

S1. Supplementary Material

420 S1.1. Measles Cases Data

Table S1: Cumulative numbers of reported measles cases by age group on those specific days.²⁹

Date	Day	C_{0-4}	C_{5-17}	C_{18+}	Total
2025-01-23	0	0	2	0	2
2025-02-05	13	0	6	0	6
2025-02-14	22	13	29	5	47
2025-02-18	26	15	33	6	54
2025-02-21	29	26	51	10	87
2025-02-26	34	32	59	13	104
2025-03-03	39	37	66	15	118
2025-03-11	47	55	84	20	159
2025-03-14	50	62	90	21	173
2025-03-18	54	67	99	22	188
2025-03-21	57	69	102	24	195
2025-03-25	61	73	107	24	204
2025-03-28	64	75	113	25	213
2025-04-01	68	79	118	27	224
2025-04-04	71	82	121	28	231
2025-04-08	75	87	129	30	246
2025-04-11	78	90	133	31	254
2025-04-15	82	93	136	33	262
2025-04-18	85	98	138	34	270
2025-04-21	88	101	140	36	277
2025-04-25	92	191	243	187	621
2025-04-29	96	200	245	194	639
2025-05-02	99	201	247	210	658
2025-05-06	103	207	255	219	681
2025-05-09	106	210	257	223	690
2025-05-13	110	211	273	229	713
2025-05-16	113	211	273	230	714
2025-05-20	117	213	272	233	718
2025-05-23	120	214	273	237	724

Date	Day	C_{0-4}	C_{5-17}	C_{18+}	Total
2025-05-27	124	214	273	238	725
2025-05-30	127	215	280	239	734
2025-06-03	131	218	281	239	738
2025-06-06	134	218	281	239	738
2025-06-10	138	218	281	241	740
2025-06-17	145	219	284	243	746

S1.2. Details about the Mathematical Model and its Assumptions

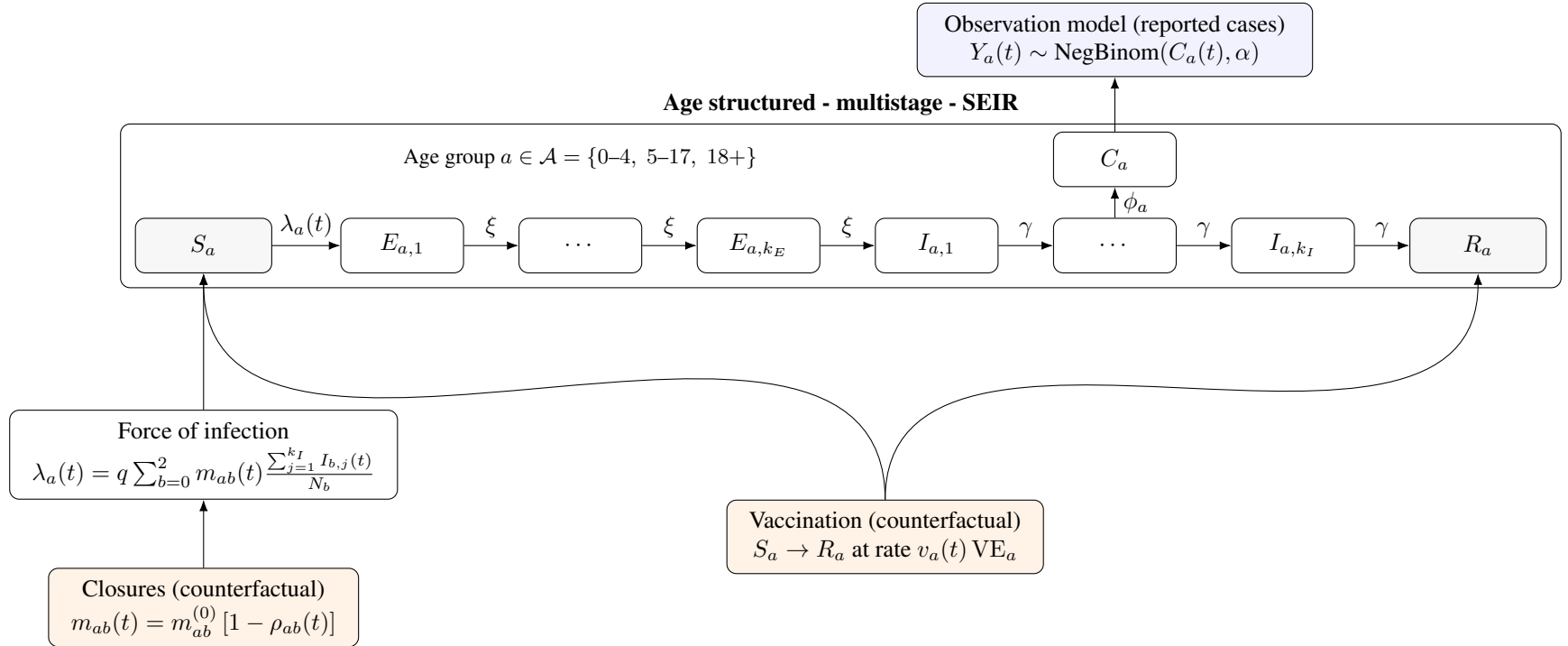


Figure S1: Schematic illustration of the age-structured multistage SEIR transmission model with an explicit observation model and intervention mechanisms.

Table S2: The main modeling assumptions.

