

Baseline menopausal hormone therapy history and subsequent recorded urticaria among UK Biobank women: a population-based prospective cohort study

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Abstract

Background

Urticaria is common and women carry a substantial share of chronic disease burden, yet population evidence on menopausal hormone therapy and later urticaria is limited. We estimated the association between baseline hormone therapy history and subsequent recorded urticaria.

Methods

This prospective cohort study included 266,560 UK Biobank women with known baseline hormone therapy history and no pre-baseline urticaria record. The outcome was first post-baseline recorded International Classification of Diseases, Tenth Revision code L50 from linked health records; the code did not identify chronic spontaneous urticaria. The primary estimand came from a multivariable Cox proportional hazards model. Urticaria and alopecia areata were prespecified co-primary outcomes with Benjamini–Hochberg correction. Sensitivity analyses examined temporality, record source, competing death, menopause, reproductive history, cross-field exposure consistency, propensity-score weighting, and unmeasured confounding.

Results

During 3.46 million person-years, 2,404 recorded urticaria events occurred. Crude incidence was 0.764 versus 0.654 per 1,000 person-years among women with versus without hormone therapy history. The adjusted hazard ratio was 1.156 (95% confidence interval, 1.051–1.270; $P = 0.00270$; co-primary $q = 0.00541$). Estimates were similar after a 2-year lag (1.133; 1.015–1.264), overlap weighting with measured-covariate balance (1.150; 1.045–1.267), restriction to postmenopausal women (1.141; 1.023–1.273), and expanded reproductive adjustment (1.146; 1.043–1.260). Conventional stabilized inverse-probability weighting failed covariate balance and was not treated as causal support. The alopecia areata co-primary estimate was imprecise (1.170; 0.832–1.647).

Conclusions

Baseline menopausal hormone therapy history was associated with a modestly higher rate of subsequent recorded urticaria. The small absolute rate difference and susceptibility to residual confounding limit immediate clinical implications. The findings warrant replication and do not establish causation or a hormonal mechanism.

Background

Urticaria is a heterogeneous group of disorders characterized by wheals, angioedema, or both. Its burden is substantial, and women are disproportionately represented in chronic urticaria in many settings.[1–3] Biological and clinical observations have prompted interest in hormonal influences on mast-cell

activation and symptom fluctuation, but those observations do not establish that exogenous menopausal hormone therapy initiates urticaria.[4, 5]

Evidence specifically linking menopausal hormone therapy to subsequent urticaria records is sparse. Published reports concerning exogenous hormones have largely involved oral contraceptives, progesterone hypersensitivity, case series or treatment-related observations rather than large longitudinal cohorts.[6, 7] A population-based analysis could therefore provide an epidemiological estimate while recognising that a coded urticaria record is not equivalent to chronic spontaneous urticaria and that prescribing decisions may be associated with age, menopause, socioeconomic position, health service use and comorbidity.

We used linked UK Biobank data to test a prespecified co-primary hypothesis: whether baseline history of menopausal hormone therapy was associated with subsequent recorded urticaria (ICD-10 L50) or alopecia areata (L63) among women. This report focuses on L50 and retains L63 as the prespecified negative co-primary outcome. Acne (L70) and rosacea (L71) remained comparison outcomes and were not used to redefine the main question. We also prespecified analyses of temporal lag, record source, health-record linkage, competing death, menopausal status, hormone-therapy recency, reproductive history, cross-field exposure consistency, propensity-score balance and unmeasured confounding.

Methods

Study design and data source

This prospective cohort study used UK Biobank, a population-based resource of approximately 500,000 participants recruited across the United Kingdom during 2006–2010, with baseline questionnaires, physical measurements and linkage to routinely collected health records.[8, 9] We report the study according to STROBE and relevant RECORD items.[10, 11] The analytic dataset contained 502,226 person-level records. Women were eligible if their baseline menopausal hormone therapy history was known, baseline age and assessment date were available, and they had no recorded outcome before the analysis time origin. Follow-up ended at the first eligible outcome, death or 31 May 2022, whichever occurred first.

