Supplementary Online Content


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This supplementary material has been provided by the authors to give readers additional information about their work.
eAppendix 1. Methods

Rationale
In our previous study, we developed a dynamic, age-structured model of pertussis transmission that incorporated different hypotheses about the mechanisms of vaccine failure. Using statistical inference methods on detailed incidence data in Massachusetts, we found evidence that both the whole-cell and the acellular pertussis vaccines conferred imperfect, but slowly waning immunity. Unexpectedly, we also found that our model-based estimates of the increase in the odds of pertussis were consistent with those that had been obtained in a number of case-control and cohort studies and had commonly been interpreted as evidence of a rapid loss of DTaP-induced immunity (Figure 3F in Ref. 1 and Refs. 2-5). To resolve this apparent paradox, we designed a simulation study to systematically compare different measures of DTaP protectiveness. Below we spell out the model structure, which is largely based on the model previously developed. Unless otherwise stated, the model parameters were fixed at the maximum likelihood estimates (MLE) obtained by applying the iterated filtering algorithm to monthly age-specific incidence data in Massachusetts, 1990–2005. We refer the readers to Ref. 1 for complete details on the estimation procedure.

Model formulation
We implemented an age-structured model of pertussis transmission, extending our previously described model in Massachusetts. Briefly, the model is an extension of the standard SEIR model that stratifies individuals according to their vaccination status: unvaccinated (superscript(1)) or vaccinated (superscript(2)). Hence, the population of susceptibles is divided into those susceptible to a naïve infection (S(1)) and to a post-vaccine infection (S(2)). Exposed (E(1) and E(2)) and infected (I(1) and I(2)) are similarly divided. Upon recovery of either type of infection, individuals move the recovered class (R). To examine differences between DTwP- and DTaP-derived immunity, vaccinated individuals are explicitly modeled and separated into two compartments, V(w) for DTwP and V(a) for DTaP. Our previous results indicated that both vaccines fail in take (at a degree controlled by the proportion of primary vaccine failure ) and in duration (at a degree controlled by the waning rate ). We therefore modeled these two modes of vaccinal failure, assuming a common failure in take but distinct waning rates for DTwP and for DTaP. A schematic of the model structure is shown in eFigure 1.

Individuals are divided into 1-yr age groups from age 1 (i.e., 1–2 yr to age 74 (i.e., 74–75) yr. The 0–1-yr age group is further divided into newborns aged 0–4 mo and infants aged 4–12 mo, assuming that newborns are fully vulnerable to infection before they receive their second dose of pertussis vaccine at age 4 mo. Altogether, the model consists of 76 age groups, labeled i = 1, ..., 76. The age groups are updated at the start of each year, except for newborns who age continuously over the year at a rate . Within each year, the mean field dynamics in newborns are given by:

\[
\begin{align*}
\frac{dV_1^{(w)}}{dt} &= 0 \\
\frac{dV_1^{(a)}}{dt} &= 0 \\
\frac{dS_1^{(1)}}{dt} &= B - (\lambda_1(t) + \delta_1)S_1^{(1)} \\
\frac{dE_1^{(1)}}{dt} &= \lambda_1(t)S_1^{(1)} - (\sigma + \delta_1)E_1^{(1)} \\
\frac{dI_1^{(1)}}{dt} &= \sigma E_1^{(1)} - (\gamma + \delta_1)I_1^{(1)} \\
\frac{dS_1^{(2)}}{dt} &= 0 \\
\frac{dE_1^{(2)}}{dt} &= 0 \\
\frac{dI_1^{(2)}}{dt} &= 0 \\
\frac{dR_1}{dt} &= \gamma I_1^{(1)} - \delta_1 R_1
\end{align*}
\]

