

Adverse Pregnancy Outcomes and Future Maternal COPD Risk

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Abstract

Background

Women are more susceptible to developing chronic obstructive pulmonary disease (COPD) than men, yet sex-specific risk factors are poorly understood. Adverse pregnancy outcomes (APOs) are established predictors of maternal cardiovascular disease. Despite shared biological mechanisms between cardiovascular disease and COPD, the relationship between APOs and COPD is unknown.

Methods

This secondary analysis of the Women's Health Initiative Observational Study (WHI-OS) included women aged 50–79 years enrolled from 1993–1998 and followed through 2024. Eligible participants had ≥ 1 pregnancy and no COPD at enrollment. In 2017, participants reported prior APOs, defined as ever having a hypertensive disorder of pregnancy (HDP), gestational diabetes, large or small for gestational age infant, or preterm delivery. Incident COPD was self-reported annually for the first 8 years and from 2010 onward. Adjusted cause-specific hazard models were used to evaluate associations between APOs and incident COPD.

Results

Among 25,541 participants, 7,226 (28.3%) reported at least one APO, with preterm delivery being the most common (50.6%). Over follow-up, 4,205 women (16.5%) reported incident COPD. Any history of APOs was associated with increased future COPD risk (adjusted hazard ratio (aHR) 1.15, 95% CI 1.07–1.22), as was having ≥ 2 different APOs (aHR 1.12, 95% CI 1.02–1.23). Specific APOs independently associated with higher COPD risk included HDP (aHR 1.27, 95% CI 1.13–1.42), small for gestational age infant (aHR 1.11, 95% CI 1.02–1.21), and preterm delivery (aHR 1.11, 95% CI 1.02–1.21).

Interpretation

This national cohort study is the first to link APOs with higher maternal COPD risk, suggesting that abnormal pregnancy may be an early indicator of long-term respiratory vulnerability. These findings underscore the need to better understand the intersection of reproductive and respiratory health in women.

BACKGROUND

Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity and currently ranks as the fourth leading cause of death worldwide.^{1–3} Once considered a disease of older men,⁴ the global prevalence of COPD is now rising more rapidly among women. One study estimated 7.2 million cases in

women, compared to 4.4 million in men.⁵ In certain countries, COPD-related mortality in women has surpassed that of men,^{5,6} and while COPD-related deaths among men have declined, this trend has not been observed in women.^{2,7} Moreover, COPD is typically diagnosed several decades after silent, progressive loss of lung function, and there are no early biomarkers of subclinical disease.^{8,9} As such, there is a need to identify early predictors of poor lung health and increased risk of COPD, particularly in women.

While smoking is a key modifiable risk factor for COPD, women with COPD are more likely than men to be never smokers.⁵ Among smokers, women experience a more rapid decline in forced expiratory volume in 1 second (FEV₁), suggesting heightened susceptibility to tobacco smoke.⁹ Other contributing factors, such as greater exposure to indoor air pollution, occupational exposures, smaller airway lumen and genetic predispositions, explain some, but not all, of the observed sex-associated differences in COPD risk among women.^{6,10–12}

Emerging evidence suggests that hormonal and reproductive factors may be associated with long-term respiratory health outcomes in women.^{9,13,14} One European longitudinal study found that higher parity, later menarche, and early menopause were independently associated with COPD-related hospitalizations and deaths, even after accounting for important confounders such as smoking history and socioeconomic indicators.¹³ Pregnancy is often one of the most significant events in a woman's reproductive lifespan and serves as an early physiologic "stress test" with the potential to forecast maternal cardiometabolic health.¹⁵ However, the impact of pregnancy on future maternal respiratory health is not established.

