

Induced Abortion and Incident Asthma in Chinese Women: A Prospective Cohort Study with Insights from Baseline Peak Expiratory Flow Measurements

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Abstract

Background.

Reproductive factors have been linked to respiratory health in Western populations, but prospective evidence from China is limited, and no study has separated the respiratory effects of induced abortion from those of spontaneous pregnancy loss.

Methods.

We analyzed 8,645 women aged 45 years or older from the China Health and Retirement Longitudinal Study (2011–2020). Reproductive exposures (parity, pregnancy loss by subtype, age at menarche and menopause) were obtained from the 2014 Life History Survey. Incident asthma and chronic obstructive pulmonary disease (COPD) were ascertained from physician-diagnosis reports across four follow-up waves; baseline peak expiratory flow (PEF) served as an objective measure of lung function. Cox models with age as the time scale and delayed entry were combined over five multiple-imputation datasets; dose–response was assessed with restricted cubic splines, and death was treated as a competing event in Fine–Gray models.

Results.

Among 6,755 ever-pregnant women, one induced abortion was associated with incident asthma (HR 1.49, 95% CI 1.04–2.14), with a similar estimate for two or more abortions (HR 1.66, 95% CI 0.94–2.96). Neither miscarriage (HR 1.05, 95% CI 0.67–1.64) nor stillbirth (HR 1.09, 95% CI 0.60–1.97) showed any association. The estimate was stable across sensitivity analyses (range 1.41–1.63), including a Fine–Gray competing-risk model (subdistribution HR 1.55, 95% CI 1.02–2.35), extended follow-up to 2020, and exclusion of events diagnosed before the exposure was reported; the E-value was 2.34. Each additional live birth was associated with 2.26 L/min lower PEF ($P = 0.015$), and each additional year before menopause with 0.84 L/min higher PEF ($P = 0.005$). Earlier menopause was paradoxically associated with lower COPD diagnosis rates, conflicting with the PEF gradient and indicating diagnostic-access bias in self-reported COPD.

Conclusions.

A history of induced abortion, but not miscarriage or stillbirth, was independently associated with incident asthma in middle-aged and older Chinese women. Parity and menopausal timing tracked objective lung function, supporting a reproductive–respiratory axis, whereas self-reported COPD diagnosis showed systematic access bias.

Introduction

Asthma and chronic obstructive pulmonary disease (COPD) together affect several hundred million people worldwide and rank among the leading causes of death and disability [1]. China carries a disproportionate share of this burden: the China Pulmonary Health study estimated 99.9 million adults with spirometry-defined COPD [2] and 45.7 million with asthma [3]. Research into respiratory risk factors has concentrated on smoking, occupational exposures and air pollution, while factors specific to women's lives have received far less attention. This imbalance is striking given that asthma is more common and more severe in women after puberty [4, 5], and given the well-documented actions of estrogens on airway smooth muscle, airway inflammation and oxidative defense in the lung [6–8].

The available evidence on reproductive factors and respiratory disease comes almost entirely from Western populations. In the COPDGene study, higher parity was linked to worse lung function and COPD-related phenotypes [9]. UK Biobank data showed elevated risks of COPD hospitalization or death among women with miscarriage, stillbirth, or early menopause [10]; a pooled analysis of three cohorts further found that shorter reproductive lifespan and earlier natural menopause predicted incident COPD [11]. Chinese data are sparser and largely cross-sectional. The CKB, the largest Chinese cohort with reproductive-history data, reported that pregnancy loss was associated with higher all-cause mortality, but did not examine respiratory outcomes separately and did not distinguish induced abortion from miscarriage [12]. Findings on menopause have been more mixed: the menopausal transition coincides with accelerated lung-function decline [13–15] and new-onset asthma in European cohorts [16], although meta-analyses remain inconclusive [17–19] and Asian evidence is notably scarce [20].

These observations point to three specific gaps in the literature. First, whether induced abortion and spontaneous pregnancy loss carry different respiratory risks is unknown, even though their physiological circumstances differ sharply. Second, no prospective cohort has tested pregnancy loss against incident asthma or COPD in Chinese women. Third, misclassification concerns persist because no study has paired self-reported respiratory diagnoses with objective lung-function measurement. We therefore used CHARLS [21] with four aims: to estimate prospective associations of parity, pregnancy-loss subtypes, and menopausal timing with incident asthma and COPD; to separate induced abortion from miscarriage and stillbirth; to benchmark self-reported diagnoses against baseline PEF; and to probe robustness through a prespecified set of sensitivity analyses.

Methods

Study design and participants

CHARLS is a nationally representative cohort of Chinese adults aged 45 years and older, described in detail elsewhere [21]. Data collection began in 2011–2012, with follow-up in 2013, 2015, 2018, and 2020. Reproductive histories were obtained from a dedicated Life History Survey conducted in 2014. Our

analyses were based on the harmonized CHARLS dataset (version D) [22] and followed STROBE reporting guidelines [23].

Women were eligible if they were aged 45 years or older at baseline and had no physician-diagnosed respiratory disease at baseline. We constructed two outcome-specific cohorts, with exclusions applied separately for each outcome: for asthma, we excluded women with baseline asthma or missing outcome data ($n = 396$) and those without any follow-up contact ($n = 489$), leaving 7,760 women; for COPD, we excluded those with baseline chronic lung disease or missing data ($n = 793$) and those lost to follow-up ($n = 450$), leaving 7,402 women (Fig. 1). The CHARLS protocol was approved by the Biomedical Ethics Review Committee of Peking University, and all participants provided written informed consent.

Reproductive exposures

Reproductive history came from the 2014 Life History Survey, which recorded each pregnancy with its outcome, cross-checked against the harmonized child-status file. The two parity sources correlated well (Spearman's $\rho = 0.71$). We assessed parity, pregnancy-loss subtypes, age at menarche, age at menopause, and reproductive lifespan. Parity was categorized as 0, 1, 2, 3, or ≥ 4 live births. Pregnancy loss was separated into induced abortion, miscarriage, and stillbirth, each grouped as 0, 1, or ≥ 2 events (stillbirth collapsed to ≥ 1 due to small numbers). Age at menarche was classified as ≤ 11 , 12, 13, 14, 15, or ≥ 16 years; age at natural menopause as < 40 , 40–44, 45–49, 50–53, or ≥ 54 years, cross-validated between the 2011 and 2018 interviews (84% agreement), with discordant values reconciled. Reproductive lifespan—menopause age minus menarche age—was categorized as ≤ 30 , 31–35, 36–38, or ≥ 39 years. Pregnancy-loss analyses were restricted to ever-pregnant women, with loss subtypes mutually adjusted and further adjusted for parity, following the approach used in large biobank studies [10]. We adopted the cut-points from the largest prior study of reproductive history and COPD [11].

