH OA.

Data abstraction

A standard approach was used to retrieve data from full text articles including research characteristics such as study title, year of publication, first author, study design, age, sex, BMI, smoking, number of cases and controls. Full-text articles for selected studies were retrieved; BMI and smoking data were recorded in a spreadsheet. We used odds ratio (OR) from multivariate model and 95% confidence interval (CI) to assess the measure of association between risk factors and GH OA.

Statistical analysis

Statistical analysis was performed using the RStudio version 4.2.2 using 'meta' and 'metafor' package (R Foundation for Statistical Computing, Vienna, Austria).[17,18] A conventional random effects mixed model was used to assess the association of BMI with GH OA, including estimating log OR and study variance followed by assuming each study ID a random effect. To measure the effect of association, we used an OR and 95% CI. We preferred a multivariable estimate over unadjusted estimates. If odds estimates were missing for a study, we manually calculated OR from a given standard error and other estimates provided in the study. Estimates of OR were pooled using a random effects mixed model. In the meta-analysis, Cochran's Q test and Higgins I^2 index statistics were used to measure heterogeneity. A two-sided p value of < 0.05 was considered statistically significant. We assumed that BMI was normally distributed. Therefore, for studies that reported BMI as a categorical variable, we calculated the average BMI in each category divided by the number of categories used to obtain an overall average BMI estimate. In studies, where BMI was reported categorically, such as in predefined ranges (e.g., underweight: < 18.5, normal: 18.5-24.9, overweight: 25-29.9, obese: ≥ 30), it was challenging to directly incorporate these data into analyses requiring continuous measurements. To address this issue, we estimated a continuous BMI value by calculating the average BMI within each category and, then, combining these estimates to derive a single representative average BMI. Furthermore, the Begg's test and Egger's test were used to address the publication bias and to provide insight into the potential bias and reliability of the included studies.[18,19] A forest plot was generated using meta and metafor package to provide a more comprehensive and reliable understanding between BMI and GH OA.

RESULTS

Literature search

Figure 1 describes the literature search process. We identified a total of 7,073 articles in our initial search and 2,867 articles were removed due to duplications. Of 4,206 articles, an additional 4,182 studies that did not match our selection criteria were removed. Further review of the remaining 24 studies, 18 studies were excluded due to reporting on age, sex and critical shoulder angle and failed to report BMI and smoking as risk factors. The remaining six studies[7,10,19-23] met the inclusion criteria. Finally, five studies reported BMI-specific, and three studies smoking-estimates were included.

Study characteristics and quality assessment

The data on 28,968 patients from six studies were reviewed. Supplementary Table 2 shows the

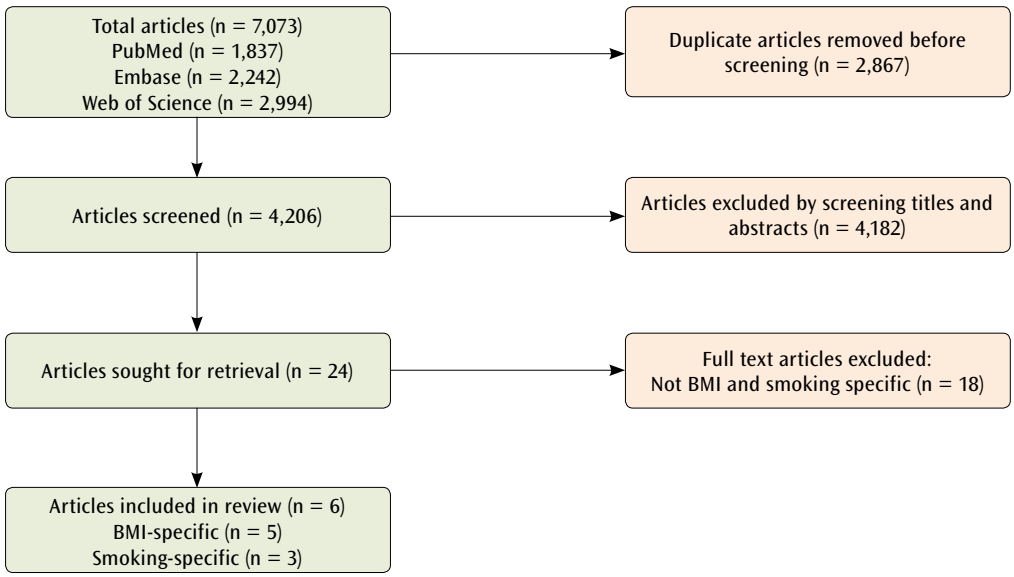

Figure 1. PRISMA flow diagram for the systematic review.
PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analyses; BMI, body mass index.

characteristics of studies included. The age of participants in our analysis ranged from 43 to 77 years. Four studies[10,20,22,23] reported OR and 95% CI to measure the association between risk factors (BMI and smoking) and GH OA. From a total of six studies, five and three studies were used to assess the association GH OA with BMI and smoking respectively. Out of six studies included, two were cross-sectional, two were case control, one was prospective, and one was retrospective. Glenohumeral OA was classified based on imaging records in each study. The included studies were either driven from population-based cohorts in China, Korea and Italy or selected participants of anterior shoulder instability who were subsequently followed for the development of GH OA in US based study (Supplementary Table 2). According to the NOS, five studies were classified as good quality, whereas one study was of fair quality (Table 1).