Adverse pregnancy outcomes (APOs) are common – occurring in approximately 20% of pregnancies¹⁶ – and have been linked to increased long-term maternal morbidity.¹⁴ APOs include gestational diabetes mellitus (GDM), hypertensive disorders of pregnancy (HDP), preterm delivery, and the delivery of a small or large for gestational age infant.¹⁹ Multiple studies have identified associations between APOs and future maternal cardiovascular diseases, including atherosclerotic coronary artery disease and heart failure.^{17–19} Cardiovascular disease and COPD share common biological pathways, and patients with one condition are at increased risk for developing the other.^{20–22} In particular, COPD is characterized by systemic inflammation beyond the airways, contributing to increased risk of cardiovascular complications.^{22,23} Thus, it is plausible that mechanisms underlying future cardiovascular risk among women with APOs may play a similar role in the development of COPD. This is supported by one recent cohort study showing that recurrent pregnancies with APOs were associated with poorer subsequent maternal lung function, compared to those without APOs.²⁴ Similarly, another large national cohort study found a significant relationship between a history of HDP and increased risk of incident COPD and asthma.²⁵ However, relationships between other APOs and the development of COPD later in life have not been studied. In this study, we examined the relationship between a history of APOs and the

subsequent development of maternal self-reported COPD among participants in the Women's Health Initiative Observational Study (WHI-OS).²⁶

METHODS

Population and study design

This is a secondary analysis of the WHI-OS. Originally, the Women's Health Initiative (WHI) study was established to investigate predictors of chronic diseases and mortality in women.²⁷ The WHI enrolled 161,808 post-menopausal women aged 50 to 79 years across 40 clinical centers in the United States (U.S.) between 1993 and 1998 and was comprised of two components: 3 randomized clinical trials (RCTs) and a longitudinal observational study.^{27,28} From 2005 to 2024, participants in the WHI-OS were invited to join the WHI Extension Studies (I and II), which continued to monitor health outcomes (Fig. 1).

For this analysis, we included women from the WHI-OS cohort and their follow-up through the WHI Extension Study II, concluding in 2024 (n = 25,541). We excluded women in the RCTs because only participants of WHI-OS were asked about incident emphysema and/or chronic bronchitis before the Extension II Study started in 2010. We included all WHI-OS participants except those who either reported a history of COPD or answered "unknown" at enrollment; had no history of pregnancy or no pregnancy lasting at least 6 months, or those missing data on pregnancy and APO history (**e-Figure 1**). All participants provided written informed consent for the WHI-OS and extension studies. IRB approval was obtained for this analysis (IRB: 2006C0007).

Assessment of APOs

Demographic and clinical characteristics were collected at baseline during an in-person visit,^{30,31} and by survey annually thereafter until 2024. Pregnancy history was recorded at WHI enrollment, whereas information on APOs was obtained in 2017 through a follow-up questionnaire that included six questions regarding specific APO history. In the survey, APO was defined as ever having had any of the following: HDP (gestational hypertension and/or pre-eclampsia), GDM, large for gestational age infant (birth weight > 9 lb 14 oz), small for gestational age infant (birth weight < 5.5 lbs), or preterm birth (born at least 3 weeks early).³² Gestational hypertension and pre-eclampsia were combined into a single HDP variable given similar pathophysiology, substantial overlap in responses, and to maintain consistency with prior analyses within the WHI cohort.^{33,34} Multiple APOs may have occurred in the same woman, though not necessarily within the same pregnancy. Due to the structure of the survey questions, it was not possible to ascertain whether respondents had experienced multiple occurrences of the same APO type. Instead, to better understand the implications of having had multiple, different APOs, we performed additional analyses comparing women who had two or more types of APOs with women who had none or one.