Outcomes

Incident asthma and COPD were defined as the first follow-up wave at which a participant reported a physician diagnosis ('Have you been diagnosed with [asthma/chronic lung disease] by a doctor?'), provided they had been negative at all preceding waves. Wave-specific questionnaire structures were harmonized across the four follow-up waves, including the restructured 2020 module. Event times were assigned to the midpoint of the interval between the last negative and first positive wave; women without events were censored at their last negative wave. We used the interval midpoint, following the standard approach for interval-censored panel data [24]. Peak expiratory flow was measured at baseline with a peak-flow meter; three readings were taken and the maximum retained (valid range 50–900 L/min) [25]. Because PEF was measured once at baseline, it was analyzed cross-sectionally as an objective correlate of cumulative reproductive exposures.

Covariates

All models were adjusted for baseline covariates, including education (primary or below, secondary, or college or above), rural residence, marital status, body mass index, smoking status (never, former, or current), household use of solid cooking fuel (coal or biomass) [26], and alcohol use. We additionally adjusted for self-reported physician-diagnosed hypertension, diabetes, and heart disease.

Statistical analysis

The primary models were Cox proportional-hazards models with age as the time scale, so that age adjustment is non-parametric; women entered the risk set at their baseline age (delayed entry, i.e., left truncation at cohort entry) and exited at the age of event or censoring [27, 28]. Under this formulation, hazard comparisons are conditional on survival to the age at baseline, and no assumption is needed about risk before study entry. Fully adjusted models included all covariates listed above. Missing values (11.5% for reproductive exposures, 17% for menarche, 21% for BMI) were handled by multiple imputation with chained equations using gradient-boosted predictive mean matching ($m = 5$ datasets, 3 iterations); estimates were combined with Rubin's rules [29–31]. We also ran complete-case models for comparison. Menopause-age analyses were restricted to postmenopausal women and imputed within that subset, since values for premenopausal women are structurally absent. Non-linear dose–response was examined with restricted cubic splines (4 knots at the 5th/35th/65th/95th percentiles) [32], with non-linearity tested by likelihood-ratio comparison against the linear specification.

We tested robustness through several pre-specified sensitivity analyses: extended follow-up through 2020; exclusion of events ascertained at the 2013 wave (the only events diagnosed before reproductive history was reported in 2014, addressing both reverse causation and differential recall); restriction to never-smokers; a Fine–Gray subdistribution hazards model with death as the competing event, using inverse-probability-of-censoring weights [33]; a composite endpoint of incident disease or death, with a comparison of diagnosis rates between decedents and survivors; tests for effect modification by rural residence and smoking; and complete-case analysis versus imputed estimates. We computed E-values [34] to assess the potential impact of unmeasured confounding on the primary estimate. In the subset with baseline blood sampling, we explored whether systemic inflammation (high-sensitivity C-reactive protein, hs-CRP) could mediate the abortion–asthma association. Analyses used Python 3.12 (lifelines, statsmodels, miceforest). Two-sided $P < 0.05$ was considered statistically significant.

Results

Baseline characteristics

Among 6,755 ever-pregnant women in the asthma cohort, 814 (12.1%) reported one induced abortion and 292 (4.3%) reported two or more (Table 1). Compared with women without abortion, those with a history of abortion were younger (mean age 55.9 and 55.7 vs 58.9 years), better educated, more often urban and married, and less likely to cook with solid fuel (42% and 38% vs 59%). They also had higher baseline PEF (272 and 274 vs 252 L/min) and lower parity (2.1 and 1.7 vs 3.6 live births)—a pattern that

later helps distinguish the effects of abortion history from those of parity and baseline lung function. Smoking prevalence was low and similar across groups (about 7% ever-smokers in all groups). Over a mean follow-up of 6.3 years, 306 women developed asthma; in the COPD cohort, 813 developed COPD over 6.1 years.

Table 1

Baseline characteristics of ever-pregnant women in the asthma cohort, by number of induced abortions

Characteristic	0 abortions (n = 5,649)	1 abortion (n = 814)	≥ 2 abortions (n = 292)	P
Age, years	58.9 ± 9.6	55.9 ± 8.0	55.7 ± 8.0	< 0.001
BMI, kg/m ²	23.8 ± 4.0	24.4 ± 4.0	24.6 ± 3.7	< 0.001
Live births, n	3.6 ± 1.4	2.1 ± 1.2	1.7 ± 1.2	< 0.001
Age at menarche, years	16.3 ± 2.2	15.9 ± 2.1	15.6 ± 2.1	< 0.001
PEF, L/min	251.5 ± 91.2	272.2 ± 97.3	273.7 ± 93.5	< 0.001
Education				< 0.001
Primary or below	5,339 (94.5)	701 (86.1)	243 (83.2)	
Secondary	278 (4.9)	98 (12.0)	41 (14.0)	
College or above	32 (0.6)	15 (1.8)	8 (2.7)	
Rural residence	3,742 (66.2)	385 (47.3)	120 (41.1)	< 0.001
Married	4,412 (78.1)	683 (83.9)	249 (85.3)	< 0.001
Smoking status				0.413
Never	5,174 (91.6)	760 (93.4)	273 (93.5)	
Former	123 (2.2)	13 (1.6)	5 (1.7)	
Current	349 (6.2)	41 (5.0)	14 (4.8)	
Solid cooking fuel	3,314 (58.7)	345 (42.4)	111 (38.0)	< 0.001
Ever drinker	796 (14.1)	118 (14.5)	55 (18.8)	0.075
Hypertension	1,524 (27.0)	222 (27.3)	84 (28.8)	0.835
Diabetes	342 (6.1)	58 (7.1)	34 (11.6)	< 0.001
Heart disease	711 (12.6)	128 (15.7)	40 (13.7)	0.046
Values are mean ± SD or n (%). P from ANOVA or chi-square test. PEF = peak expiratory flow.				

Induced abortion and incident asthma

In our primary imputed models, fully adjusted for covariates, one induced abortion was associated with a 49% higher hazard of incident asthma (HR 1.49, 95% CI 1.04–2.14; $P = 0.028$); the estimate for two or more abortions was similar in magnitude but less precise (HR 1.66, 95% CI 0.94–2.96; $P = 0.083$) (Fig. 2, Table 2). Modelled as a continuous count, each additional abortion carried a 34% higher hazard (HR 1.34, 95% CI 1.10–1.63; $P = 0.004$). By contrast, neither miscarriage (one: HR 1.05, 95% CI 0.67–1.64; two or more: HR 0.86, 95% CI 0.34–2.19) nor stillbirth (HR 1.09, 95% CI 0.60–1.97) was associated with asthma—the association was specific to induced abortion, not to pregnancy loss in general. Spline analysis showed risk rising from the first abortion and peaking around one to two abortions (HR ≈ 1.6), with wide confidence intervals beyond two abortions where data were sparse; however, the non-linearity test was not significant ($P = 0.26$), so we interpret the continuous linear trend as the more reliable summary of the dose–response, while the apparent peak remains speculative (Fig. 3).