Body mass index and glenohumeral osteoarthritis

Five studies reported BMI-specific risk estimates for GH OA either as a categorical or continuous variable.[7,10,20-23] Of five studies, two were cross-sectional, one was case-control, one prospective cohort, and one retrospective study. Wall et al.[23] reported BMI in categorical form (< 19, 19–24, 25–29, 30–34, 35–39, and > 39), BMI in a group 25–29 and less was not associated (OR = 1.11; 95% CI 0.93–1.31) with GH OA. However, BMI in categories 30–34 (OR = 1.22; 95% CI 1.04–1.43), 35–39 (OR = 1.59; 95% CI 1.36–1.85) and > 39 (OR = 1.56; 95% CI 1.34–1.82) reported a significant association between BMI

Table 1. Quality assessment using the Newcastle-Ottawa Quality Assessment scale of included studies

	Selection				Comparability	Outcome/exposure			
Article	1	2	3	4	1	1	2	3	Total score
Siviero et al.[22] 2009	*	*	*	*	*	*	*	*	8 (Good)
Cho et al.[20] 2015	*	*	*		**	*	*	*	8 (Good)
Zhang et al.[21] 2016	*	*	*		**	*	*	*	8 (Good)
Wall et al.[23] 2020	*	*	*		*	*			5 (Fair)
Kruckeberg et al.[10] 2020	*	*	*	*	*	*	*	*	8 (Good)
Plachel et al.[7] 2023	*	*	*	*	*	*	*	*	8 (Good)

Table 2. Characteristics of studies included in the meta-analysis

Trial name	Year	Country	Total population	Study design	OR (95% CI) for BMI	OR (95% CI) for smoking
Siviero et al.[22]	2009	Italy	851	Prospective cohort	1.11 (0.76–1.64)	
Cho et al.[20]	2015	South Korea	36	Cross-sectional	1.7 (0.8–3.8)	1.26 (1.15–1.37)
Zhang et al.[21]	2016	China	211	Cross-sectional	1.6 (1.40–3.06)	
Wall et al.[23]	2020	USA	27,803	Retrospective cohort	1.28 (1.22–1.33)	
Kruckeberg et al.[10]	2020	USA	35	Case control	1.2 (1.03–1.3)	4.3 (1.1–16.5)
Plachel et al.[7]	2023	Germany	32	Case control		3.88 (1.44–10.27)

OR, Odds ratio; CI, confidence interval.

with GH OA. Zhang et al.[21] categorized BMI as low, normal, overweight, and obese and reported increased prevalence of GH OA along with an increase in BMI (OR = 1.6; 95% CI 1.40–3.06; p < 0.001). All remaining studies reported BMI as a continuous variable.[7,10,20,22] Cho et al.[20] reported a mean BMI of 24.5 with a prevalence of 5.2% for shoulder GH OA. A high BMI > 25 kg/m^2 had no association with GH OA (OR = 1.7; 95% CI 0.8–3.8; p = 0.162). Similarly, Siviero et al.[22] also did not find any significant association between high BMI versus GH OA (OR = 1.11; 95% CI 0.76–1.64; p = 0.58). Similar findings were also reported by Kruckeberg et al.[10] where high BMI (27 ± 1) was significantly associated with GH OA (OR = 1.2; 95% CI 1.03–1.3; p = 0.012) (Table 2).

Meta-analysis

We pooled the results from all five studies and analyzed the effect of BMI on GH OA. A pooled OR of 1.35 (95% CI 1.04–2.22) was observed with negligible heterogeneity (I^2 = 0.00%)

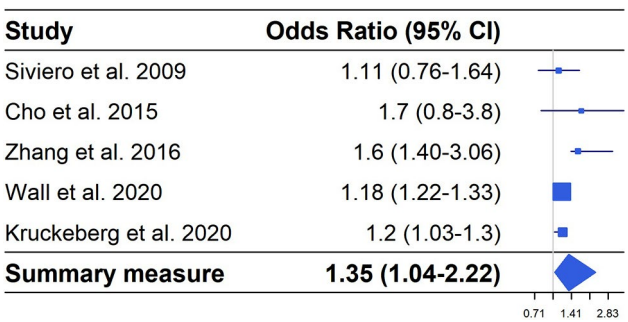

Figure 2. Forest plot showing association between BMI and glenohumeral osteoarthritis. Figure showing effect estimates (odds ratios), 95% CI and the summary measure of contributing studies evaluating the association between BMI and glenohumeral osteoarthritis.

