

Dry Eye Disease Across the Menopausal Transition: A Systematic Review and Meta-Analysis

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Abstract

BACKGROUND/OBJECTIVES:

Dry eye disease (DED) disproportionately affects women, and hormonal changes across the menopausal transition have been proposed as contributors. This prospectively registered (PROSPERO: CRD420261390285) systematic review and meta-analysis examined whether the odds of prevalent DED differs between premenopausal, perimenopausal, and postmenopausal women.

SUBJECTS/METHODS:

MEDLINE, Embase, and Cochrane Library were searched to September 17, 2025. Sixteen observational studies met inclusion criteria, of which ten provided extractable data for meta-analysis. Study designs were cross-sectional, and no randomized studies were identified. The analyses compared postmenopausal women with (1) combined premenopausal and perimenopausal women, (2) perimenopausal women alone, and (3) strictly premenopausal women. Risk of bias was assessed using ROBINS-E. Random-effects Mantel-Haenszel models generated pooled odds ratios (ORs) comparing the odds of prevalent DED across menopausal stages.

RESULTS:

Postmenopausal women had significantly higher odds of prevalent DED than pre/perimenopausal women (OR 2.05, 95% CI 1.28–3.31, $p = 0.003$; $I^2=82\%$). Compared with strictly premenopausal women, postmenopausal women had higher odds of prevalent DED (OR 2.42, 95% CI 1.23–4.75, $p = 0.010$; $I^2=82\%$). The comparison with perimenopausal women showed a nonsignificant association in the same direction (OR 1.32, 95% CI 0.79–2.18, $p = 0.287$; $I^2=88\%$). Non-pooled studies supported increased DED burden with advancing menopausal stage. Overall certainty of evidence was low due to residual confounding, cross-sectional designs, and substantial heterogeneity.

CONCLUSIONS:

DED appears more prevalent in postmenopausal women than in earlier reproductive stages, although heterogeneity and confounding limit causal inference. Longitudinal studies with standardized menopausal staging and definitions are needed to clarify hormonal and age-related contributions.

SUMMARY

What was known before

- Dry eye disease disproportionately affects women, particularly in midlife and older age, and sex-steroid changes during the menopausal transition have been proposed as contributors.
- Prior observational studies suggested higher dry eye burden after menopause, but findings were limited by variable dry eye definitions, inconsistent menopausal staging, and lack of pooled quantitative estimates.

What this study adds

- In this meta-analysis, postmenopausal women had higher odds of prevalent dry eye disease than pre/perimenopausal women combined (OR 2.05, 95% CI 1.28–3.31; $I^2=82\%$) and strictly premenopausal women (OR 2.42, 95% CI 1.23–4.75; $I^2=82\%$).
- The comparison between postmenopausal and perimenopausal women showed a smaller, non-significant association in the same direction (OR 1.32, 95% CI 0.79–2.18; $I^2=88\%$), suggesting perimenopause may represent an intermediate-risk state.
- Although the direction of effect was generally consistent across pooled and non-pooled studies, the certainty of evidence was low because of cross-sectional study designs, substantial heterogeneity, and residual confounding from age and comorbidities.

INTRODUCTION

Dry eye disease (DED) is a multifactorial disorder of the ocular surface characterized by loss of tear-film homeostasis, tear-film instability, hyperosmolarity, ocular surface inflammation, and neurosensory abnormalities recognized as central features of its pathophysiology.¹ DED represents a significant public health concern due to its substantial impact on vision-related functioning, including fluctuating vision, ocular discomfort, and reduced quality of life.^{2,3} Epidemiologic studies consistently demonstrate that DED disproportionately affects women, with large population-based cohorts reporting higher prevalence among middle-aged and older female individuals.^{4,5} The TFOS DEWS II Epidemiology Report further identifies female sex and increasing age as key determinants of DED burden globally.⁶

Hormonal influences are increasingly recognized as critical drivers of this sex-based disparity. According to the TFOS DEWS II Sex, Gender, and Hormones Report, fluctuating and declining sex-steroid levels, particularly estrogen and androgens, affect the lacrimal functional unit by modulating lacrimal gland secretion, meibomian gland lipid production, and ocular surface inflammatory pathways.⁷ Supporting this biological framework, human lacrimal gland tissue demonstrates expression of both androgen and estrogen receptors, suggesting a direct endocrine influence on ocular surface physiology.⁸ Furthermore, experimental and clinical data demonstrate that androgen deficiency is associated with meibomian gland dysfunction and altered lipid profiles, contributing to evaporative forms of DED.^{9,10}

