Supplemental Methods

We developed a three-stage population-based model to estimate childhood wheeze/asthma burden attributable to infant respiratory syncytial virus (RSV) lower respiratory tract infection (LRTI) in the United States. The first stage estimated medically attended RSV LRTI encounters across outpatient, emergency department, and hospitalization settings during infancy under three RSV prevention scenarios using age-specific incidence inputs from national surveillance and epidemiologic data and previously validated modeling methods. The second stage derived unique patient-level RSV LRTI episodes from modeled healthcare encounter estimates using national insurance claims data. The third stage estimated wheeze/asthma burden at age 6 under each prevention scenario based on modeled infant RSV LRTI incidence.

Stage 1. RSV LRTI Incidence and Prevention Scenarios

The first stage estimated medically attended RSV LRTI encounters under three RSV prevention scenarios. Population estimates were obtained from recent US Census data,[1] whereas RSV incidence inputs were based on epidemiologic estimates from periods preceding the COVID-19 pandemic to reflect typical RSV circulation patterns.[2-7] We limited analyses to RSV LRTI because it has the strongest and most consistently reported association with subsequent childhood wheeze/asthma, supported by longitudinal cohort studies, mechanistic data, and systematic reviews.[8-14] Because any-severity RSV illness during infancy has also been associated with subsequent wheeze/asthma,[10] restricting analyses to medically attended RSV LRTI may underestimate the total RSV-attributable burden. However, prospective cohort studies have shown a severity-dependent relationship, with more severe illness associated with greater subsequent risk.[10,15]

Monthly RSV LRTI episode estimates for an annual US birth cohort were generated using age-specific incidence inputs from national surveillance and epidemiologic studies and previously validated modeling methods developed for evaluating infant RSV prevention strategies in the United States.[16,17] The model incorporated 2021 US birth cohort estimates and age-specific RSV episode estimates derived from national surveillance and epidemiologic studies.[3,5,6,18] We assumed a uniform monthly distribution of births. RSV-related medically attended encounters were modeled separately across outpatient, emergency department, and hospitalization settings using prospective population-based surveillance studies and CDC New Vaccine Surveillance Network data spanning 2002–2020.[3,6] Seasonal variation in RSV circulation was informed by National Respiratory and Enteric Virus Surveillance System (NREVSS) data from 2015–2019.[4]

Incidence and seasonal inputs were derived from prepandemic surveillance data to reflect established, stable RSV circulation patterns. Recent NREVSS data indicate that RSV seasonality has largely returned toward prepandemic norms following pandemic-era disruption,[2,19] and the age distribution of severe RSV illness driving infant LRTI incidence in our model remains consistent with prepandemic patterns.[7] Modeled age- and setting-specific probabilities were used to estimate the proportion of medically attended RSV encounters classified as RSV LRTI.[20] Repeated RSV LRTI episodes within the same RSV season were not modeled.

Next, we modeled RSV LRTI incidence within this cohort under three prevention scenarios: 1) no RSV prevention; 2) infant RSV immunoprophylaxis program using nirsevimab; and 3) a counterfactual scenario assuming complete prevention of RSV LRTI. The no RSV LRTI prevention scenario served as the baseline comparator. Nirsevimab was selected as an illustrative prevention strategy because robust clinical trial efficacy and real-world effectiveness data are available. Real-world effectiveness estimates closely align with clinical trial efficacy.[21-23] Optimization of RSV prevention strategies, including comparative effectiveness across products or coverage scenarios, was beyond the scope of this analysis. The nirsevimab scenario incorporated seasonal administration assumptions corresponding to 50% immunoprophylaxis coverage, selected to approximate overall US infant RSV protection coverage during the 2025–26 season.[24] Seasonal administration assumptions followed the previously published model framework,[16] with infants born outside the RSV season receiving protection timed to routine well-child visits before the subsequent RSV season. Modeled nirsevimab efficacy was derived from clinical trial data,[21,22] following a sigmoidal waning function from the time of administration, with effectiveness against RSV LRTI remaining approximately 79% during the first 5 months after administration and declining progressively thereafter to 0% by month 10. The counterfactual scenario assumed complete prevention of medically attended RSV LRTI and was included to estimate the corresponding attributable wheeze/asthma burden.

