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Household transmission of influenza A and B within a prospective cohort during the 2013-2014 and 2014-2015 seasons

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Abstract

People living within the same household as someone ill with influenza are at increased risk of infection. Here, we use Markov chain Monte Carlo methods to partition the hazard of influenza illness within a cohort into the hazard from the community and the hazard from the household. During the 2013-2014 influenza season, 49 (4.7%) of the 1044 people enrolled in a community surveillance cohort had an acute respiratory illness (ARI) attributable to influenza. During the 2014-2015 influenza season, 50 (4.7%) of the 1063 people in the cohort had an ARI attributable to influenza. The secondary attack rate from a household member was 2.3% for influenza A (H1) during 2013-2014, 5.3% for influenza B during 2013-2014, and 7.6% for influenza A (H3) during 2014-2015. Living in a household with a person ill with influenza increased the risk of an ARI attributable to influenza up to 350%, depending on the season and the influenza virus circulating within the household.

KEYWORDS

epidemiology, household transmission, influenza

1 | INTRODUCTION

> In accepting the family as a semiclosed group one must believe that infections in successive family members within a short period of time are more likely to be related to each other than to have been separately acquired from outside sources. This is essentially an act of faith and nearly impossible to establish in terms of exact probability. –Carol Buck, 1956[1]

For almost a century, researchers have prospectively followed households to elucidate many of the intricacies of the natural history of influenza and other respiratory illnesses: the range of severity, the collection of symptoms, the diversity

Abbreviations: ARI, acute respiratory illness; CAR, community attack rate; MoSAIC, Mobile Surveillance for Acute Respiratory and Influenza-like Illness in the Community; RR, risk ratio; RT-PCR, reverse transcription polymerase chain reaction; SAR, secondary attack rate

of etiologies, the spatiotemporal trends, the incidence within subgroups, and the transmission within the household.[1-64] Additionally, case-ascertained household studies have sought to also answer similar questions about influenza by following the household contacts of a case series.[65-133] To summarize the transmissibility of influenza, studies of both types often report the secondary attack rate (SAR), a descriptive statistic that quantifies the risk of an exposed person becoming ill from an infectious person living within the same household.[1-24,65-72,73-104,105-124] The SAR is the *de facto* measure of transmissibility of influenza, and trials of antivirals have used SAR to quantify changes in the transmissibility of influenza attributable to a therapeutic regimen given to infectious household members or a prophylactic regimen given to susceptible household members.[13,14,21,66,71,72,111] Here, we estimate the SAR of influenza A and B virus using data from a prospective cohort in New York City during the 2013-2014 and 2014-2015 seasons.[55] Counting the number of cases within a household following the index case may overestimate the SAR of seasonal influenza, as household members may be infected outside the home, for example, at work or at school.[37,79,81,93,96,134-139] To avoid overcounting, we fit a compartmental model that quantifies both community transmission and household transmission of influenza.

2 | METHODS

The Mobile Surveillance for Acute Respiratory and Influenza like Illness in the Community (MoSAIC) study was a prospective cohort study enrolling households in New York City to investigate the incidence, etiology, and risk factors for acute respiratory infections.[55] Inclusion in the study required a household with at least three people, of whom at least 1 was a child under 18 years old. Investigators defined an episode of an acute respiratory illness (ARI) as the presence of at least two of the following symptoms: rhinorrhea, congestion, sore throat, cough, and fever. Investigators also included rhinorrhea among children under 1 year of age as an episode of ARI. Enrollees reported possible ARI within their household via text message. After confirming an episode of ARI over the telephone, researchers collected mid-turbinate nasal swabs from enrollees with an ARI and tested swabs for a panel of 20 viral and bacterial respiratory pathogens using PCR.[140] The institutional review boards of the Columbia University Medical Center (New York, NY) and the Centers for Disease Control and Prevention (Atlanta, GA) approved this study.

Here, we investigated a secondary objective of the study: to characterize the transmission of influenza within households enrolled in the study. We defined a case as an enrollee

1. who had a confirmed episode of ARI with onset of symptoms between October 5, 2013 and October 5, 2015 and
2. whose nasal swab was positive for influenza A, influenza A (H1), influenza A (H3), or influenza B by RT-PCR.

We divided the study period into two influenza seasons. The 2013-2014 influenza season started October 5, 2013 and ended September 27, 2014, and the 2014-2015 influenza season started September 28, 2014 and ended October 5, 2015. For the 2013-2014 and 2014-2015 seasons, we tabulated the number of households with 0, 1, 2, 3, 4, or 5 cases for

- influenza A (H1);
- influenza A (H3);
- influenza A (H1) or A (H3);
- influenza B; and,
- influenza A (H1), A (H3), or B.

For each of these season-virus combination, we considered enrollees at risk of influenza if they (1) were enrolled on or before the onset date of the first case for that season and (2) remained enrolled by the onset date of the last case of the season. Because investigators enrolled households into the cohort on a rolling basis, this time frame ensured all cases were included while also including as many households as possible. As this time frame was different for each season-virus combination, a different number of households was at risk for each analysis. The attack rate of influenza was the proportion of cases among those at risk. We used Wilson's interval to compute 95% confidence intervals for the attack rate.[141]

For the primary analysis of household transmission, we only considered pairs of cases both PCR-positive for the same type or subtype of influenza. As a secondary analysis of household transmission, we included additional episodes of ARI up to 10 days after the onset date of a PCR-positive case within the household, regardless of the test results for these additional episodes of ARI. The results from this secondary analysis simplify comparison of our results to previous work

which used a syndromic case definition for secondary cases. For both analyses, we separately analyzed cases of influenza A (H1), influenza A (H3), and influenza B to reduce spurious household transmission in the data. We computed 3-, 5-, and 7-day SARs by counting the number of apparent secondary cases among those at risk, as follows. For each household, we defined the index case (or index cases) as the enrollee(s) who met the case definition with the earliest onset date within that household. The enrollees at risk of secondary transmission were those enrollees who were not index cases but lived in a household with at least 1 case. We denoted the length of time at risk in days as r and consider $r = 3,\ 5,$ or 7. For our primary analysis, the r-day secondary cases were those cases with an onset date at least 1 day and at most r days following the date of onset of the index case within their household. For our secondary analysis, we also counted the episodes of ARI regardless of test result within 10 days of the onset of a case when counting secondary cases. The r-day SAR was the proportion of r-day secondary cases among those enrollees at risk of secondary transmission. We used Wilson's interval to compute 95% confidence intervals for the r-day SAR.[141]

We continued our analysis of SAR using Markov chain Monte Carlo methods. We modeled the transmission of influenza within our cohort using a transmission model—built on a susceptible-infectious-recovered (SIR) compartmental model—modified from the work of Cauchemez and colleagues.[125] Details follow in the remainder of this section.

2.1 | Household transmission model

We built a compartmental SIR model for the transmission of influenza into and within a cohort of households. We used the framework that Cauchemez and colleagues developed to analyze data from a case-ascertained study.[125] They assumed the length of the infectious period followed a gamma distribution and imputed the start and end of each case's infectious period. Additionally, they allowed for different susceptibility and infectiousness for children and adults. However, we needed to reduce the number of parameters in our model because cohort data have substantially fewer cases, and we wanted to avoid technical issues when imputing sparse data which leads to implausible results.[142] So, we assumed the shape parameter of the gamma distribution is 1; the infectious period began with symptom onset; and, we did not estimate separate parameters for children and adults. These simplifications allowed estimation with sparse data from a prospective cohort. In addition to information available from case-ascertained studies, the prospective cohort has richer information on the hazard from the community over time. So, we also wanted to take advantage of this information in the model.

Now, we make the following assumptions to begin building our model.

- The time to infection follows an exponential distribution with a time-dependent rate parameter.
- The time from infection to recovery follows an exponential distribution.
- The hazard from the community is proportional to the smoothed hazard within the cohort.
- The hazard within the household is proportional to the percent of household members who are infectious.
- The overall hazard is the sum of the community hazard and the household hazard.
- The observed onset dates are conditionally independent given the first case of the season, the overall hazard within the cohort, the onset dates of other household members, and the model parameters.
- The prior probability distribution of our parameters follows a uniform distribution on $\left[1 \times 10^{-2}, 1 \times 10^{3}\right]$.