Clinically, several observational studies and reviews have suggested that DED is more prevalent and often more symptomatic among women undergoing the menopausal transition.^{11–14} These findings are consistent with the endocrine changes characteristic of reproductive aging, in which declining and fluctuating hormone levels may disrupt tear-film stability and ocular surface homeostasis. However, the degree to which DED prevalence differs among premenopausal, perimenopausal, and postmenopausal women remains uncertain due to heterogeneity in diagnostic criteria, variation in menopausal staging, and differences in how comparative groups are defined across studies.^{4, 5, 11–14}

The objective of this systematic review and meta-analysis is to synthesize the current evidence on both the prevalence and relative odds of DED across distinct menopausal stages. By examining premenopausal, perimenopausal, and postmenopausal groups separately, we aim to clarify whether DED burden follows a progressive gradient across the menopausal transition. We hypothesize that DED is most likely to occur among postmenopausal women, reflecting underlying hormonal changes affecting the lacrimal functional unit.

MATERIALS/SUBJECTS AND METHODS

This study adhered to the Meta-analyses Of Observational Studies in Epidemiology (MOOSE) and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. Need for ethics review was waived since this data relied entirely on published primary research. This study adhered to the tenets of the Declaration of Helsinki. The study was prospectively registered on PROSPERO (CRD420261390285).

Search and Study Selection

A MEDLINE, Embase, and Cochrane Library search was conducted up to September 17th, 2025, to identify studies reporting DED counts for any of the following menopausal stage comparisons: (1) premenopausal + perimenopausal vs postmenopausal, (2) perimenopausal vs postmenopausal, or (3) strictly premenopausal vs postmenopausal. The librarian-informed search strategy for this search is available in Supplementary Table 1. Reference lists of included studies were examined to identify candidate studies of any kind that were not initially captured in the search. We also included grey literature since evidence supports that including such works promotes more conservative outcome analysis.¹⁵ There were no restrictions on study language, population age, menopausal stage definitions, or duration of follow-up. In cases where multiple studies reported on overlapping or identical cohorts, the most recent or most comprehensive report was used for data extraction. Where applicable, we attempted to contact study authors to obtain relevant study data or publication copies. Study records were managed using Covidence (Veritas Health Innovation, Melbourne, Australia). The titles, abstracts, and full texts of each record were screened by two independent reviewers (two of F.N., MA., or A.K.). Conflicts were resolved through discussion with a third reviewer (B.T.) and where a consensus was not reached, the final decision was adjudicated by the senior author (M.K.).

Data Extraction

A standardized pre-piloted data extraction form was prepared in Microsoft Excel (v16.62; Redmond, Washington, United States). Data extraction proceeded in an independent pairwise fashion by a combination of two out of three reviewers (two of F.N., MA., or A.K.). Study risk of bias (ROB) was evaluated using the ROB In Non-randomized Studies of Exposure tool (ROBINS-E) in the same fashion. Conflicts were resolved through discussion with a third reviewer (B.T.), and where a consensus was not reached, the final decision was adjudicated by the senior author (M.K.).

From each included study, we extracted data on study characteristics (e.g., title, journal, year, country, and city), study design (e.g., design type, population setting, sample size), and participant characteristics (e.g., mean or range of age, sex distribution, and criteria used to define menopausal status). We also collected details on DED definitions, diagnostic methods, total number of women enrolled, and the stratification of participants by menopausal stage (premenopausal, perimenopausal, and postmenopausal), with and without DED, according to each study. Menopausal-stage definitions were accepted as reported by the original studies and varied across the literature. Comparator groups were synthesized according to the classifications used in the primary literature.

Statistical Analysis

The primary outcome was the literature-pooled odds ratio (OR; with 95% confidence interval, CI) comparing DED prevalence between menopausal stage groups. Three pairwise comparisons were evaluated: (1) postmenopausal versus premenopausal women (including perimenopausal counts), (2) postmenopausal versus perimenopausal women, and (3) postmenopausal versus premenopausal women (excluding perimenopausal counts). Comparator groups were derived directly from the menopausal-stage classifications and counts reported in each study, without reclassification. Studies that reported only premenopausal (and not perimenopausal) participants contributed to both the combined pre/perimenopausal comparator, representing a broader reference group, and the strictly premenopausal comparator, resulting in overlapping study inclusion and identical counts across these analyses where applicable. A pairwise random-effects meta-analysis using the Mantel–Haenszel method was conducted, with between-study variance estimated using the DerSimonian-Laird method. For all outcomes an I^2 was calculated to assess for statistical heterogeneity, following these thresholds: i) might not be significant (0–30%); ii) moderate (30–60%); substantial (50–90%); and considerable (75–100%) heterogeneity.¹⁶ Funnel plots were generated to assess publication bias. All analysis and figure generation were completed with Review Manager (RevMan) Version 7.2.0 (Cochrane Collaboration, London, United Kingdom). The GRADE tool was used to evaluate for certainty of evidence.¹⁷