Table 2

Pregnancy-loss subtypes and incident asthma among ever-pregnant women (MICE-pooled, m = 5)

Exposure	Events/N	HR (95% CI)	P
Any pregnancy loss			
0	197/5,074	1.00 (ref)	
1	66/1,473	1.31 (0.97–1.76)	0.075
≥2	13/208	1.47 (0.85–2.55)	0.166
Induced abortion			
0	226/5,649	1.00 (ref)	
1	36/814	1.49 (1.04–2.14)	0.028
≥2	14/292	1.66 (0.94–2.96)	0.083
Miscarriage			
0	252/6,198	1.00 (ref)	
1	19/472	1.05 (0.67–1.64)	0.824
≥2	5/85	0.86 (0.34–2.19)	0.755
Stillbirth			
0	265/6,564	1.00 (ref)	
≥1	11/191	1.09 (0.60–1.97)	0.788
Age-as-timescale Cox models, fully adjusted (education, residence, marital status, BMI, smoking, solid fuel, alcohol, hypertension, diabetes, heart disease). Loss subtypes mutually adjusted and adjusted for parity. Events/N from complete-case data. All pregnancy-loss models are restricted to ever-pregnant women: the reference category (“0”) comprises only ever-pregnant women without that loss subtype, and never-pregnant women are excluded entirely.			

Other reproductive factors and incident asthma

Parity showed a borderline inverse pattern: women with one birth had a higher hazard than those with two (HR 1.64, 95% CI 0.98–2.75; P = 0.060), with no clear trend across higher parity categories. Age at menarche, age at menopause and reproductive lifespan showed no association with asthma (Table 2 and Supplementary Table S3).

Reproductive factors and incident COPD

No association was found for parity, pregnancy loss of any subtype, menarche age or reproductive lifespan (Table 3). Age at menopause, however, showed an unexpected inverse gradient: compared with menopause at 54 years or later, women with menopause before 40 had a lower hazard of COPD diagnosis (HR 0.65, 95% CI 0.42–0.99; P = 0.042), as did women in the 45–49 group (HR 0.76, 95% CI

0.58–0.99; $P = 0.043$). The inverse association persisted in extended follow-up and among never-smokers, and contrasts with the estrogen-protection hypothesis and with findings from Western cohorts [10, 11].

Table 3
Reproductive factors and incident COPD (MICE-pooled, m = 5)

Exposure	HR (95% CI)	P
Parity (ref: 2)		
0	0.95 (0.55–1.67)	0.870
1	1.23 (0.89–1.70)	0.211
3	1.07 (0.87–1.32)	0.542
≥4	1.08 (0.85–1.35)	0.536
Any pregnancy loss (ref: 0)		
1	1.12 (0.93–1.35)	0.216
≥2	1.19 (0.78–1.83)	0.422
Induced abortion (ref: 0)		
1	1.09 (0.86–1.37)	0.486
≥2	1.03 (0.70–1.52)	0.897
Miscarriage (ref: 0)		
1	1.15 (0.88–1.50)	0.299
≥2	1.02 (0.62–1.69)	0.933
Stillbirth ≥ 1 (ref: 0)	0.94 (0.65–1.37)	0.756
Age at menarche, years (ref: 13)		
≤11	0.63 (0.15–2.58)	0.517
12	1.23 (0.73–2.06)	0.431
14	1.42 (0.96–2.09)	0.079
15	1.13 (0.77–1.65)	0.529
≥16	1.11 (0.82–1.52)	0.492
Age at menopause, years (ref: ≥54)*		
<40	0.65 (0.42–0.99)	0.042
40–44	0.85 (0.61–1.19)	0.343
45–49	0.76 (0.58–0.99)	0.043
Model specifications as in Table 2. *Restricted to postmenopausal women; menopause-age and reproductive-lifespan models additionally adjusted for parity.		

Exposure	HR (95% CI)	P
50–53	0.74 (0.54–1.03)	0.072
Reproductive lifespan, years (ref: 36–38)*		
≤30	1.00 (0.78–1.27)	0.988
31–35	1.01 (0.79–1.28)	0.960
≥39	1.16 (0.84–1.60)	0.368
Model specifications as in Table 2. *Restricted to postmenopausal women; menopause-age and reproductive-lifespan models additionally adjusted for parity.		

Objective lung function

Both parity and menopausal timing were associated with PEF in directions consistent with prior literature (Table 4). Each additional live birth was associated with 2.26 L/min lower PEF (95% CI – 4.08 to – 0.43; $P = 0.015$); the LOWESS curve fell from about 270 L/min at two births to a trough near 181 L/min at five to six births (Fig. 4). A longer premenopausal period was also associated with higher PEF: each additional year of premenopausal reproductive life corresponded to 0.84 L/min higher PEF (95% CI 0.26–1.42; $P = 0.005$), and each year of reproductive lifespan to 0.71 L/min (95% CI 0.12–1.29; $P = 0.018$). Induced abortion count was not associated with PEF after adjustment for parity ($\beta = 3.88$, 95% CI – 0.58 to 8.33; $P = 0.088$). This finding argues against reverse causation, as it shows that women who had abortions did not have poorer baseline lung function.

Table 4
Reproductive factors and baseline peak expiratory flow (linear regression, fully adjusted)

Exposure	β , L/min (95% CI)	P	n
Live births, per birth	–2.26 (– 4.08 to – 0.43)	0.015	5,492
Age at menarche, per year	0.05 (– 1.03 to 1.13)	0.927	5,520
Age at menopause, per year*	0.84 (0.26 to 1.42)	0.005	4,062
Reproductive lifespan, per year*	0.71 (0.12 to 1.29)	0.018	3,283
Any pregnancy loss, per event**	5.39 (0.38 to 10.39)	0.035	5,425
Induced abortion, per event**	3.88 (– 0.58 to 8.33)	0.088	5,425
Miscarriage, per event**	–1.96 (– 8.39 to 4.47)	0.550	5,425
Stillbirth, per event**	1.87 (– 8.95 to 12.69)	0.735	5,423
Adjusted for age, education, residence, marital status, BMI, smoking, solid fuel, alcohol, hypertension, diabetes, heart disease. *Postmenopausal women only. **Ever-pregnant women, adjusted for parity and the other loss subtypes.			

Dissociation between lung function and COPD diagnosis

Figure 5 displays the central inconsistency of the COPD results. Mean PEF rose across menopause-age groups, from 222 L/min in women with menopause before 40 to 246 L/min in the 45–49 group. Incident COPD diagnosis rates followed the opposite pattern: the < 40 group, with the worst measured lung function, had the lowest diagnosis rate (9.4%), while the ≥ 54 group, with better PEF, had the highest (15.1%). The group with objectively worse lungs was the least likely to carry a COPD label—suggesting differential access to diagnosis rather than differential disease.