Stage 2. Derivation of Unique RSV LRTI Episodes

The second stage derived unique patient-level RSV LRTI episodes from modeled healthcare encounter estimates. Because RSV episode estimates were modeled separately across outpatient, emergency department, and hospitalization settings, encounter-level estimates were adjusted to account for multiple healthcare encounters associated with the same illness episode. RSV LRTI encounters were summed across all healthcare settings during the first year of life. The relationship between healthcare encounters and unique RSV LRTI episodes was

derived from a retrospective observational study of healthcare utilization during acute RSV LRTI episodes among US infants during their first RSV season.[25] Using outpatient, emergency department, and hospitalization encounters identified from the Merative MarketScan Commercial Claims and Encounters and MarketScan Multistate Medicaid databases, that study estimated healthcare encounter-to-episode ratios separately for commercially insured and Medicaid-insured infants.[25] We weighted these ratios using national estimates of insurance coverage among US children[26] to derive a correction factor. The resulting correction factor was applied to modeled healthcare encounters to estimate unique RSV LRTI episodes:

$$Cf = (Vc/Ec) \times Pc + (Vm/Em) \times Pm$$

where Cf is the weighted encounter-to-episode correction factor; Vc and Ec denote the numbers of healthcare encounters and unique RSV LRTI episodes, respectively, in the MarketScan Commercial Claims and Encounters database; Pc is the proportion of US children with commercial insurance; Vm and Em denote the numbers of healthcare encounters and unique RSV LRTI episodes, respectively, in the MarketScan Multistate Medicaid database; and Pm is the proportion of US children with Medicaid insurance.

Across 239,813 RSV LRTI episodes and 363,833 associated healthcare encounters, 65.3% of medically attended RSV LRTI encounters corresponded to unique RSV LRTI episodes. Because the encounter-to-episode relationship was derived from healthcare utilization data during the first RSV season of life, analyses were restricted to RSV LRTI exposures occurring during infancy.

Stage 3. Wheeze/Asthma Outcomes and RSV-Attributable Burden

The third stage of the model estimated wheeze/asthma outcomes at age 6 associated with infant RSV LRTI exposure. Age 6 was selected because both national wheeze/asthma prevalence estimates and published effect estimates relating infant RSV LRTI to subsequent wheeze/asthma were available at this age.[27] The association between infant RSV LRTI and subsequent wheeze/asthma was obtained from a systematic review and meta-analysis of 35 studies of laboratory-confirmed RSV LRTI, most involving medically attended illness.[8] We selected the adjusted odds ratio estimate of 2.45 (95% CI 1.23–4.88) from analyses accounting for genetic influences and family predisposition to wheezing illness (77 effect estimates), representing the most conservative summary estimate reported by the investigators. Adjustment for these factors reduces the potential for confounding by shared respiratory vulnerability.

Baseline wheeze/asthma prevalence at age 6 was obtained from CDC national asthma surveillance estimates, which are derived from nationally representative household surveys of the US population.[27] For children aged 6 years, asthma prevalence was defined from parent or guardian responses indicating that a healthcare provider had ever diagnosed the child with asthma and that the child still had asthma at the time of the survey. Estimates of RSV LRTI during infancy were linked to wheeze/asthma outcomes at age 6 within the modeled birth cohort. Mortality adjustments were not applied because cumulative mortality before age 6 represented less than 1% of the modeled birth cohort.

Because attributable burden calculations require a relative risk, and no suitable external estimate of wheeze/asthma prevalence among children without prior RSV LRTI was identified, wheeze/asthma prevalence among RSV-exposed and RSV-unexposed children was estimated simultaneously by solving the reported odds ratio relationship together with the national wheeze/asthma prevalence estimate at age 6.[8] Wheeze/asthma prevalence among children without prior RSV LRTI (P0) and among children with prior RSV LRTI (P1) were estimated simultaneously by solving the standard odds ratio relationship:

$$OR = [P1/(1 - P1)] / [P0/(1 - P0)]$$

together with the equation describing the overall national wheeze/asthma prevalence:

$$PrevalenceTotal = [(P1 \times PopulationRSV) + (P0 \times PopulationNoRSV)] / PopulationTotal$$

where PrevalenceTotal is the observed prevalence of wheeze/asthma at age 6, PopulationTotal is the total modeled population surviving to age 6, PopulationRSV is the number of children with prior RSV LRTI, and PopulationNoRSV is the number of children without prior RSV LRTI.