Assessment of incident COPD

Incident self-reported COPD was the primary outcome. Participants were asked about a history of self-reported COPD during their baseline enrollment visit and in the first 8 annual follow-up questionnaires through the question, “Has a doctor or other healthcare provider told you that you have emphysema or chronic bronchitis?” There were no questionnaires on COPD history in the Extension I study (2005 to 2010). In the Extension II study beginning in 2010, the question was slightly modified such that participants were asked specifically about COPD in addition to emphysema and chronic bronchitis (Fig. 1). Similar to previous studies within the WHI cohort, for this analysis, incident COPD was defined as reporting an affirmative answer to the annual follow-up questionnaires, but not at baseline, as we could not confirm whether a self-report of COPD at baseline (outcome) occurred after pregnancy (exposure).³⁵ Although self-reported COPD was not adjudicated in the WHI-OS, prior studies have demonstrated that self-reported questionnaire data can reliably identify COPD.^{36,37}

Covariates and statistical analysis

Covariates were determined *a priori* based on established literature. These included age at enrollment, pack-years of smoking, secondhand smoke exposure (defined as ever having lived and/or worked with a smoker), history of self-reported asthma, self-identified race and ethnicity, education level, household income, geographic region of enrollment, personal history of premature birth and low birthweight, and parity. All covariate data were obtained from baseline demographic information collected at the time of WHI-OS enrollment. To address missing covariate data, multiple imputation was performed, generating 20 imputed datasets. Fully conditional specification methods were used for the imputation models, and included the previously listed covariates, BMI at enrollment, physical activity level at enrollment (as energy expenditure per week), history of breastfeeding, history of any APO, incident COPD, and each participant’s Nelson-Aalen estimator of overall survival.³⁸

Demographics were compared between women with no APOs and those with at least one APO. Continuous comparisons were made using Wilcoxon rank-sum tests, and categorical comparisons were made using Chi-square tests. Unadjusted and adjusted cause-specific proportional hazard models were used to evaluate the association between a history of APOs and incident COPD. Similar models were used to examine the independent effect of each APO (HDP, GDM, large for gestational age infant, small for gestational age infant and preterm birth) on incident COPD. Due to differences in data collection, specifically the period between the end of the WHI-OS follow-up and the start of Extension II when data on COPD was not captured, the exact timing of self-reported COPD incidence was unknown. We accounted for this using interval censoring in the models. The interval censored proportional hazards models were fit using the PROC ICPHREG statement in SAS, with the baseline hazard function modeled as cubic splines with three knots and death before COPD treated as a censoring event. Adjusted hazard ratios (aHR) were estimated within each imputed dataset and subsequently combined using Rubin’s rules to appropriately account for imputation-related variability.⁶¹

We also conducted a stratified analysis to determine whether associations between APOs and incident COPD differed across the two follow-up intervals (< 10 years for the original WHI-OS study follow-up, and $\geq$ 10 years for the extension study follow-up) to account for the two distinct periods of outcome assessment (Fig. 1). For the main analysis, a sensitivity analysis was also conducted with the addition of weekly physical activity energy expenditure at enrollment as a covariate and excluding maternal history of premature birth and low birth weight, due to possible recall bias. Finally, we examined potential effect modification by lactation status, smoking status, and race/ethnicity on the relationship between history of APOs and incident COPD through interaction analyses. P-values < 0.05 were considered statistically significant. All statistical analyses were performed using SAS version 9.4 (SAS Institute, Inc., Cary, NC).

RESULTS

Population characteristics

A total of 25,541 women met our eligibility criteria (**e-Figure 1**). At the time of enrollment, the median (interquartile range) age of participants was 60.3 (56.1–65.2) years. Most were never-smokers (53.0%), although 94.1% reported exposure to secondhand smoke. The participants were predominantly White (92.7%) with a high level of education (76.9% attended college). Compared to women without APOs, those with APOs had statistically significantly greater pack years of smoking, greater BMI, lower annual household income, and more reported a history of asthma, grand multiparity, low personal birth weight and personal prematurity (Table 1). In this cohort 7,226 women (28.3%) reported at least one APO, with preterm birth being the most frequent (50.6%) (Table 2). A total of 4,205 (16.5%) participants self-reported incident COPD, with the condition identified a median (range) of 18 (0.02–28.9) years following enrollment. Among individuals with a history of APOs, 1,335 (18.5%) reported incident COPD, compared with 2,870 (15.7%) among those without prior APOs.