Sensitivity analyses

The induced-abortion estimate was stable across all prespecified checks (Fig. 6, Table 5). The estimate was 1.52 in complete-case analysis, 1.52 with follow-up extended through 2020, and 1.41 among never-smokers. A Fine–Gray model treating death as the competing event (346 competing deaths) gave a nearly identical subdistribution HR of 1.55 (1.02–2.35; $P = 0.042$) for one abortion and 1.44 (0.72–2.91; $P = 0.30$) for two or more, so competing mortality does not account for the association. Because reproductive history was reported in 2014, the 2013-wave events ($n = 22$) are the only outcomes that preceded exposure assessment and are thus most susceptible to differential recall. Excluding them strengthened the estimate (HR 1.63, 1.07–2.51; $P = 0.022$), whereas restricting to those early events alone was uninformative (HR 0.45, 0.06–3.40; 14 events). The composite endpoint including death attenuated the estimate to 1.16 (0.87–1.56), as expected when deaths unrelated to the exposure dominate the endpoint; women who died during follow-up had in fact been diagnosed with asthma at higher rates than survivors while alive (6.2% vs 3.7%), despite shorter observation (4.1 vs 6.3 years), consistent with death not masking diagnoses. No effect-measure modification was detected by rural residence (P for interaction 0.77) or smoking status ($P = 0.17$). In an exploratory smoking-stratified analysis, the association appeared stronger among ever-smokers with two or more abortions (HR 13.6, 95% CI 3.15–58.8), but this stratum held only 545 women and 43 events and should be read as hypothesis-generating (Supplementary Figure S1). Robustness to unmeasured confounding was moderate: the E-value for the primary estimate was 2.34, meaning an unmeasured confounder would need to be associated with both abortion history and incident asthma by risk ratios of at least 2.34-fold each to fully explain the result. The confidence-limit E-value of 1.24 indicates that weaker confounding could shift the interval across the null [34]. Finally, in the blood-sampling subset ($n = 4,759$ after excluding hs-CRP above 10 mg/L), abortion history was weakly and non-significantly associated with higher hs-CRP ($\beta = 0.07$ log mg/L; $P = 0.087$), hs-CRP did not predict incident asthma (HR per SD 0.99, 0.84–1.17; $P = 0.92$), and the abortion estimate was unchanged by hs-CRP adjustment (HR 1.77 in both models), which argues against mediation by systemic inflammation (Supplementary Table S4).

Table 5
Sensitivity analyses for pregnancy loss and incident asthma, HR (95% CI)

Analysis	Any pregnancy loss, 1 event	Any pregnancy loss, ≥ 2 events	Induced abortion, 1 event
Primary (MICE, m = 5)	1.31 (0.97–1.76)	1.47 (0.85–2.55)	1.49 (1.04–2.14)
Complete-case	1.48 (1.05–2.09)	1.93 (0.94–3.96)	1.52 (1.00–2.32)
Extended follow-up to 2020	1.45 (1.03–2.04)	1.85 (0.91–3.78)	1.52 (1.00–2.32)
Excluding events diagnosed by 2013*	1.55 (1.09–2.20)	2.08 (1.02–4.27)	1.63 (1.07–2.51)
Never-smokers (MICE)	1.21 (0.87–1.67)	1.24 (0.63–2.42)	1.41 (0.96–2.07)
Composite endpoint (disease or death)	1.01 (0.80–1.26)	0.78 (0.44–1.37)	1.16 (0.87–1.56)
Fine–Gray competing-risk model†	—	—	1.55 (1.02–2.35)
Exposure modelled continuously, per event	—	—	1.34 (1.10–1.63)
Events ascertained at the 2013 wave only	—	—	0.45 (0.06–3.40)
All models fully adjusted. Extended follow-up, 2013-exclusion, composite, Fine–Gray and 2013-only analyses are complete-case; the continuous exposure estimate is MICE-pooled. *The 2013 wave preceded the 2014 reproductive-history report, so this analysis also removes the events most susceptible to differential recall. †Death as the competing event (346 competing deaths); subdistribution HR. The 2013-only analysis includes 14 events in complete-case data and is underpowered by design.			

Discussion

Principal findings

In this nationally representative cohort of middle-aged and older Chinese women, a history of induced abortion was associated with roughly a 50% higher hazard of incident asthma over six years of follow-up, while miscarriage and stillbirth carried no risk. Parity and menopausal timing were unrelated to disease incidence but tracked objective lung function: higher parity was associated with lower PEF, whereas later menopause was associated with higher PEF. Finally, the inverse association between menopause age and COPD diagnosis contradicted the PEF gradient, exposing a systematic diagnostic-access bias in self-reported COPD.

Comparison with previous work

Our COPD-null results for pregnancy loss contrast with UK Biobank, where miscarriage and stillbirth predicted COPD hospitalization and death [10], and with the pooled analysis of Liang and colleagues

[11]. Several differences may explain the discrepancy. First, our endpoint was self-reported diagnosis rather than hospital records. Second, smoking prevalence among Chinese women is very low, removing the main effect amplifier seen in Western cohorts. Third, our diagnostic-access analysis suggests that self-reported COPD in China measures healthcare contact as much as disease. In contrast, the PEF findings were consistent with previous reports. Multiparity was linked to worse lung function in COPDGene [9]; menopause coincided with accelerated decline in FEV1 and FVC in European data [14]; and in UK Biobank, lower lung function accompanied earlier cessation of menstruation [15]. Our estimate of -2.26 L/min per birth extends this consistency to a low-middle-income setting with high parity and universal pregnancy experience. The asthma finding itself has no direct precedent. The closest evidence comes from cross-sectional studies: pregnancy loss was associated with all-cause mortality in the China Kadoorie Biobank [12], and reproductive indicators including pregnancy loss predicted COPD outcomes in UK Biobank [10]. However, neither study distinguished induced abortion from miscarriage, and neither examined asthma prospectively—gaps that our study directly addresses.

Why induced abortion specifically?

The specificity of the finding is what makes it informative: miscarriage and stillbirth share the 'loss' but not the circumstances. Several mechanisms—not mutually exclusive—could explain why induced abortion, specifically, might influence later asthma risk. One involves abrupt endocrine withdrawal: surgical termination removes the conceptus and its hormonal support in a single event, producing a sharper drop in estrogen and progesterone than most spontaneous losses. Sex steroids modulate airway smooth muscle and immune tone [6, 7], and estrogen shifts can tilt T-helper balance toward the Th2-dominant responses that favor asthma [35, 36]. Another is uterine immune injury: the pregnant uterus is immunologically active [37, 38], and surgical evacuation adds mechanical decidual injury and a burst of inflammatory signaling that spontaneous loss often does not. A third possibility is psychological stress. In the context of China's family-planning era, abortion was frequently involuntary or socially fraught, and although the mental-health literature remains debated [40], chronic stress is known to amplify airway inflammation through neuroendocrine–immune pathways [41, 42]. These proposed pathways remain unconfirmed. The negative hs-CRP result, however, suggests that systemic inflammation is unlikely to be the main mediator—though local airway inflammation cannot be excluded.

The COPD paradox and diagnostic access

The inverse menopause–COPD gradient deserves emphasis because it is stable and significant, but almost certainly artifactual. Women with early menopause had the lowest PEF yet the fewest COPD diagnoses; women with late menopause had better PEF yet the most diagnoses. Late menopause in this generation of Chinese women is a marker of better nutrition and urban upbringing—the same characteristics that predict whether a woman with chronic cough ever reaches a hospital and receives a diagnosis. Underdiagnosis of COPD in China is well documented: in national spirometry surveys, most cases had not been previously diagnosed [2, 43], and underdiagnosis concentrates in rural and poorer populations internationally [44]. Methodologically, this finding has a clear implication: studies using self-

reported COPD in CHARLS and similar surveys should treat the endpoint as 'diagnosed and reported COPD' and, where possible, anchor their inferences to measured lung function.