In the remainder of this subsection, we will implement these assumptions to build a posterior, and we will detail our Metropolis algorithm.

2.1.1 | Hazard within the cohort

Here, we construct smoothed estimates of the hazard within the cohort for use in our model. In the next subsection, we will use these smoothed estimates when estimating the hazard within each household.

We take the earliest onset date of the season among laboratory confirmed cases as $t = 1$ and the last onset date of the season as day $t = T$. Then, we compute the Kaplan-Meier estimates of survival $\hat{S}(t)$ for $t = 0, 1, \ldots, T$: the probability of not meeting the case definition before time t.[143] We estimate the cummulative hazard for $t = 0, 1, \ldots, T$ with

$$\hat{H}(t) = -\log(\hat{S}(t)).$$

We estimate the instantaneous hazard for $t = 1, 2, \ldots, T$ with

$$\hat{h}(t) = \hat{H}(t) - \hat{H}(t-1).$$

We use LOESS to smooth our estimates of $\hat{h}$.[144] We name these locally smoothed estimates $\tilde{h}$.

2.1.2 | Hazard within the household

The time-dependent hazard within each household underlays the results from the model. Suppose we follow a cohort of households for T days and document the days on which household members first meet the case definition. For an individual, we take the sample space of the first day when a household member meets the case definition to be

$$\Omega = \{1, 2, 3, \ldots, T-1, T, \infty\},$$

where individuals who do not meet the case definition during followup are assigned an onset date of ∞. Suppose a household has n members. Then, we may write the set of observed onset dates for the household in increasing order as

$$A = \{a_1 \leq a_2 \leq \cdots \leq a_n\} \in \Omega^n.$$

We write the onset dates in order to simplify our indices. We fit three parameters to our data: α, β, and γ.

- If h is the instantaneous hazard within the cohort, then αh is the hazard attributable to community transmission.
- If π is the proportion of household members who are infectious, then $\beta\pi$ is the hazard attributable to household transmission.
- The rate of recovery among infectious individuals is γ.

Next, we need an estimate of the proportion of infectious household members at day t. For individual $i = 1, \ldots, n$, we define characteristic functions

$$\chi_i(t) = \begin{cases} 1 & \text{if } t > a_i \\ 0 & \text{if } t \leq a_i \end{cases}$$

The characteristic function reflects the fact that an individual is not infectious before their onset date. Using these characteristic functions, we define the expected proportion of infectious people in the household at time t as

$$\pi(t) = \frac{1}{n}\sum_{i=1}^{n} \chi_i(t) e^{-\gamma(t-a_i)},$$

where $e^{-\gamma(t-a_i)}$ is the probability that the ith case in the household is still infectious at time t for $t \geq a_i$. Finally, we may define the time-dependent hazard within the household:

$$\lambda(t) = \alpha\tilde{h}(t) + \beta\pi(t).$$

We summarize the model with Figure 1.

2.1.3 | Conditional probability of onset dates

We do not want the first case of the season to contribute to our likelihood. Instead, we want to condition our probability on the first case of the season. We use the symbol $\mathbf{1}$ to denote the fact that the season's index case is $1 \in \Omega$. Recall that $A = \{a_1 \leq a_2 \leq \cdots \leq a_n\} \in \Omega^n$ are ordered onset dates of a household with n people. Suppose $a \in A$ is the onset date of

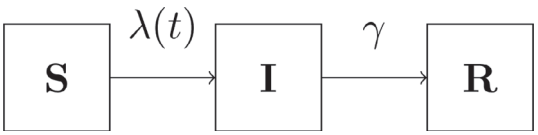

FIGURE 1 Compartmental SIR model. Susceptible individuals become infectious with a time-dependent, household-specific hazard $\lambda(t)$, and recover at a constant rate of γ

a household member. Because we want to condition our analysis on the season's index case, if $a = 1$, then the household member is an index case for the season. So, if $a = 1$, we take $p(a = 1|\mathbf{1}) = 1$. If $2 \leq a \leq T$, we take the conditional probability of onset on day a as

$$p(a|A \setminus \{a\}, \alpha\tilde{h}, \beta, \gamma, \mathbf{1}) = \int_{a-1}^{a} \lambda(t) e^{-\int_0^t \lambda(u)du} dt. \tag{1}$$

If $a = \infty$, we take the conditional probability of not meeting the case definition during followup as

$$p(a|A \setminus \{a\}, \alpha\tilde{h}, \beta, \gamma, \mathbf{1}) = \int_{T}^{\infty} \lambda(t) e^{-\int_0^t \lambda(u)du} dt. \tag{2}$$

The integrals in (1) and (2) are integrals of an exponential distribution with a time-dependent rate parameter λ. Because χ is not continuous for cases, λ is not continuous for households with at least one case. However, λ is continuous between onset dates. So, we want to rewrite the integrals in (1) and (2) so that we may compute them easily. For a household with d unique onset dates, we order the unique onset dates in increasing order $a_{i_1} < a_{i_2} < \cdots < a_{i_d}$. Then, we define a non-decreasing sequence of times $\{s_{2d+3}\}$ as $\left\{0, a_{i_1} - 1, a_{i_1}, \ldots, a_{i_d} - 1, a_{i_d}, T, \infty\right\}$. Now, for every $t \in \{s_{2d+3}\}$ we may compute the integral

$$\int_0^{t=s_j} \lambda(u)du = \sum_{k=1}^{j-1} \int_{s_k}^{s_{k+1}} \lambda(u)du. \tag{3}$$

Using (3) allows us to compute the integral in (1) for each onset date after day 1 and the integral in (2) for household members who do not meet the case definition during followup.

We assume conditional independence of onset dates within a household to estimate the conditional probability of onset dates A

$$p(A|\alpha\tilde{h}, \beta, \gamma, \mathbf{1}) = \prod_{l=1}^{n} p(a_l|A \setminus \{a_l\}, \alpha\tilde{h}, \beta, \gamma, \mathbf{1}).$$

Similarly, we assume conditional independence of onset dates between households in order to compute the conditional probability of the onset dates within the cohort. We index the households with $1, \ldots, m$. Then, we label the onset dates within the hth household as $A_h = (a_{h1}, a_{h2}, \ldots, a_{hn_h}) \in \Omega^{n_h}$. We define the onset dates for the cohort as $\mathcal{A} = \{A_1, \ldots, A_m\}$. The overall conditional probability of $\mathcal{A}$ is

$$p(\mathcal{A}|\alpha\tilde{h}, \beta, \gamma, \mathbf{1}) = \prod_{h=1}^{m} \prod_{l=1}^{n_h} p(a_{hl}|A_h \setminus \{a_{hl}\}, \alpha\tilde{h}, \beta, \gamma, \mathbf{1}).$$

In summary, we have a conditional probability of the observed onset dates. This is the likelihood we use when estimating our model parameters.

2.1.4 | Metropolis algorithm

We use Markov chain Monte Carlo methods to estimate $\alpha, \beta,$ and γ. Specifically, we use a stepwise Metropolis algorithm.[145] We use a uniform prior with a large support for our parameters

$$p(\alpha,\beta,\gamma) \propto \begin{cases} 1 & \text{if } \alpha,\beta,\gamma \in [10^{-2}, 10^{3}] \\ 0 & \text{otherwise} \end{cases}$$

Before the algorithm begins, we tune mixing parameters using small chains such that the acceptance of the Metropolis algorithm is about 1/2. Before the first iteration of the algorithm, we initialize the parameters using random draws from the uniform distribution on $[0.1, 1]$. Then, we compute the conditional probability of the onset dates within the cohort $\mathcal{A}$ given these parameters and given the date of the first observed case of the season, $p(\mathcal{A}|\alpha\tilde{h}, \beta, \gamma, \mathbf{1})$.