For the no-prevention scenario, PrevalenceTotal was 8.8%, PopulationTotal was 3,664,292, PopulationRSV was 379,659, and PopulationNoRSV was 3,281,111. For the no-prevention scenario, substitution of the model parameters into these equations yielded wheeze/asthma prevalences of 7.8% among children without prior RSV LRTI and 17.3% among children with prior RSV LRTI.

The corresponding relative risk was derived from the adjusted odds ratio using:

$$RR = OR / [1 - P0 + (P0 \times OR)]$$

where OR is the adjusted odds ratio reported in the meta-analysis and P0 is the estimated wheeze/asthma prevalence among children without prior RSV LRTI.[28] Using P0 = 7.8%, the

corresponding relative risk was 2.21. This relative risk was used in all subsequent burden calculations. Wheeze/asthma prevalence among children with and without RSV LRTI exposure was then estimated under each prevention scenario using the derived relative risk and modeled RSV LRTI incidence.

For each prevention scenario, prevalent wheeze/asthma cases at age 6 were estimated as:

$$\text{Cases} = (P1 \times \text{PopulationRSV}) + (P0 \times \text{PopulationNoRSV})$$

Overall wheeze/asthma prevalence was calculated by dividing total prevalent cases by the total population surviving to age 6.

RSV-attributable wheeze/asthma burden was estimated by comparing modeled wheeze/asthma prevalence under the no-prevention scenario with modeled prevalence under complete prevention of RSV LRTI. This counterfactual framework is similar to that used in a previous model-based analysis of RSV-attributable wheeze/asthma burden.[29] The primary outcome, the population attributable fraction (PAF), was calculated as the proportional reduction in all-cause wheeze/asthma prevalence between the no-prevention scenario and the complete-prevention counterfactual scenario:[30]

$$\text{PAF} = (\text{CasesNoPrevention} - \text{CasesCompletePrevention}) / \text{CasesNoPrevention}$$

Absolute wheeze/asthma case counts, prevalence per 100,000 children, and relative reductions between prevention scenarios were calculated.

Uncertainty Analysis

Uncertainty was incorporated using probabilistic Monte Carlo simulation with 1,000 independent trials. For each simulation, model input parameters were sampled from probability distributions parameterized using published point estimates and corresponding 95% confidence intervals (CIs). Sampled values were propagated through all stages of the model to generate distributions of RSV LRTI incidence, wheeze/asthma prevalence, and RSV-attributable burden. Ninety-five percent uncertainty intervals were defined as the 2.5th and 97.5th percentiles of simulated values. The proportion of healthcare encounters representing unique RSV LRTI episodes was modeled using a beta distribution derived from Merative MarketScan Commercial Claims and Encounters and MarketScan Multistate Medicaid database estimates.[25] Parameter values and distributional assumptions are summarized in Supplemental Table 1.

Contextualization of RSV-Attributable Burden

To contextualize our PAF estimate, we identified potentially modifiable childhood asthma risk factors from a published review summarizing systematic reviews and meta-analyses and paired pooled odds ratio estimates with US exposure prevalence estimates from national databases and published literature.[31] For each risk factor, relative risks were derived from the reported odds ratios and exposure prevalence using the same calibration approach applied in the primary RSV analysis. We then calculated crude PAF estimates and 95% CIs using the calibrated relative risks according to the standard population attributable fraction formula:[32]

$$PAF = P \times (RR - 1) / (1 + P \times [RR - 1])$$

where P is the prevalence of risk factor exposure and RR is the calibrated relative risk. Comparator PAFs were calculated for contextualization only, were not incorporated into the wheeze/asthma model, were derived from heterogeneous literature sources, are unadjusted, and assume independence among risk factors. Source data and calculated estimates are provided in Supplemental Table 2. Analyses were performed using R version 4.4.1 (R Foundation for Statistical Computing, Vienna, Austria).

