Original Research

Association of Short-Term Pollen Exposure With Lung Function in COPD Patients

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Running Head: **Short-Term Pollen and Lung Function in COPD**

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Abbreviations: COPD - Chronic Obstructive Pulmonary Disease; SPACE - Study of Air Pollution and COPD Exacerbation; FEV_1 - Forced Expiratory Volume in 1 second; FVC - Forced Vital Capacity; NO_2 - Nitrogen Dioxide; O_3 - Ozone; $PM_{2.5}$ - Particulate Matter Diameter of 2.5 micrometers or less

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Pre-Proof

Abstract

Background: Environmental exposures such as air pollution are well established triggers for Chronic obstructive pulmonary disease (COPD) exacerbation and impaired lung function. However, the role of aeroallergens, particularly pollen, has not been thoroughly examined in COPD, despite evidence that pollen inhalation can cause airway inflammation. We investigated whether short-term exposure to higher concentrations of ambient pollen is associated with changes in lung function in people with COPD.

Methods: Thirty COPD participants, contributing 1,808 observations, were enrolled in the Study of Pollution and COPD Exacerbation (SPACE) and completed four clinic visits over one year while performing daily spirometry during four 30￼day seasonal periods. Ambient total pollen was measured regionally. Associations of short-term pollen exposure (lag 1-7 days and cumulative 3- and 7-day concentration) with FEV_1 and FVC were assessed using distributed lag non-linear models with a Generalized Additive Mixed Model. We tested if inflammatory biomarkers, inhaler medications, asthma history and concurrent exposure ambient pollutants (NO_2, $PM_{2.5}$, O_3) modified associations between pollen exposure and lung function.

Results: Pollen exposure at lag 3 day was associated with lower FEV_1 (–5.5 mL per IQR pollen increase; 95% CI: –9.6, –1.3). Cumulative 3-day pollen exposure was similarly associated with lower FEV_1 (–4.9 mL; 95% CI: –9.6, –0.2). Associations of pollen and FEV1 were greater in those with higher CRP and diagnosis of asthma.

Conclusions: In this longitudinal study of people with COPD, higher short-term exposure to ambient pollen in the preceding 3 days was associated with lower FEV_1. Our findings suggest that aeroallergen exposure may worsen airflow obstruction in people with COPD.

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Introduction

Chronic obstructive pulmonary disease (COPD) is an inflammatory lung disease that is characterized by progressive persistent airflow limitation and recurring exacerbations. Over 390 million individuals are affected by COPD globally [1], and it is the third leading cause of death worldwide [2]. COPD cases and related exacerbations are predicted to rise due to an aging global population and worsening climatic and environmental conditions [3, 4]. Acute exacerbations from various factors such as infections, air pollutants, and environmental triggers can rapidly worsen lung function and contribute to morbidity, hospitalization, and even mortality [5, 6]. Previous studies, including our own work in the SPACE study, have found that exposure to air pollutants is associated with lower Forced Expiratory Volume 1 (FEV_1), worse respiratory symptoms, and increased healthcare reliance in COPD sufferers [7, 8, 9, 10, 11]. While the impact of air pollution on COPD is well-studied, less is understood about the effect of aeroallergens on the respiratory health of people with COPD.

Most research on the respiratory effect of pollen exposure has concentrated on allergic rhinitis or asthma. Pollen is a known trigger of airway inflammation, bronchoconstriction, and symptom exacerbation in allergic asthma [12,13]. However, a notable proportion of COPD patients exhibit allergic sensitization or atopy, with studies indicating that COPD who are sensitized to environmental allergens may experience more frequent symptoms and poorer lung function [14,15]. Mechanistically, pollen exposure can induce neutrophilic and eosinophilic inflammation, mucous hypersecretion, and increased airway reactivity, processes that could worsen airflow obstruction in COPD [10,16]. Further, pollen is an increasingly important exposure in the context of climate change, with higher temperatures and variable precipitation leading to extended pollen seasons, higher concentrations within the season, and more potent allergenic particles, elevating

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population-level respiratory risk [17, 18, 19, 20]. As previous ecological studies have linked aeroallergen exposure to increased COPD hospitalizations [21, 22], and a time-series analysis in Beijing showed associations between ambient pollen and COPD-related clinical visits [23]. However, few studies have examined objective lung function as a measure of airway response to pollen exposure in COPD.