Component	Assumption
Population age groups	The population is stratified into 3 age groups: 0–4, 5–17, and 18+ years. Within each age group, contact is homogeneous.
Spatial aggregation	The main model is fit to aggregated data over the 37 focus counties in Texas.
Transmission mechanism	Infections arise via frequency-dependent transmission with age-specific mixing described by a contact matrix $M(t) = [m_{ab}(t)]$, and a per-contact transmission probability q is estimated.
Contact structure	Contact is assumed to be homogeneous over Texas. Baseline contacts are fixed to an external benchmark matrix adapted from ¹⁴ and re-aggregated to match the analysis age groups. Reciprocity is enforced during re-aggregation so that contacts between groups satisfy $N_a m_{ab} = N_b m_{ba}$.
Natural history (latent and infectious periods)	Latent and infectious dwell times follow Erlang (gamma) distributions, with k_E exposed stages and k_I infectious stages, using the linear-chain trick. The numbers k_E and k_I are selected based on the Leave-One-Out cross-validation predictive performance measure. Mean durations are governed by ξ and γ and are not fixed to single values.
Initial immunity $R_a(0)$	Baseline immunity by age is unknown and treated as a parameter with informative bounds; for children, upper bounds are informed by school vaccine coverage (Kindergarten and 7th grade) and vaccine efficacy assumptions.
Vaccine efficacy	MMR vaccine efficacy against infection is fixed.
Interventions (closures)	Daycare/school closures are modeled as time-varying reductions in specific elements of $M(t)$ (age-to-age contacts), with the intensity and duration specified by scenarios. Closure durations are multiples of the measles generation time.
Interventions (vaccination)	MMR vaccines are administered only once during the reactive vaccination campaign.

S1.3. Details about the Bayesian Estimation

Age-specific Contact Matrix

We model age-structured transmission using an explicit age-specific contact matrix. Let the model age groups be

$$\mathcal{A} = \{0, 1, 2\} = \{0-4, 5-17, 18+\}.$$

We start from the Texas benchmark contact matrix reported by,¹⁴ which is defined on the age partition

$$\mathcal{B} = \{0, 1, 2\} = \{0-4, 5-19, 20+\}.$$

Let $M^{\mathcal{B}} \in \mathbb{R}_+^{3 \times 3}$ denote the contact matrix in,¹⁴ where $M_{ij}^{\mathcal{B}}$ is the expected number of (potentially infectious) contacts per day that an individual in age group $i \in \mathcal{B}$ makes with individuals in age group $j \in \mathcal{B}$. In,¹⁴

$$M^{\mathcal{B}} = \begin{pmatrix} 1.4101 & 2.5282 & 4.9100 \\ 0.9290 & 11.0091 & 6.0771 \\ 0.4884 & 1.6387 & 8.8461 \end{pmatrix},$$

with rows indexing the age group of the *contact-maker* and columns indexing the age group of the *contactee*.

425 The case data used for our model fitting are provided in the age groups 0–4, 5–17, and 18+, whereas $M^{\mathcal{B}}$ is specified for 0–4, 5–19, and 20+. To ensure that the force of infection and all counterfactual intervention effects are

computed using the same age partition as the observed data, we convert $M^{\mathcal{B}}$ to a matrix M defined on $\mathcal{A}$.

The only mismatch is that¹⁴ aggregate ages 5–19, while our model separates 18–19 from 5–17. We resolve this by splitting contacts involving the middle group (5–19) in $\mathcal{B}$ proportionally using single-year-of-age population counts for 2024. Let N_a denote the population size of exact age a (with $a = 0, 1, \dots, 95$ and $95+$ treated as $a = 95$).¹² Define

$$N_{5-17} = \sum_{a=5}^{17} N_a, \quad N_{18-19} = \sum_{a=18}^{19} N_a, \quad N_{5-19} = N_{5-17} + N_{18-19}.$$

We then define the splitting proportions

$$\pi_{5-17} = \frac{N_{5-17}}{N_{5-19}}, \quad \pi_{18-19} = \frac{N_{18-19}}{N_{5-19}} = 1 - \pi_{5-17}.$$

These proportions can be computed at the county level and aggregated to obtain a single set consistent with the spatial aggregation of the case data.

430 We define $M \in \mathbb{R}_+^{3 \times 3}$ with indices of the row 0–4 in M with the split as follows:

$$\begin{aligned} M_{0-4, 0-4} &= M_{0-4, 0-4}^{\mathcal{B}}, \\ M_{0-4, 5-17} &= \pi_{5-17} M_{0-4, 5-19}^{\mathcal{B}}, \\ M_{0-4, 18+} &= \pi_{18-19} M_{0-4, 5-19}^{\mathcal{B}} + M_{0-4, 20+}^{\mathcal{B}}, \end{aligned}$$

For the row corresponding to 5–17, we take it to be the same as the 5–19 row in $M^{\mathcal{B}}$ as follows:

$$\begin{aligned} M_{5-17, 0-4} &= M_{5-19, 0-4}^{\mathcal{B}}, \\ M_{5-17, 5-17} &= \pi_{5-17} M_{5-19, 5-19}^{\mathcal{B}}, \\ M_{5-17, 18+} &= \pi_{18-19} M_{5-19, 5-19}^{\mathcal{B}} + M_{5-19, 20+}^{\mathcal{B}}. \end{aligned}$$

Finally, the adult row of (18+) in M is derived from the adult row of (20+) in $M^{\mathcal{B}}$ plus the contribution of ages 18–19. Since ages 18–19 are included within our adult group, we incorporate their contacts by adding a proportion of the $M^{\mathcal{B}}$'s middle row:

$$\begin{aligned} M_{18+, 0-4} &= \pi_{18-19} M_{5-19, 0-4}^{\mathcal{B}} + M_{20+, 0-4}^{\mathcal{B}}, \\ M_{18+, 5-17} &= \pi_{5-17} M_{20+, 5-19}^{\mathcal{B}} + \pi_{18-19} \pi_{5-17} M_{5-19, 5-19}^{\mathcal{B}}, \\ M_{18+, 18+} &= \pi_{18-19} M_{20+, 5-19}^{\mathcal{B}} + \pi_{18-19} M_{5-19, 20+}^{\mathcal{B}} + \pi_{18-19}^2 M_{5-19, 5-19}^{\mathcal{B}} + M_{20+, 20+}^{\mathcal{B}}. \end{aligned}$$

The result is

$$M = \begin{pmatrix} 1.4101 & 2.4226239 & 5.7905445 \\ 0.8314527 & 9.311699 & 7.554599 \\ 0.46766597 & 1.7777724 & 9.041952 \end{pmatrix}$$

435 which satisfies reciprocity.