We begin the first iteration of our stepwise algorithm. To begin the first step, we take a random draw from a standard normal distribution and call this value u. Using u and the tuning parameter δ_α, we propose a new value for α as

$$\overline{\alpha} = \alpha e^{u\delta_\alpha}.$$

As $p(u) = p(-u)$, we have $p(\alpha|\overline{\alpha}) = p(-u) = p(u) = p(\overline{\alpha}|\alpha)$. So, our proposal distribution is symmetric. Next, we decide whether to accept or reject our proposal $\overline{\alpha}$.

- If $p(\overline{\alpha}, \beta, \gamma) = 0$, then we reject $\overline{\alpha}$.
- Otherwise, we compute $p(\mathcal{A}|\overline{\alpha}\ \tilde{h}, \beta, \gamma, \mathbf{1})$.
  - If $p(\mathcal{A}|\overline{\alpha}\ \tilde{h}, \beta, \gamma, \mathbf{1}) \geq p(\mathcal{A}|\alpha\tilde{h}, \beta, \gamma, \mathbf{1})$, then we accept $\overline{\alpha}$ as the new value of α.
  - Otherwise, we accept $\overline{\alpha}$ as the new value of α with probability

$$\frac{p(\mathcal{A}|\overline{\alpha}\ \tilde{h}, \beta, \gamma, \mathbf{1})}{p(\mathcal{A}|\alpha\tilde{h}, \beta, \gamma, \mathbf{1})}.$$

This completes the first step. For the second step, we propose $\overline{\beta}$ and either accept or reject using the same method as the first step. For the third step, we propose $\overline{\gamma}$ and either accept or reject using the same method as the first step. This completes the first iteration of the algorithm. The algorithm continues for 10^6 iterations.

2.1.5 | Estimates

We assume the parameter values from the Metropolis algorithm are a sample from the posterior probability distribution with density $p(\alpha, \beta, \gamma|\mathcal{A}, \mathbf{1})$. For each iterate, we compute the community attack rate (CAR) within the cohort by setting $\beta = 0$, and we have

$$1 - e^{-\int_0^T \alpha\tilde{h}(t)dt} = 1 - e^{-\sum_{t=1}^{T} \alpha\tilde{h}(t)}.$$

So, the CAR is the probability of influenza from the community during followup when no household members are infectious. We compute the SAR within a household of size n by setting $\alpha = 0$, and we have

$$1 - e^{-\int_0^\infty \frac{\beta}{n} e^{-\gamma t} dt} = 1 - e^{-\frac{\beta}{n\gamma}}.$$

So, the SAR is the probability of influenza from a single infectious household member when no community members are infectious.

For each iterate, we compute the relative attack rate of influenza within a household

- with a single infectious member and
- $n - 1$ susceptible members

TABLE 1 Number of households with 0, 1, 2, 3, 4, or 5 cases; number of cases; number of people enrolled; and attack rate by virus and season

Influenza virus	Season	Households enrolled	Cases in household						Cases	People enrolled	Attack rate % (95% CI[a])
			0	1	2	3	4	5			
A and B	2013-2014	230	189	34	6	1			49	1044	4.7 (3.6, 6.2)
A and B	2014-2015	238	206	19	10	2	0	1[b]	50	1063	4.7 (3.6, 6.1)
B	2013-2014	234	217	14	2	1			21	1062	2.0 (1.3, 3.0)
B	2014-2015	238	234	4					4	1064	0.4 (0.1, 1.0)
A	2013-2014	233	207	23	3				29	1067	2.7 (1.9, 3.9)
A	2014-2015	242	212	18	10	1	1		45	1088	4.1 (3.1, 5.5)
A (H1)	2013-2014	241	223	16	2				20	1144	1.7 (1.1, 2.7)
A (H1)	2014-2015	242[c]	242						0	1088[c]	0 (0, 0.4)[c]
A (H3)	2013-2014	233	225	8					8	1067	0.7 (0.4, 1.5)
A (H3)	2014-2015	242	213	21	6	1	1		40	1088	3.7 (2.7, 5.0)

[a] Confidence interval.
[b] One enrollee met the case definition twice: once for influenza A (H3) and once for influenza B.
[c] Based on the number of people enrolled in the cohort for the influenza A (H3) analysis during 2014-2015.

vs. within a household

- without any infectious people and
- of size n

using the formula

$$\mathrm{RR} = \frac{1 - e^{-\int_0^\infty \frac{\beta}{n} e^{-\gamma t} dt - \int_0^T \alpha \tilde{h}(t) dt}}{1 - e^{-\int_0^T \alpha \tilde{h}(t) dt}} = \frac{1 - e^{-\frac{\beta}{n\gamma} - \sum_{t=1}^T \alpha \tilde{h}(t)}}{1 - e^{-\sum_{t=1}^T \alpha \tilde{h}(t)}}.$$

So, the denominator of our RR is simply the CAR. The numerator of the RR is the risk of influenza: the competing risk from the community and from a single infectious household member.

We use the median value as our point estimate. We create 95% credible intervals with the 2.5% and 97.5% quantiles.

2.2 | Computer software

We used R version 3.5.1"Feather Spray" (Copyright 2018 The R Foundation for Statistical Computing) and GCC version 4.9.3 (Copyright 2015 Free Software Foundation, Inc.) for all computations. We used the R package Rcpp to execute our C++ functions within R.[146]

3 | RESULTS

The number of households enrolled for our analyses ranged from a low of 230 for influenza A and B in 2013-2014 to a high of 242 for influenza A (H3) in 2014-2015 (Table 1). During the 2014-2015 season, a single enrollee met the case definition twice: once for influenza A (H3) and once for influenza B. Otherwise, enrollees met the case definition at most once per season. The attack rate of influenza A and B was 4.7% during 2013-2014 and 4.7% during 2014-2015 seasons (Table 1). Most cases were attributable to community transmission (Tables 1 and 2). Two households had at least 2 cases of influenza A (H1) in 2013-2014; 8 households had at least 2 cases of influenza A (H3) in 2014-2015; and 3 household had at least 2 cases of influenza B in 2013-2014 (Table 1).

TABLE 2 Attack rate, community attack rate, and secondary attack rate for seasons and influenza virus with at least one case

Influenza virus	Season	Attack rate % (95% CI[c])	Community attack rate % (95% CI[b])	Secondary attack rate[a] % (95% CI[b])	Relative attack rate[a] RR (95% CI[b])
A (H1)	2013-2014	1.7 (1.1, 2.7)	1.5 (0.9, 2.3)	2.3 (0.3, 7.7)	2.6 (1.2, 6.8)
A (H3)	2013-2014	0.7 (0.4, 1.5)	0.6 (0.3, 1.2)	0.2 (0.0, 6.8)	1.3 (1.0, 13.3)
A (H3)	2014-2015	3.7 (2.7, 5.0)	2.7 (1.8, 3.8)	7.6 (3.7 13.3)	3.8 (2.2, 6.5)
B	2013-2014	2.0 (1.3, 3.0)	1.5 (0.9, 2.4)	5.3 (1.5, 12.7)	4.5 (1.9, 10.9)
B	2014-2015	0.4 (1.3, 3.0)	0.3 (0.1, 0.7)	0.3 (0.0, 14.3)	2.3 (1.0, 78.9)

Note: The relative attack rate is the attack rate of influenza in households with a single case relative to the attack rate of influenza in households with no cases.

[a] Computed for the average household size.

[b] Credible interval.

[c] Confidence interval.