RESULTS

The initial search strategy across all included databases identified 3545 studies, of which 114 were assessed further in full text (Fig. 1). A total of 98 full-text articles were excluded for the following

reasons: 1 was not accessible; 8 were non-primary articles; 2 were ongoing clinical trials; 6 included participants on hormone therapy; and 81 did not include counts of the comparison groups. Overall, 16 studies met the inclusion criteria for review.^{18–33}

Among the 16 included studies, ten provided distinct counts suitable for meta-analysis.^{19, 22, 23, 25, 27–29, 31–33} Nine of these included studies were cross-sectional in design.^{19, 22, 23, 25, 27–29, 31, 33} However, one study (Bergamaschi 2019) described by authors as “prospective observational” but conducted as a single-timepoint self-reported OSDI survey, was assessed to be more cross-sectional in nature, and included.³² Additionally, one study (Garcia-Marques 2022) did not have explicit reporting of the amount of menopausal women with DED in their manuscript, however, the exact amount was verified through data request from the corresponding author.²⁷ Among the six studies not included in the meta-analysis, five were observational cross-sectional designs, with one study being a secondary cross-sectional analysis of a randomized controlled trial (the DREAM study).^{18, 20, 21, 24, 26, 30} Although these studies could not contribute quantitative data to the meta-analysis, their findings will be narratively summarized to contextualize, complement, or, where applicable, challenge the patterns observed in the pooled analyses. The specific characteristics of each included study are summarized in Supplemental Table 2.

Table 1 summarizes the ROBINS-E assessments across the 16 included studies. Overall, 12/16 studies (75%) were rated as “high” risk of bias, and 4/16 (25%) were rated at “some concerns”. Many studies were downrated to “high” due to bias due to confounding, as studies lacked a consistent definition or adjustment for for key determinants of both menopausal status and DED; particularly age, systemic comorbidities, hormone therapy, and tear-affecting medications. Other domains showed fewer issues: exposure measurement was low risk in 13/16 studies (81%), participant selection showed some concerns in 14/16 (88%), post-exposure interventions were low risk in 10/16 (62%), missing data was low risk in 15/16 (94%), outcome measurement was low risk in 9/16 (56%), and selection of reported results showed some concerns in 15/16 (94%). Taken together, confounding, rather than measurement or reporting quality, was the dominant limitation in this literature.

Postmenopausal compared to Premenopausal, including Perimenopausal

For the first comparator group, which compared postmenopausal women with premenopausal women (with perimenopausal women included in the premenopausal category), 10 studies contributed data, comprising 7,607 participants in total (3,462 postmenopausal and 4,145 premenopausal).^{19, 22, 23, 25, 27–29, 31–33} Across these, 2,760 women had DED, of whom 1,532 (55.5%) were postmenopausal and 1,228 (44.5%) were pre/perimenopausal. Overall, postmenopausal status was associated with a significantly higher odds of DED compared with pre/perimenopausal status (OR: 2.05, 95% CI [1.28, 3.31]; $p = 0.003$; $I^2 = 82.0\%$ [considerable heterogeneity]) (Fig. 2).

Postmenopausal compared to Perimenopausal, strictly

A second comparator meta-analysis evaluated postmenopausal versus perimenopausal women across three studies, encompassing 2,794 participants (1,687 postmenopausal and 1,107 perimenopausal).^{22, 28, 33} Of the 980 women with DED in this analysis, 627 (64.0%) were postmenopausal and 353 (36.0%) were perimenopausal. Although the point estimate favored a higher odds of DED among postmenopausal women, the pooled effect did not reach statistical significance (OR: 1.32, 95% CI [0.79, 2.18]; $p = 0.287$; $I^2 = 88.3\%$ [considerable heterogeneity]) (Fig. 3).

Postmenopausal compared to Premenopausal, excluding Perimenopausal

A third comparator analysis restricted the reference group to strictly premenopausal women and excluded perimenopausal observations, yielding eight studies with 5,143 participants (2,105 postmenopausal and 3,038 premenopausal).^{19, 23, 25, 27, 29, 31–33} In this analysis, 1,949 women had DED, including 1,074 (55.1%) postmenopausal and 875 (44.9%) strictly premenopausal women. The pooled odds ratio demonstrated a significantly higher odds of DED among postmenopausal women (OR: 2.42, 95% CI [1.23, 4.75]; $p = 0.010$; $I^2 = 82.3\%$ [considerable heterogeneity]) (Fig. 4). Visual inspection of the funnel plots for all three analyses (Fig. 5A-C) suggested only mild asymmetry overall; for the first comparator group, neither Egger's test ($p = 0.158$) nor Harbord's test ($p = 0.181$) indicated significant small-study effects, and regression-based asymmetry tests were not performed for the second and third comparator groups due to the limited number of contributing studies.