Exposure

The primary exposure was self-reported ever use of menopausal hormone therapy before or at baseline (UK Biobank Field 2814), contrasted with never use. A supporting baseline recency variable used Field 3546: current use was represented by the UK Biobank current-use code, former use by a valid age at last use, and unknown recency otherwise. Current versus never and former versus never contrasts were specified before the paper-level enhancement. Because Field 2814 captures history rather than treatment during follow-up, the analysis does not estimate effects of continued treatment, formulation,

dose, or route. Women classified as never users could subsequently initiate treatment (Supplementary Methods S1).

Outcomes

The primary outcome for this report was the first post-baseline recorded urticaria event in the ICD-10 L50 category. The confirmed definition used first-occurrence records whose source indicated primary care, hospital admission, death registration, or combinations of those sources; self-report-only first-occurrence records were excluded. Hospital records contained L50 subcodes, but the UK Biobank first-occurrence category did not encode acute versus chronic disease and hospital subcodes did not establish chronicity. We therefore use the term recorded urticaria throughout and do not label the endpoint chronic spontaneous urticaria. Alopecia areata (L63) was the prespecified co-primary outcome. Acne (L70) and rosacea (L71) were prespecified comparison outcomes (Supplementary Methods S1 and Table S5).

Covariates

The primary model adjusted for baseline age; menopause status; age at menopause; body mass index; smoking; alcohol use; ethnicity; Townsend deprivation index; household income; education; oral contraceptive history; log-transformed pre-baseline hospital-record burden; pre-baseline atopy; autoimmune disease; hormone-therapy contraindication proxies; pre-baseline menopausal-disorder records (ICD-10 N95); and assessment-center strata. Continuous variables with missing values were median-imputed and accompanied by missingness indicators; categorical variables included an unknown category where applicable. A complete-case analysis was performed. C-reactive protein was added in a separate expanded model (Supplementary Methods S1 and Table S1).

Statistical analysis

Cox proportional hazards models used time since baseline as the analysis scale, Efron handling of ties, and assessment-center strata. Hazard ratios (HRs), 95% confidence intervals (CIs), and two-sided P values are reported. The proportional hazards test for the primary exposure in the L50 model was nonsignificant ($P = 0.639$). L50 and L63 were co-primary outcomes; their primary-model P values were corrected by the Benjamini–Hochberg procedure, with $q < 0.05$ considered statistically significant.[12] L70 and L71 remained contextual comparisons. No a priori sample-size calculation was performed because all eligible records meeting the prespecified criteria were included.

Sensitivity analyses applied 2- and 5-year landmark lags; restricted to general-practice-linked participants; used hospital-only outcomes; additionally adjusted for C-reactive protein; used complete cases; and treated death as a competing event using Fine–Gray regression.[13] Paper-level enhancements restricted the cohort to postmenopausal women; contrasted current, former, and

unknown-recency users with never users; restricted to participants whose Field 3546 status was consistent with current hormone medication reported in Field 6153; and adjusted for live births, ages at first and last live birth, stillbirths, spontaneous abortions, and terminations, each with missingness indicators. The high missingness of some reproductive variables, especially pregnancy terminations, limits interpretation of this final model (Supplementary Methods S2, Table S1, Table S2, and Table S3).

A conventional stabilized inverse-probability weighting model was audited using absolute standardized mean differences (SMDs). It failed the prespecified balance criterion (maximum weighted absolute SMD 0.404) and was retained only as a transparent negative diagnostic. A corrective overlap-weighted analysis used a flexible propensity model with nonlinear age, age-by-menopause interactions, and all measured baseline covariates.[14] The overlap-population estimand achieved a maximum absolute SMD of 2.16×10^{-8} . We did not interpret either weighting analysis as establishing causality. An E-value quantified the minimum joint association that an unmeasured confounder would need with exposure and outcome, on the risk-ratio scale and conditional on measured covariates, to move the estimate to the null.[15] Analyses were conducted in R, and an independent script rederived participant-level variables and refitted the primary and lag models (Supplementary Methods S2 and Table S6).