Bounds and priors for initial immunity $R_a(0)$

Let $R_a(0)$ denote the fraction immune at time $t = 0$ in age group $a \in \mathcal{A} = \{0-4, 5-17, 18+\}$. Because measles transmission dynamics and the inferred effective reproduction number are sensitive to the initial immune fraction, we use county-level school immunization coverage data to construct empirically grounded *upper bounds* for $R_a(0)$ in
 440 infants and children, while keeping adult immunity weakly constrained.¹³

We use reported county-level MMR vaccination coverage for (i) kindergarten (KG) and (ii) 7th grade for years 2019–2024, see Figures S2 and S3 made based on DSHS Texas School Coverage data.¹³ Let $c_{y,k}^{(\text{KG})}$ and $c_{y,k}^{(7)}$ denote the reported coverage proportions in county k and year y for KG and 7th grade, respectively. To aggregate the coverage over years, we summarize the six-year coverage histories by the geometric mean

$$\bar{c}_k^{(\text{KG})} = \left(\prod_{y=2019}^{2024} c_{y,k}^{(\text{KG})} \right)^{\frac{1}{6}}, \quad \bar{c}_k^{(7)} = \left(\prod_{y=2019}^{2024} c_{y,k}^{(7)} \right)^{\frac{1}{6}}$$

since geometric mean is the most appropriate in aggregating fractions and probabilities, as it is sensitive to low values.

We treat school coverage as an empirical upper bound on vaccine-induced immunity for children, adjusted by vaccine effectiveness. Let VE denote the effectiveness against infection for a vaccinated child, which is $\text{VE}_1 = 0.93$ for one-dose MMR and $\text{VE}_2 = 0.97$ for two-dose MMR. We define county-specific upper bounds for immunity in the
 445 infants and children age groups:

$$U_{k,0-4} = \min\{1, \text{VE}_1 \bar{c}_k^{(\text{KG})}\}, \quad U_{k,5-17} = \min\{1, \text{VE}_2 \bar{c}_k^{(7)}\}.$$

Since the measles case data are spatially aggregated across the 37 counties, we convert county-level bounds to aggregated bounds via weighted averages based on 2024 age-specific population sizes.¹² In particular, let $N_{k,a}$ denote the 2024 population size of age group a in county k . The aggregated upper bounds are

$$U_a = \frac{\sum_k N_{k,a} U_{k,a}}{\sum_k N_{k,a}}, \quad a \in \{0-4, 5-17\}.$$

In conclusion, U_a is found to be 0.8834 for infants and 0.9516 for children. The lower boundaries L_a are chosen to be broad to avoid over-constraining inference ($L_a = 0.40$ for all a).

For adults in the age group 18+, prior immunity is expected to be high but heterogeneous; we use a weakly informative upper bound U_{18+} close to 1 ($U_{18+} = 0.99$) and again a comparatively broad lower bound.

We use independent priors for each age group:

$$R_a(0)/N_a \sim \text{Uniform}(L_a, U_a), \quad a \in \mathcal{A} = \{0-4, 5-17, 18+\},$$

450 where U_a and L_a are as defined above.

MMR Vaccine Coverage Maps

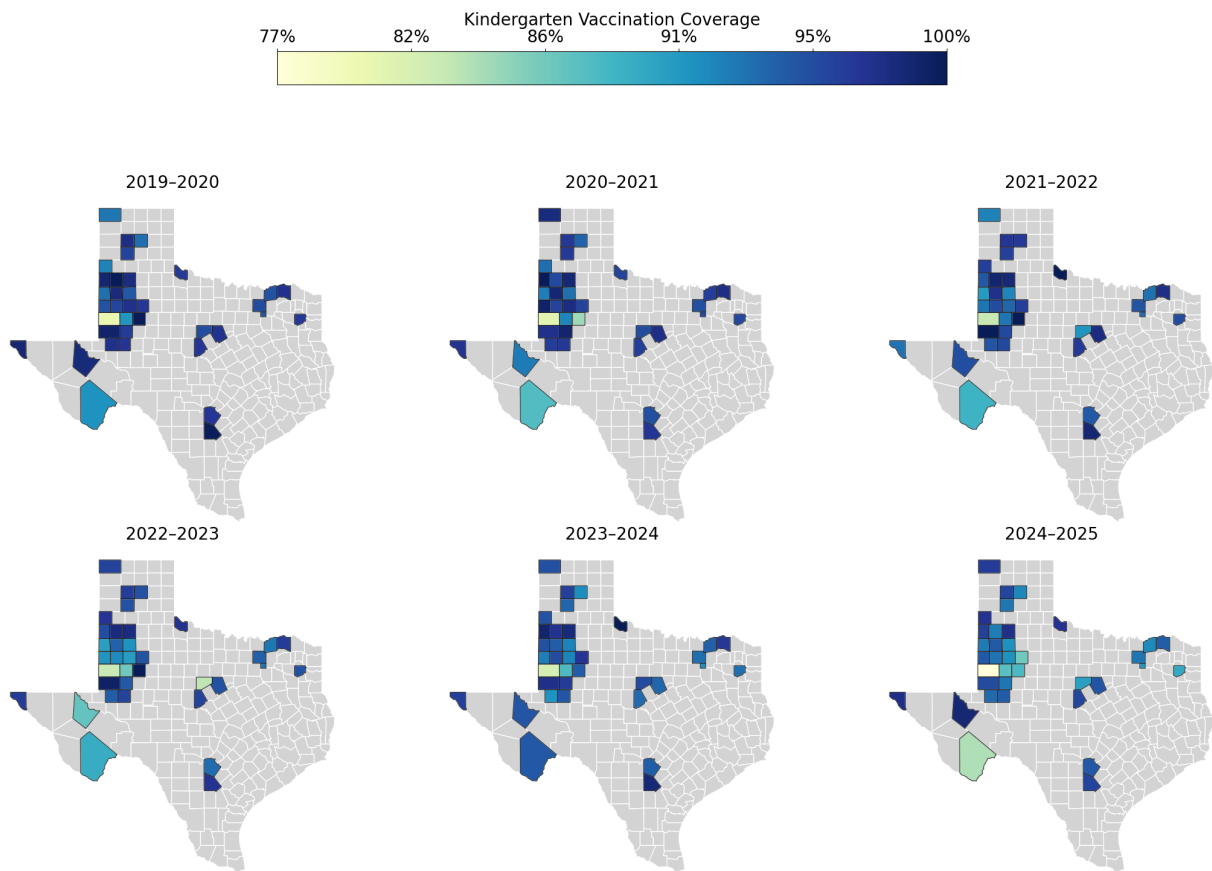


Figure S2: Maps of MMR vaccine percentage coverage of Kindergarten in the past 6 school years of the 37 focus counties.¹³