Among studies that could not be incorporated into the meta-analysis, several offered relevant findings. Shakoor et al. (2022) stratified women as pre- or postmenopausal and then further by Schirmer test severity categories (mild, moderate, severe) but did not report the number of women with normal Schirmer values in each group, precluding construction of a full 2×2 table.¹⁸ However, they noted that abnormal Schirmer test results and clinically defined DED were more frequent among postmenopausal women. Ayaki et al. (2024) examined DED prevalence across age bands chosen to approximate pre-, peri-, and postmenopausal stages (36–45, 46–55, and 56–65 years), but did not use hormonal or menstrual criteria to classify menopausal status and therefore did not meet our definition.²⁰ Nevertheless, they observed an age-related increase in the proportion of women with DED (approximately 55%, 54%, and 60% across these age groups, respectively), suggesting a directional trend, albeit with a non-significant overall p value ($p = 0.19$). Chen et al. (2013, grey literature) reported that 163 of 371 female patients with DED (43.9%) had presentations attributed to hormonal changes, and that meibomian gland-related DED increased with age, supporting a hormonal contribution but without providing stratified counts by menopausal stage.³⁰ Li et al. (2015) found that 163 of 371 female participants with clinically diagnosed DED (43.9%) were in a menopausal or postmenopausal state, indicating that this group constituted a large proportion of affected women, although the absence of corresponding non-DED counts by menopausal status prevented inclusion in the pooled analysis.²⁶ Pereira et al. (2017, grey literature) identified menopause as one of three factors significantly associated

with DED in multivariable analysis, with an odds ratio of 2.39 (95% CI 1.07–5.35).²⁴ Finally, Zhao et al. (2024) demonstrated that postmenopausal women had significantly worse objective DED signs than premenopausal women, including higher corneal and conjunctival staining scores, increased tear osmolarity, shorter tear break up time (TBUT), more severe meibomian gland dysfunction, and higher composite DED sign scores, while reporting no statistically significant differences in symptom scores by menopausal status.²¹ Collectively, these non-pooled studies generally align with, and in some cases strengthen, the observed association between menopausal status and increased DED burden.

Based on the GRADE framework, the certainty of evidence for the association between menopausal stage and DED was rated as low.¹⁷ All studies were observational, yielding a low starting certainty, and most carried some risk of bias that was driven primarily by residual confounding and non-random sampling. Across the three comparator meta-analyses, postmenopausal women consistently demonstrated higher odds of DED, with two comparisons reaching statistical significance; however, all analyses showed substantial to considerable heterogeneity ($I^2 = 82\text{--}88\%$), indicating variability in effect estimates. Funnel plots suggested mild asymmetry, although no significant small-study effects were detected in the only analysis with sufficient study count, and tests were not feasible for the others. While the overall direction of effect was relatively consistent, the cross-sectional nature of nearly all studies, combined with heterogeneity and potential residual confounding, limits causal inference. Consequently, the overall certainty of evidence remains low.

DISCUSSION

In this systematic review and meta-analysis, we synthesized evidence on how DED varies across menopausal stages. In the broadest comparison, where pre- and perimenopausal women were grouped together and contrasted against postmenopausal women, postmenopause was associated with roughly double the odds of DED (OR = 2.05) with 55.5% of all DED cases occurring in the postmenopausal group. When restricting the comparison to strictly premenopausal versus postmenopausal women, excluding perimenopausal participants, the association strengthened (OR = 2.42), again with a higher proportion of DED cases among postmenopausal women despite fewer total participants in this group. In contrast, the comparison between postmenopausal and perimenopausal women showed a smaller, non-significant increase in odds (OR = 1.32), albeit with the same direction of effect. Taken together, these findings are consistent with a pattern in which DED burden is higher in postmenopausal women than in earlier reproductive stages, with the strongest signal emerging when strictly premenopausal women are used as the reference group. The attenuation of the pooled OR when perimenopausal women are combined with premenopausal women suggests that perimenopause may represent an intermediate-risk state rather than a low-risk category. Non-pooled studies reinforce this gradient: several report menopause or postmenopause as an independent predictor of DED or show worsening signs with advancing age and hormonal transition, even when formal menopausal staging is not fully standardized.^{20, 21, 24–27, 30} Collectively, this supports a model in which DED risk increases along the reproductive aging spectrum, with perimenopause potentially occupying a transitional zone between pre- and postmenopausal risk.