Strengths and limitations

This study has several strengths. The cohort is nationally representative with up to nine years of follow-up. Reproductive histories were collected through a dedicated life-history module with cross-source validation. We also separated pregnancy loss by subtype, included an objective lung-function correlate, and used a statistical framework aligned with current best practice [11].

Our findings should be interpreted in light of several limitations. Outcomes relied on self-reported physician diagnosis; PEF mitigates but does not eliminate the resulting misclassification, and PEF itself is effort-dependent and less specific than spirometry. Event times are interval-censored and were approximated by midpoints. Induced abortion is likely under-reported; if under-reporting is non-differential with respect to later asthma, our estimates are biased toward the null, so the true association may be stronger. Differential recall—women diagnosed with asthma reporting abortions more completely—is constrained by the design: reproductive history was collected in 2014, before most outcomes occurred, and removing the minority of events diagnosed before that report strengthened rather than weakened the association. Residual confounding remains possible: induced abortion was more common among urban, better-educated women, and we lacked data on childhood asthma, passive smoking, occupational exposures and ambient pollution, although solid-fuel use and urbanicity partially capture these [26, 45]. The E-value analysis indicates that only a confounder of moderate strength (risk ratio ≥ 2.34 with both exposure and outcome) could fully explain the estimate; however, the confidence-limit E-value of 1.24 means that modest unmeasured confounding could move the interval across the null. The findings therefore warrant confirmation. The ever-smoker interaction rests on small numbers. PEF was available for 63% of women, and those without measurements were older and less educated (Supplementary Table S2); PEF analyses adjusted for age and education, but selection into PEF measurement may still bias the cross-sectional estimates toward healthier women. The hs-CRP analysis, although reassuring, relied on a single baseline measurement and cannot exclude localized airway inflammation. Finally, the observational design precludes causal claims.

Implications

If confirmed, abortion history could contribute to asthma risk stratification in middle-aged women — a group in whom adult-onset asthma is rising and frequently misattributed to COPD [4, 46, 47]. The result also adds abortion to the list of reproductive events with possible long-term respiratory sequelae, an area not currently addressed by clinical guidelines [48, 49]. For respiratory epidemiology in China, our diagnostic-bias analysis is a caution: the rural–urban gradient in COPD labeling is steep enough to invert exposure–disease relationships. Expanding spirometry and peak-flow screening in primary care would address both the research artifact and the underlying clinical gap [43, 50].

Conclusions

Induced abortion, but not miscarriage or stillbirth, was independently associated with incident asthma in Chinese women aged 45 and older. Parity and menopausal timing were reflected in objective lung function, supporting a reproductive–respiratory axis. Self-reported COPD diagnoses appeared to be systematically biased by healthcare access and should be interpreted with caution in this population.

Declarations

Ethics approval and consent to participate

The CHARLS project was approved by the Ethical Review Committee of Peking University (IRB identification number: IRB00001052-11015). All original CHARLS participants provided written informed consent before data collection. The present secondary data analysis was conducted in full accordance with the principles outlined in the Declaration of Helsinki. This study uses publicly released fully de-identified CHARLS datasets without access to personal identifiable information, so additional institutional ethical review and separate participant informed consent were waived. All analytical procedures strictly followed ethical standards for human observational research.

Consent for publication

Not applicable. This manuscript only reports aggregated population-level statistical results and does not contain any identifiable individual information, clinical images, or personal case records.

Competing interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Author Contribution

Jiaping Lu performed data cleaning, descriptive statistics, primary regression analysis, and drafted the original manuscript. Qiulian Xu assisted in participant screening according to inclusion and exclusion criteria, sorted baseline characteristic data, and revised the results section. Heng Xu proposed the research hypothesis, designed the overall analytical framework, supervised all statistical procedures,

repeatedly revised the full manuscript, and served as the corresponding author. All authors have read and approved the final version to be submitted.

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Data Availability

The original CHARLS datasets used in this study are publicly accessible after official registration and application via the CHARLS official website (<http://charls.pku.edu.cn/>). Researchers can apply for data access following the platform's standard application procedures. The analytical code generated during the current study is available from the corresponding author upon reasonable request.