In infants aged 4–12 mo (i = 2), we have:
\[
\frac{dV_t^{(w)}}{dt} = p_1(t)(1 - \epsilon_A)\mathbb{I}(t \in T^{(w)})\delta_i S_t^{(1)} - \alpha_i^{(w)}V_t^{(w)}
\]
\[
\frac{dV_t^{(a)}}{dt} = p_1(t)(1 - \epsilon_A)\mathbb{I}(t \in T^{(a)})\delta_i S_t^{(1)} - \alpha_i^{(a)}V_t^{(a)}
\]
\[
\frac{dS_t^{(1)}}{dt} = [1 - p_1(t)]\delta_i S_t^{(1)} - \lambda_i(t)S_t^{(1)}
\]
\[
\frac{dE_t^{(1)}}{dt} = \delta_i E_t^{(1)} + \lambda_i(t)S_t^{(1)} - \sigma E_t^{(1)}
\]
\[
\frac{dI_t^{(1)}}{dt} = \delta_i I_t^{(1)} + \sigma E_t^{(1)} - \gamma I_t^{(1)}
\]
\[
\frac{dS_t^{(2)}}{dt} = \epsilon_0 p_1(t)\delta_i S_t^{(1)} + \alpha_i^{(w)}V_t^{(w)} + \alpha_i^{(a)}V_t^{(a)} - \lambda_i(t)S_t^{(2)}
\]
\[
\frac{dE_t^{(2)}}{dt} = \lambda_i(t)S_t^{(2)} - \sigma E_t^{(2)}
\]
\[
\frac{dI_t^{(2)}}{dt} = \sigma E_t^{(2)} - \gamma I_t^{(2)}
\]
\[
\frac{dR_t}{dt} = \delta_i R_t + \gamma(I_t^{(1)} + I_t^{(2)})
\]

The dynamics in older age groups \((i \geq 3)\) are given by:

\[
\frac{dV_t^{(w)}}{dt} = -\alpha_i^{(w)}V_t^{(w)}
\]
\[
\frac{dV_t^{(a)}}{dt} = -\alpha_i^{(a)}V_t^{(a)}
\]
\[
\frac{dS_t^{(1)}}{dt} = -\lambda_i(t)S_t^{(1)}
\]
\[
\frac{dE_t^{(1)}}{dt} = \lambda_i(t)S_t^{(1)} - \sigma E_t^{(1)}
\]
\[
\frac{dI_t^{(1)}}{dt} = \sigma E_t^{(1)} - \gamma I_t^{(1)}
\]
\[
\frac{dS_t^{(2)}}{dt} = \alpha_i^{(w)}V_t^{(w)} + \alpha_i^{(a)}V_t^{(a)} - \lambda_i(t)S_t^{(2)}
\]
\[
\frac{dE_t^{(2)}}{dt} = \lambda_i(t)S_t^{(2)} - \sigma E_t^{(2)}
\]
\[
\frac{dI_t^{(2)}}{dt} = \sigma E_t^{(2)} - \gamma I_t^{(2)}
\]
\[
\frac{dR_t}{dt} = \gamma(I_t^{(1)} + I_t^{(2)})
\]

where \(\sigma\) represents the rate of progression from the exposed to the infected compartment, \(\gamma\) the recovery rate, \(p_1(t)\) the time-varying fraction of newborns that receive 3 doses of pertussis vaccine (i.e., the primary course), and \(\mathbb{I}(t \in T^{(w)})\) (respectively \(\mathbb{I}(t \in T^{(a)})\)) an indicator function equal to 1 when DTwP (respectively DTaP) is in use, 0 otherwise. In all simulations, we assumed a type-I mortality (whereby all individuals reach age 74 and die at age 75) and no migration. Given these assumptions, the total population size, \(N = 5 \times 10^6\), was held approximately constant by fixing \(B = N/75\) per year.