Biologically, these patterns are plausible given what is known about sex hormones and the ocular surface. Estrogen and androgen receptors are expressed in the lacrimal and meibomian glands, and changes in sex-steroid milieu can alter aqueous production, lipid secretion, and inflammatory tone at the ocular surface.^{7, 8, 10, 34} Perimenopause is characterized by fluctuating and often erratic estrogen levels, along with gradual androgen decline, which may destabilize tear-film homeostasis and precipitate subclinical or intermittent DED.^{12–14} Postmenopause is defined by a more sustained hypoestrogenic and relative androgen-deficient state, which could drive more persistent meibomian gland dysfunction, increased tear evaporation, and chronic ocular surface inflammation.^{7, 9, 10, 13} Data from non-pooled studies, including the DREAM cohort analysis by Zhao et al., demonstrate worse objective DED signs in postmenopausal compared with premenopausal women, including higher corneal staining (3.74 vs 2.58), higher conjunctival staining (2.80 vs 2.22), increased tear osmolarity (304 vs 299 mOsm/L), and shorter TBUT (3.37 vs 3.93 seconds), supporting a pathophysiologic shift in ocular surface integrity as women transition into and through menopause.²¹ Although several studies reported objective measures of DED (e.g., Schirmer testing, tear break-up time, and tear osmolarity), these outcomes were not amenable to pooled analysis due to substantial heterogeneity in measurement methods, diagnostic thresholds, and reporting formats, as well as limited availability of stratified data by menopausal stage. As such, our analysis focused on prevalence-based outcomes, which were more consistently reported across studies. Following this, an important consideration is that most included studies did not systematically assess the nasolacrimal drainage system; this is relevant because primary acquired nasolacrimal duct obstruction, which is more prevalent in postmenopausal women, can produce epiphora and ocular discomfort leading to elevated symptom scores on instruments such as the OSDI, yet is generally considered a distinct clinical entity from dry eye disease.³⁵

At the same time, age itself, independent of hormonal status, is likely an important contributor to the observed associations. Older women are more likely to accumulate systemic comorbidities, use polypharmacy (including antidepressants, antihistamines, and antihypertensives known to exacerbate DED), and experience environmental and occupational exposures that affect the ocular surface.^{3, 5, 6} Many of the included studies did not fully adjust for these factors, and menopausal status is highly collinear with age, making it difficult to disentangle age-related from hormone-related effects. This limitation is supported by longitudinal data from the Beaver Dam Eye Study, which reported a 5-year incidence of dry eye of 13.3% and demonstrated a clear age gradient, increasing from 10.7% among individuals aged 48–59 years to 17.9% among those aged ≥ 80 years ($p < 0.001$).³⁶ In that cohort, the apparent sex difference in dry eye incidence was attenuated and no longer statistically significant after adjustment for age, whereas age itself remained independently associated with incident disease (OR 1.14 per 10-year increase, 95% CI 1.01–1.30). Notably, increased incidence was also observed among users of antihistamines and diuretics, further supporting the contribution of age-related medication burden. Taken together, we acknowledge transparently that a proportion of the menopausal-stage signal observed in our pooled analyses may reflect age-related effects. However, given the limitations of the current literature, these findings should be interpreted as associative rather than causal.

An additional limitation relates to residual confounding from factors that were inconsistently measured or adjusted for across studies. Beyond age, which is highly collinear with menopausal status, other important variables such as systemic comorbidities, dermatologic conditions (e.g., rosacea), medication use (including antidepressants, antihistamines, and topical ocular therapies), prior ocular surgery, and underlying ocular surface or eyelid disease were not uniformly reported or controlled. Many of these factors increase with age and may be more prevalent in postmenopausal populations, potentially contributing to the observed associations. Furthermore, the included studies varied in their underlying populations, with some including general community samples and others clinic-based cohorts and often did not clearly define exclusion criteria related to comorbidities or surgical history. As such, these unmeasured or inconsistently reported variables represent important sources of residual confounding that limit causal interpretation.

Heterogeneity in DED definitions (symptom questionnaires alone vs combined signs and symptoms, differing thresholds for Schirmer, TBUT, or staining) and in menopausal staging (age cut-offs vs self-reported menstrual history vs gynecologic evaluation) likely further contributed to the substantial I^2 values observed.

Overall, our results may provide considerations for both interpretation and practice. From a clinical standpoint, our findings may support heightened vigilance for DED in midlife and postmenopausal women, particularly those reporting ocular discomfort, fluctuating vision, or known risk factors such as meibomian gland dysfunction or systemic hormone-related symptoms.^{6, 7, 12, 13} However, given the low overall certainty of evidence, these results should be viewed at best as hypothesis generating. Future research will need well-characterized longitudinal cohorts with standardized menopausal definitions, detailed hormone therapy, systemic medication data, and harmonized DED diagnostic criteria to more clearly delineate the relative contributions of age, hormonal transition, and comorbidity. Mechanistic studies linking clinical DED phenotypes with hormonal profiles, meibomian gland structure and function, and tear-film biomarkers will also be essential to refine pathophysiologic models and guide targeted preventive or therapeutic strategies.