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Figures

Figure 1. Participant flow diagram

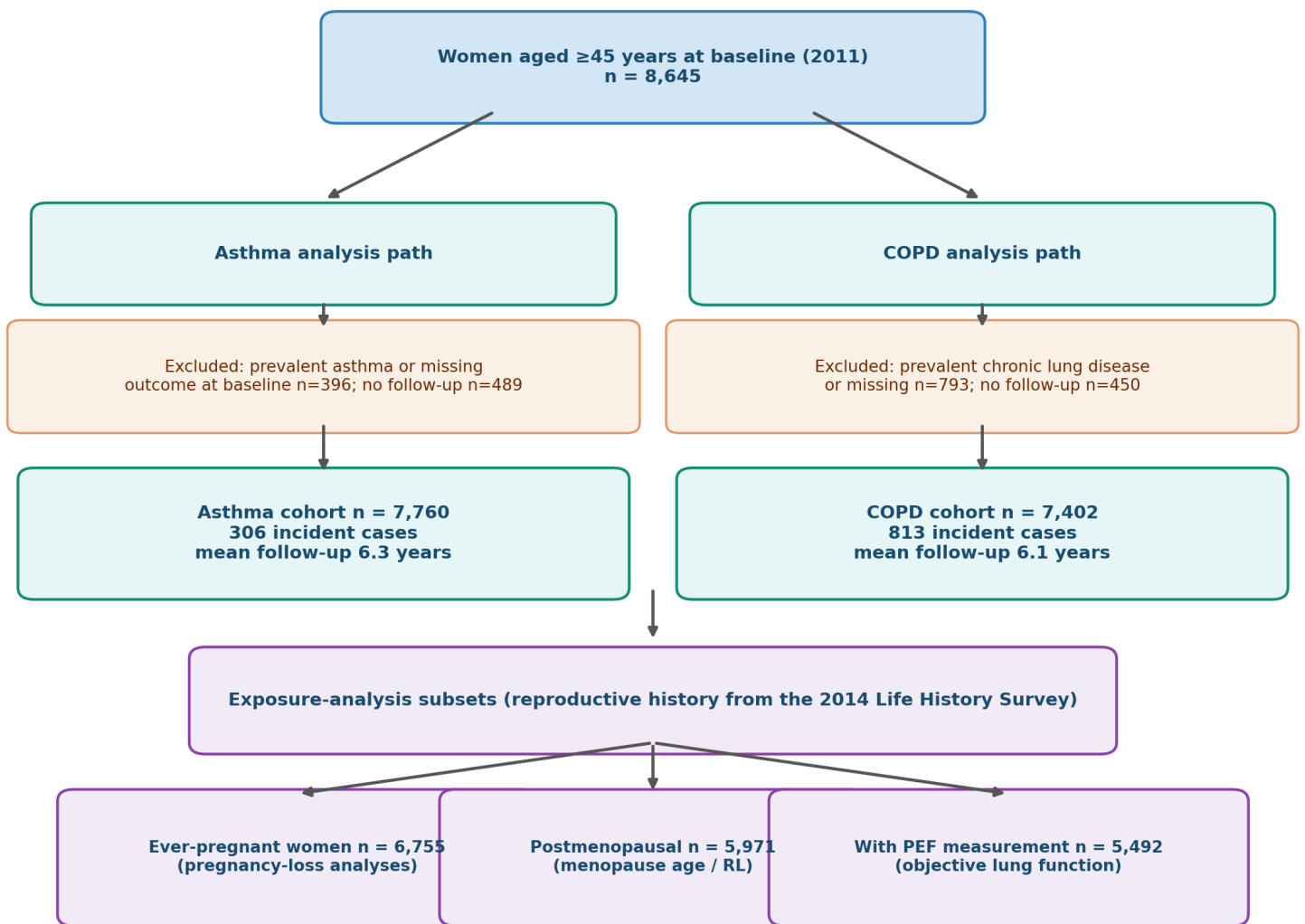


Figure 1

Participant flow diagram. Two outcome-specific cohorts were derived from 8,645 women aged ≥45 years at the 2011 baseline; exposure-analysis subsets were defined by availability of the 2014 Life History reproductive module.

**Figure 2. Pregnancy-loss subtypes and incident asthma
(ever-pregnant women; MICE-pooled, m=5; age-as-timescale Cox; mutually adjusted + parity)**

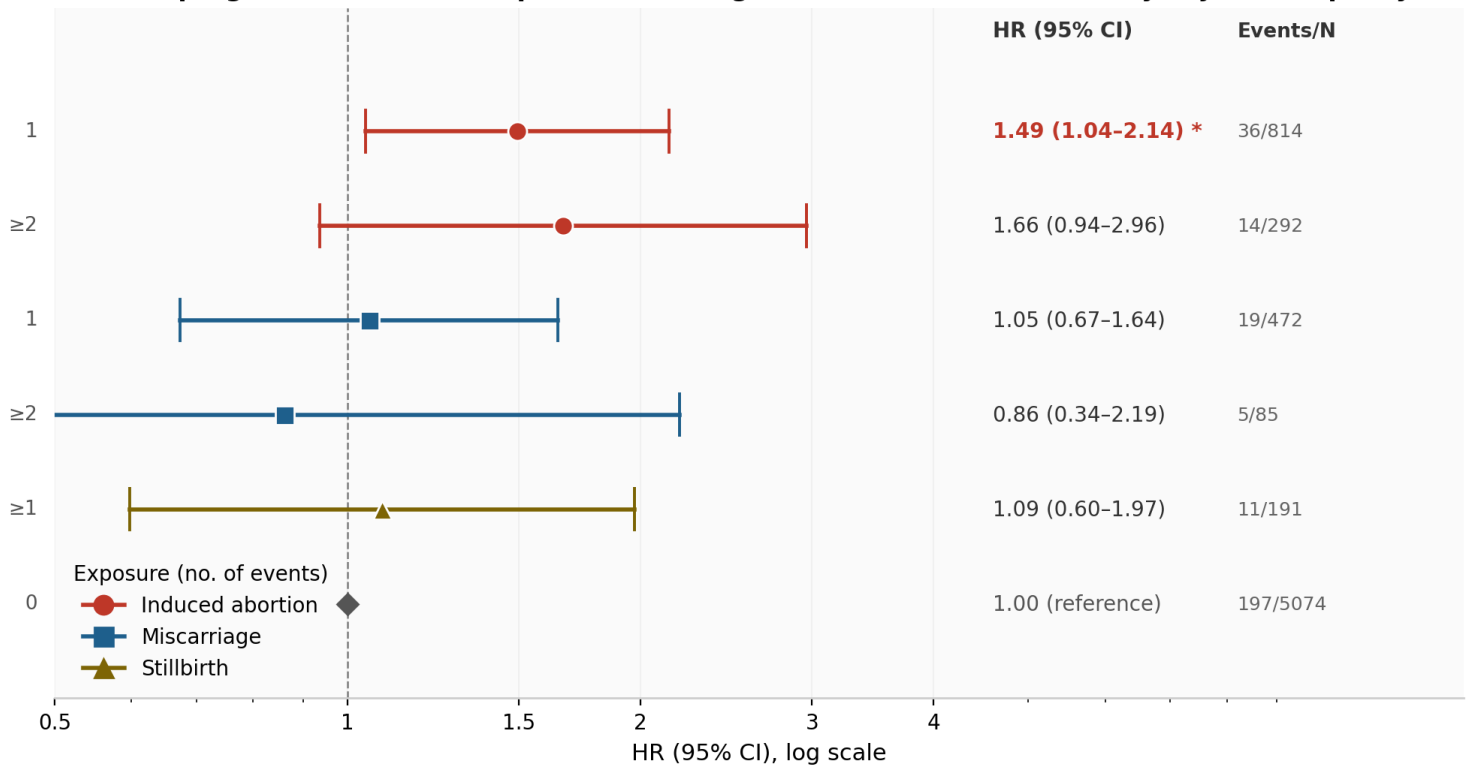


Figure 2

Pregnancy-loss subtypes and incident asthma among ever-pregnant women. Hazard ratios from age-as-timescale Cox models pooled over five imputed datasets, with subtypes mutually adjusted and adjusted for parity. Only induced abortion was associated with incident asthma. *P<0.05.

Figure 3. Dose-response between induced abortion and incident asthma (restricted cubic spline, df=3; reference = 0; adjusted for parity, miscarriage, stillbirth + full covariates)