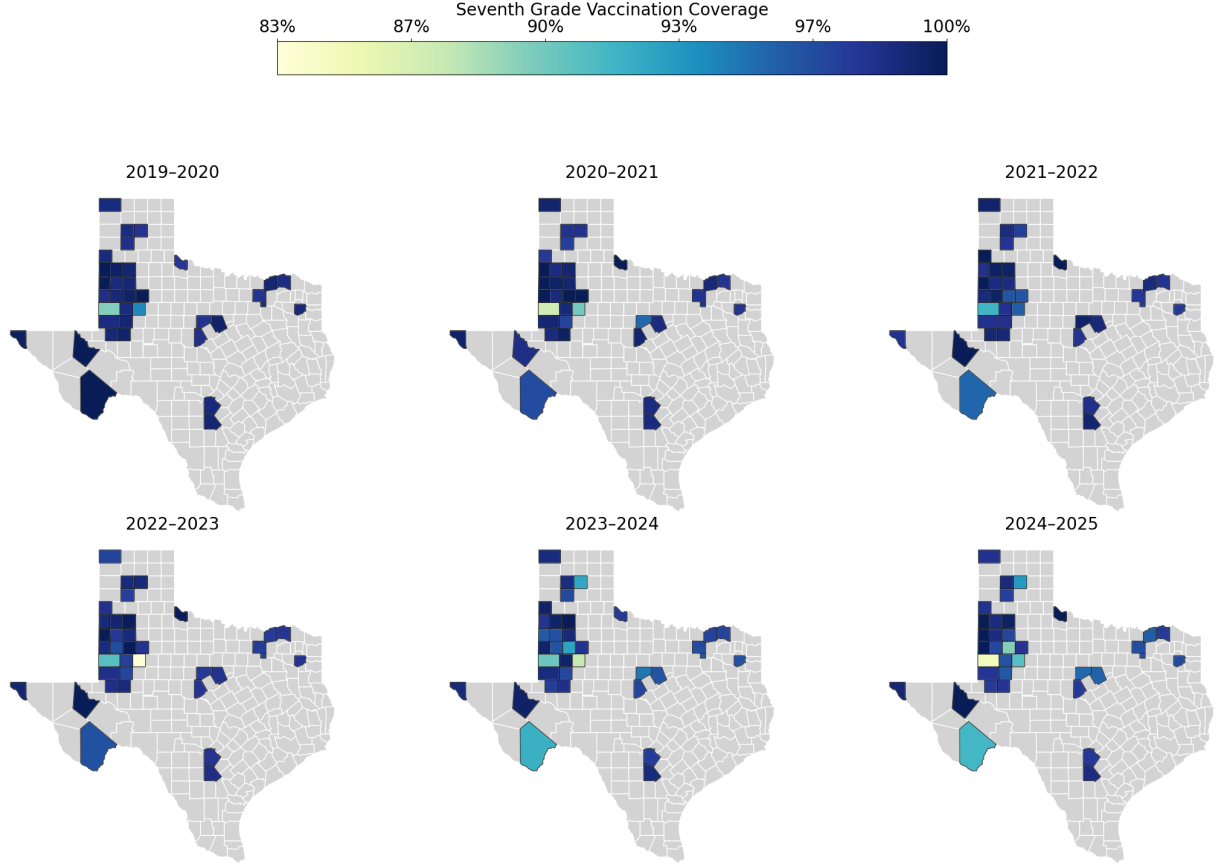


Figure S3: Maps of MMR vaccine percentage coverage of seventh-graders in the past 6 school years of the 37 focus counties.¹³

Selecting Shape Parameters k_E and k_I of Erlang (Gamma) Distributions

The classical SEIR model assumes exponentially distributed latent and infectious periods (single-stage compartments). To relax this assumption, we model the latent and infectious dwell times using Erlang (gamma) distributions via k_E exposed stages and k_I infectious stages.^{30,31} The population is stratified into age groups with state variables

$$S_a(t), E_{a,1}(t), \dots, E_{a,k_E}(t), I_{a,1}(t), \dots, I_{a,k_I}(t), R_a(t)$$

for $a \in \mathcal{A} = \{0, 1, 2\}$. As mentioned above, we use an Erlang (gamma) dwell-time representation for the latent and infectious periods of length T_E and T_I , respectively, via the linear-chain trick with k_E exposed stages and k_I infectious stages. Denote by ξ the progression rate through exposed stages and by γ the progression rate through infectious stages, so that the mean latent and infectious periods are

$$L_E := \mathbb{E}[T_E] = \frac{k_E}{\xi}, \quad L_I := \mathbb{E}[T_I] = \frac{k_I}{\gamma}.$$

The integers k_E and k_I represent the shape (variance) of the waiting-time distributions while preserving the mean durations through the rates ξ and γ . We treat k_E and k_I as model-design parameters and perform a structured sensitivity

analysis over a small grid of plausible values, which is

$$k_E \in \{1, 2\}, \quad k_I \in \{1, 2\}.$$

For each (k_E, k_I) pair, we re-fit the model with appropriate priors on the mean latent and infectious periods (see below), and compare goodness-of-fit and predictive performance using out-of-sample or information-criterion met-
 455 rics; Leave-One-Out (LOO) and Watanabe-Akaike Information Criterion (WAIC), in addition to checking posterior predictive adequacy.