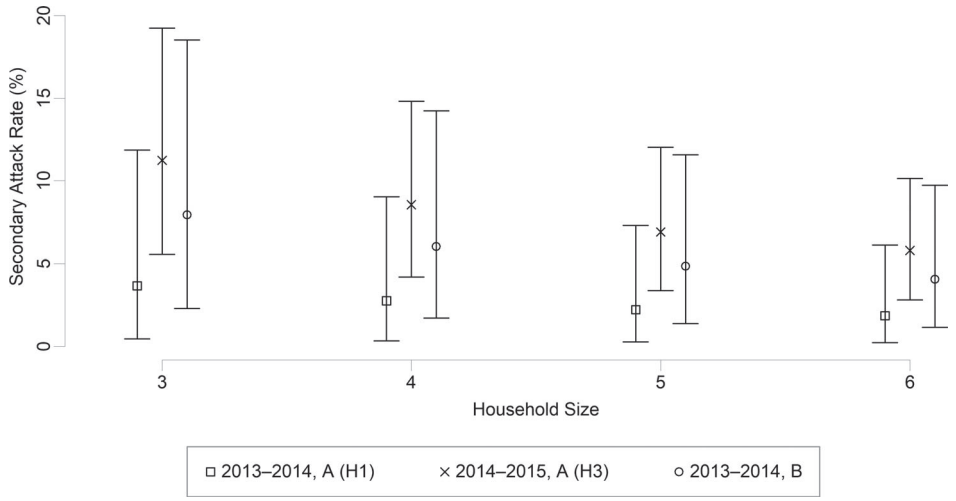

FIGURE 2 The estimated secondary attack rate by household size for influenza by selected type and subtype during 2013-2014 and 2014-2015. Vertical bars represent 95% credible intervals

Secondary attack rates varied depending on the season and the circulating virus type (Figure 2 and Table 2). Similarly, the CAR varied by season and circulating virus type (Figure 3 and Table 2). For a household of average size (4.7 people), the SAR of influenza A (H1) was 2.3% during 2013-2014 (Table 2). Similarly, the SAR of influenza A (H3) was 7.6% during 2014-2015 for a household of average size (4.5 people). The SAR of influenza B was 5.3% during 2013-2014 for a household of average size (4.5 people).

For an average-sized household during 2013-2014, if a household member became infectious with influenza A (H1), then the attack rate of influenza for the other household members increased 160% (Table 2). Similarly, the attack rate increased by 280% during 2014-2015 if a household member became infectious with influenza A (H3). The attack rate increased by 350% during 2013-2014 if a household member became infectious with influenza B.

The estimates of the SAR from the transmission model differed from the 3-day, 5-day, and 7-day r-day SAR (Table 3). As expected, estimates of SAR from the analysis, which also included episodes of ARI regardless of test result, were higher than when limited to only laboratory-confirmed cases (Table 4).

Estimates of model parameters are in Table 5. The 95% credible intervals of β and γ were wide, especially for those analyses with no apparent secondary cases. The point estimates and 95% credible intervals for α scale the overall hazard within the cohort, which we present for those analyses with at least one apparent secondary case (Figure 3).

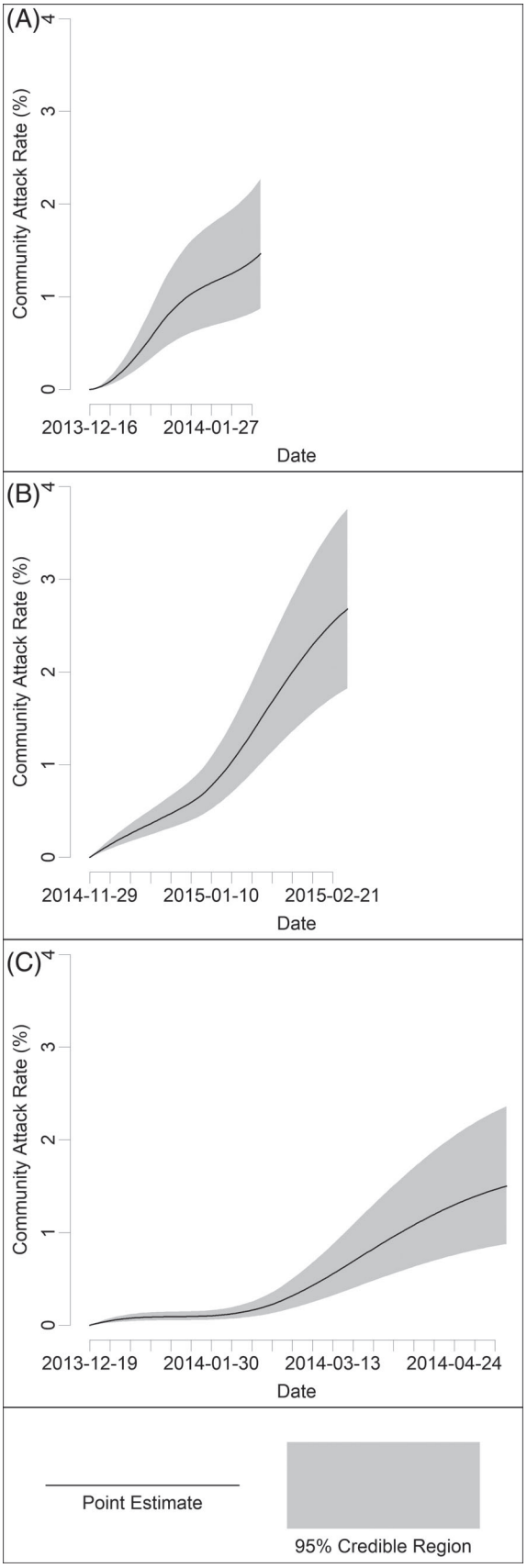

FIGURE 3 The estimated community attack rate over the followup period of (**A**) influenza A (H1) during 2013-2014, (**B**) influenza A (H3) during 2014-2015, and (**C**) influenza B during 2013-2014. The black lines represent point estimates, and the shaded areas represent 95% credible regions

TABLE 3 Comparing secondary attack rate of PCR confirmed influenza from the transmission model with 3-, 5-, and 7-day *r*-day secondary attack rate

Influenza virus	Season	Transmission model secondary attack rate [a] % (95% CI[b])	*r*-day secondary attack rate		
			3-day % (95% CI[c])	5-day % (95% CI[c])	7-day % (95% CI[c])
A (H1)	2013-2014	2.3 (0.3, 7.7)	2.5 (0.7, 8.8)	2.5 (0.7, 8.8)	2.5 (0.7, 8.8)
A (H3)	2013-2014	0.2 (0.0, 6.8)	0.0 (0.0, 12.9)	0.0 (0.0, 12.9)	0.0 (0.0, 12.9)
A (H3)	2014-2015	7.6 (3.7 13.3	7.4 (3.8, 13.9)	9.2 (5.1, 16.2)	9.2 (5.1, 16.2)
B	2013-2014	5.3 (1.5, 12.7)	3.4 (1.0, 11.7)	5.2 (1.8, 14.1)	6.9 (2.7, 16.4)
B	2014-2015	0.3 (0.0, 14.3)	0.0 (0.0, 24.2)	0.0 (0.0, 24.2)	0.0 (0.0, 24.2)

[a]Computed for the average household size.
[b]Credible interval
[c]Confidence interval

TABLE 4 Comparing secondary attack rate of episodes of ARI regardless of test results from the transmission model with 3-, 5-, 7-, and 10-day *r*-day secondary attack rate

Influenza virus	Season	Transmission model secondary attack rate[a] % (95% CI[b])	*r*-day secondary attack rate			
			3-day % (95% CI[c])	5-day % (95% CI[c])	7-day % (95% CI[c])	10-day % (95% CI[c])
A (H1)	2013-2014	5.7 (2.0, 12.2)	6.3 (2.7, 14.0)	6.3 (2.7, 14.0)	6.3 (2.7, 14.0)	6.3 (2.7, 14.0)
A (H3)	2013-2014	0.2 (0.0, 6.8)	0.0 (0.0, 12.9)	0.0 (0.0, 12.9)	0.0 (0.0, 12.9)	0 (0.0, 12.9)
A (H3)	2014-2015	13.5 (8.6, 19.7)	12.0 (7.2, 19.5)	16.7 (10.8, 24.8)	17.6 (11.6, 25.8)	19.4 (13.1, 27.9)
B	2013-2014	6.5 (2.1, 14.0)	5.2 (1.8, 14.1)	6.9 (2.7, 16.4)	8.6 (3.7, 18.6)	8.6 (3.7, 18.6)
B	2014-2015	5.6 (0.2, 26.0)	8.3 (1.5, 35.4)	8.3 (1.5, 35.4)	8.3 (1.5, 35.4)	8.3 (1.5, 35.4)

[a]Computed for the average household size.
[b]Credible interval.
[c]Confidence interval.