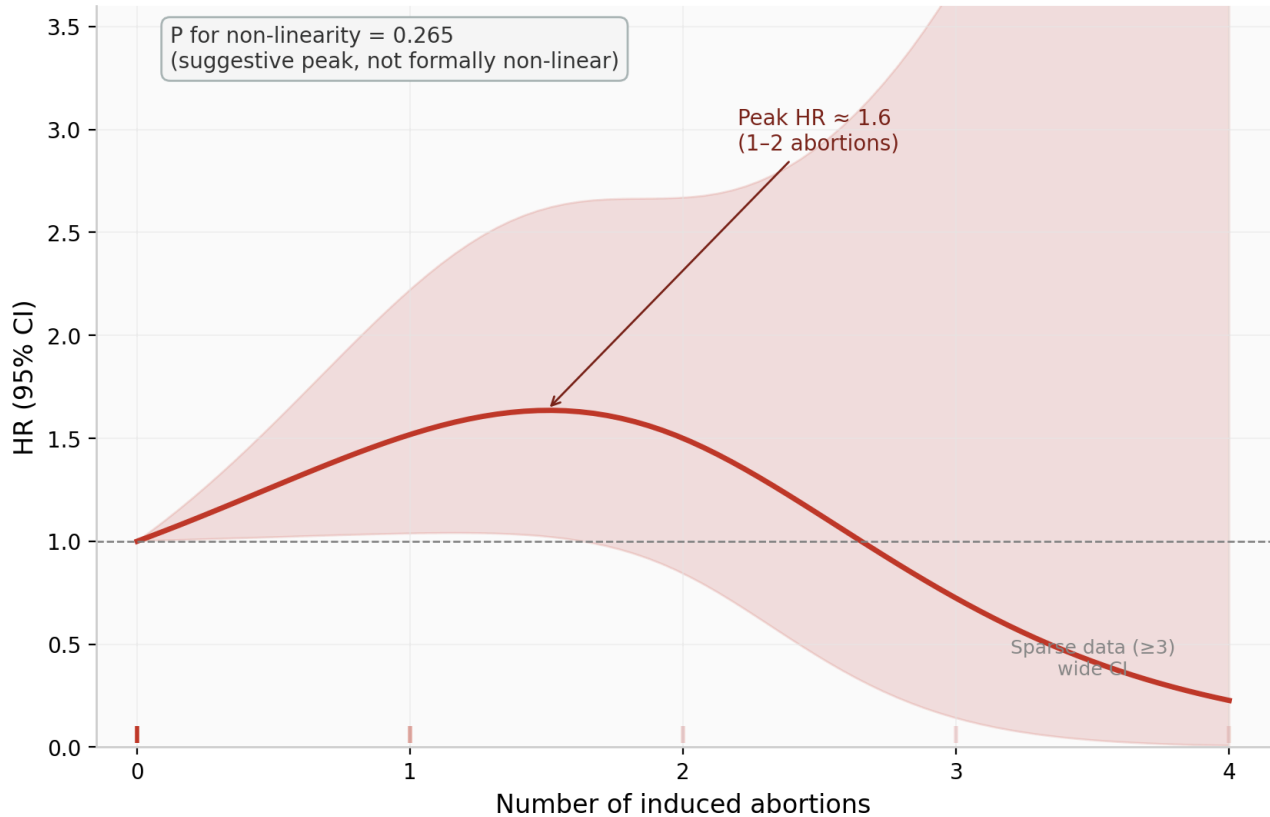


Figure 3

Dose-response between number of induced abortions and incident asthma (restricted cubic spline, df=3; reference = 0 abortions; adjusted for parity, miscarriage, stillbirth and full covariates; likelihood-ratio P for non-linearity = 0.26). Tick marks on the x-axis show the data distribution; estimates beyond two abortions rest on sparse data.

Figure 4. Parity and baseline peak expiratory flow
(individual values, n=5,492; red curve = LOWESS; yellow points = parity-specific means)

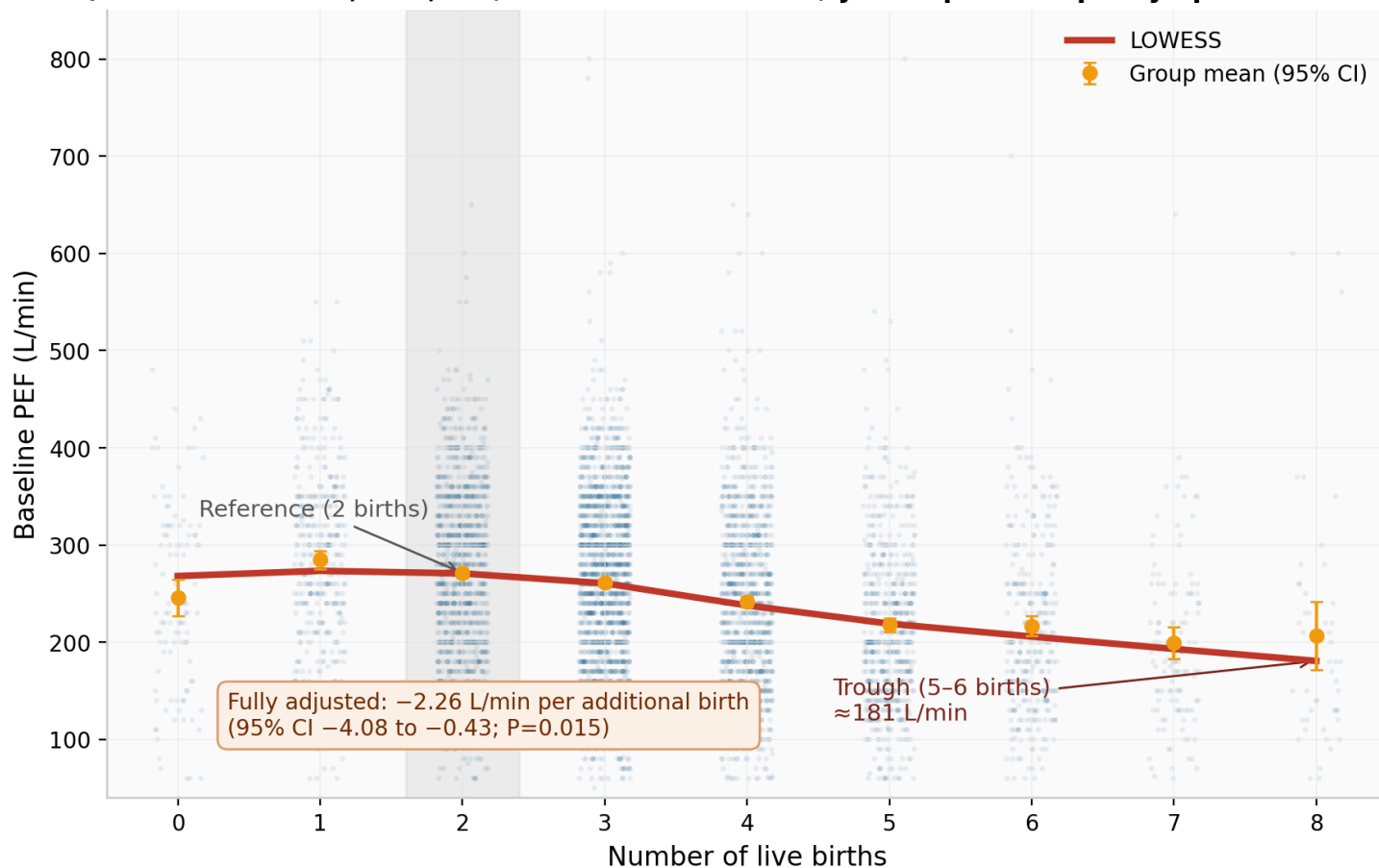


Figure 4

Parity and baseline peak expiratory flow (n=5,492). Grey points are individual measurements (jittered); the red curve is a LOWESS smooth; yellow points are parity-specific means with 95% CIs. The fully adjusted linear estimate was -2.26 L/min per additional birth (P=0.015).