Effective Reproduction Number $R_e(t)$

Transmission is mediated by an age-specific contact matrix $M(t) = [m_{ab}(t)]$ and a per-contact transmission probability q . The force of infection for age group a is

$$\lambda_a(t) = q \sum_{b=0}^2 m_{ab}(t) \frac{\sum_{j=1}^{k_I} I_{b,j}(t)}{N_b}, \quad \text{where } N_b = S_b(t) + \sum_{i=1}^{k_E} E_{b,i}(t) + \sum_{j=1}^{k_I} I_{b,j}(t) + R_b(t).$$

When vaccine efficacy against infection is included, we model the effective susceptible pool as reduced by immunity and vaccination, such that the instantaneous fraction susceptible is $s_a(t) = \frac{S_a(t)}{N_a}$, where $S_a(t)$ already accounts
 460 for protection (i.e., vaccinated/immune individuals are not counted in S_a ; see model equations and vaccine efficacy specification).

We compute the time-varying effective reproduction number $R_e(t)$ using the next-generation matrix (NGM) evaluated at time t .^{32,15} For our age-structured Erlang-chain model, the expected total infectiousness generated in group b is proportional to the mean infectious duration $\mathbb{E}[T_I] = k_I/\gamma$, so the NGM at time t is

$$K(t) = [K_{ab}(t)]_{a,b=0}^2, \quad \text{where } K_{ab}(t) = q m_{ab}(t) s_a(t) \frac{k_I}{\gamma}.$$

The effective reproduction number is then defined as the spectral radius $R_e(t) = \rho(K(t))$, where $\rho(\cdot)$ denotes the dominant eigenvalue magnitude. We report $R_e(t)$ for the baseline fit and for each counterfactual scenario by computing $K(t)$ along the simulated trajectories, using the scenario-specific contact matrix $M(t)$ and the corresponding $S_a(t)$
 465 states.

Finally, we define the baseline effective reproduction number over the analysis window as the posterior distribution of $R_e(t)$ evaluated at the initial observation time (or day $t = 0$), i.e., $R_e = R_e(0)$, and we summarize R_e and $R_e(t)$ using posterior medians and 95% highest density intervals (HDIs).

Model Parameters, Fixed Values, and Prior Distributions

Table S3: Model parameters, fixed values, and prior distributions.

Symbol	Description	Units	Type	Value / Prior (hyperparameters)
N_a	Population size in age group $a \in \mathcal{A} = \{0-4, 5-17, 18+\}$	persons	Fixed	From 2024 county-age population estimates, aggregated to the study region. ¹²
$m_{ab}(t)$	Age-specific contact rate from group a (contact-maker) to b (contactee)	contacts/day	Fixed + scenario	Baseline $M^{(0)}$ adapted from ¹⁴ and re-aggregated to 0-4, 5-17, 18+. Interventions apply multiplicative reductions to selected entries.
q	Per-contact transmission probability	–	Estimated	$\log q \sim \mathcal{N}(0.05, 1)$.
k_E	Number of Erlang stages in exposed (latent) chain	–	Model choice	Grid search: $k_E \in \{1, 2\}$.
k_I	Number of Erlang stages in infectious chain	–	Model choice	Grid search: $k_I \in \{1, 2\}$.
ξ	Progression rate through exposed stages	day ⁻¹	Estimated	Prior on mean latent period $L_E = k_E/\xi \sim \text{Truncated Normal}(12, 3 6 \leq L_E \leq 25)$.
γ	Progression rate through infectious stages	day ⁻¹	Estimated	Prior on mean infectious period $L_I = k_I/\gamma \sim \text{Truncated Normal}(9, 3 4 \leq L_I \leq 20)$.
$R_a(0)$	Initial immune number in age group a	fraction	Estimated	$R_a(0)/N_a \sim \text{Beta}(I_a, U_a)$. For infants and children, U_a is informed by school coverage and vaccine efficacy (see text above).
$S_a(0)$	Initial susceptible number in age group a	fraction	Derived	$S_a(0) = N_a - R_a(0) - \sum_{i=1}^{k_E} E_{a,i}(0) - \sum_{j=1}^{k_I} I_{a,j}(0)$.
$E_{a,1}(0), I_{a,1}(0)$	Initial infected numbers	persons	Estimated	$E_{a,1}(0)/N_a \sim \text{Beta}(2, 2)$, $I_{a,1}(0)/N_a \sim \text{Beta}(2, 2)$. Remaining stages initialized at 0.
ϕ_a	Diagnosis/Reporting probability for cases in age group a	fraction	Estimated	$\phi_a \sim \text{Beta}(2, 8)$.
α	Overdispersion in the Negative Binomial observation model	–	Estimated	$\alpha \sim \text{HalfNormal}(0, 2)$.
VE_i	Vaccine efficacy against infection for a number $i = 1, 2$ doses	fraction	Fixed	$\text{VE}_1 = 0.93$ one dose, $\text{VE}_2 = 0.97$ two doses.
$\rho_{ab}(t)$	Reduction in contact rate due to closure	day ⁻¹	Counterfactual Scenario	Base levels are 15%, 26%, and 9% for the three age groups.
$v_a(t)$	Vaccination campaign rate	day ⁻¹	Counterfactual Scenario	Levels are 0.1%, 0.2%, and 0.3%.

S1.4. Interventions

Daycare and School Closure Adjustment of the Contact Matrix.

We follow the policy implementation described in²⁴ during the COVID-19 pandemic. We model daycare and school closures as a reduction in daily age-specific contact rates $m_{ab}^{(0)}$ by $\rho_{ab}(t)$ leading to contact rates of $m_{ab}(t) = m_{ab}^{(0)} [1 - \rho_{ab}(t)]$. Specifically, school-specific contacts include interactions among students and teachers across all educational levels, from elementary schools through colleges and universities, and closures are represented by decreasing each age group's total daily contacts by the corresponding school-contact fraction. In,²⁴ the reported reductions in

daily contacts under school closure were 15% for ages 1–4 years, 26% for ages 5–17 years, 9% for ages 18–49 years, and 9% for ages 50–64 years. To align these reductions with our three age classes (0–4, 5–17, and 18+), we assign one of the following reduction levels RL_i , $i = 1, 2, 3$, see Table S4.

