

Wildfire seasons, prenatal PM_{2.5} exposure, and respiratory infections by age 1 year:

A population-based case-control analysis of critical developmental windows

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Supplementary tables

Table S1. Specific time windows that are critical to significant physiological changes experienced by the developing foetus.

Critical time windows for the development of the Eustachian tube	
1 st stage (0-9 week)	Tubal lumen can be observed.
2 nd stage (10-12 week)	First appearance of the Levator Veli Palatini (LVP) and Tensor Veli Palatini (TVP) muscles.
3 rd stage (13-18 week)	From the pharyngeal region medially, the cartilaginous condensations have spread out to include a region superior and medial to the lumen. There are glandular tissues present. Glandular tissues can be seen.
4 th stage (19-28 week)	The cartilage and paratubal muscle structure are well-formed; the perichondrium developed along the entire tubal length; and the lumens showed differentiation of some seromucous glandular extensions.
5 th stage (>28 weeks)	Continued expansion of the lumen, which is mostly correlated with an extension of the osseous section of the tube.
Critical time windows for the development of the lower respiratory tract	
Embryonic stage (0-7 week)	The trachea and bronchi start to appear; Occurrence of two lungs.
Pseudoglandular stage (8-17 week)	All preacinar airways and blood vessels are formed; The occurrence of centrifugal differentiation of the airway wall structure and epithelium.
Canalicular stage (18-27 week)	Differentiation in type I and type II cells takes place and an alveolar-capillary barrier is formed.
Saccular stage (28-36 week)	Enlargement of the peripheral airways and thinning of the gas-blood barrier.
Alveolar stage (>36 weeks)	Between 100 million and 150 million alveoli are formed; the process of alveologenesis will extend until birth.

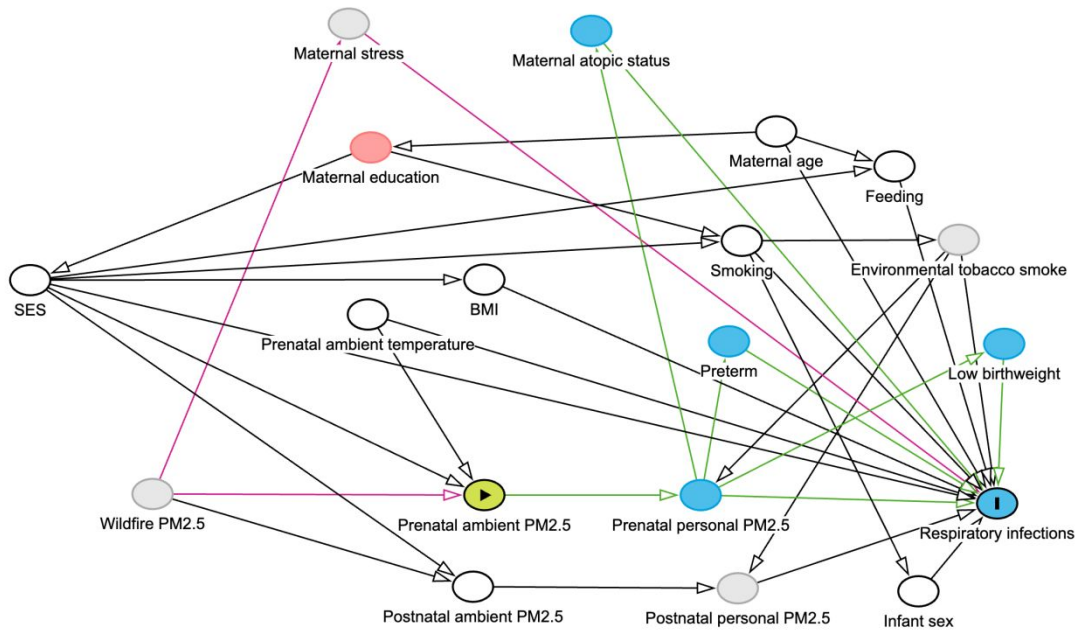


Figure S1. Causal diagram showing the relationship between prenatal exposure to wildfire smoke and respiratory infections (otitis media and lower respiratory tract infection) in the first year of life. The variables in white bubbles were identified as confounders and included in the analyses. Variables in blue bubbles connected green lines were identified as mediators and were not included in the analyses. Maternal education in the red bubble is an unmeasured variable but not a confounder based on the disjunctive cause criterion. Maternal stress, environmental tobacco smoke, and wildfire PM_{2.5} in the grey bubbles are unmeasured confounders. Maternal atopic status in the blue bubble, connected by green lines, is an unmeasured mediator.