To address this gap, we investigated the association between short-term pollen exposure over the preceding days and differences in FEV_1 and FVC among former smokers with moderate-to severe COPD.

Methods

Study Population

The study population comprised 30 participants diagnosed with COPD who were former smokers.

These participants were recruited as part of the Study of Air Pollution and COPD Exacerbation (SPACE) conducted at the Beth Israel Deaconess Medical Center in Boston, Massachusetts [11,24]. Eligibility criteria for participation included a clinical diagnosis of COPD, former smoking status with at least six months smoke-free, and moderate airflow obstruction. Specifically, participants required an FEV_1/FVC ratio <0.70 and an FEV_1 between 50 and 80% of the predicted value, consistent with Global Initiative for Chronic Obstructive Lung Disease (GOLD) Stage II [25] A distinctive eligibility requirement was that participants' home addresses be within 50 km of the Harvard Supersite air pollution monitor at Harvard Medical School. The study was approved by the Committee on Clinical Investigations at Beth Israel Deaconess Medical Center.

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Data Collection

Data collection for the study spanned from February 24, 2017 to January 17, 2019. Upon entry into the study, comprehensive baseline information was collected for each participant, including demographic details, height, weight, past medical history, and initial measures of lung function. Participants were subsequently observed for up to four non-consecutive 30-day periods, distributed across four different seasons over a 12-month duration.

Exposure Assessment

Total pollen count was obtained as a regional daily average from Asthma and Allergy Affiliates, Inc. based in Salem, Massachusetts, US. A Rotorod sampler was used to record daily pollen concentrations, measured in grains per cubic meter (grains/m^3). Total pollen included tree, grass, and weed pollen, along with "other" or unclassified pollen particles [26]. Our measured pollen season was defined as March through October, coinciding with the pollen season in the Northeastern United States. Observations outside the pollen season were excluded. Pollen collected over weekends and holidays was counted on the following weekday; therefore, weekend and holiday values reflect the pollen concentrations averaged across the days of collection (e.g., Saturday, Sunday, and Monday pollen concentrations are the average across those 3 days). Moving averages are defined as the average exposure over the period of days preceding the lung function measurement (e.g., a 3-day moving average is the sum of pollen over 3 days divided by 3). Temperature was defined as the daily mean temperature. Daily ambient air pollution data were obtained from state-owned stationary monitors in the Boston area. For $PM_{2.5}$ and NO_2, we averaged daily concentrations from multiple Boston monitors; for O_3, we used data from the Boston (Roxbury neighborhood) state monitor as previously described [11].

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Outcome Assessment

Participants performed daily lung function measurements each morning, prior to taking any medications or inhalers, utilizing a portable EasyOne™ Plus Diagnostic Spirometer. This device meets the guidelines established by the American Thoracic Society and incorporates built-in quality assurance and incentive software to ensure data reliability. By the conclusion of the follow-up period, the 30 participants collectively contributed a total of 1,808 observations during the pollen season (March to October).

Statistical Analysis

To assess the relationship between short-term total pollen exposure at lag 1 to 7 days and lung function in COPD, as well as the cumulative association over lag 1–3 days, we used distributed lag non-linear models (DLNM) combined with a generalized additive mixed model (GAMM). We adjusted for potential confounders including age, sex, height, weight, education, seasonality (sine and cosine terms of date), and 3-day moving averages of temperature (lag 1–3 days) prior to the spirometry assessment.

We assessed potential effect modification by markers of Type 2 inflammation (blood absolute eosinophils ≥ 150 vs. < 150 cells/µL), systemic inflammation (C-reactive protein [CRP] > 3 vs. ≤ 3 mg/L), history of CVD, history of asthma, and baseline use of maintenance medications (inhaled corticosteroids [ICS], long-acting β-agonists [LABA], long-acting muscarinic antagonists [LAMA], and beta-blockers). Moreover, we assessed effect modification by ambient air pollutants ($PM_{2.5}$, NO_2, and O_3), categorized as high versus low based on the median 3-day moving average of each pollutant prior to the spirometry assessment. Differences in the estimated effects between subgroup strata were formally evaluated for statistical significance

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using a Z-test.

Sensitivity analyses were performed by adding other environmental exposures (humidity, NO_2, O_3, $PM_{2.5}$) to the primary model. Additionally, we performed sensitivity analyses: excluding Sundays through Tuesdays to account for weekend/holiday pollen averaging; and removing all covariates from our model. All analyses were conducted in R version 4.3.1.