Use of generative artificial intelligence in manuscript preparation

OpenAI Codex (OpenAI) was used during manuscript preparation to assist with literature-search query development, English-language editing, restructuring for journal requirements, consistency checks, and preparation and formatting of document and figure code from author-supplied aggregate results. It was not treated as an author or a source of evidence. All references were checked against primary bibliographic records, numerical statements and figure values were checked against locked analysis outputs, and the interpretation remained author-led. No participant-level UK Biobank data were entered into external web searches or included in the submission package. The authors reviewed and edited all assisted content and accept full responsibility for the manuscript.

Results

Cohort and baseline characteristics

Among 502,226 person-level records, 273,216 were women, 271,642 had known baseline hormone-therapy history, and 266,560 entered the L50 risk set (Fig. 1). Of these, 101,733 (38.2%) reported previous hormone therapy and 164,827 reported never use. Ever users were older (mean 60.6 versus 53.7 years), were more often postmenopausal (74.7% versus 52.1%) and had higher recorded prevalences of pre-baseline atopy and autoimmune disease (Table 1). These differences motivated extensive adjustment and the propensity-score balance audit.

Table 1
Baseline characteristics by menopausal hormone therapy history

Characteristic	Overall	Never use	Ever use
Participants, n	266560	164827	101733
Age, years, mean (SD)	56.4 (8.0)	53.7 (8.1)	60.6 (5.6)
Postmenopausal, n (%)	161860 (60.7)	85895 (52.1)	75965 (74.7)
Age at menopause, years, mean (SD)	49.7 (5.1)	50.2 (4.4)	49.0 (5.8)
Body mass index, kg/m ² , mean (SD)	27.1 (5.2)	26.9 (5.3)	27.3 (4.9)
Current smoker, n (%)	23783 (8.9)	14453 (8.8)	9330 (9.2)
White ethnicity, n (%)	251799 (94.5)	153149 (92.9)	98650 (97.0)
Townsend deprivation index, median (IQR)	-2.1 (-3.6 to 0.5)	-2.1 (-3.6 to 0.6)	-2.3 (-3.7 to 0.2)
Ever oral contraceptive use, n (%)	215578 (80.9)	133161 (80.8)	82417 (81.0)
Live births, mean (SD)	1.8 (1.2)	1.8 (1.2)	1.9 (1.2)
Age at first live birth, years, mean (SD)	25.3 (4.6)	26.0 (4.7)	24.4 (4.3)
Pre-baseline atopy, n (%)	9431 (3.5)	4785 (2.9)	4646 (4.6)
Pre-baseline autoimmune disease, n (%)	5712 (2.1)	2991 (1.8)	2721 (2.7)
Pre-baseline HRT contraindication, n (%)	8485 (3.2)	4675 (2.8)	3810 (3.7)
GP-linked participant, n (%)	119675 (44.9)	73968 (44.9)	45707 (44.9)
Data are mean (SD), median (IQR), or n (%). Values describe the outcome-specific risk set and are not standardized comparisons. GP, general practice; MHT, menopausal hormone therapy; IQR, interquartile range; SD, standard deviation.			

Recorded urticaria

Over 3.46 million person-years, 2,404 confirmed incident L50 records occurred: 1,002 among women with and 1,402 among women without hormone-therapy history. Median follow-up was 13.25 years in both groups. Crude incidence rates were 0.764 and 0.654 per 1,000 person-years, respectively (Table 2). In the primary adjusted model, hormone-therapy history was associated with a modestly higher rate of recorded urticaria (HR 1.156, 95% CI 1.051–1.270; P = 0.00270; co-primary BH q = 0.00541).

Table 2
Crude incidence of recorded urticaria

Exposure group	Participants	Events	Person-years	Median follow-up, years	Incidence per 1000 person-years
Never hormone therapy	164827	1402	2143790	13.25	0.654
Ever hormone therapy	101733	1002	1311380	13.25	0.764
Incidence rates are unadjusted. The primary effect estimate is the multivariable Cox-model hazard ratio in Table 3.					