TABLE 5 Summary of parameter estimates from the household transmission models

Season	Virus	Case definition	α (95% CI)	β (95% CI)	γ (95% CI)
2013-2014	A (H1)	PCR confirmed	0.82 (0.48, 1.29)	3.3 (0.04, 139)	33 (0.7, 845)
2013-2014	A (H3)	PCR confirmed	0.80 (0.33, 1.58)	0.16 (0.01, 39)	59 (0.1, 886)
2014-2015	A (H1)	PCR confirmed	0.73 (0.49, 1.03)	0.23 (0.08, 0.56)	0.65 (0.29, 1.29)
2013-2014	B	PCR confirmed	0.73 (0.42, 1.16)	0.06 (0.01, 0.22)	0.24 (0.06, 0.63)
2014-2015	B	PCR confirmed	0.60 (0.13, 1.63)	0.20 (0.01, 78)	46.9 (0.09, 878))
2013-2014	A (H1)	ARI episode	0.72 (0.43, 1.11) 11.0	(0.3, 282)	40.5 (1.4, 845)
2013-2014	A (H3)	ARI episode	0.80 (0.33, 1.58) 0.16	(0.01, 41)	59.3 (0.1, 886)
2014-2015	A (H1)	ARI episode	0.60 (0.41, 0.83) 0.29	(0.14, 0.54)	0.44 (0.25, 0.74)
2013-2014	B	ARI episode	0.70 (0.41, 1.11)	0.09 (0.01, 0.29)	0.30 (0.09, 0.71)
2014-2015	B	ARI episode	0.47 (0.10, 1.29)	4.9 (0.03, 386)	24.3 (0.2, 828)

4 | DISCUSSION

We found an attack rate of 4.7% for influenza illness each season and a SAR of 2.3% for influenza A (H1) in 2013-2014, 5.3% for influenza B in 2013-2014, and 7.6% for influenza A (H3) 2014-2015. When the first person in an average sized household was infected with influenza, the attack rate increased up to 350% for the rest of the household, depending on the season and virus circulating in the home. Our results are qualitatively similar to published estimates of the attack rate and SAR of PCR-confirmed influenza infections from household cohort studies in the United States. The Flu Watch cohort study reported the attack rate was on average 4% per season for the 2006-2007 through 2008-2009 seasons.[53] The Household Influenza Vaccine Effectiveness (HIVE) study reported SARs of 2.9% for influenza A (H1), 7.7% for influenza B, and 15.3% for influenza A (H3) during the 2010-2011 season and reported an attack rate of 8.7%.[18] For the 2014-2015 season, the HIVE study reported a SAR of 17% for influenza A (H3), a SAR of 6% for influenza B, and an attack rate of 12%.[23]

When we included episodes of ARI regardless of test results, our estimates of SAR often increased substantially. For example, the estimate of SAR for influenza A (H3) during 2014-2015 increased from 7.6% to 13.5% by including additional episodes of ARI regardless of test result. While we know that including all episodes of ARI likely overestimates the SAR, the opposite is true when we restricted analysis to laboratory-confirmed secondary cases. The more inclusive definition of a secondary case allows comparison with previously published work from case ascertained household studies. The earliest reports from the United States on the household transmission of pandemic H1N1 2009 influenza reported a SAR of 13% and 27.3%.[76,147] Subsequent reports of estimates of the SAR ranged from 3.5% to 22.8%; and, our analogous estimate of SAR of influenza A (H1) during 2013-2014 was 5.7%, which is in the middle of this range.[81,82,85,93,94,148]

As accurate counts of the number of secondary cases are rarely available, researchers rely on statistical methods to estimate SARs. If we relied only on counting apparent secondary cases, then neither the 3-, nor 5-, nor 7-day SAR could capture quantitatively similar estimates as our method. In effect, shortening the time at risk for these SARs corrects for over counting, but specifying the optimal time interval is difficult. Specifically, the number of uncounted cases attributable to household transmission must equal the number of over counted cases attributable to community transmission. While the 3-, 5-, or 7-day SAR was similar to that from the transmission model for influenza A (H1) during 2013-2014, neither the 3-, 5-, nor 7- day SAR was similar to the SAR from the transmission model for both influenza A (H3) during 2014-2015 and influenza B during 2013-2014 (Table 3). As risk from the community relative to the risk from the household increases, this bias increases. Using the model-based approach obviates choosing the appropriate length of time at risk for household transmission, in exchange for a more cumbersome process of estimation. Additionally, the model-based approach provides credible intervals for the CAR and the SAR, while simultaneously adjusting for uncertainty in both.

Our results may not be generalizable. In order to participate in this study, households must have at least one child. The dynamics of influenza transmission in homes without children may be qualitatively different than what we present here.[76] In order to participate in this study, households must have at least 3 people. Smaller households account for about 60% of households in the United States (U.S. Census Bureau, 2012-2016 American Community Survey 5-Year Estimates). Our case definition relied on molecular evidence of influenza virus in a nasal swab as detected by RT-PCR using a benchtop system.[140] While highly specific, our methods may not have detected every infection with influenza virus among enrollees with an ARI. Our study did not capture asymptomatic infections with influenza, which would violate our assumptions that enrollees who never met the case definition remained susceptible for the entire influenza season. Although we analyzed the data separately for cases of influenza A (H1), influenza A (H3), and influenza B, more than one influenza virus may have circulated in some households. For example, most of the circulating influenza A (H3) viruses belonged to the 3C.2a genetic group during the 2014-2015 season in New York, but influenza A (H3) viruses from other genetic groups also circulated within the state.[148]

Estimates of the transmissibility of influenza, measured by SAR within a household, ranges widely over recent history. The estimates we present here reinforce the variation of the expected transmissibility of influenza viruses circulating in the population from historical data (Table S3). Future data about this cohort may enable analyses which account for differential susceptibility within the cohort, for example, by extending the compartmental model to allow for immunity. The changing susceptibility of the population to the circulating influenza viruses could explain part of the observed dynamics of the transmissibility of influenza. Quantifying this relationship could strengthen public health messaging about preventing influenza.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data are not available due to privacy or ethical restrictions.