The force of infection in age group \(i\) is given by:

\[
\lambda_i = q_i \sum_{j=1}^{16} F_{ij}(t)\tilde{C}_{ij} \frac{\tilde{I}_j^{(1)} + \theta \tilde{I}_j^{(2)} + \epsilon}{N_j}
\]

where \(q_i\) is the probability of infection given exposure (i.e., susceptibility) in age group \(i\), \(\theta\) the infectiousness of
post-vaccine infections relative to that of naïve infections, $N_j$ the total population in age group $j$, and $t$ an immigration term, fixed to 1 in all simulations. The contact matrix $C = (C_{i,j})$ is shown in eFigure 2, was available from the POLYMOD study in Great Britain, after application of a correction to ensure reciprocity of contacts between age groups, as detailed in Refs. 1, 10. The matrix $C_{i,j}$ represents the full contact matrix, augmented to incorporate the extra age groups in the model, under the assumption that all individuals within a 5-yr age block have similar contact rates. The terms $f_j^{(1,2)}$ represent the number of infected individuals, aggregated over 5-yr age blocks (0–5, 5–10, …, 70–75, $j = 1, \ldots, 15$) to match the age groups in the POLYMOD study.

The seasonality in children’s contact was modeled using the age-dependent seasonal transmission terms $F_j(t)$. Following Ref. 1, this was assumed additive and applied only within the same 5-yr age group, that is, only for contacts between children 5–10 yr and 5–10 yr, between 10–15 yr and 10-15 yr, and between 15–20 yr and 15–20 yr. The seasonality coefficients were fixed according to the estimates obtained in Ref. 1, resulting in the functional forms depicted in eFigure 3. Although a mechanistic interpretation of these variations is yet to be found, they are consistent with previous observations (in Massachusetts11,12 and in the US overall13) that higher proportions of cases are reported during July–August in young children and during October–December (with a peak in November) in adolescents.

The model was implemented as a continuous-time Markov process, approximated via a multinomial modification of the tau-leap algorithm with a fixed time step $\Delta t = 10^{-3}$ yr. The model was coded using the pomp package, operating in the R environment.

Model parametrization

**Vaccinal parameters.** According to the US immunization schedule, 5 doses of pertussis should be administered: 3 doses at 2, 4, and 6 mo of age (primary course), one dose at age 15–18 mo (pediatric booster), and one dose at age 4–6 yr (preschool booster). This schedule was modeled by moving a fraction of susceptibles to the vaccinated class when individuals reached the following age groups:

- **Age 4–12 months ($i = 2$, primary course):** fraction $p_1(t)$ vaccinated in unvaccinated susceptibles $S^{(1)}_2$, comprising a fraction $p_2(t)\epsilon_A$ transitioning to $S^{(2)}_2$ (primary vaccine failures) and a fraction $p_1(t)(1-\epsilon_A)$ transitioning to $V_2^{(w)}$ (DTwP era) or to $V_2^{(a)}$ (DTaP era).
- **Age 1–2 yr ($i = 3$, pediatric booster):** total fraction $v_2$ vaccinated in previously vaccinated susceptibles $S^{(2)}_3$, comprising a fraction $v_2\epsilon_A$ transitioning to $S^{(2)}_3$ and a fraction $v_2(1-\epsilon_A)$ transitioning to $V_3^{(w)}$ or to $V_3^{(a)}$.
- **Age 5–6 yr ($i = 7$, preschool booster):** total fraction $v_2$ vaccinated in previously vaccinated susceptibles $S^{(6)}_7$, comprising a fraction $v_2\epsilon_A$ transitioning to $S^{(2)}_7$ and a fraction $v_2(1-\epsilon_A)$ transitioning to $V_7^{(w)}$ or to $V_7^{(a)}$.

In all simulations, we assumed that mass vaccination with DTwP had started in 1940 and that the vaccine coverage for the primary course linearly increased to reach $v_2 = 0.95$ from 1955. Hence,

$$ p_1(t) = \begin{cases} 0, & t < 1940 \\ 0.95 \frac{t-1940}{1955-1940}, & t \in [1940, 1955] \\ 0.95, & t > 1955 \end{cases} $$

For simplicity, we further assumed full coverage for the booster doses ($v_2 = 1$), so that all children who received a primary course of vaccine were also assumed to receive their booster doses. Booster vaccination was assumed to start in 1967, based on the earliest record we could find in the literature.