Table 3
Main and enhanced analyses of hormone therapy history and recorded urticaria

Analysis	N	Events	Measure	Estimate (95% CI)	P value
Primary adjusted Cox model	266560	2404	HR	1.156 (1.051 to 1.270)	0.002703
Death as competing event	266560	2404	subdistribution HR	1.140 (1.041 to 1.248)	0.004756
Overlap weighting	266560	2404	HR	1.150 (1.045 to 1.267)	0.004402
Postmenopausal women	161860	1428	HR	1.141 (1.023 to 1.273)	0.01761
Expanded reproductive adjustment	266560	2404	HR	1.146 (1.043 to 1.260)	0.004714
Field 3546/6153 consistency	260187	2359	HR	1.156 (1.050 to 1.272)	0.003091
The primary Cox model adjusted for demographic, menopause, reproductive, lifestyle, socioeconomic, health care-use, and comorbidity variables and was stratified by assessment center. Co-primary Benjamini–Hochberg $q = 0.00541$ for the primary L50 estimate. Overlap weighting targets the propensity-score overlap population. CI, confidence interval; HR, hazard ratio.					

Sensitivity and enhanced analyses

Results remained directionally consistent with a 2-year lag (HR 1.133, 95% CI 1.015–1.264) and 5-year lag (1.171, 1.002–1.368), in the general-practice-linked subset (1.156, 1.046–1.277), after C-reactive protein adjustment (1.150, 1.046–1.264), among complete cases (1.260, 1.102–1.440), and with death as a competing event (subdistribution HR 1.140, 1.041–1.248). The hospital-only estimate was less precise and crossed the null (1.219, 0.973–1.527).

The association was similar among postmenopausal women (HR 1.141, 95% CI 1.023–1.273), after expanded reproductive adjustment (1.146, 1.043–1.260), and under strict Field 3546/6153 consistency

(1.156, 1.050–1.272). Relative to never use, the adjusted HR was 1.341 (1.135–1.583) for current use and 1.123 (1.014–1.244) for former use; these recency categories were baseline classifications and were not time-updated (Table S2).

Overlap weighting yielded HR 1.150 (95% CI 1.045–1.267) in the overlap population, with measured-covariate balance below the prespecified SMD threshold. By contrast, conventional stabilized weighting yielded a numerically similar HR but failed balance and was not used as support for a causal interpretation. The E-value was 1.58 for the point estimate and 1.28 for the confidence-limit nearest the null, indicating that modest residual confounding could move the lower confidence limit to the null.

Prespecified co-primary and comparison outcomes

The prespecified co-primary alopecia areata estimate was imprecise and compatible with the null (HR 1.170, 95% CI 0.832–1.647; $P = 0.367$; $q = 0.367$). Lagged L63 results were not used to reinterpret the co-primary hypothesis. Acne and rosacea remained comparison outcomes; their findings were not promoted into the main research question, particularly because a directly overlapping UK Biobank rosacea study had already been identified (Table S4).

Discussion

Principal findings

In this large UK Biobank cohort of women, baseline history of menopausal hormone therapy was associated with a 16% higher adjusted rate of subsequent recorded urticaria. The absolute difference in crude incidence was approximately 0.11 events per 1,000 person-years. Estimates were stable across analyses addressing delayed onset, competing death, record source, general-practice linkage, menopause restriction, reproductive history, exposure-field consistency and overlap weighting. The association was therefore statistically reproducible within the available data, but its magnitude was modest and its interpretation remains observational.

Comparison with existing evidence

The study adds longitudinal population evidence to a literature in which hormonal involvement has mainly been inferred from female predominance, symptom fluctuation, mechanistic discussion and uncommon hormone-associated hypersensitivity syndromes.[1–7] Those lines of evidence make the question plausible but do not identify a causal pathway for menopausal hormone therapy. We found no directly overlapping longitudinal cohort of baseline menopausal hormone-therapy history and subsequent recorded L50 during the final search update; this should be understood as a search-based assessment rather than proof that no such study exists.