Low-certainty evidence suggests that postmenopausal women experience a higher burden of DED than women in earlier reproductive stages, with the strongest association observed when strictly premenopausal women were used as the reference group and with perimenopausal women showing an intermediate pattern. The direction of effect across all meta-analyses and non-pooled studies was generally consistent, although effect sizes varied and heterogeneity was considerable. Interpretation remains limited by the predominance of cross-sectional designs, residual confounding related to age, comorbidities, and medication use, and variability in both DED diagnostic criteria and menopausal staging. These factors reduce the certainty of the evidence and prevent specific clinical recommendations. Rather than favouring one interpretation, we present the pooled and narrative results transparently and encourage readers to consider the strengths and limitations of each. Overall, menopausal stage appears to be an important contributor to DED burden in midlife women, and future longitudinal and mechanistic studies are needed to clarify the roles of hormonal transition, aging, and

comorbidity in shaping DED risk and to determine whether stage-specific screening or treatment strategies may offer benefit.

Declarations

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CONFLICT OF INTEREST:

Author conflict of interest disclosures: M.K. has received honoraria from Laboratoires Théa, Alcon Inc., Bausch + Lomb Corporation, Kowa Company, Ltd., Allergan and Sun Pharmaceutical Industries Ltd., and has received research grant funding from Alcon Inc. All funding not relevant to this publication.

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AUTHOR CONTRIBUTION STATEMENT:

FN contributed to study conception and design, literature search, study screening, data extraction, data analysis, interpretation of results, and drafting of the manuscript. MA and AK contributed to study screening and data extraction. CL contributed to drafting and critical revision of the manuscript. BKT and SZ contributed to critical revision of the manuscript. MK contributed to study supervision, critical revision, and overall oversight of the project.

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Tables

Table 1 is available in the Supplementary Files section.