According to reports from the Advisory Committee on Immunization Practices, DTaP was recommended for the 4th and the 5th doses in December 1991 and for the full course of vaccination in July 1996. To model the effect of this progressive switch, susceptible children receiving their booster doses were moved the DTaP-vaccinated class from 1992, and those receiving their primary course from 1997. In spring 2005, a single dose of Tdap was recommended to vaccinate adolescents at age 11–12 yr. According to national estimates from the Centers for Disease Control and Prevention, Tdap vaccine coverage was 10% in 2006 and gradually increased to plateau at 85% from 2012. This adolescent booster dose was modeled by moving a fraction of susceptibles reaching age
Epidemiological parameters. Epidemiological model parameters were fixed using the values estimated in Ref. 1, summarized in eTable 1. In particular, we assumed perfect infection-derived immunity, based on studies that showed that repeat infections were extremely rare in the prevaccine period and in the vaccine era. In a sensitivity analysis, we tested alternative hypotheses regarding the duration of infection-derived immunity after a postvaccine infection (eFigure 9).

Simulation protocol
All simulations were started 100 yr before the start of mass vaccination with DTwP, assuming that the system was at equilibrium in the pre-vaccine era. To compare the model simulations with epidemiological studies in the US, we simulated the dynamics of pertussis in 5 cohorts of children born between 2001 and 2005, until they reached ages 5–9 yr. As shown in eTable 2, the simulated study period was therefore 2006–2014. To determine which degree of DTaP waning resulted in odds ratio estimates most consistent with those of empirical studies, we tested 100 values of DTaP waning rate (\( \alpha_{a}^{(b)} \)) between 0.01 and 1 per yr, uniformly distributed on a log._2-scale. In the main text, we quantify the degree of waning as the probability that immunity wanes within 5 yr, \( p_{5} = 1 - e^{-5a_{a}^{(b)}} \). For each value, 100 replicate stochastic simulations were run, for a grand total of 10,000 simulations.

Estimation of DTaP protectiveness measures
Odds ratios. For every simulation, we estimated the relative change in the odds of acquiring pertussis using linear regression. Let \( R_{b,y}^{(V)} \) be the annual incidence (not corrected for under-reporting) of infections in children born during year \( b \) (\( b = 2001, ..., 2005 \)), \( y \) years after they receive their preschool booster (\( y = 0, ..., A \), i.e., ages 5, ..., 9 yr), calculated as the ratio of the annual number of cases to the size of the population vaccinated. The corresponding odds of acquiring pertussis is given by:

\[
O_{b,y}^{(V)} = \frac{R_{b,y}^{(V)}}{1 - R_{b,0}^{(V)}}
\]

We fitted the linear model:

\[
\log O_{b,y}^{(V)} = \alpha + \beta y
\]

where \( \alpha \) is the intercept and \( \beta \) the slope. Hence, \( e^{\beta} \) represents the average relative yearly change in the odds of acquiring pertussis after receipt of the fifth dose, a quantity directly comparable to that estimated in epidemiological studies in the US. In the main text, we also refer to \( e^{\beta} \) as an odds ratio, since it equals the average year-on-year ratio of odds.

Vaccine effectiveness in children aged 5 to 9 yr. We estimated the effectiveness of DTaP in children aged 5 to 9 yr using the standard formula: \( VE = 1 - \frac{R_{b,y}^{(V)}}{R_{b,y}^{(NV)}} \), where \( R_{b,y}^{(V)} \) is the risk of pertussis in vaccinated children, and \( R_{b,y}^{(NV)} \) that in unvaccinated children. For each birth cohort \( b \), we calculated \( C_{b}^{(V)} = \sum_{y=0}^{4} C_{b,y}^{(V)} \) the total number of infections occurring between ages 5 and 9 yr in vaccinated children, and \( A_{b,y}^{(V)} = R_{b,y}^{(V)} + S_{b,y}^{(V)} \) the initial population at risk of infection. We defined the risk of pertussis as the fraction of at-risk vaccinated children contracting pertussis between ages 5 and 9 yr, that is, \( R_{b,y}^{(V)} = \frac{C_{b,y}^{(V)}}{A_{b,y}^{(V)}} \). We similarly defined the quantities \( C_{b}^{(NV)} \), \( A_{b}^{(NV)} = S_{b,y}^{(1)} + R_{b,y}^{(NV)} \) in unvaccinated children. We then fitted the binomial regression model with log-link:

\[
\begin{aligned}
&c_{b}^{(V)} \sim \text{Bin}(A_{b}^{(V)}, \theta) \\
&\log \theta = \text{offset}(\log R_{b,y}^{(NV)}) + \gamma
\end{aligned}
\]

where \( \gamma \) is the intercept. The estimate of vaccine effectiveness is therefore given by \( VE = 1 - e^{\gamma} \).

Vaccine impact. We calculated the vaccine impact \( \phi \), a population-wide measure of vaccine impact that quantifies the overall reduction of transmission caused by DTaP. This was calculated as:
\[
\phi = \frac{1}{v_1} \left( 1 - \frac{R_{v_1}}{R_0} \right)
\]

where \( R_{v_1} \) is the reproduction number with vaccination at coverage \( v_1 \) and \( R_0 \) the basic reproduction number, in the absence of vaccination. Both reproduction numbers were calculated using the next-generation method on a simplified model with no exposed class and vaccination at birth, at coverage \( v_1 \).

**Effect of under-reporting on DTaP protectiveness measures.** All the above protectiveness measures were estimated assuming complete reporting of cases. Although under-reporting is inevitable in practice, we here (qualitatively) argue that it would affect our estimates little, if the reporting probability is assumed to be constant over age in children aged 5–9 yr. Regarding the OR estimates, considering the magnitude of pertussis incidence rates in children 5–9 years of age (below a few percents, even in the high-waning regime, see Figure 2A), the odds can be approximated by the incidence rate. Under this assumption, the reporting probability would be absorbed in the intercept of the log-linear model on under-reported data, leaving the estimate of the slope unchanged. Regarding the VE estimates, our estimates in Massachusetts indicated that post-vaccine infections were less reported than naive infections¹, in keeping with the common view that infections are less symptomatic in individuals previously primed by vaccination²⁵. Thus, if the VE is estimated on under-reported data, the denominator would be inflated compared with the numerator in the VE formula above. In that situation, we expect our "true" estimates of VE to under-estimate those based on under-reported data, so that our main conclusions would hold.
eAppendix 2. Results

Association between the odds ratio and the vaccine impact

In the main text Figure 2A, we presented the association between the odds ratio and the vaccine effectiveness. In eFigure 4, we show the association between the odds ratio and the vaccine impact. Based on the meta-analysis estimate in Ref. [3], we predict that the vaccine impact of DTaP exceeds 50%. (Please note that the upper bound of the vaccine impact in eFigure 4 is constrained by the lower bound of the waning rate fixed in model simulations. Lower waning rates would result in higher values of vaccine impact and would still be consistent with the empirical range of odds ratios, considering the large estimation uncertainty when the vaccine is assumed to be highly effective.)

Association between the odds ratio and waning vaccine effectiveness

In the discussion of Ref. [2], Klein et al. estimated the waning of vaccine effectiveness using the formula:

\[ 1 - VE_y = (1 - VE_0) \text{OR}^y \]

where \( VE_y \) represents the vaccine effectiveness \( y \) years after receipt of the last DTaP dose. This calculation assumes that the relative increase (over time since last DTaP vaccination) in the odds of acquiring pertussis (OR) equates that in 1–VE. To determine the validity of this assumption, we extended the binomial regression model described above to calculate age-specific VEs:

\[
\begin{align*}
    C_{k,y}^{(V)} &\sim \text{Bin}(A_{k,y}^{(V)}, \theta) \\
    \log \theta &= \text{offset}(\log R_{k,y}^{(NV)}) + \gamma + \xi y
\end{align*}
\]

According to this model, we have \( VE_y = 1 - e^{\gamma + \xi y} \), so that \( e^{\xi} \) estimates the relative increase in 1–VE over time since last DTaP. The results of this analysis are presented in Figure 3 and discussed in the main text.