Research Ethics

Because this study used publicly available aggregate data without direct human participant involvement, institutional review board approval was not required.

Supplemental Table 1. Parameters Included in Probabilistic Uncertainty Analysis

Parameter	Point Estimate and Rationale	95% CI/UI	Distribution	Source
RSV hospitalization incidence	4.0 per 1,000 children; prospective surveillance studies, children <5 years, 2016–2020; monthly age-specific rates, highest at 1 month declining thereafter. See source data for month-by-month base case rate.	3.8–4.1 per 1,000; See source data for month-by-month CI	Lognormal	3,16
RSV emergency department incidence	Age-specific rates modeled separately for 0–5 months and 6–11 months; prospective population-based surveillance, children <5 years, 2002–2004. From 0–5 months 7,500 and from 6–11 months 5,800 per 100,000 children.	1–5 mo: 5,500–7,500 per 100,000; 6–11 mo: 5,700–5,800 per 100,000	Lognormal	5,16
RSV outpatient incidence	Age-specific rates modeled separately for 0–5 months and 6–11 months; prospective population-based surveillance, children <5 years, 2002–2004. From 0–5 months 21,600 and from 6–11 months 24,600 per 100,000 children.	1–5 mo: 13,200–21,600 per 100,000; 6–11 mo: 17,700–24,600 per 100,000	Lognormal	5,6
Probability that a medically attended RSV encounter represented RSV LRTI	All hospitalized encounters assumed RSV LRTI; age- and setting-specific probabilities for outpatient and ED encounters from previously published model. For outpatient encounters: 0.65 from 0–5 months and 0.3 from 6–11 months. For ED encounters: 0.65 from 0–5 months and 0.5 from 6–11 months	ED – 0–11 mo: 0.25–1.0; Outpatient: 0–5 mo: 0.25–1.0, 6–11 mo: 0.1–1.0	Beta	16
Baseline wheeze/asthma prevalence at age 6	8.8%; CDC survey-based estimate of current asthma/wheeze prevalence among US children	8.1–9.6%	Lognormal	27
RSV-associated wheeze/asthma effect estimate (adjusted OR)	2.45; systematic review and meta-analysis of laboratory-confirmed RSV LRTI, studies adjusting for genetic predisposition and major confounders	1.23–4.88	Lognormal	8
Estimated wheeze/asthma prevalence without prior RSV LRTI (P0)	7.8%; derived from the odds ratio relationship and national wheeze/asthma prevalence estimate	6.7–8.9	Derived	--
Estimated wheeze/asthma prevalence with prior RSV LRTI (P1)	17.3%; derived from the same calibration equations as P0	10.3–26.8	Derived	--
Relative risk of wheeze/asthma following RSV LRTI	2.21; derived from the adjusted odds ratio and estimated P0	1.20–3.83	Derived	--
US population and birth cohort size	Total population 331,893,745; annual birth cohort 3,664,292 infants	N/A	Fixed	1,18
Nirsevimab effectiveness against RSV LRTI	79.0% during months 0–5; sigmoidal decline to 25.0% by day 150, 0% by month 10; derived from pooled results of phase 2b and phase 3 clinical trial data	1–5 mo: 68.5–86.1%; 6–10 mo: 0.0–50.0%	Beta	21,22
Proportion of medically attended RSV encounters representing unique RSV LRTI episodes	0.653; derived from MarketScan Commercial Claims and Multistate Medicaid analyses; 65.3% of medically attended RSV encounters represented unique RSV LRTI episodes	0.651–0.655	Beta	25
Insurance status of children	Commercial insurance: 49.1%; Medicaid: 37.4%. Weighted: Commercial insurance: 56.8%; Medicaid: 43.2%.	N/A	N/A	26

CI = confidence interval; UI = uncertainty interval; OR = odds ratio; RR = relative risk; ED = emergency department; mo = months; RSV = respiratory syncytial virus; LRTI = lower respiratory tract infection. P0 and P1 denote estimated wheeze/asthma prevalences among children without and with prior RSV LRTI, respectively. RR was derived from the adjusted OR and estimated P0.