Table 1. Participant demographics and characteristics at baseline by APO status.

Baseline characteristic ^a	Total n = 25,541	No APOs n = 18,315	≥1 APO n = 7,226	p-value ^b
Age (years)	60.3 (56.1-65.2)	60.3 (56.0-65.3)	60.3 (56.1-65.2)	0.41
Pack years of smoking				<0.001
Never	13,121 (53.0)	9,522 (53.6)	3,599 (51.6)	
<5	4,286 (17.3)	3,109 (17.5)	1,177 (16.9)	
5-19.9	3,752 (15.2)	2,607 (14.7)	1,145 (16.4)	
20+	3,596 (14.5)	2,536 (14.3)	1,060 (15.2)	
Missing	786	541	245	
Secondhand smoke exposure ^c				0.17
No	1,489 (5.9)	1,091 (6.0)	398 (5.5)	
Yes	23,941 (94.1)	17,144 (94.0)	6,797 (94.5)	
Missing	111	80	31	
History of asthma				<0.001
No	23,779 (93.1)	17,125 (93.5)	6,654 (92.1)	
Yes	1,762 (6.9)	1,190 (6.5)	572 (7.9)	
Body mass index	25.4 (22.9-28.9)	25.3 (22.8-28.7)	25.8 (23.2-29.6)	<0.001
Race				0.17
Black	1,033 (4.1)	705 (3.9)	328 (4.6)	
White	23,493 (92.7)	16,883 (92.9)	6,610 (92.2)	
Other ^d	1,015 (3.2)	727 (3.2)	288 (3.2)	
Ethnicity				0.76
Not Hispanic/Latino	24,812 (97.3)	17,791 (97.3)	7,021 (97.4)	
Hispanic/Latino	677 (2.7)	489 (2.7)	188 (2.6)	
Missing	52	35	17	
Education				0.10
High school and below	5,852 (23.1)	4,145 (22.8)	1,707 (23.8)	
College and above	19,516 (76.9)	14,042 (77.2)	5,474 (76.2)	

Missing	173	128	45	
Annual household income (\$)				<0.001
<20,000	1,634 (6.8)	1,134 (6.5)	500 (7.3)	
20,000-74,000	15,137 (62.5)	10,717 (61.8)	4,420 (64.4)	
≥75,000	7,447 (30.8)	5,504 (31.7)	1,943 (28.3)	
Missing	1,323	960	363	
Region of enrollment				0.33
Northeast	6,215 (24.3)	4,447 (24.3)	1,768 (24.5)	
South	6,066 (23.8)	4,403 (24.0)	1,663 (23.0)	
Midwest	6,192 (24.2)	4,405 (24.1)	1,787 (24.7)	
West	7,068 (27.7)	5,060 (27.6)	2,008 (27.8)	
Personal history of prematurity				<0.001
No	24,055 (97.5)	17,345 (97.8)	6,710 (96.5)	
Yes	626 (2.5)	385 (2.2)	241 (3.5)	
Unknown	860	585	275	
Personal history of low birth weight				<0.001
No (6+ lbs)	20,788 (89.5)	15,036 (90.4)	5,752 (87.2)	
Yes (<6 lbs)	2,442 (10.5)	1,594 (9.6)	848 (12.9)	
Unknown	2,311	1,685	626	
Parity				<0.001
1	2,519 (9.9)	2,027 (11.1)	492 (6.8)	
2	8,452 (33.1)	6,531 (35.7)	1,921 (26.6)	
3	7,423 (29.1)	5,316 (29.0)	2,107 (29.2)	
4	4,056 (15.9)	2,667 (14.6)	1,389 (19.2)	
≥5	3,091 (12.1)	1,774 (9.7)	1,317 (18.2)	
Physical activity (MET-hours/week)	11.8 (4.5-22.3)	12.2 (4.5-22.6)	11.0 (3.8-21.3)	<0.001

^aMedian (interquartile range) is reported for continuous variables. Frequency (%) is reported for categorical variables and excludes missing/unknown responses.