Sensitivity analyses

To assess the robustness of our results, we conducted five sensitivity analyses. First, the simulation results presented in the main text were obtained assuming 95% vaccine coverage for the primary course and identical vaccine traits for DTaP and Tdap. Because some evidence suggests that Tdap protectiveness is lower than that of DTaP [26,27], we considered alternative scenarios in which Tdap vaccination resulted in a higher fraction of primary vaccine failures, fixed to 10, 25, or 50%. As shown in eFigure 5, we found comparable results under these assumptions, with empirical values of odds ratios more consistent with a highly effective DTaP.

Second, we conducted simulations in which we assumed lower vaccine coverage, fixed to \( v_1 = 0.85 \). Our main results were also robust in this scenario (eFigure 6), although the variability in odds ratio estimates at high values of vaccine effectiveness was less pronounced, presumably because the lower vaccine coverage resulted in more circulation of pertussis and, therefore, in more post-vaccine cases.

Third, we tested an alternative simulation protocol in which, while varying the waning rate of DTaP, the other model parameters were fixed to values different from their maximum likelihood estimates. Specifically, for every waning rate value tested, we selected from the bootstrap distribution (obtained in Ref. [1]) the parameter set with the waning rate estimate closest to the value being tested. A comparable picture emerged in this case, although the lower bound of the predicted VEs was lower (eFigure 7). This difference may be interpreted as a consequence of the negative correlation between the waning rate and the basic reproduction number \( R_0 \): as the waning rate is increased, \( R_0 \) becomes smaller, resulting in a lower intercept and in a higher slope in the age-specific incidence curve.

Fourth, because our study aimed to explain an epidemiological signature that has been reported in different locations, we made the simplest assumptions about demography, so that the simulated birth cohorts had approximately equal size. Nevertheless, we sought to test the robustness of our results by carrying out new simulations using actual birth rates in the US (obtained from Ref. [28] during years 1933–2008 and completed with data from 2009 to 2014, the last year of the simulated study period in our study). As shown in eFigure 8, our results were almost unchanged, suggesting that demographic changes had little dynamic impact on the epidemiological signature we examined here.

Fifth, we tested an alternative model structure, in which protection after a post-vaccine infection was assumed to wane faster than that after a naïve infection. The base model was therefore extended to incorporate two recovered
classes, following a naïve infection ($R_1$) or a post-vaccine infection ($R_2$). We assumed that infection-derived immunity after a naïve infection was perfect (waning rate $\alpha_{R,1} = 0$), and we tested three different values for the degree of waning infection-derived immunity after a post-vaccine infection (waning rate $\alpha_{R,2} \in \{0, 0.02, 0.05\}$ per yr). (The base model therefore corresponds to the hypothesis $\alpha_{R,1} = \alpha_{R,2} = 0$.) As shown in eFigure 9, our main results remained robust under these different hypotheses. The OR estimates increased with $\alpha_{R,2}$, but the change was modest. Of note, higher values of $\alpha_{R,2}$ tended to increase the number of post-vaccine cases and, therefore, to decrease the variability of the OR estimates.
eFigure 1. Model Schematic

For simplicity, age is omitted.
eFigure 2. Matrix of Age-Specific Contact Rates
**eFigure 3.** Age-Specific Seasonal Forcing in Children 5-20 y
eFigure 4. Comparison of the Vaccine Impact With the Odds Ratio
**eFigure 5.** Comparison of DTaP Protectiveness Measures, Assuming Other Values of Primary Vaccine Failure for Tdap
eFigure 6. Comparison of DTaP Protectiveness Measures, Assuming 85% Coverage for the Primary Course of Vaccination
eFigure 7. Comparison of DTaP Protectiveness Measures With a Bootstrap Simulation Protocol
eFigure 8. Comparison of DTaP Protectiveness Measures, Assuming Nonconstant Birth Rates
**eFigure 9. Pertussis Incidence as a Function of Time Since Last Receipt of DTaP, Assuming Waning Infection-Derived Immunity**