Results

Study Population and Exposure

In this longitudinal study, we included 30 COPD participants with 1,808 observations. Table 1 summarizes baseline population characteristics. Participants had a mean age of 71.1 years (SD 8.4), those as female from birth were the majority (53.3%), were predominantly white (82%), and most had at least some post-secondary education. Across all study visits, the mean FEV_1 was 1.3 L, and FVC was 2.6 L. The average within-participant variation was 0.13L for FEV_1 and 0.24L for FVC. Table 2 outlines the distribution of exposures encountered by the study participants. Median exposure to pollen was 32.4 grains/m^3 with an IQR of 145.4 grains/m^3.

Pollen Exposure and Lung Function Associations

We observed an association between higher total pollen exposure at lag 3-day and lower FEV_1 (Figure 1). Each IQR (145.4 grains/m^3) difference in total pollen exposure was associated with a 5.5 mL lower FEV_1 (95% CI: -9.6, -1.3). A cumulative association between higher total pollen exposure at lag 1-3 days with a lower FEV1 of 4.9 mL (95% CI: - 9.6, -0.2). There was a similar pattern but non-significant cumulative association between higher total pollen exposure at lag 1-3 days and lower FVC of 7.9 (-15.9, 0.1) (Supplemental Table 2). For the subtypes of pollen, we observed a negative pattern between higher tree and other pollen exposure at a 7-day lag and

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lower FEV_1, although this association did not reach statistical significance, nor did we see similar negative patterns at Lag 1 or 3 days (Supplemental Table 3).

Effect modification

We observed that the association between total pollen exposure at lag 3 day and lower FEV_1 was more pronounced among individuals with elevated baseline systemic inflammation (CRP > 3 mg/L) and a history of asthma (Figure 2, *P*-interactive<0.05 for both tests of effect modification). Similarly, the use of maintenance inhaler therapies (ICS) appeared to slightly attenuate the association between pollen and lower FEV_1 compared to non-use, but this modifying effect was not statistically significant (*P*-interactive: 0.15). Furthermore, the negative association between pollen and 1 appeared slightly more pronounced on days characterized by high ambient NO_2 exposure compared to low NO_2 days, although this difference was not significant (*P*-interactive: 0.11). We found no evidence of interactive effects regarding eosinophils, CVD, other inhalers, and concurrent exposure to other ambient air pollutants ($PM_{2.5}$ and O_3) on FEV_1.

For FVC the patterns of effect modification were similar to FEV_1. However, no tests of effect modification were statistically significant, except for NO_2 exposure. We found a stronger negative association with pollen exposure and FVC on days characterized by high concurrent ambient NO_2 (-19.1mL; 95% CI: -30.9 to -7.3) compared to low NO_2 exposure (-0.8mL; 95% CI: -12.4 to 10.8) per IQR increased in pollen (*P*-interaction < 0.05).

Sensitivity Analyses

The cumulative association between pollen exposure at lag 1-3 days and FEV_1 remained robust across multiple sensitivity analyses (Supplemental Table 4). Upon adjusting for humidity, NO_2, O_3, and $PM_{2.5}$, a significant negative association between total pollen and FEV_1 was still

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observed. Our results also remained robust upon removing all covariates from our. Further, we performed a sensitivity analysis excluding Sundays through Tuesdays to account for weekend/holiday pollen averaging. We found that the direction and effect size of FEV_1 (cumulative associations with lag 1-3 days) remained similar (-4.6 mL 95% CI: -10.6 mL, 1.3 mL).

Discussion

In this longitudinal study of 30 COPD patients with 1,808 observations, we found that higher short-term pollen exposure was associated with lower FEV_1. Short-term exposure to pollen at lag 3 days was associated with a 5.5 mL lower FEV_1, and the cumulative lag 1-3 day association yielded a similar effect size. This finding suggests that pollen may impair expiratory airflow in patients with COPD, with a possible cumulative effect that takes more than a day to develop.

While we found associations of pollen and lung function in our study of COPD patients overall, associations predominated among those with asthma-COPD overlap. Studies in asthmatic populations demonstrate that pollen can increase bronchial hyperresponsiveness in individuals, not classically "allergic" [10, 27, 28]. Our results are consistent with such past asthma literature, especially that of Erbas et al. whose meta-analysis showed that acute pollen exposure is associated with reduced lung function and increased hospitalizations for emergency presentations due to asthma exacerbations [17]. Previous evidence has also suggested that non-asthmatic people are susceptible to airway inflammation and respiratory effects of pollen exposure. A study of acute pollen exposure and respiratory hospital admissions found an association with COPD patients but not people with asthma [18].