^bWilcoxon rank-sum tests were used for continuous variables and chi-square tests of independence were used for categorical variables.

^cDefined as ever having had secondhand smoke exposure at home or work.

^dOther includes those who self-identified as American Indian/Alaska native, Asian, Native Hawaiian/Pacific Islander, more than one race, or those whose race identification was missing.

APO: adverse pregnancy outcomes; MET: Metabolic equivalent of task.

Table 2
Summary of type of APOs among women with a history of APOs and all participants

APO category	≥ 1 APO n = 7,226	All included women n = 25,541
Hypertensive disorders of pregnancy, n (%)	1,576 (21.8)	1,576 (6.2)
Gestational diabetes, n (%)	511 (7.1)	511 (2.0)
Large for gestational age infant ^a , n (%)	1,418 (19.6)	1,418 (5.6)
Small for gestational age infant ^b , n (%)	3,071 (42.5)	3,071 (12.0)
Preterm delivery ^c , n (%)	3,659 (50.6)	3,659 (14.3)
^a Defined as birth weight of > 9 lb 14 oz or 4.08 kg		
^b Defined as birth weight of < 5.5 lbs or 2.49 kg		
^c Defined as delivery at least 3 weeks early		
APO: Adverse pregnancy outcome.		

Relationships between APOs and incident COPD

In both unadjusted and adjusted models, the presence of any APO was significantly associated with an increased maternal risk of future incident COPD (aHR 1.15, 95% CI 1.07–1.22) (Fig. 2). Significant associations were also independently observed between HDP (aHR 1.27, 95% CI 1.13–1.42), delivery of a small for gestational age infant (aHR 1.11, 95% CI 1.02–1.21), preterm delivery (aHR 1.11, 95% CI 1.02–1.21) and incident COPD. A history of two or more different APO types was similarly associated with a significantly elevated risk of incident COPD (aHR 1.12, 95% CI 1.02–1.23) (Fig. 2). There were no significant independent associations between a history of GDM or delivery of a large for gestational age infant and incident COPD. These results remained robust after a sensitivity analysis including physical activity as a covariate and excluding self-reported personal history of prematurity and low birthweight (**e-Table 1**). When stratified by time since enrollment, adjusted associations between APOs and incident

COPD were generally only significant after 10 years of follow-up (**e-Figure 2**). Finally in interaction analyses, the association between APO and incident COPD did not significantly differ by any of the following effect modifiers: lactation status ($p_{\text{int}} = 0.21$), race ($p_{\text{int}} = 0.81$), ethnicity ($p_{\text{int}} = 0.56$), or pack years of smoking ($p_{\text{int}} = 0.20$) (**e-Table 2**).

DISCUSSION

To our knowledge, this is the first national cohort study to examine the association between APOs and the subsequent maternal risk of COPD. Women with a history of any APO had a significantly higher incidence of self-reported COPD later in life, compared to women without APOs, even after accounting for key confounders. The same pattern was observed independently for HDP, delivery of a small for gestational age infant, and preterm delivery. These findings suggest a potentially novel link between gestational complications and long-term maternal respiratory health, warranting further mechanistic and confirmatory studies. While additional research is needed to elucidate causal pathways, our findings contribute to growing evidence that prenatal health may be an underrecognized predictor of respiratory outcomes in women, with implications extending beyond pregnancy into mid- and late life.

COPD is a heterogenous disease with important sex-specific differences, with women experiencing greater disease prevalence, symptom burden and exacerbations, compared to men.^{4–7} Reasons underlying these sex disparities remain incompletely understood, but may reflect genetic and epigenetic influences in combination with cumulative internal and external environmental exposures across the life course (the “exposome”).³⁹ Reproductive health is an important yet understudied aspect of the exposome. Certain reproductive factors, such as a history of multiple pregnancies (multiparity), early menarche and early menopause, have recently been shown to be associated with reduced lung function.^{13,40–42} Recognizing reproductive factors as potential determinants of respiratory health may facilitate earlier identification of women at increased risk for COPD. This is important as COPD is often diagnosed late in the disease course – after years or decades of subclinical decline in lung function – without consensus on screening strategies for early detection.^{9,43} Our findings add to evidence that reproductive health is a core component of the female exposome with potential implications for respiratory health later in life.