Several non-causal explanations remain credible. Hormone-therapy users were older and more often postmenopausal, and they may differ in healthcare engagement, symptom reporting, prescribing history and comorbidity. Adjustment and overlap weighting addressed measured differences but cannot remove

residual confounding or detection bias. The E-value for the confidence limit was only 1.28, compatible with a comparatively modest unmeasured association. The stronger baseline current-versus-never estimate may reflect exposure recency, selection or measurement differences, but treatment was not updated and no formulation, dose, route or adherence data were available. It should not be interpreted as a dose-response relation or as evidence that stopping therapy would change risk.

Strengths and limitations

Strengths include the large prospective cohort, long follow-up, frozen co-primary analysis plan, multiplicity correction, transparent retention of the negative L63 co-primary outcome, multiple source and temporal analyses, explicit balance diagnostics, corrective overlap weighting and independent participant-level recalculation. The Field 3546/6153 restriction reduced concern about contradictory baseline treatment status, and expanded reproductive adjustment addressed additional measured confounding.

The study also has important limitations. First, L50 is a broad recorded urticaria category. The first-occurrence field did not distinguish acute from chronic disease, and hospital subcodes did not supply duration or the clinical work-up required to identify chronic spontaneous urticaria. Second, hormone therapy was self-reported at baseline and lacked formulation, dose, route, indication, adherence and time-updated use; exposure misclassification and post-baseline crossover are likely. Third, UK Biobank is not representative of the general population, so absolute rates may not transport directly. Fourth, outcome ascertainment depends on healthcare contact and coding. Fifth, some covariates had substantial missingness; the fertility-expanded analysis is particularly limited by 97.5% missingness for pregnancy terminations. Sixth, conventional stabilized weighting failed covariate balance, underscoring limited whole-cohort exchangeability. Finally, residual confounding, selection and detection bias remain possible despite consistent sensitivity estimates.

Implications

At the population level, even modest relative associations may merit investigation when both an exposure and an outcome are encountered in routine care. Here, however, the absolute crude rate difference was small and the data do not support a clinical prediction rule, treatment recommendation or public-health intervention. Replication should use cohorts with time-updated prescriptions, formulation and route, validated urticaria phenotyping, duration sufficient to identify chronic disease, and explicit assessment of health-care utilization. Causal or mechanistic studies would require designs and measurements not available here.

Conclusions

Baseline history of menopausal hormone therapy was associated with a modestly higher rate of subsequent recorded urticaria in UK Biobank women, with similar estimates in multiple sensitivity analyses. The findings do not demonstrate that hormone therapy causes urticaria, do not establish an estrogen mechanism, and do not identify chronic spontaneous urticaria.

Abbreviations

BH	
Benjamini–Hochberg	
CI	
confidence interval	
CRP	
C-reactive protein	
GP	
general practice	
HR	
hazard ratio	
ICD-10	
International Classification of Diseases,Tenth Revision	
IPW	
inverse-probability weighting	
MHT	
menopausal hormone therapy	
RECORD	
REporting of studies Conducted using Observational Routinely-collected health Data	
SMD	
standardized mean difference	
STROBE	
Strengthening the Reporting of Observational Studies in Epidemiology.	

Declarations

Ethics approval and consent to participate

UK Biobank has approval as a Research Tissue Bank from the North West–Haydock Research Ethics Committee (reference 21/NW/0157). All participants provided written informed consent. The study was conducted in accordance with the Declaration of Helsinki, applicable UK regulations, and the UK Biobank Ethics and Governance Framework. This secondary analysis was conducted under UK Biobank application 206581. The study involved no participant recontact or new data collection and was covered by UK Biobank's Research Tissue Bank approval; therefore, no separate local research ethics committee approval was required.

Consent for publication

Not applicable. No identifiable participant information is reported.

Availability of data and materials

Individual-level UK Biobank data cannot be shared directly by the authors. Eligible researchers may apply for access through UK Biobank at <https://www.ukbiobank.ac.uk/enable-your-research/apply-for-access>. Analysis code and non-disclosive aggregate outputs may be requested from the corresponding author; execution of the code requires authorized access to the relevant UK Biobank data.