Figures

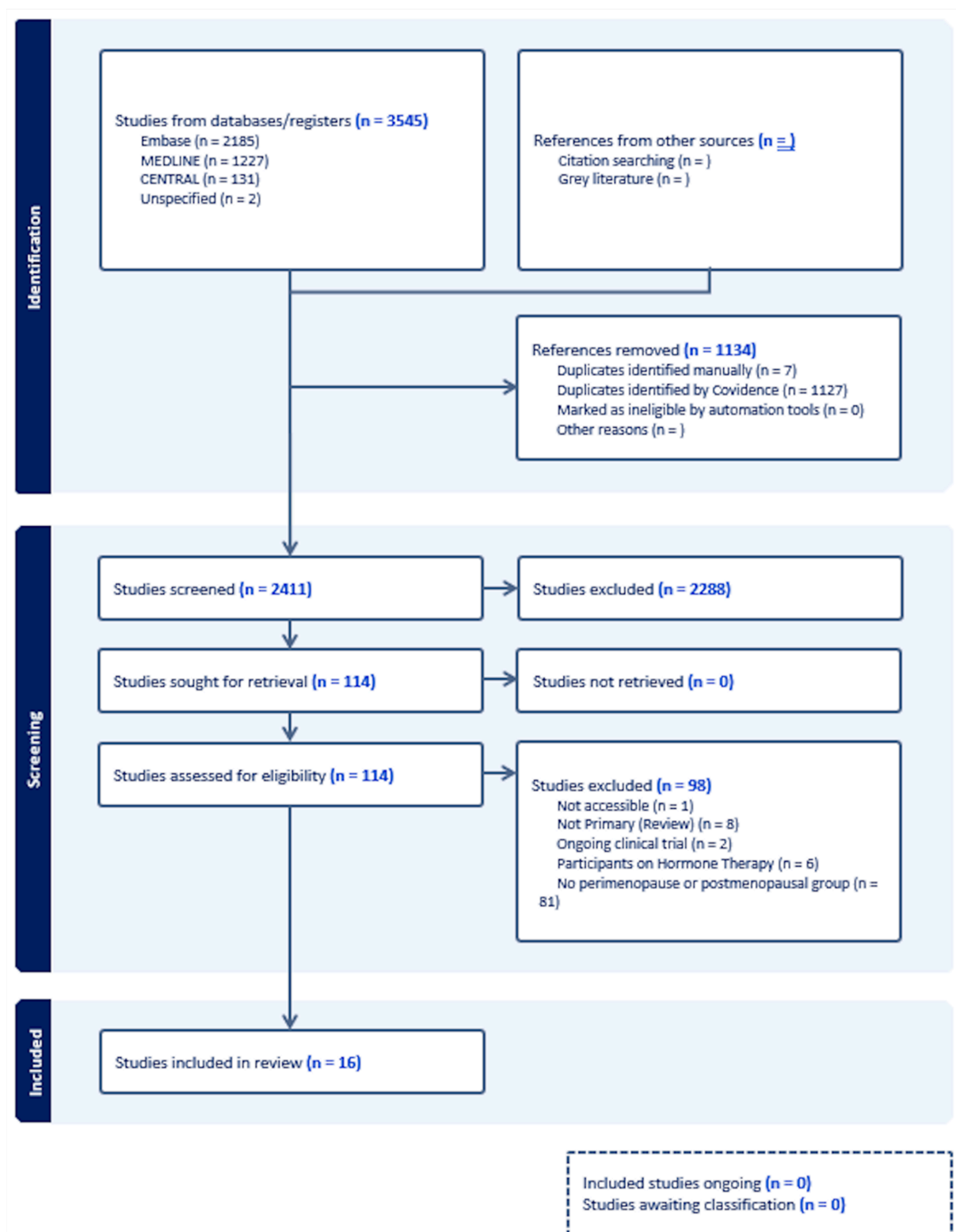


Figure 1

PRISMA flow diagram

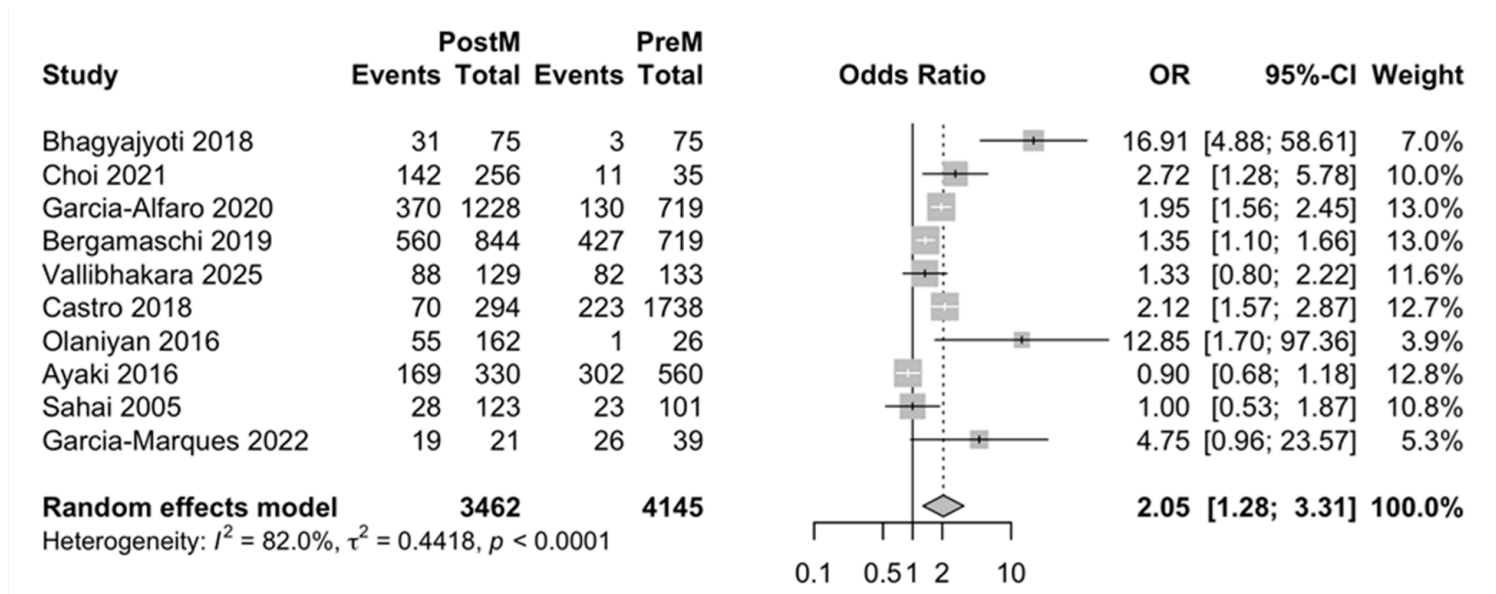


Figure 2

Forest plot comparing the odds of prevalent dry eye disease in postmenopausal women versus pre/perimenopausal women combined.