In this study, we show that APOs are linked to a greater risk of incident maternal COPD later in life. The individual APOs most strongly associated with incident COPD were HDP, premature birth, and the delivery of a small for gestational age infant. These conditions share overlapping inflammatory and immune-related pathways. For example, HDP is associated with abnormal cytotrophoblast invasion, impaired oxygen delivery, and a dysregulated immune profile characterized by Th1/Th2 imbalance, elevated cytokines, and oxidative stress.^{54–57} Oxidative stress has independently been linked with HDP, preterm delivery, and intrauterine growth restriction. Chronic inflammation and oxidative stress have also been implicated in the development of COPD.^{58–60} Women with a history of APOs – particularly HDP and small for gestational age infant – may exhibit abnormal placentation, heightened systemic inflammation,

immune dysregulation, and vascular dysfunction. When these abnormalities persist post-partum, they may increase susceptibility to both cardiovascular and pulmonary disease.^{19,54} Gestational complications, such as HDP, are well known to predict higher long-term maternal risk of heart failure, ischemic heart disease, and stroke.^{16,17,19,33,34,47} This has prompted major cardiac and obstetric societies to recommend earlier screening and longitudinal follow-up beyond the postpartum period.^{48, 49, 50, 51, 52} As APOs may signal an early, heightened systemic inflammatory burden that fails to fully resolve postpartum and can persist sub-clinically, the same pathophysiologic mechanisms linking APOs to cardiovascular disease may also increase COPD risk later in life.

We also found that a history of two or more different APO types was associated with a significantly elevated risk of incident COPD, even after adjusting for multiple pregnancies and other important confounders. This suggests that multiple and/or repeated abnormal gestational outcomes may perhaps signal persistent underlying physiologic and immunologic abnormalities, portending greater risk of future COPD. This is supported by a recent cohort study, which found that a history of multiple pregnancies affected by APOs was associated with lower FEV₁ and greater lung function loss in the decades to follow, compared to women without APOs.²⁴ Additional research is needed to elucidate the specific biological mechanisms linking APOs with respiratory health. As there is no single determinant for the development of COPD, these findings emphasize for the first time that APOs may be sex-specific predictors – in addition to the array of other genetic and environmental determinants – that inform COPD risk assessment in early adulthood. Integrating obstetric history into pulmonary risk assessment may facilitate earlier identification of women more susceptible to developing COPD, particularly among those with other risk factors, such as smoking or occupational exposures.

This study has multiple strengths. The use of the WHI-OS provides a robust dataset with a long follow-up period of over 3 decades, enhancing assessment of long-term outcomes throughout the life course. Our analysis also benefits from a large sample size drawn from centers across the U.S., comprehensive adjustment for important confounders, and the evaluation of multiple, different APOs on incident COPD. However, there are also important limitations. The reliance on self-reported data for both APOs and COPD introduces the potential for recall bias and misclassification. Additionally, the retrospective collection of obstetric history (in 2017) long after most pregnancies occurred may have increased the risk of reporting errors. The diagnosis of COPD was also not confirmed with clinical testing such as spirometry. The cohort consisted of predominantly White women, which may affect the generalizability of the findings to more diverse populations. Lastly, residual confounding is possible, particularly regarding environmental exposures, socioeconomic status, and access to healthcare.

CONCLUSION

In this large, national cohort of postmenopausal women, a history of APOs was associated with a significantly increased maternal risk of developing COPD later in life. These findings suggest that APOs

may serve as early-adulthood markers of future respiratory vulnerability, highlighting a novel avenue for future research integrating obstetric and reproductive history into COPD risk assessment in women.