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Throughout this study, we saw that the association of pollen on lower FEV_1 appeared at lag 3 but not lag 1. This is consistent with asthma and atopy studies showing delayed airway responses to pollen. For example, Idrose et al. found that grass pollen exposure was associated with airway obstruction 2–3 days after exposure in a cohort with atopy [29]. Further, grass pollen was shown to have a lagged inflammatory response suggesting that pollen related inflammation may peak after a short lag [30].

We saw that the association between pollen and lower FEV_1 was modified by elevated systemic inflammation (CRP) and history of asthma. Systemic inflammation measures such as CRP have been found to be a raised in COPD patients and demonstrate an inverse correlation with FEV_1 [31, 32]. It is therefore plausible that higher CRP could modify and even exacerbate the pollen-reduced FEV_1 relationship through augmented inflammatory pathways. While not statistically significant, we found that those who used steroid inhalers had a less pronounced association between pollen and FEV1, suggesting that palliation of inflammatory pathways in the airways may help prevent some of these effects.

We also explored potential modification by air pollutants, as prior studies have found that pollutants can synergistically enhance allergenicity and respiratory effects of pollen [33, 34, 35]. We observed that the negative association between pollen and FVC was more pronounced on high-NO_2 days for FVC with a similar pattern for FEV1. Previous experimental studies have shown that traffic-related air pollution can enhance the allergenic potency of pollen through nitration, synergistic oxidative stress, fragmentation or a combination of all previously mentioned [33,36].

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Further, studies of diesel adjuvants have also shown that exhaust particles can increase IgE production and airway inflammation upon allergen co-exposure [37, 38]. These findings indicate that individuals in urban residences with a multiple exposure profile of aeroallergens and pollutants may face compounded respiratory risks.

The study's strengths include its longitudinal design with daily repeated spirometry using standardized home equipment. Further, the daily regional data on temperature and air pollutants allowed us to control environmental confounders. However, this study has limitations worth acknowledging. Despite multiple daily lung function measurements, the modest sample size of only 30 individuals limits statistical power and generalizability. Our use of a single regional pollen monitor may have introduced some exposure misclassification [39], although pollen levels tend to be consistent across urban environments [29]. Further, exposure error may be related to the averaging of weekend values. While Sunday to Tuesday sensitivity tests showed no change in direction or effect size, such averaging may have introduced noise. Clinically, the low magnitude of FEV_1 changes observed may be of limited concern in well managed patients but could become more consequential in those with advanced COPD with severely impaired lung function or multitrigger exacerbations. Finally, our reliance on a single regional monitoring station to estimate ambient pollen exposure may introduce spatial exposure misclassification. High-resolution spatiotemporal pollen models (e.g., 3 km x 3 km grids) were unfortunately not available for our specific study period (2017-2019). Importantly, however, our analysis focuses on acute, short-term health effects, which rely on day-to-day temporal variations in exposure rather than long-term spatial differences. Therefore, a central monitor may adequately capture the day-to-day temporal exposure variability required to assess acute respiratory effects.

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Our findings have significance in the context of climate change. Pollen season are lengthening and pollen concentrations are higher as a result of climate change [15, 16, 17, 18]. For people with COPD and asthma-COPD overlap whose lung function is already impaired, any further obstruction or inflammation caused by pollen exposure could have clinical consequences. Patients with COPD may benefit from awareness of potential aeroallergen risks, particularly during high-pollen days and high pollen days in combination with urban air pollution. For individual patients preventive interventions such as indoor air filtration and possible pharmacological therapies may be beneficial [40].

Conclusion

Short-term increases in ambient pollen exposure were associated with lower FEV_1 among COPD patients. As pollen seasons are lengthening and levels are increasing due to climate change, greater attention to the respiratory effects of pollen on COPD and the evaluation of potential prevention strategies are warranted.