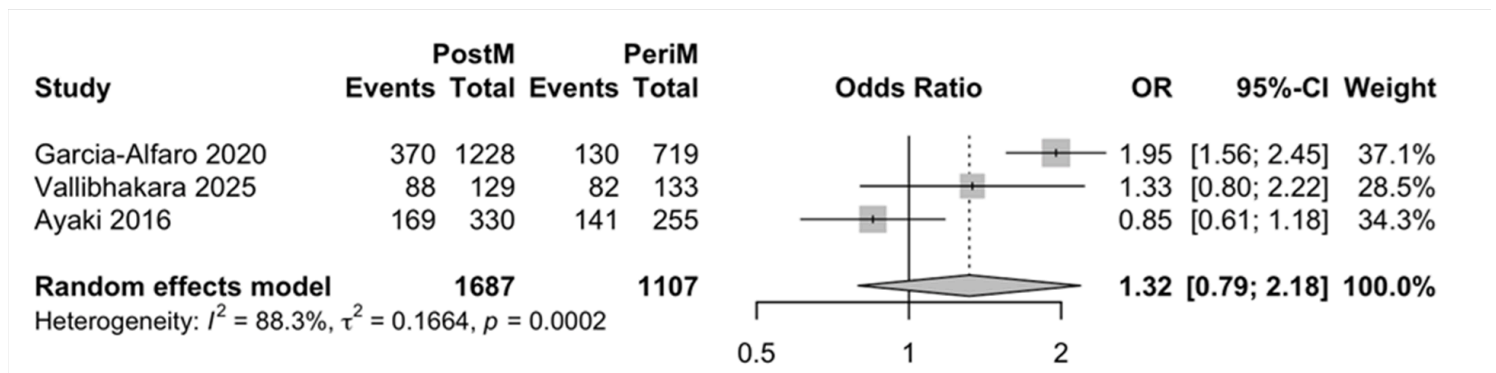


Figure 3

Forest plot comparing the odds of prevalent dry eye disease in postmenopausal women versus perimenopausal women.

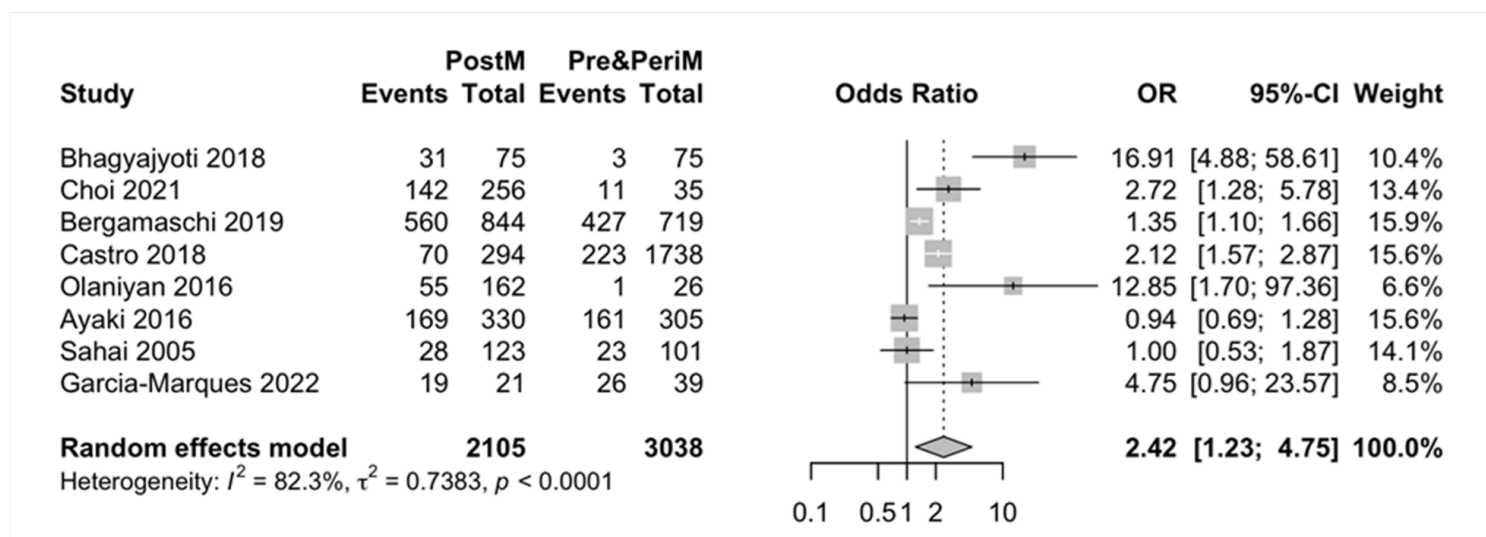


Figure 4

Forest plot comparing the odds of prevalent dry eye disease in postmenopausal women versus strictly premenopausal women.

Supplementary Files

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