Abbreviations

APO
Adverse Pregnancy Outcomes
BMI
Body mass index
COPD
Chronic Obstructive Pulmonary Disease
FEV1
Forced expiratory volume in one second
HDP
Hypertensive disorders of pregnancy
GDM
Gestational diabetes mellitus
LGA
Large for gestational age
SGA
Small for gestational age
WHI
Women's Health Initiative
WHI-OS
Women's Health Initiative – Observational Study

Declarations

IRB Approval: IRB approval was obtained for this analysis (IRB: 2006C0007)

Clinical trial number: not applicable

Ethics Approval: Ethics approval for the Women's Health Initiative study was obtained by the original investigators, and all participants provided written informed consent. The present study is a secondary analysis of de-identified Women's Health Initiative data. This secondary analysis was reviewed and granted IRB approval by the Ohio State University (IRB: 2006C0007).

All procedures involving human participants in the original study were conducted in accordance with relevant ethical guidelines and regulations, including the Declaration of Helsinki and its later amendments.

Consent for Participation: All participants in the Women's Health Initiative provided written informed consent at enrollment in the parent study.

Consent for Publication: Not applicable

Availability of Data and Materials: The data that support the findings of this study are available from the Women's Health Initiative but restrictions apply to the availability of these data, which were obtained via proposal for the current study, and are not publicly available. In order to obtain the data used, submit a request through WHI to access the raw data used in the study by contacting the WHI Help Desk (<https://www.whi.org/helpdesk>).

Competing Interests: Not applicable

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Guarantor and author contributions:

JW is the author responsible for all content of the manuscript and conceived the study idea. SS contributed to data interpretation and drafted the manuscript. EM and MP performed data analyses. PM, AS, RY, EP, RM, LG, JR and SB contributed to data interpretation.

All authors revised the manuscript for intellectual content and approved the final version of the manuscript.

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Figures

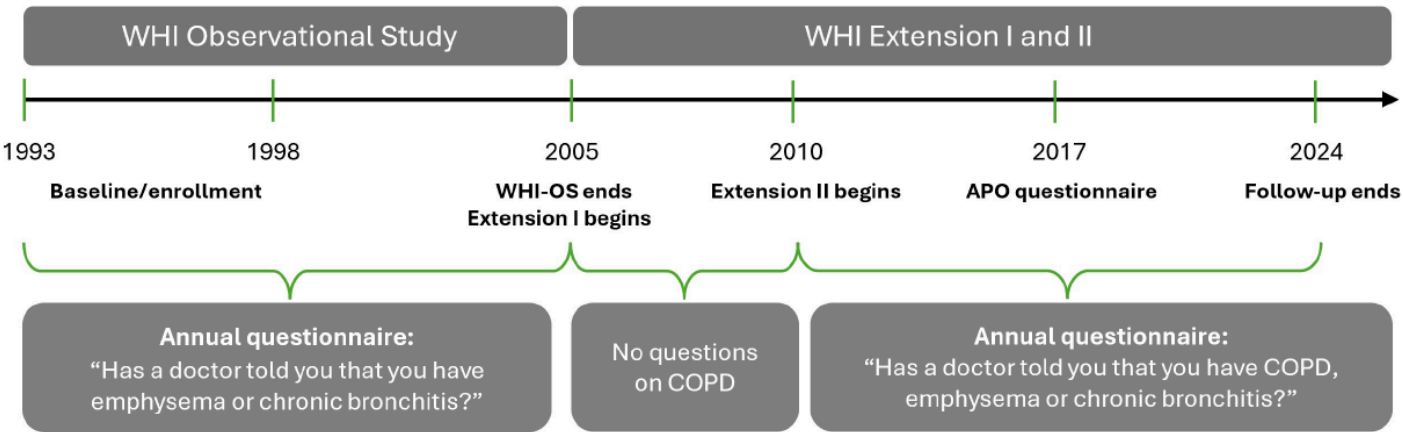


Figure 1

Study timeline. WHI: Women’s Health Initiative. APO: Adverse pregnancy outcome.