BMI, body mass index; CI, confidence interval.

(Figure 2 and Table 3). The Begg's regression test and Egger's test did not show any publication bias (p = 0.083 and p = 0.201, respectively) (Table 3). Since there was no publication bias and heterogeneity were negligible, the findings support the robustness of the pooled estimates (OR = 1.35; 95% CI 1.04–2.22).

Smoking and glenohumeral osteoarthritis

Three studies reported smoking-specific risk estimates for GH OA.[7,10,21] Out of these, two were case-control and one was cross-sectional studies. Zhang et al.[21] reported an association between smoking and GH OA with an OR of 1.26 (95% CI 1.15–1.37). Kruckeberg et al.[10] reported an OR of 4.3 (95% CI 1.1–16.5) for current or former smokers and a significant association between smoking and GH OA. Furthermore, Plachel et al.[7] also reported a significant association between smoking and GHOA (OR = 3.88; 95% CI 1.44–10.27) (Table 2).

Meta-analysis

We pooled the results from three studies to analyze the effects of smoking on GH OA. The pooled OR was 3.14 (95% CI 1.23–9.38) (Figure 3) with a substantial heterogeneity (I^2 = 71.72%). The odds of having GH OA in smokers was 3.14 times greater than the odds of GH OA in non-smokers. The Begg's regression and Egger's test did not show any publication bias (p = 1.00 and p = 0.95, respectively) (Table 3). Although considerable heterogeneity was observed for smoking, there was no evidence of publication bias. However, the small number of included studies limits the reliability of these assessments and suggests variability across studies in the pooled effect size (OR = 3.14; 95% CI 1.23–9.38).

Table 3. Meta-analysis for BMI and smoking with GH OA

Studies	Number of studies	Total population	Pooled OR (95% CI)	Heterogeneity (I^2)	Begg's test, Egger's test
BMI	5	28,936	1.35 (1.04–2.22)	0.00%	0.08, 0.20
Smoking	3	103	3.14 (1.23–9.38)	71.72%	1, 0.95

BMI, body mass index; GH, glenohumeral; OA, osteoarthritis; OR, Odds ratio; CI, confidence interval.

DISCUSSION

We performed a systematic review and meta-analysis on the association of BMI and smoking with GH OA. Our meta-analysis revealed a significant association between higher BMI and increased risk of GH OA with a pooled OR of 1.35 (95% CI 1.04–2.22) and observed no heterogeneity. Additionally, smoking was strongly associated with GH OA (pooled OR: 3.14 95% CI 1.23–9.38), although substantial heterogeneity was present.

Our findings suggest that higher BMI increases the risk of developing GH OA by 35% and is significantly associated with the overall burden of the disease. Previous studies have also explored the relationship between BMI and GH OA.[7,10] Plachel et al.[7] reported a significant association between higher BMI (higher in cases than control) and GH OA (p = 0.017) in a retrospective cohort study. However, the small sample size limited the reliability of the findings. Similarly, a prospective study reported a 20% increased risk of GH OA in individuals with higher BMI, but again, the sample size posed limitations to the robustness of the results.[10] A third study with a larger sample size found a 28% increased risk of GH OA associated with elevated BMI.[23] Despite the adequate sample size, this study did not meet quality standards upon assessment.

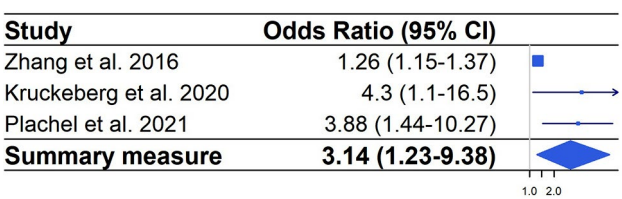

Figure 3. Forest plot showing association between smoking and glenohumeral osteoarthritis. Figure showing effect estimates (odds ratios), 95% CI and the summary measure of contributing studies evaluating the association between smoking and glenohumeral osteoarthritis.

CI, confidence interval.