Table S4: Reduction levels scenarios due to daycare and school closures for $(\rho_{0-4,b}, \rho_{5-17,b}, \rho_{18+,b})$ for $b \in \{0-4, 5-17, 18+\}$.

Closure Scenarios	Reduction Level RL_1	Reduction Level RL_2	Reduction Level RL_3
DC	(15%, 0%, 4.5%)	(20%, 0%, 4.5%)	(25%, 0%, 4.5%)
SC	(0%, 26%, 4.5%)	(0%, 33%, 4.5%)	(0%, 40%, 4.5%)
DSC	(15%, 26%, 9%)	(20%, 33%, 9%)	(25%, 40%, 9%)

Given that the mean generation time for measles (the mean length of the period between the time of infection and the time of infecting another person) is about 14 days,²³ and we presume that day cares and schools would have closures in multiples of weeks, we assume the scenarios of closures lasting for 14 days, 21 days and 28 days.

Reactive Vaccination Campaign Design

To reflect realistic vaccination capacity, we used vaccination rates from the COVID-19 vaccination campaign across the 37 counties by estimating the time-varying vaccination rate $\nu(t)$. We constructed a time-varying vaccination rate using daily county-level vaccine administration data and annual population estimates for 37 selected Texas counties.³³ The data provides the cumulative doses CVAD by county and date. Daily administered doses were obtained by differencing cumulative counts $VAD(t) = CVAD(t) - CVAD(t - 1)$. To reduce reporting noise and day-of-week effects, we applied a 7-day moving average to the fraction $VAD(t)/N(t)$. See Figure S4 for the resulting smooth vaccination rate.

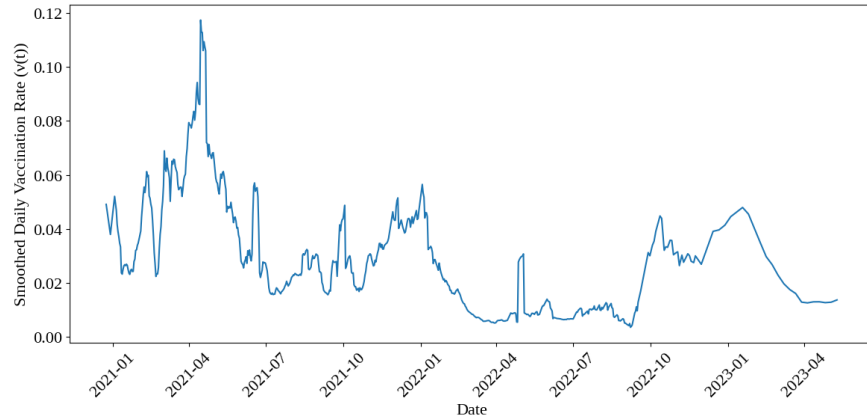


Figure S4: COVID-19 vaccination rates based on vaccine doses administered in the 37 focus counties.³³

The statistics of the smoothed vaccination rate $\nu(t)$ in Table S5 show that the vaccination rate can be as low as 0.0036 and as high as 0.1173.

Table S5: Summary statistics for $\nu(t)$.

Statistic	$\nu(t)$
Count	624
Mean	0.029005
Std	0.020787
Min	0.003638
25%	0.011019
50%	0.025558
75%	0.040604
Max	0.117293

We design a time-limited reactive vaccination campaign, with the statistics in Table S5 in mind, by adjusting the age-specific susceptible fractions to approach the herd immunity target:

$$\frac{dS_a}{dt} = -\nu_a(S_a - S_{\infty,a})\mathbf{1}_{[0,T]}(t), \quad a \in \mathcal{A} = \{0, 1, 2\}.$$

In isolation, this yields

$$S_a(t) = S_{\infty,a} + (S_a(0) - S_{\infty,a})e^{-\nu_a t},$$

and therefore

$$\nu_a = -\frac{1}{T} \ln \left(\frac{S_a(T) - S_{\infty,a}}{S_a(0) - S_{\infty,a}} \right) = -\frac{1}{T} \ln \left(\frac{s_a(T) - s_{\infty,a}}{s_a(0) - s_{\infty,a}} \right).$$

Let $K_0 = cM$ represent the baseline next-generation matrix, where M is the empirical contact matrix. Herd immunity is achieved through the spectral-radius condition $\rho(\text{diag}(s_\infty)K_0) \leq 1$. Under uniform susceptibility reduction $s_{\infty,a} \equiv s_\infty$, this reduces to $s_\infty \leq 1/\rho(K_0) = 1/R_0$. With an approximate value of $R_0 = 15$,¹⁶ we can assign $s_\infty \approx \frac{1}{15} = 0.067$, we can then assume that $s_0(0) = 1 - R_0(0)/N_0 = 1 - 0.67 = 0.33$ and $s_1(0) = 1 - R_1(0)/N_1 = 1 - .78 = 0.22$ based on the estimations below. Thus,

$$\nu_0 = -\frac{1}{T} \ln \left(\frac{s_0(T) - .067}{.33 - .067} \right),$$

and

$$\nu_1 = -\frac{1}{T} \ln \left(\frac{s_1(T) - .067}{.22 - .067} \right).$$

A 150-day vaccination campaign can reduce susceptibility by half, i.e., to 0.165 and 0.11, at rates 0.00658 and 0.00846, respectively.

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In conclusion, we use three vaccination rates that align with the calculations and the estimated COVID-19 capacity: 0.1%, 0.2%, and 0.3%. These scenarios may also account for healthcare capacity, population sizes, and vaccine hesitancy.