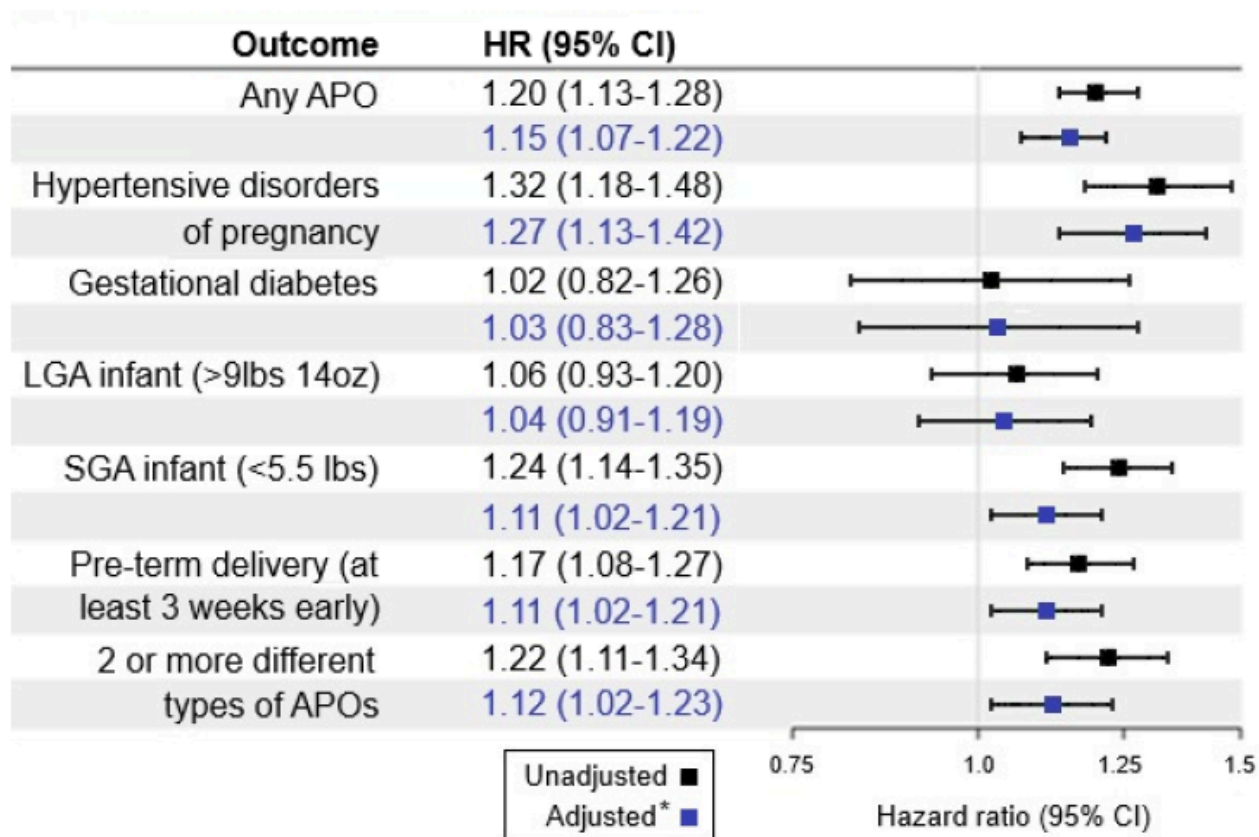


Figure 2

Associations between a history of APOs and future incident COPD.

*Models adjusted for age at enrollment, race, ethnicity, pack years of smoking, secondhand smoke exposure, history of asthma, education, household income, geographic region, parity, born at low birthweight, and born premature. APO: Adverse pregnancy outcome; LGA: Large for gestational age; SGA: Small for gestational age.

Supplementary Files

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