Competing interests

The authors declare that they have no financial or non-financial competing interests relevant to this work.

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Authors' contributions

JZ, YL, YG and WKM contributed equally to the work overall. All authors contributed to the conception and design, methodology, validation, interpretation of results, drafting or critical revision, and final accountability. JZ and YL performed data curation, formal analysis, software implementation and visualization. YG and WKM provided supervision and resources. JZ and YL contributed equally and share first authorship. All authors read and approved the final manuscript.

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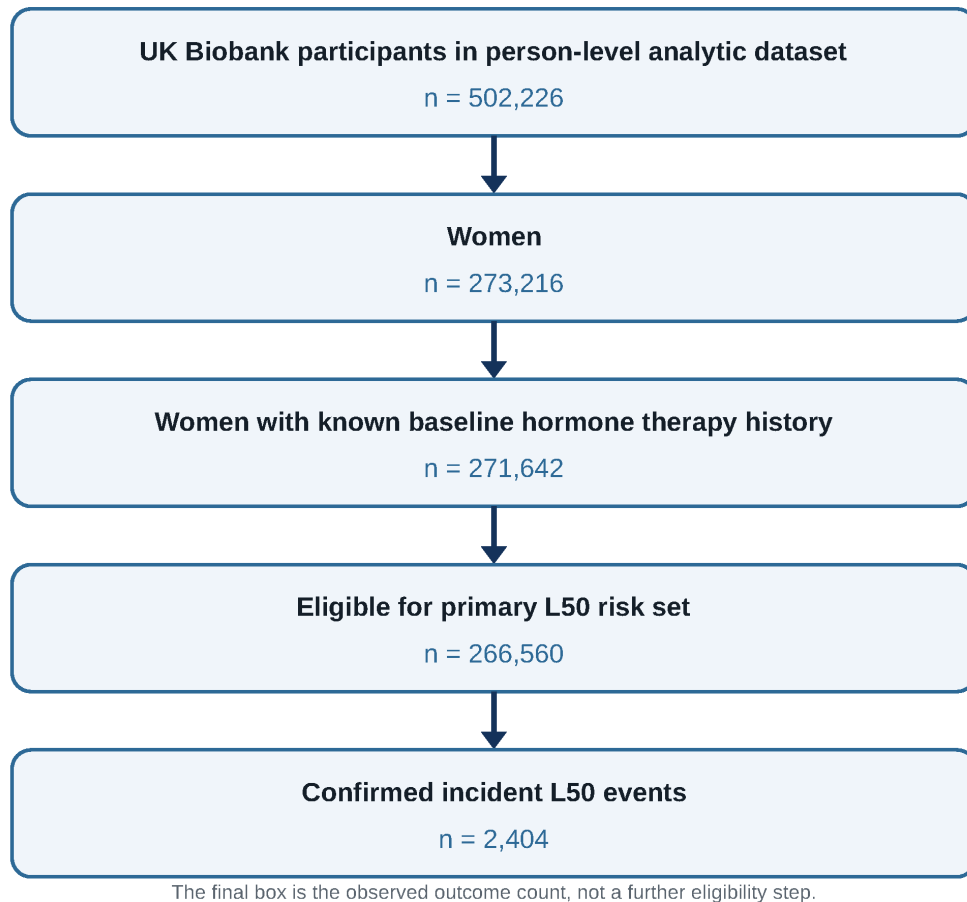
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Figures