S1.5. Bayesian MCMC Results

We performed prior predictive diagnostics to ensure that the selected prior distributions are consistent with the observed data and do not yield uncertainty intervals that are either too wide or too narrow (see Figure S5). Bayesian MCMC diagnostics also show convergence and perfect mixing of all of the MCMC for all parameters and reliability on the posterior estimates generated by MCMC, supported by estimated rank-normalization of $\hat{r} = 1$, with significantly large bulk and tail effective sample sizes (ESS) and small Monte Carlo standard errors for the mean and standard deviation relative to the posteriors' standard deviations; see Table S6. The traces and the posterior distributions overlap in Figure S6, indicating that MCMC has converged to the same posterior distribution. The autocorrelation also showed almost no correlation; the data is not shown.

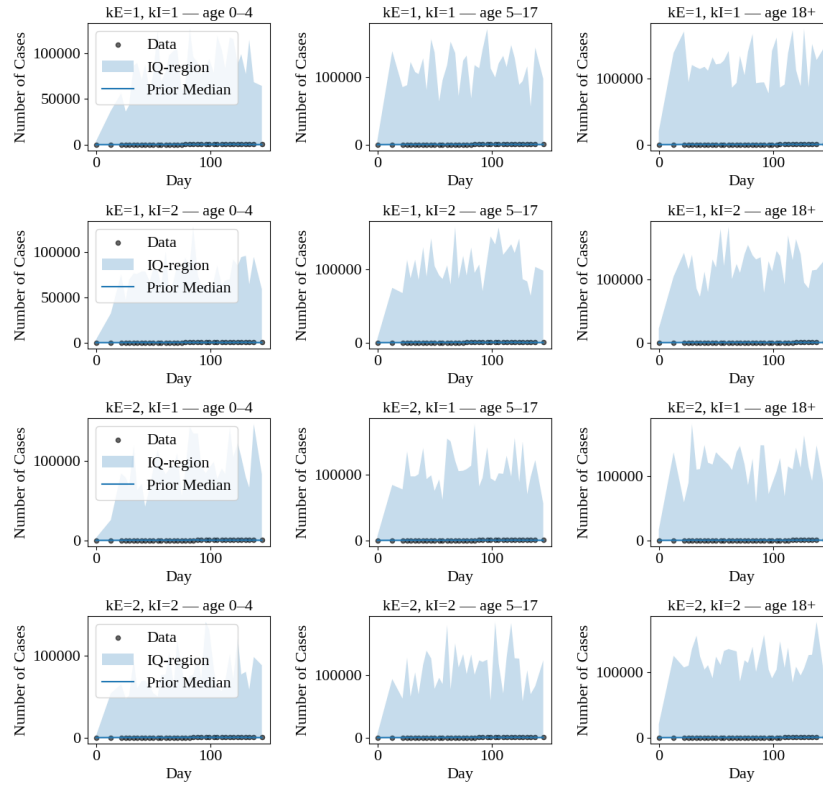


Figure S5: Prior median and interquartile range (IQR) of the prior predictive versus data of the number of measles cases in West, North, and Central Texas with parameters estimated using Bayesian methods. The figure shows acceptable choices of prior distributions.

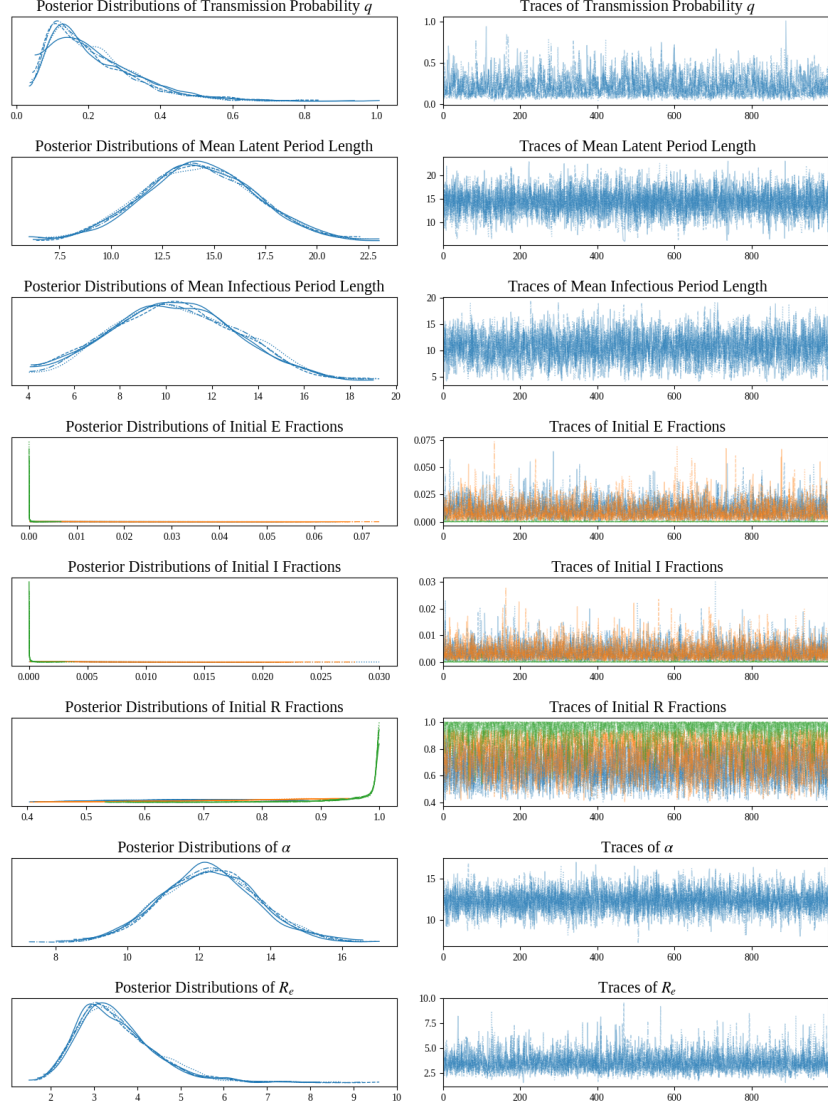


Figure S6: MCMC traces and posterior distributions of the estimated parameters indicate convergence of the five independent MCMC chains after the burn-in period, starting from five randomly selected initial parameter vectors. In the first panel, the posterior distributions and traces of the transmission probability q are shown. In the second and third panels, the mean length of the latent and infectious periods is shown, respectively. In the following panels of the initial fractions of E , I , and R , the blue color corresponds to the age group 0-4, the orange color corresponds to the age group 5-17, and the green color corresponds to the age group 18+. The following panels show the overdispersion parameter α and the effective reproduction number R_e at day 0.