Supplemental Table 2. Estimated Population Attributable Fractions for Selected Modifiable Risk Factors Associated with Childhood Asthma

Risk Factor	Exposure Window	Prevalence (%)	Odds ratio (95% CI)	PAF using OR (95% CI)	Relative risk (95% CI)	PAF using calibrated RR (95% CI)
Cesarean section	Prenatal	32.0%	1.16 (1.14–1.29)	4.9% (4.3–8.5%)	1.14 (1.13–1.26)	4.3% (4.0–7.7%)
Maternal smoking, prenatal	Prenatal	8.4%	1.85 (1.35–2.53)	6.7% (2.9–11.4%)	1.73 (1.31–2.26)	5.8% (2.5–9.6%)
Antibiotic use, infant	Age 0–1 year	66.0%	1.27 (1.12–1.43)	15.1% (7.3–22.1%)	1.24 (1.11–1.39)	13.7% (6.8–20.5%)
Allergic rhinitis, mold	Age <5 years	4.3%	1.09 (0.90–1.32)	0.4% (−0.4–1.4%)	1.08 (0.91–1.28)	0.3% (−0.4–1.2%)
Food sensitization, age ≤2 years	Age <5 years	5.8%	2.80 (2.10–3.90)	9.4% (6.0–14.4%)	2.44 (1.92–3.18)	7.7% (5.1–11.2%)
Obesity, BMI >95th percentile	Age <5 years	9.4%	1.46 (1.36–1.57)	4.1% (3.3–5.1%)	1.41 (1.32–1.50)	3.7% (2.9–4.5%)
Overweight, BMI 85–94th percentile	Age <5 years	13.9%	1.23 (1.17–1.29)	3.1% (2.3–3.9%)	1.21 (1.15–1.26)	2.8% (2.0–3.5%)
Traffic pollution, black carbon	Age <5 years	3.8%	1.20 (1.05–1.38)	0.8% (0.2–1.4%)	1.18 (1.05–1.34)	0.7% (0.2–1.3%)
Traffic pollution, nitrogen dioxide	Age <5 years	3.8%	1.09 (0.96–1.23)	0.3% (−0.2–0.9%)	1.08 (0.96–1.21)	0.3% (−0.2–0.8%)
Traffic pollution, particulate matter	Age <5 years	3.8%	1.14 (1.00–1.30)	0.5% (0.0–1.1%)	1.13 (1.00–1.27)	0.5% (0.0–1.0%)
Gas stove cooking	Age 5–13 years	9.3%	1.32 (1.18–1.48)	2.9% (1.6–4.3%)	1.28 (1.16–1.42)	2.5% (1.5–3.8%)
Physical activity, inadequate	Age 5–13 years	57.4%	1.32 (0.95–1.84)	15.5% (−3.0–32.6%)	1.29 (0.95–1.75)	14.3% (−3.0–30.1%)
Secondhand smoke exposure	Age 5–13 years	40.6%	1.32 (1.23–1.42)	11.5% (8.5–14.6%)	1.29 (1.21–1.38)	10.5% (7.9–13.4%)

Prevalence and effect estimates were obtained from published literature sources.[31] Comparator effect estimates were drawn from systematic reviews and meta-analyses and paired with US exposure prevalence estimates obtained from national databases and published literature. Population attributable fractions (PAFs) were calculated from exposure prevalence and calibrated relative risks. Relative risks were derived from reported odds ratios using the same calibration approach applied in the primary RSV analysis, and the PAF estimates presented in Figure 2 were calculated using the calibrated relative risks. Comparator PAFs were calculated for contextualization only, were derived from heterogeneous literature sources, are unadjusted, assume independence among risk factors, and should not be summed. Negative lower confidence bounds reflect statistical uncertainty when the underlying effect estimate confidence interval includes the null value and were not truncated at zero.

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