In each panel, the 5 lines represent 5 cohorts of children born between 2001 and 2005, tracked 0 to 4 yr after receipt of the last dose of DTaP (that is, during ages 5 to 9 yr). The rows correspond to the three values of waning DTaP-induced immunity ($p_5 \in \{0.05, 0.32, 0.99\}$, cf. Figure 2B); the columns correspond to the three values of waning infection-derived immunity after a post-vaccine infection ($\alpha_{R,2} \in \{0.02, 0.05\}$ per yr).
### eTable 1. Model Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Meaning</th>
<th>Value</th>
<th>Source/comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>$N$</td>
<td>Total population size</td>
<td>5 million</td>
<td>Assumption</td>
</tr>
<tr>
<td>$B$</td>
<td>Birth rate</td>
<td>$N/75$ per year</td>
<td>Fixed to keep population size approximately constant</td>
</tr>
<tr>
<td>$v_1$</td>
<td>Vaccine coverage for primary course</td>
<td>95%</td>
<td>Assume linear ramp-up from 0 in 1940 to 95% from 1955</td>
</tr>
<tr>
<td>$v_2$</td>
<td>Vaccine coverage for booster doses</td>
<td>100%</td>
<td>Assume that all children receiving the first 3 doses also receive the 4th and the 5th doses</td>
</tr>
<tr>
<td>$\delta_1$</td>
<td>Aging rate in 0–4 mo</td>
<td>3 per yr</td>
<td>—</td>
</tr>
<tr>
<td>$D_E$</td>
<td>Latent period</td>
<td>8 days</td>
<td>1</td>
</tr>
<tr>
<td>$D_I$</td>
<td>Infectious period</td>
<td>15 days</td>
<td>1</td>
</tr>
<tr>
<td>$q_1, \ldots, q_{11}$</td>
<td>Susceptibility in 0–10 yr</td>
<td>0.094</td>
<td>1</td>
</tr>
<tr>
<td>$q_{12}, \ldots, q_{21}$</td>
<td>Susceptibility in 10–20 yr</td>
<td>0.048</td>
<td>1</td>
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<td>$q_{22}, \ldots, q_{76}$</td>
<td>Susceptibility in ≥20 yr</td>
<td>0.008</td>
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<tr>
<td>$\theta$</td>
<td>Relative infectiousness</td>
<td>0.99</td>
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<td>$\epsilon_A$</td>
<td>Primary vaccine failure</td>
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<td>1</td>
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<tr>
<td>$\alpha^{(w)}_{V}$</td>
<td>Waning rate of DTwP</td>
<td>0.011 per yr</td>
<td>1</td>
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<tr>
<td>$\alpha^{(a)}_{V}$</td>
<td>Waning rate of DTaP</td>
<td>Varied in [0.01, 1] per yr</td>
<td>Assumed identical for DTwP and DTaP</td>
</tr>
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<td>$C_{ij}$</td>
<td>Age-specific contact rates</td>
<td>eFigure 2</td>
<td>9</td>
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**eTable 2. Synoptic Table of the Five Simulated Cohorts of Children**

<table>
<thead>
<tr>
<th>Year</th>
<th>Age</th>
<th>5 yr</th>
<th>6 yr</th>
<th>7 yr</th>
<th>8 yr</th>
<th>9 yr</th>
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<tbody>
<tr>
<td>2006</td>
<td>Cohort₁</td>
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<tr>
<td>2007</td>
<td>Cohort₂</td>
<td>Cohort₁</td>
<td></td>
<td></td>
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<td>2008</td>
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<td>Cohort₁</td>
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<td>Cohort₃</td>
<td>Cohort₂</td>
<td>Cohort₁</td>
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<td>Cohort₃</td>
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<td>Cohort₁</td>
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eReferences