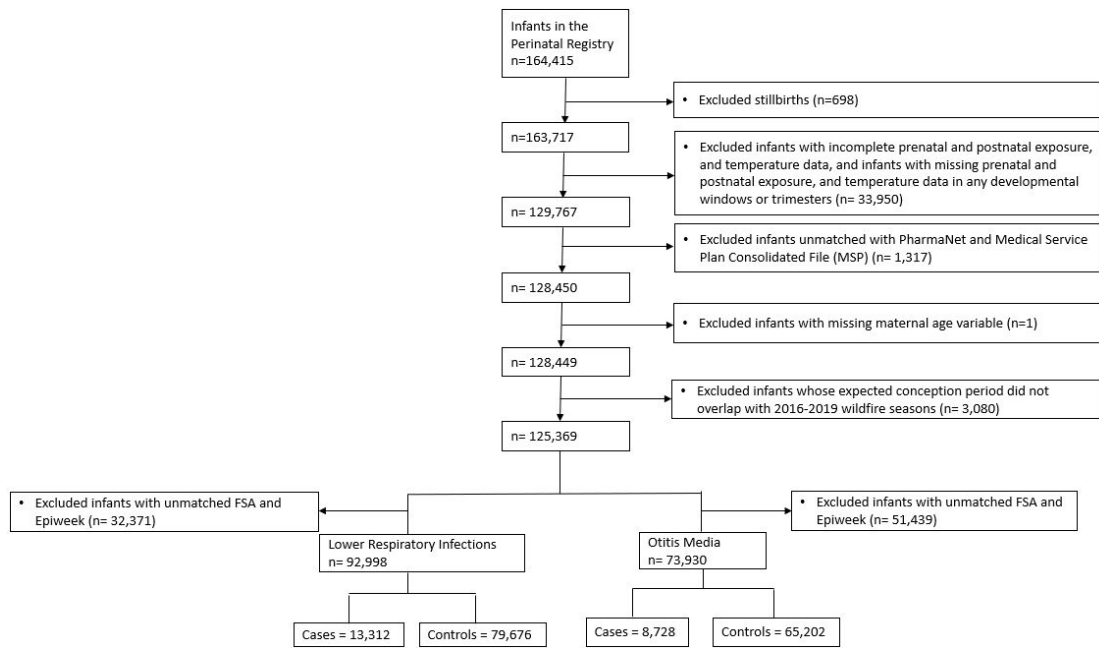


Figure S2. Flowchart for the inclusion and exclusion of infants in the population-based cohort covering all the infants prenatally exposure to wildfire seasons from 2016 to 2019 for sensitivity analyses using alternate residential histories for exposure estimates.