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REFERENCES

1. Buck C. Acute upper respiratory infections in families. *Am J Hyg*. 1956;63(1):1-12. https://doi.org/10.1093/oxfordjournals.aje.a119786
2. Lidwell OM, Sommerville T. Observations on the incidence and distribution of the common cold in a rural community during 1948 and 1949. *J Hyg (Lond)*. 1951;49(4):365-381. https://doi.org/10.1017/s0022172400066699
3. Badger GF, Dingle JH, Feller AE, Hodges RG, Jordan J, Rammelkamp J. A study of illness in a group of Cleveland families. IV. the spread of respiratory infections within the home. *Am J Hyg*. 1953;58(2):174-178. https://doi.org/10.1093/oxfordjournals.aje.a119598
4. Brimblecombe FSW, Cruickshank R, Masters PL, Reid DD, Stewart GT. Family studies of respiratory infections. *Br Med J*. 1958;1(5063):119-128. https://doi.org/10.1136/bmj.1.5063.119
5. Hall CE, Cooney MK, Fox JP. The seattle virus watch. IV. comparative epidemiologic observations of infections with influenza A and B viruses, 1965–1969, in families with young children. *Am J Epidemiol*. 1973;98(5):365-380. https://doi.org/10.1093/oxfordjournals.aje.a121566
6. Foy HM, Cooney MK, Allan I. Longitudinal studies of types A and B influenza among Seattle schoolchildren and families, 1968–1974. *J Infect Dis*. 1976;134(4):362-369. https://doi.org/10.1093/infdis/134.4.362
7. Jennings LC, Miles JAR. A study of acute respiratory disease in the community of Port Chalmers. II. influenza A/Port Chalmers/1/73: intrafamilial spread and the effect of antibodies to the surface antigens. *J Hyg (Lond)*. 1978;81(1):67-75. https://doi.org/10.1017/s0022172400053778
8. The Royal College of General Practitioners. The Public Health Laboratory Service. Long-term study of influenza in families. *J R Coll Gen Pract*. 1981;31(227):351-356.
9. Fox JP, Cooney MK, Hall CE, Foy HM. Influenzavirus infections in Seattle families, 1975–1979. II. pattern of infection in invaded households and relation of age and prior antibody to occurrence of infection and related illness. *Am J Epidemiol*. 1982;116(2):228-242. https://doi.org/10.1093/oxfordjournals.aje.a113408
10. Longini J, Koopman JS, Monto AS, Fox JP. Estimating household and community transmission parameters for influenza. *Am J Epidemiol*. 1982;115(5):736-751. https://doi.org/10.1093/oxfordjournals.aje.a113356
11. Frank AL, Taber LH, Glezen WP, Geyer EA, McIlwain S, Paredes A. Influenza B virus infections in the community and the family. the epidemics of 1976–1977 and 1979–1980 in Houston, Texas. *Am J Epidemiol*. 1983;118(3):313-325. https://doi.org/10.1093/oxfordjournals.aje.a113638
12. Longini J, Monto AS. Efficacy of virucidal nasal tissues in interrupting familial transmission of respiratory agents. a field trial in Tecumseh, Michigan. *Am J Epidemiol*. 1988;128(3):639-644. https://doi.org/10.1093/oxfordjournals.aje.a115011
13. Hayden FG, Gubareva LV, Monto AS, et al. Inhaled Zanamivir for the prevention of influenza in families. *N Engl J Med*. 2000;343(18):1282-1289. https://doi.org/10.1056/NEJM200011023431801
14. Hayden FG, Belshe R, Villanueva C, et al. Management of influenza in households: a prospective, randomized comparison of oseltamivir treatment with or without postexposure prophylaxis. *J Infect Dis*. 2004;189(3):440-449. https://doi.org/10.1086/381128
15. Klick B, Nishiura H, Ng S, et al. Transmissibility of seasonal and pandemic influenza in a cohort of households in Hong Kong in 2009. *Epidemiology*. 2011;22(6):793-796. https://doi.org/10.1097/EDE.0b013e3182302e8e
16. Nukiwa-Souma N, Burmaa A, Kamigaki T, et al. Influenza transmission in a community during a seasonal influenza A(H3N2) outbreak (2010–2011) in Mongolia: a community-based prospective cohort study. *PLoS One*. 2012;7(3):e33046. https://doi.org/10.1371/journal.pone.0033046

17. Ohmit SE, Petrie JG, Malosh RE, et al. Influenza vaccine effectiveness in the community and the household. *Clin Infect Dis*. 2013;56(10):1363-1369. https://doi.org/10.1093/cid/cit060
18. Petrie JG, Ohmit SE, Cowling BJ, et al. Influenza transmission in a cohort of households with children: 2010-2011. *PLoS One*. 2013;8(9):e75339. https://doi.org/10.1371/journal.pone.0075339
19. Cauchemez S, Ferguson NM, Fox A, et al. Determinants of influenza transmission in South East Asia: insights from a household cohort study in Vietnam. *PLoS Pathog*. 2014;10(8):e1004310. https://doi.org/10.1371/journal.ppat.1004310
20. Thai PQ, Mai LQ, Welkers MRA, et al. Pandemic H1N1 virus transmission and shedding dynamics in index case households of a prospective Vietnamese cohort. *J Infect*. 2014;68(6):581-590. https://doi.org/10.1016/j.jinf.2014.01.008
21. Fry AM, Goswami D, Nahar K, et al. Effects of oseltamivir treatment of index patients with influenza on secondary household illness in an urban setting in Bangladesh: secondary analysis of a randomised, placebo-controlled trial. *Lancet Infect Dis*. 2015;15(6):654-662. https://doi.org/10.1016/S1473-3099(15)70041-1
22. Judd MC, Emukule GO, Njuguna H, et al. The role of HIV in the household introduction and transmission of influenza in an urban slum, Nairobi, Kenya, 2008–2011. *J Infect Dis*. 2015;212(5):740-744. https://doi.org/10.1093/infdis/jiv106
23. Petrie JG, Malosh RE, Cheng CK, et al. The household influenza vaccine effectiveness study: lack of antibody response and protection following receipt of 2014–2015 influenza vaccine. *Clin Infect Dis*. 2017;65(10):1644-1651. https://doi.org/10.1093/cid/cix608
24. Buchwald AG, Tamboura B, Haidara FC, et al. Maternal influenza vaccination and the risk of laboratory-confirmed influenza among household contacts under the age of five in Mali. *Am J Trop Med Hyg*. 2019;100(1):159-164. https://doi.org/10.4269/ajtmh.18-0450
25. Malosh RE, Noppert GA, Zelner J, Martin ET, Monto AS. Social patterning of acute respiratory illnesses in the Household Influenza Vaccine Evaluation (HIVE) study 2014–2015. *Epidemiol Infect*. 2019;147:e185. https://doi.org/10.1017/S0950268819000748
26. Ranjeva S, Subramanian R, Fang VJ, et al. Age-specific differences in the dynamics of protective immunity to influenza. *Nat Commun*. 2019;10(1):1660. https://doi.org/10.1038/s41467-019-09652-6
27. Sydenstricker E. The incidence of illness in a general population group. General results of a morbidity study from December 1, 1921, through March 31, 1924, in Hagerstown, MD. *Public Health Rep*. 1925;40(7):279-293.
28. Sydenstricker E. A study of illness in a general population group. Hagerstown morbidity studies no. I: the method of study and general results. *Public Health Rep*. 1926;41(39):2069-2088.
29. Van Volkenburgh VA, Frost WH. Acute minor respiratory diseases prevailing in a group of families residing in Baltimore, Maryland, 1928–1930. Prevalence, distribution and clinical description of observed cases. *Am J Epidemiol*. 1933;17(1):122-153. https://doi.org/10.1093/oxfordjournals.aje.a117900
30. Dingle JH, Badger GF, Feller AE, Hodges RG, Jordan J, Rammelkamp J. A study of respiratory infections in families. *Trans Assoc Am Phys*. 1949;62:99-104.
31. Tucher D, Coulter JE, Downes J. Incidence of acute respiratory illness among males and females at specific ages; study no. 5. *Milbank Mem Fund Q*. 1952;30(1):42-60.
32. Badger GF, Dingle JH, Feller AE, Hodges RG, Jordan J, Rammelkamp J. A study of illness in a group of Cleveland families. V. Introductions and secondary attack rates as indices of exposure to common respiratory diseases in the community. *Am J Hyg*. 1953;58(2):179-182. https://doi.org/10.1093/oxfordjournals.aje.a119599
33. Dingle JH, Badger GF, Feller AE, Hodges RG, Jordan J, Rammelkamp J. A study of illness in a group of Cleveland families. I. Plan of study and certain general observations. *Am J Hyg*. 1953;58(1):16-30. https://doi.org/10.1093/oxfordjournals.aje.a119587
34. Jordan J, Badger GF, Dingle JH. A study of illness in a group of Cleveland families. XVI. The epidemiology of influenza, 1948-1953. *Am J Hyg*. 1958;68(2):169-189. https://doi.org/10.1093/oxfordjournals.aje.a119961
35. Dunn FL, Carey DE, Cohen A, Martin JD. Epidemiologic studies of Asian influenza in a Louisiana parish. *Am J Hyg*. 1959;70:351-371. https://doi.org/10.1093/oxfordjournals.aje.a120083
36. Philip RN, Bell JA, Davis DJ, et al. Epidemiologic studies on influenza in familial and general population groups, 1951–1956. II. Characteristics of occurrence. *Am J Hyg*. 1961;73:123-137. https://doi.org/10.1093/oxfordjournals.aje.a120171
37. Dingle JH, Badger GF, Jordan J. *Illness in the Home. A Study of 25,000 Illnesses in a Group of Cleveland Families*. Cleveland: The Press of Western Reserve University; 1964.
38. Fox JP, Elveback LR, Spigland I, Frothingham TE, Sevens DA, Huger M. The virus watch program: a continuing surveillance of viral infections in metropolitan New York families. I. Overall plan, methods of collecting and handling information and a summary report of specimens collected and illnesses observed. *Am J Epidemiol*. 1966;83(3):389-412. https://doi.org/10.1093/oxfordjournals.aje.a120594
39. Spigland I, Fox JP, Elveback LR, et al. The Virus Watch program: a continuing surveillance of viral infections in metropolitan New York families. II. Laboratory methods and preliminary report on infections revealed by virus isolation. *Am J Epidemiol*. 1966;83(3):413-435. https://doi.org/10.1093/oxfordjournals.aje.a120595
40. Elveback LR, Fox JP, Ketler A, Brandt CD, Wassermann FE, Hall CE. The Virus Watch program: a continuing surveillance of viral infections in metropolitan New York families. III. Preliminary report on association of infections with disease. *Am J Epidemiol*. 1966;83(3):436-454. https://doi.org/10.1093/oxfordjournals.aje.a120596
41. Hall CE, Brandt CD, Frothingham TE, Spigland I, Cooney MK, Fox JP. The virus watch program: a continuing surveillance of viral infections in metropolitan New York families. IX. A comparison of infections with several respiratory pathogens in New York and New Orleans families. *Am J Epidemiol*. 1971;94(4):367-385. https://doi.org/10.1093/oxfordjournals.aje.a121332
42. Monto AS, Napier JA, Metzner HL. The Tecumseh study of respiratory illness. I. Plan of study and observations on syndromes of acute respiratory disease. *Am J Epidemiol*. 1971;94(3):269-279. https://doi.org/10.1093/oxfordjournals.aje.a121320