Cohort construction



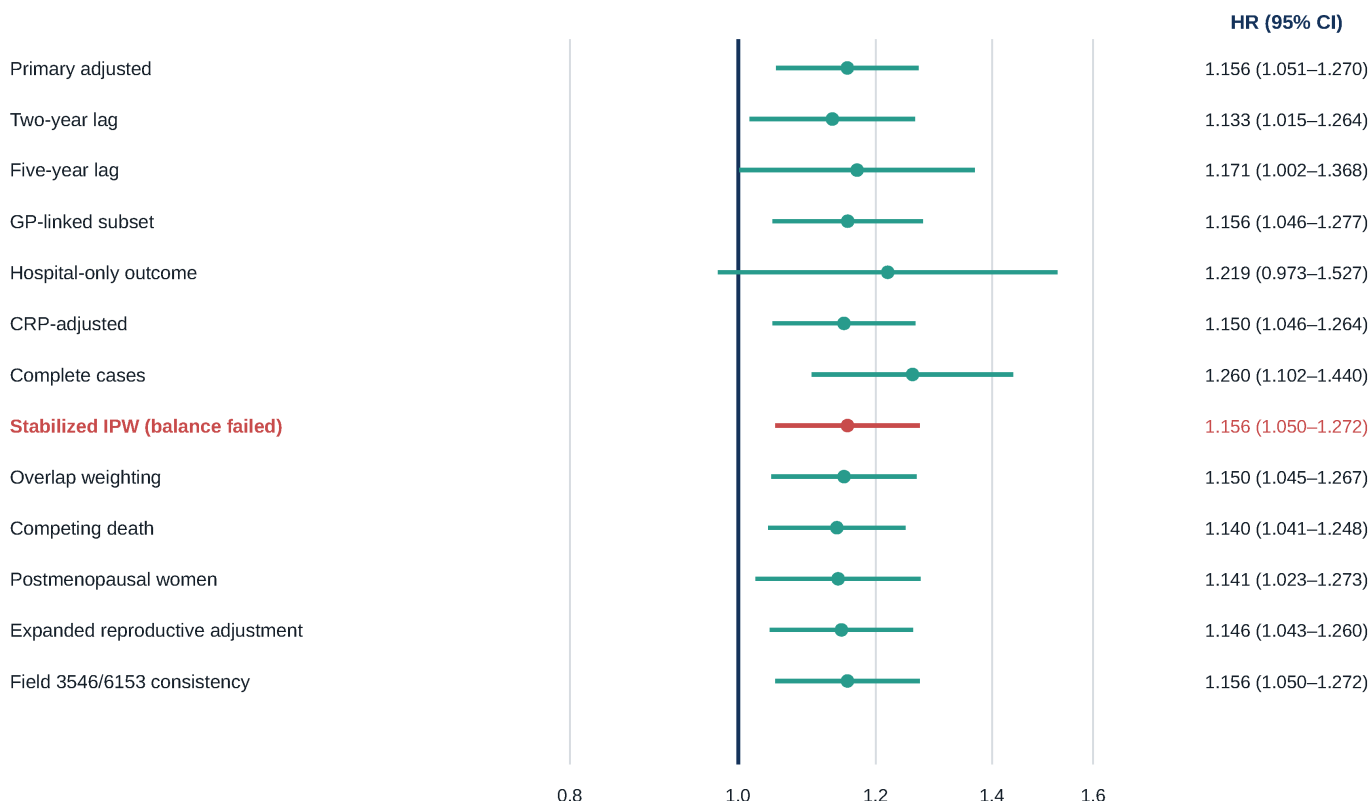
The final box is the observed outcome count, not a further eligibility step.

Figure 1

Cohort construction for the recorded urticaria analysis. MHT denotes menopausal hormone therapy. Exclusions between known exposure and the L50 risk set comprised a pre-baseline L50 record or an invalid outcome time origin. OpenAI Codex assisted with figure-code formatting; the authors checked all counts against validated aggregate outputs.

Recorded urticaria: main and sensitivity estimates

Hazard ratio (95% confidence interval)



Red row: conventional stabilized weighting failed measured-covariate balance and is not causal support.

Figure 2

Main and sensitivity estimates for recorded urticaria. Points are hazard ratios and lines are 95% confidence intervals; the vertical line is the null. The Fine–Gray estimate is a subdistribution hazard ratio, and overlap weighting targets the overlap population. Conventional stabilized inverse-probability weighting is flagged because balance failed (maximum weighted absolute standardized mean difference 0.404). OpenAI Codex assisted with figure-code formatting; the authors checked all plotted values against validated aggregate outputs.

Supplementary Files

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