Figure 5. Dissociation between objective lung function and COPD diagnosis by age at menopause (bars = mean PEF $\pm$ 95% CI; line = incident COPD diagnosis rate $\pm$ 95% CI; postmenopausal women)

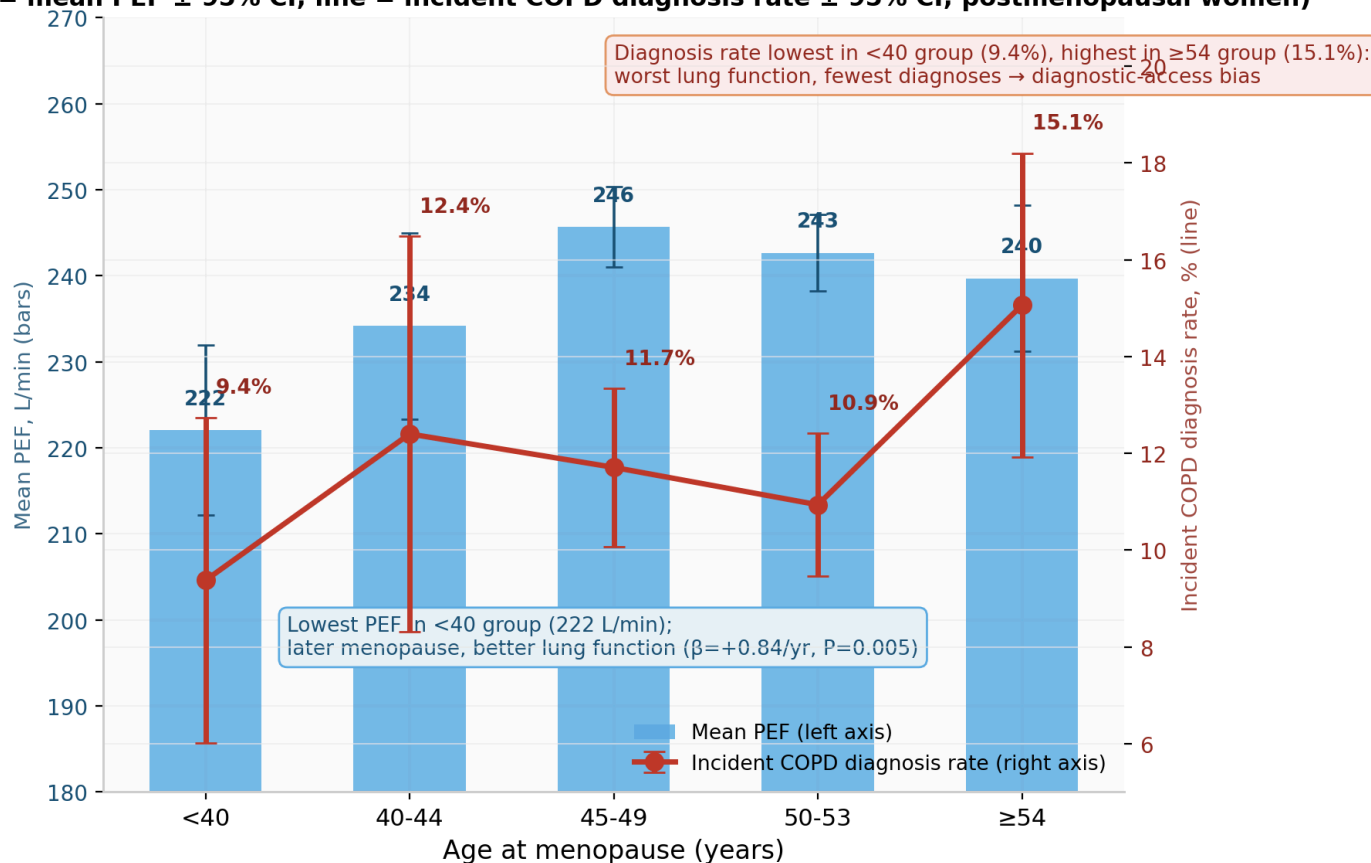


Figure 5

Dissociation between objective lung function and COPD diagnosis across menopause-age groups in postmenopausal women. Bars show mean baseline PEF (left axis); the red line shows the incident COPD diagnosis rate during follow-up (right axis). Women with the worst measured lung function (menopause <40 years) had the fewest diagnoses, indicating diagnostic-access bias.

Figure 6. Sensitivity analyses: one induced abortion and incident asthma (age-as-timescale Cox, fully adjusted, except Fine-Gray subdistribution model; * P<0.05)

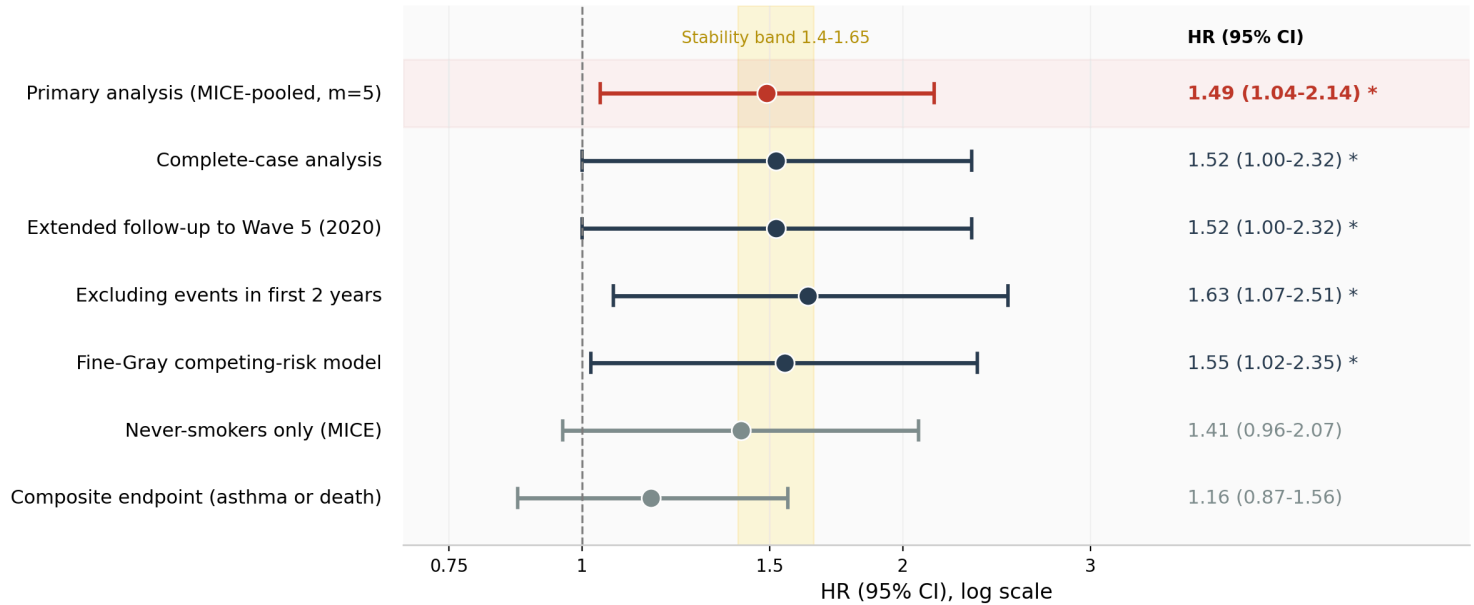


Figure 6

Sensitivity analyses for the association between one induced abortion and incident asthma, including a Fine-Gray subdistribution model with death as the competing event. The primary imputed estimate (1.49) is highlighted; five of seven alternative specifications produced estimates between 1.41 and 1.63. The composite endpoint is diluted by deaths unrelated to the exposure.

Supplementary Files

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