43. Monto AS, Cavallaro JJ. The Tecumseh study of respiratory illness. II. Patterns of occurrence of infection with respiratory pathogens, 1965–1969. *Am J Epidemiol*. 1971;94(3):280-289. https://doi.org/10.1093/oxfordjournals.aje.a121321
44. Fox JP, Hall CE, Cooney MK, Luce RE, Kronmal RA. The Seattle virus watch. II. Objectives, study population and its observation, data processing and summary of illnesses. *Am J Epidemiol*. 1972;96(4):270-285. https://doi.org/10.1093/oxfordjournals.aje.a121458
45. Cooney MK, Hall CE, Fox JP. The Seattle virus watch. III. Evaluation of isolation methods and summary of infections detected by virus isolations. *Am J Epidemiol*. 1972;96(4):286-305. https://doi.org/10.1093/oxfordjournals.aje.a121459
46. Monto AS, Higgins MW, Ross HW. The Tecumseh study of respiratory illness. VIII. Acute infection in chronic respiratory disease and comparison groups. *Am Rev Respir Dis*. 1975;111(1):27-36. https://doi.org/10.1164/arrd.1975.111.1.27
47. Frank AL, Taber LH, Glezen WP, Paredes A, Couch RB. Reinfection with influenza A (H3N2) virus in young children and their families. *J Infect Dis*. 1979;140(6):829-836. https://doi.org/10.1093/infdis/140.6.829
48. Taber LH, Paredes A, Glezen WP, Couch RB. Infection with influenza A/Victoria virus in Houston families, 1976. *J Hyg (Lond)*. 1981;86(3):303-313. https://doi.org/10.1017/s0022172400069059
49. Monto AS, Koopman JS, Longini J. Tecumseh study of illness. XIII. Influenza infection and disease, 1976-1981. *Am J Epidemiol*. 1985;121(6):811-822. https://doi.org/10.1093/oxfordjournals.aje.a114052
50. Monto AS, Sullivan KM. Acute respiratory illness in the community. Frequency of illness and the agents involved. *Epidemiol Infect*. 1993;110(1):145-160. https://doi.org/10.1017/s0950268800050779
51. Carrat F, Sahler C, Rogez S, et al. Influenza burden of illness: estimates from a national prospective survey of household contacts in France. *Arch Intern Med*. 2002;162(16):1842-1848. https://doi.org/10.1001/archinte.162.16.1842
52. Horby P, Mai LQ, Fox A, et al. The epidemiology of interpandemic and pandemic influenza in Vietnam, 2007–2010: the Ha Nam household cohort study I. *Am J Epidemiol*. 2012;175(10):1062-1074. https://doi.org/10.1093/aje/kws121
53. Hayward AC, Fragaszy EB, Bermingham A, et al. Comparative community burden and severity of seasonal and pandemic influenza: results of the Flu Watch cohort study. *Lancet Respir Med*. 2014;2(6):445-454. https://doi.org/10.1016/S2213-2600(14)70034-7
54. Monto AS, Malosh RE, Petrie JG, Thompson MG, Ohmit SE. Frequency of acute respiratory illnesses and circulation of respiratory viruses in households with children over 3 surveillance seasons. *J Infect Dis*. 2014;210(11):1792-1799. https://doi.org/10.1093/infdis/jiu327
55. Stockwell MS, Reed C, Vargas CY, et al. MoSAIC: mobile surveillance for acute respiratory infections and influenza-like illness in the community. *Am J Epidemiol*. 2014;180(12):1196-1201. https://doi.org/10.1093/aje/kwu303
56. Delabre RM, Lapidus N, Salez N, Mansiaux Y, de Lamballerie X, Carrat F. Risk factors of pandemic influenza A/H1N1 in a prospective household cohort in the general population: results from the CoPanFlu-France cohort. *Influenza Other Respir Viruses*. 2015;9(1):43-50. https://doi.org/10.1111/irv.12294
57. Mansiaux Y, Salez N, Lapidus N, et al. Causal analysis of H1N1pdm09 influenza infection risk in a household cohort. *J Epidemiol Community Health*. 2015;69(3):272-277. https://doi.org/10.1136/jech-2014-204678
58. Ip DKM, Lau LLH, Chan KH, et al. The dynamic relationship between clinical symptomatology and viral shedding in naturally acquired seasonal and pandemic influenza virus infection. *Clin Infect Dis*. 2016;62(4):431-437. https://doi.org/10.1093/cid/civ909
59. Smithgall M, Vargas CY, Reed C, et al. Influenza vaccine effectiveness in a low-income, urban community cohort. *Clin Infect Dis*. 2016;62(3):358-360. https://doi.org/10.1093/cid/civ867
60. Vargas CY, Wang L, de Belliard YC, et al. Pilot study of participant-collected nasal swabs for acute respiratory infections in a low-income, urban population. *Clin Epidemiol*. 2016;8:1-5. https://doi.org/10.2147/CLEP.S95847
61. Zachariah P, Whittier S, Reed C, et al. Community -and hospital laboratory-based surveillance for respiratory viruses. *Influenza Other Respir Viruses*. 2016;10(5):361-366. https://doi.org/10.1111/irv.12387
62. Petrie JG, Eisenberg MC, Ng S, et al. Application of an individual-based transmission hazard model for estimation of influenza vaccine effectiveness in a household cohort. *Am J Epidemiol*. 2017;186(12):1380-1388. https://doi.org/10.1093/aje/kwx217
63. Tinoco YO, Azziz-Baumgartner E, Uyeki TM, et al. Burden of influenza in 4 ecologically distinct regions of Peru: household active surveillance of a community cohort, 2009–2015. *Clin Infect Dis*. 2017;65(9):1532-1541. https://doi.org/10.1093/cid/cix565
64. Ainslie KEC, Haber MJ, Malosh RE, Petrie JG, Monto AS. Maximum likelihood estimation of influenza vaccine effectiveness against transmission from the household and from the community. *Stat Med*. 2018;37(6):970-982. https://doi.org/10.1002/sim.7558
65. Hope Simpson RE, Sutherland I. Does influenza spread within the household? *Lancet*. 1954;266(6814):721-726. https://doi.org/10.1016/s0140-6736(54)92126-1
66. U.S. National Communicable Disease Center Respiratory Viral Diseases Unit, U.S. National Communicable Disease Center Laboratory Branch Respiratory Virology Unit, United States Public Health Service Office of the Surgeon General, Advisory Committee on Immunization Practice, World Health Organization International Influenza Center for the Americas. Influenza-respiratory disease surveillance report number 85. *MMWR Morb Mortal Wkly Rep*. 1969;18(25):22-32.
67. Hope-Simpson RE. First outbreak of Hong Kong influenza in a general practice population in Great Britain. A field and laboratory study. *Br Med J*. 1970;3(5714):74-77. https://doi.org/10.1136/bmj.3.5714.74
68. Hope-Simpson RE. Epidemic mechanisms of type A influenza. *J Hyg (Lond)*. 1979;83(1):11-26. https://doi.org/10.1017/s002217240002578x
69. Katz JM, Lim W, Bridges CB, et al. Antibody response in individuals infected with avian influenza A (H5N1) viruses and detection of anti-H5 antibody among household and social contacts. *J Infect Dis*. 1999;180(6):1763-1770. https://doi.org/10.1086/315137
70. Hurwitz ES, Haber M, Chang A, et al. Effectiveness of influenza vaccination of day care children in reducing influenza-related morbidity among household contacts. *JAMA*. 2000;284(13):1677-1682. https://doi.org/10.1001/jama.284.13.1677