Table S6: Posterior summary statistics.

Parameter	Mean	Std	HDI 2.5%	HDI 97.5%	MC SE (Mean)	MC SE (Std)	Bulk ESS	Tail ESS	$\hat{r}$
q	0.21	0.12	0.05	0.44	0.00	0.00	1484.87	2700.35	1.0
L_E	14.29	2.72	9.18	19.36	0.04	0.04	3737.72	2421.89	1.0
L_I	10.39	2.77	5.33	15.56	0.05	0.03	2842.48	2462.47	1.0
α	12.30	1.33	9.88	14.96	0.02	0.02	5032.57	3668.45	1.0
$R_0(0)/N_0$	0.67	0.11	0.48	0.86	0.00	0.00	2953.84	3283.25	1.0
$R_1(0)/N_1$	0.78	0.12	0.55	0.95	0.00	0.00	1614.14	3144.21	1.0
$R_2(0)/N_2$	0.96	0.06	0.85	1.00	0.00	0.00	2152.00	3511.83	1.0
R_e	3.52	0.93	2.07	5.23	0.02	0.02	3082.39	2818.38	1.0

Table S7: Leave-One-Out (LOO) and Watanabe-Akaike Information Criterion (WAIC) model comparison results across (k_E, k_I) .

k_E	k_I	Expected Log Predictive Density	Effective Number of Parameters (D.F.)	WAIC
1	1	-501.86	3.81	1003.73
1	2	-502.76	3.74	1005.52
2	1	-502.17	3.85	1004.33
2	2	-502.72	3.99	1005.43

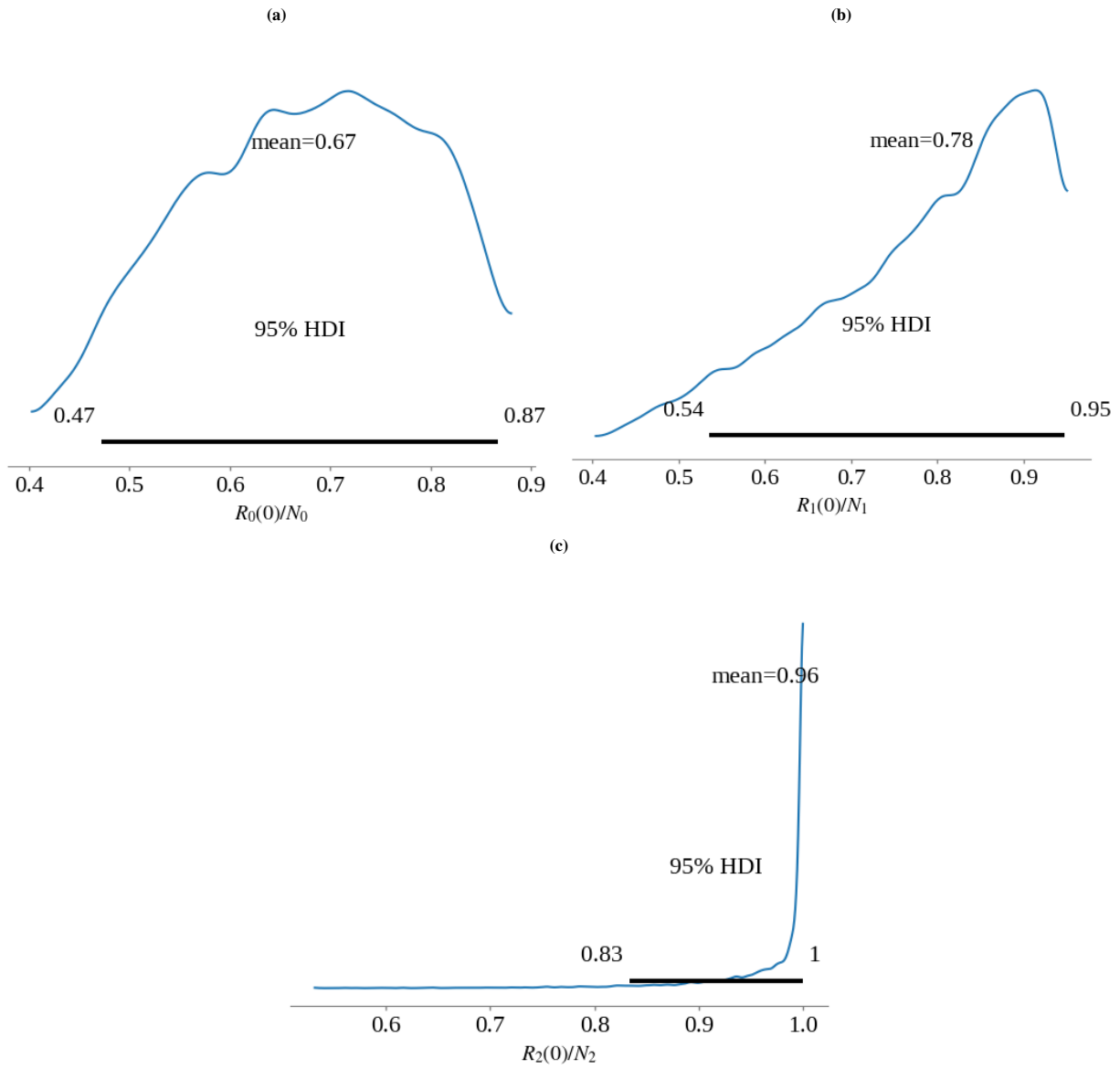


Figure S7: Posterior distributions of $R_0(0)/N_0$ in (a), $R_1(0)/N_1$ in (b), and $R_2(0)/N_2$ in (c).