Our meta-analysis reflects a similar trend showing that higher BMI is significantly associated with GH OA, with an OR of 1.35. The underlying biological mechanism may involve metabolic and inflammatory pathways. Obesity is often characterized by excessive nutrient intake, leads to insulin resistance, and the accumulation of adipose tissue which disrupts the balance between innate and adaptive immune responses.[24,25] A few studies have indicated that accumulation of adipokines, leptin and adipose tissue in synovial fluid of GH joint also mediates in the cartilage degeneration.[26-29] Emerging evidence suggests that pathophysiology of OA varies across different joints, driven by joint-specific molecular and biomechanical functions.[12] These differences likely influence susceptibility, progression and response to treatment. It is reported that high inflammation also leads to disease progression and limits the functional ability of OA patients.[23]

While the association between higher BMI and OA is well-established in weight-bearing joints such as knee and hip, our findings suggest a similarly significant association in the non-weight-bearing GH joint.[30] This implies that distinct molecular mechanisms may be at play and underscores the need for further clinical and mechanistic studies to better understand and address this relationship.

Our study found a significant association between smoking and the development of GH OA. However, existing literature on this relationship is limited, with only a few studies available with a small sample size. One study reported that current smokers had a significantly higher prevalence of concentric GH OA compared to healthy controls, while individuals with eccentric GH OA demonstrated higher shoulder activity level than both healthy controls and those with concentric GH OA. Similar to our findings, a prospective cohort study also reported higher

odds of developing GH OA among smokers, suggesting that smoking may pose a serious risk.[10] However, the small sample sizes and heterogeneity in study design across these studies reduce the reliability of the evidence. Potential selection bias due to participant recruitment from small cohorts, along with ethnic and geographical variations influencing lifestyle factors, may also have contributed to inconsistencies in the data. However, the likely mechanism linking smoking to GH OA may involve nicotine, which is known to alter the expression of multiple proteins including proteases and cytokines. Nicotine has been observed to increase the production of matrix metalloproteinase-1 and secretion of chitinase 3-like protein fibronectin, all of which contribute to joint inflammation.[14] Additionally, nicotine negatively affects cartilage metabolism, which may accelerate cartilage degeneration and contribute to the development of OA in the GH joint.[31]

Although our study found significant associations between BMI, smoking and GH OA, it is important to consider that these relationships may be influenced by confounding factors such as physical activity, occupational exposures and host genetics not fully accounted for in the included studies. Repetitive use of the shoulder, overhead activities and heavy weightlifting are mechanical risk factors and may correlate with smoking and BMI potentially acting as confounders. Similarly, genetic susceptibility may contribute to the development of GH OA and influence body composition indirectly increasing the risk for GH OA.

Our study has major strength. This is the first meta-analysis to examine the association of BMI and smoking with GH OA. This meta-analysis included all the English language studies between the years 2000 through March 2023, thereby covering two decades of the studies published in this field. However, our study has certain limitations that should be acknowledged. First, the number of studies that met our eligibility criteria was relatively small, and the included studies varied substantially in sample size, which may have influenced the precision of the pooled estimates despite use of a random effects model. Second, BMI was reported inconsistently across studies, both as categorical and continuous variables which required us to estimate average BMI values by assuming normal distribution within categories. This introduces uncertainty and may not fully capture within category variance. Third, in studies where ORs were not directly reported, we derived estimates using available statistical data (e.g., standard error and estimates or counts) which may be less robust than adjusted, author-reported values. These findings underscore the importance of managing modifiable risk factors, such as weight management and smoking cessation as a potential preventive strategy for GH OA. Although causality cannot be established given the nature of observational studies included, these associations are clinically relevant. They offer a strong rationale for future prospective studies and clinical trials to further evaluate the efficacy of structured lifestyle interventions in reducing the incidence or delaying the progression of GH OA.

In conclusion, we observed a significant association of both BMI and smoking with the risk of developing GH OA. Our meta-analysis revealed that increasing BMI was associated with higher odds of GH OA, and smokers exhibited a 3.14-fold increased likelihood of being diagnosed with GH OA compared to non-smokers. While our findings provide robust evidence supporting these associations, future research should focus on larger, more diverse populations and assess the efficacy of structured lifestyle interventions in reducing the incidence or delaying the progression of GH OA.

Declaration of Conflicting Interests

The authors declare that there are no conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Author Contributions

R.P.: Analyzed the data and wrote the manuscript; N.B.J.: Designed and facilitated the study and reviewed the final draft of the manuscript; F.D.A: Provided essential construct in the analysis of the data and reviewed the final version of the manuscript; J.E.G.: Performed the literature

search, extracted the data and reviewed final version of the manuscript; U.B.P.: Extracted the data and approved final version of the manuscript; R.P.: Designed and supervised extraction of the data, wrote this manuscript, reviewed and approved the final version of the manuscript.

Data Availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

AI Disclosure

The authors declare that artificial intelligence (AI) tools were not used, or were used solely for language editing, and had no role in data analysis, interpretation, or the formulation of conclusions. All scientific content, data interpretation, and conclusions are the sole responsibility of the authors. The authors further confirm that AI tools were not used to generate, fabricate, or 'hallucinate' references, and that all references have been carefully verified for accuracy.

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