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Table

Table 1. Baseline Characteristics of Study Participants (N = 30)

Characteristic	**Mean ± SD or No. (%)**
Age, years	71.11 (7.98)
BMI, kg/m²	31.84 (7.10)
Sex, n (%)	
Male	13 (43.3)
Female	17 (56.7)
Race, n (%)	
Black, non-Hispanic	5 (18.0)
White, non-Hispanic	25 (82.0)
Education, n (%)	
Up to grade 12 or GED	8 (26.7)
Some college/Associate degree	13 (43.3)
Bachelor’s or above	9 (30.0)
Baseline lung function	
FEV_1, L	1.17 (0.55)
FVC, L	2.39 (0.85)
History of asthma, n (%)	
Yes	15 (51.7)
No	14 (48.3)
Cardiopulmonary comorbidities (CVD), n (%)	
Yes	11 (36.7)
No	19 (63.3)
Type 2 inflammation (blood eosinophils), n (%)	
≥150 cells/μL	12 (60.0)
<150 cells/μL	8 (40.0)
Systemic inflammation (CRP), n (%)	
>3 mg/L	12 (63.2)
≤3 mg/L	7 (36.8)
Inhaled corticosteroids (ICS), n (%)	
Yes	18 (60.0)
No	12 (40.0)
Long-acting β-agonists (LABA), n (%)	
Yes	21 (70.0)
No	9 (30.0)
Long-acting muscarinic antagonists (LAMA), n (%)	

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Characteristic	**Mean ± SD or No. (%)**
Yes	16 (53.3)
No	14 (46.7)
Beta-blockers (BBL), n (%)	
Yes	7 (23.3)
No	23 (76.7)

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Table 2. Exposure descriptive statistics. (n=1,808 observations)

Exposure Variable		**Median**	**IQR**
Total Pollen (1–3 day avg, grains/m^3)	1808	32.4	145.4
Temperature (1–3 day avg, °C)	1808	12.7	15.4
NO_2 (1–3 day avg, ppb)	1808	7.3	3.7
Humidity (1–3 day avg, %)	1808	65.9	16.4

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Figure 1. Difference in FEV1 (mL) associated with IQR Increase in Total Pollen (95% CI)