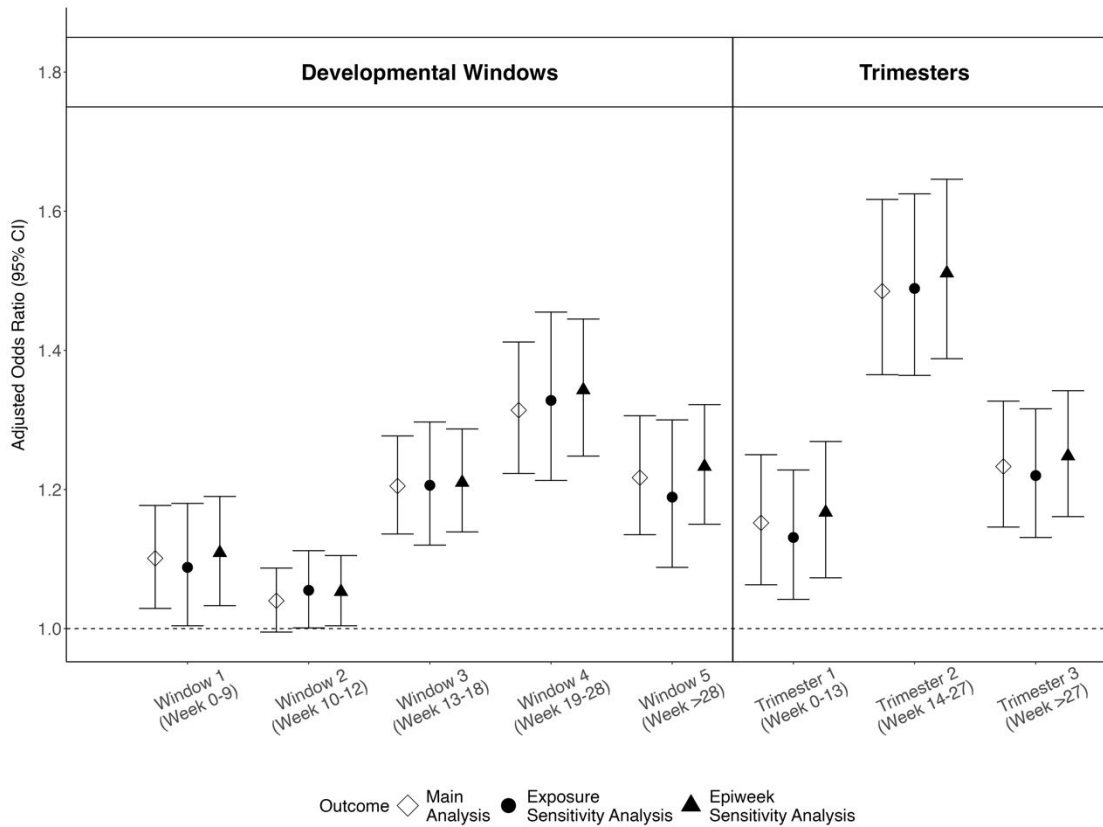


Figure S3. Adjusted odds ratios for prenatal exposure to PM_{2.5} for the main analysis (diamond), the sensitivity analysis using alternate residential histories for exposure estimates (filled circle), and the sensitivity analysis conditioned on the epidemiologic week of conception (filled triangle point up) for all diagnoses of otitis media in the first year of life. Plot shows the effects of exposures during critical windows of Eustachian tube development (left) and trimesters (right).

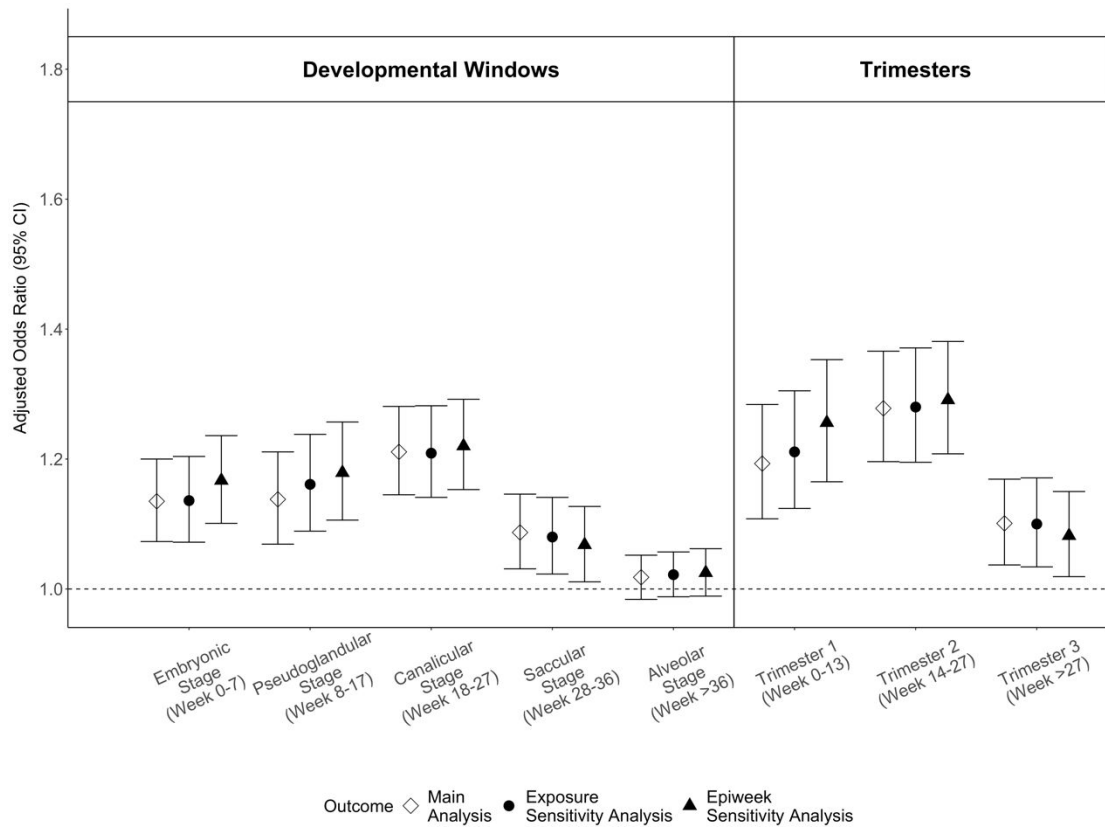


Figure S4. Adjusted odds ratios for prenatal exposure to $PM_{2.5}$ for the main analysis (diamond), the sensitivity analysis using alternate residential histories for exposure estimates (filled circle), and the sensitivity analysis conditioned on the epidemiologic week of conception (filled triangle point up) for all diagnoses of lower respiratory infection in the first year of life. Plot shows the effects of exposures during critical windows of lower respiratory tract development (left) and trimesters (right).