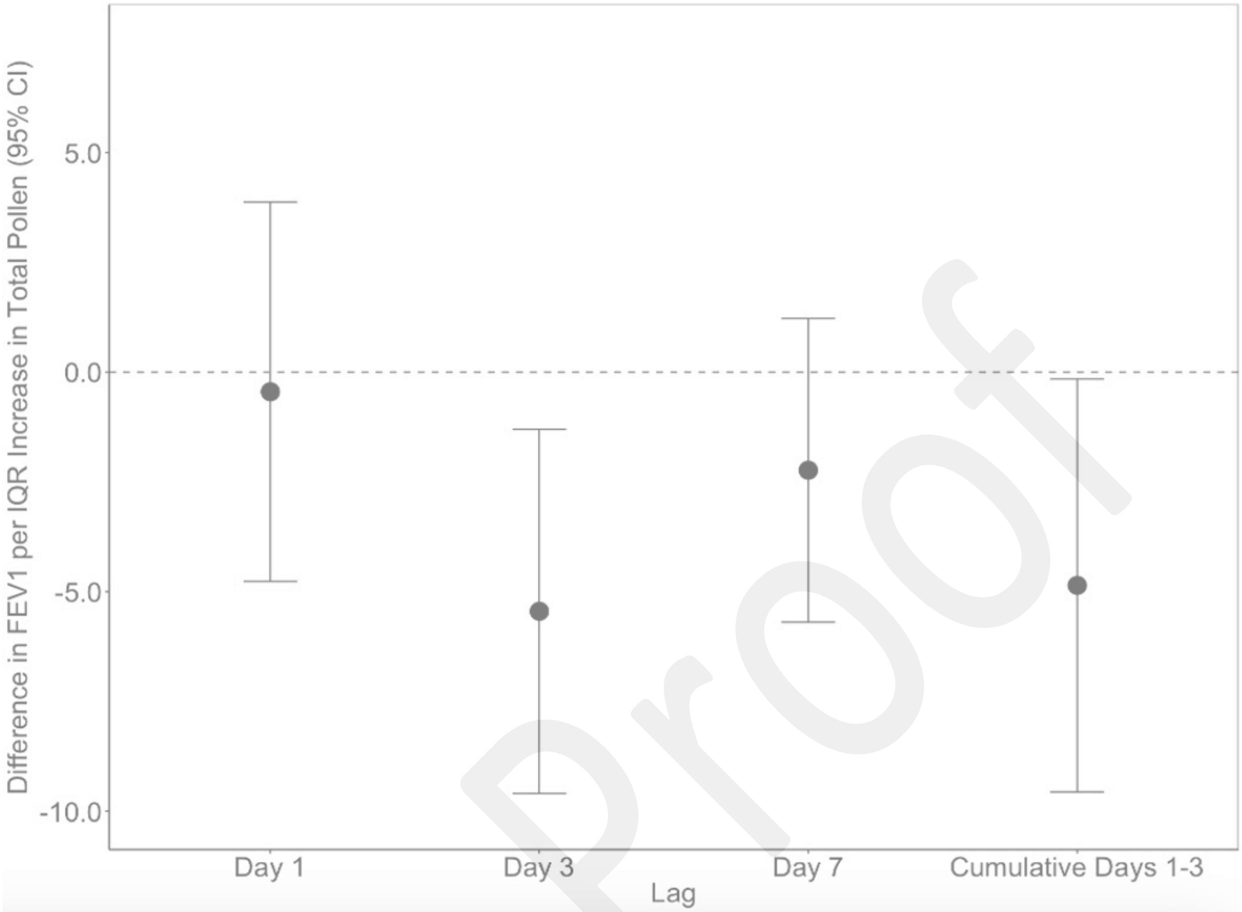

Figure 1 Note: The estimated difference in FEV_1 (mL) associated with an interquartile range (IQR) increase in total ambient pollen. Estimates were generated using GAMMs incorporating a DLNM with linear function. All models were adjusted for baseline age, gender, height, weight, education level, seasonality, and ambient temperature, with random intercepts for participant ID nested within sequence number. Central points represent the estimated difference in FEV_1 (mL), and horizontal error bars represent the 95% confidence intervals (CI).

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Figure 2. Association between lag 3-day pollen exposure and FEV_1 by CRP level, co-morbid asthma, ICS use, and ambient NO2.

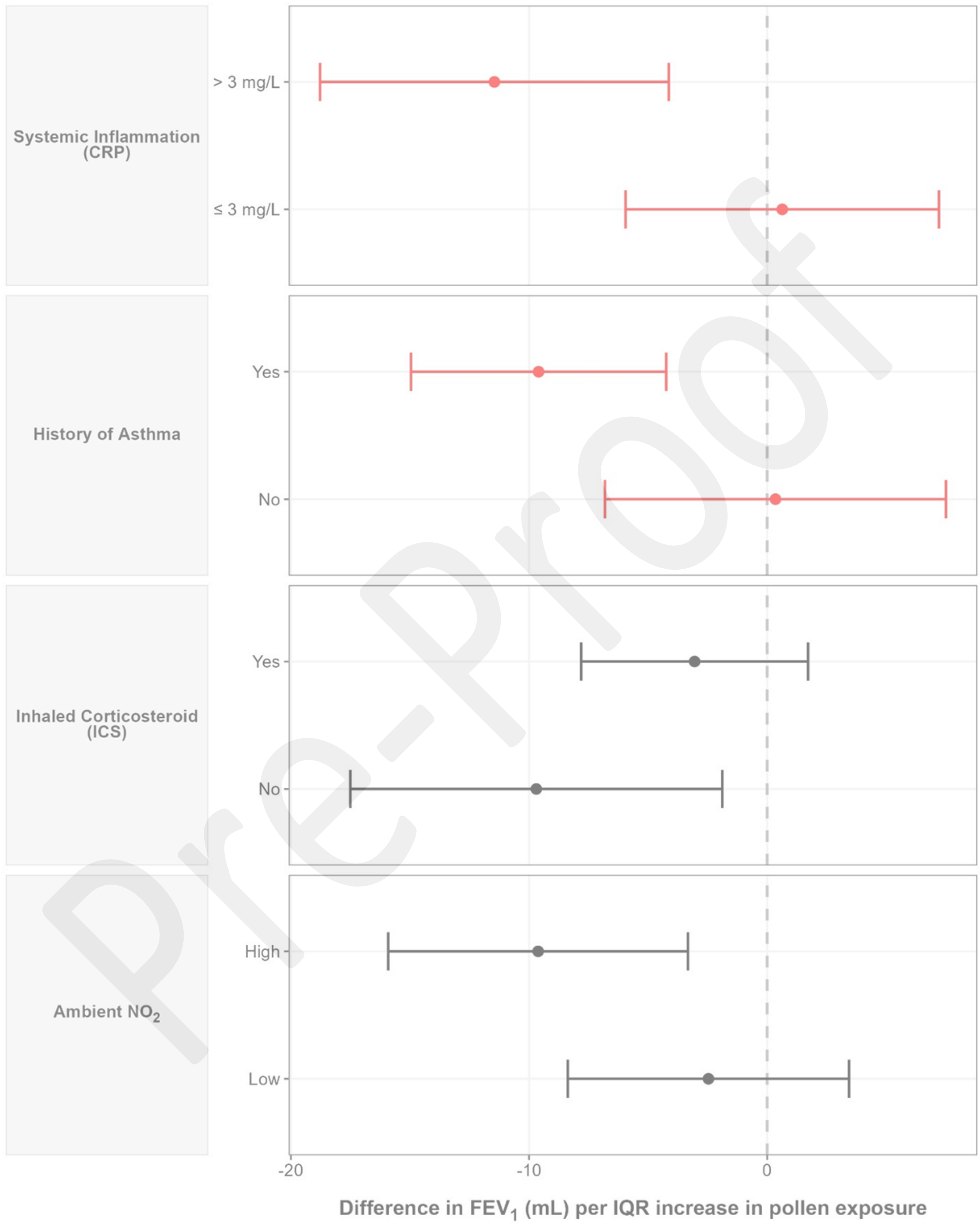

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Figure 2 Note: The estimated cumulative difference in FEV_1 (mL) associated with an interquartile range (IQR) increase in total ambient pollen at lag 3 day. Estimates were generated using GAMMs incorporating a DLNM with linear function. All models were adjusted for baseline age, gender, height, weight, education level, seasonality, and ambient temperature, with random intercepts for participant ID nested within sequence number. Central points represent the estimated difference in FEV_1 (mL), and horizontal error bars represent the 95% confidence intervals (CI). Red coloring indicates a statistically significant difference between the subgroup strata (P-interaction < 0.05).

Pre-Proof

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Online Supplement

Supplemental Table 1. Exposure descriptive statistics

Variable	n	Median	IQR
Total Pollen (1–3 day avg, grains/m³)	1808	32.4	145.4
Temperature (1–3 day avg, °C)	1808	12.7	15.4
NO_2 (1–3 day avg, ppb)	1808	7.3	3.7
Humidity (1–3 day avg, %)	1808	65.9	16.4

Supplemental Table 2: The association of Total Pollen on FVC over lag day 1, 3, 7 and a cumulative 3-day lag.

Exposure	Lag	Change	95% CI
Total Pollen	Day 1	−1.3	(−9.7, 7.1)
Total Pollen	Day 3	−7.9	(−15.9, 0.1)
Total Pollen	Day 7	-1.5	(−4.6, 1.6)
Total Pollen	Days 1–3 Cumulative	−1.9	(−11, 7.1)

Supplemental Table 3: The association of Pollen types on FEV1 over lag day 1, 3, 7 and a cumulative 3-day lag.

Exposure	Lag	Change	95% CI
Grass	Day 1	0.3	(−1.5, 2.2)
Grass	Day 3	0.9	(−0.9, 2.8)
Grass	Day 7	0.5	(−0.3, 1.3)
Other	Day 1	0.2	(−1.6, 2.0)
Other	Day 3	−0.5	(−2.4, 2.5)
Other	Day 7	0.1	(−0.6, 0.9)
Tree	Day 1	-0.8	(−5.1, 3.5)
Tree	Day 3	−5.4	(−9.5, 1.3)
Tree	Day 7	−1.0	(−2.6, 0.6)

Supplemental Table 4: Sensitivity analyses on the association between Total Pollen on FEV1 on a cumulative 3-day lag.

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Confounder	**Lag**	**Change**	**95% CI**
Humidity	Days 1-3 Cumulative	−5.3	(−10.1, −0.6)
NO_2	Days 1-3 Cumulative	−4.8	(−9.6, −0.1)
O_3	Days 1-3 Cumulative	−4.9	(−9.5, −0.2)
$PM_{2.5}$	Days 1-3 Cumulative	−4.5	(−9.2, 0.2)
No Covariates	Days 1-3 Cumulative	−5	(−9.3, −0.7)
Sun-Tues Removed	Days 1-3 Cumulative	−4.6	(-10.6, 1.3)

Note: Humidity, NO_2, O_3, $PM_{2.5}$ were added individually to the pre-existing model to test for confounding. No covariates was a model which only looked at the relationship between pollen and FEV1 without controlling for covariates. Sun-Tues Removed, excluded cumulative weekend values.

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