71. Welliver R, Monto AS, Carewicz O, et al. Effectiveness of oseltamivir in preventing influenza in household contacts: a randomized controlled trial. *JAMA*. 2001;285(6):748-754. https://doi.org/10.1001/jama.285.6.748
72. Monto AS, Pichichero ME, Blanckenberg SJ, et al. Zanamivir prophylaxis: an effective strategy for the prevention of influenza types A and B within households. *J Infect Dis*. 2002;186(11):1582-1588. https://doi.org/10.1086/345722
73. Neuzil KM, Hohlbein C, Zhu Y. Illness among schoolchildren during influenza season: effect on school absenteeism, parental absenteeism from work, and secondary illness in families. *Arch Pediatr Adolesc Med*. 2002;156(10):986-991. https://doi.org/10.1001/archpedi.156.10.986
74. Viboud C, Boëlle PY, Cauchemez S, et al. Risk factors of influenza transmission in households. *Br J Gen Pract*. 2004;54(506):684-689.
75. Cowling BJ, Fung ROP, Cheng CKY, et al. Preliminary findings of a randomized trial of non-pharmaceutical interventions to prevent influenza transmission in households. *PLoS One*. 2008;3(5):e2101. https://doi.org/10.1371/journal.pone.0002101
76. Cauchemez S, Donnelly CA, Reed C, et al. Household transmission of 2009 pandemic influenza A (H1N1) virus in the United States. *N Engl J Med*. 2009;361(27):2619-2627. https://doi.org/10.1056/NEJMoa0905498
77. Odaira F, Takahashi H, Toyokawa T, et al. Assessment of secondary attack rate and effectiveness of antiviral prophylaxis among household contacts in an influenza A(H1N1)v outbreak in Kobe, Japan, May–June 2009. *Eurosurveillance*. 2009;14(35):19320.
78. van Boven M, Donker T, van der Lubben M, et al. Transmission of novel influenza A(H1N1) in households with post-exposure antiviral prophylaxis. *PLoS One*. 2010;5(7):e11442. https://doi.org/10.1371/journal.pone.0011442
79. Calatayud L, Kurkela S, Neave PE, et al. Pandemic (H1N1) 2009 virus outbreak in a school in London, April-May 2009: an observational study. *Epidemiol Infect*. 2010;138(2):183-191. https://doi.org/10.1017/S0950268809991191
80. Cowling BJ, Chan KH, Fang VJ, et al. Comparative epidemiology of pandemic and seasonal influenza A in households. *N Engl J Med*. 2010;362(23):2175-2184. https://doi.org/10.1056/NEJMoa0911530
81. France AM, Jackson M, Schrag S, et al. Household transmission of 2009 influenza A (H1N1) virus after a school-based outbreak in New York City, April–May 2009. *J Infect Dis*. 2010;201(7):984-992. https://doi.org/10.1086/651145
82. Goldstein E, Cowling BJ, O'Hagan JJ, et al. Oseltamivir for treatment and prevention of pandemic influenza A/H1N1 virus infection in households, Milwaukee, 2009. *BMC Infect Dis*. 2010;10:211. https://doi.org/10.1186/1471-2334-10-211
83. Komiya N, Gu Y, Kamiya H, et al. Household transmission of pandemic 2009 influenza A (H1N1) virus in Osaka, Japan in May 2009. *J Infect*. 2010;61(4):284-288. https://doi.org/10.1016/j.jinf.2010.06.019
84. Looker C, Carville K, Grant K, Kelly H. Influenza A (H1N1) in Victoria, Australia: a community case series and analysis of household transmission. *PLoS One*. 2010;5(10):e13702. https://doi.org/10.1371/journal.pone.0013702
85. Morgan OW, Parks S, Shim T, et al. Household transmission of pandemic (H1N1) 2009, San Antonio, Texas, USA, April–May 2009. *Emerg Infect Dis*. 2010;16(4):631-637. https://doi.org/10.3201/eid1604.091658
86. Ng S, Cowling BJ, Fang VJ, et al. Effects of oseltamivir treatment on duration of clinical illness and viral shedding and household transmission of influenza virus. *Clin Infect Dis*. 2010;50(5):707-714. https://doi.org/10.1086/650458
87. Papenburg J, Baz M, Hamelin MÈ, et al. Household transmission of the 2009 pandemic A/H1N1 influenza virus: elevated laboratory-confirmed secondary attack rates and evidence of asymptomatic infections. *Clin Infect Dis*. 2010;51(9):1033-1041. https://doi.org/10.1086/656582
88. Pedroni E, Garcia M, Espinola V, et al. Outbreak of 2009 pandemic influenza A(H1N1), Los Lagos, Chile, April-June 2009. *Eurosurveillance*. 2010;15(1):19456. https://doi.org/10.2807/ese.15.01.19456-en
89. Sikora C, Fan S, Golonka R, et al. Transmission of pandemic influenza A (H1N1) 2009 within households: Edmonton. *Canada J Clin Virol*. 2010;49(2):90-93. https://doi.org/10.1016/j.jcv.2010.06.015
90. Suess T, Buchholz U, Dupke S, et al. Shedding and transmission of novel influenza virus A/H1N1 infection in households—Germany, 2009. *Am J Epidemiol*. 2010;171(11):1157-1164. https://doi.org/10.1093/aje/kwq071
91. Carcione D, Giele CM, Goggin LS, et al. Secondary attack rate of pandemic influenza A(H1N1) 2009 in Western Australian households, 29 May–7 August 2009. *Eurosurveillance*. 2011;16(3):19765.
92. Chang LY, Chen WH, Lu CY, et al. Household transmission of pandemic (H1N1) 2009 virus. *Taiwan Emerg Infect Dis*. 2011;17(10):1928-1931. https://doi.org/10.3201/eid1710.101662
93. Doyle TJ, Hopkins RS, Transmission Investigation Team. Low secondary transmission of 2009 pandemic influenza A (H1N1) in households following an outbreak at a summer camp: relationship to timing of exposure. *Epidemiol Infect*. 2011;139(1):45-51. https://doi.org/10.1017/S095026881000141X
94. Jackson ML, France AM, Hancock K, et al. Serologically confirmed household transmission of 2009 pandemic influenza A (H1N1) virus during the first pandemic wave—New York City, April–May 2009. *Clin Infect Dis*. 2011;53(5):455-462. https://doi.org/10.1093/cid/cir437
95. Van Gemert C, Hellard M, McBryde ES, et al. Intrahousehold transmission of pandemic (H1N1) 2009 virus, Victoria, Australia. *Emerg Infect Dis*. 2011;17(9):1599-1607. https://doi.org/10.3201/eid1709.101948
96. Leung YH, Li MP, Chuang SK. A school outbreak of pandemic (H1N1) 2009 infection: assessment of secondary household transmission and the protective role of oseltamivir. *Epidemiol Infect*. 2011;139(1):41-44. https://doi.org/10.1017/S0950268810001445
97. Nishiura H, Oshitani H. Household transmission of influenza (H1N1-2009) in Japan: age-specificity and reduction of household transmission risk by Zanamivir treatment. *J Int Med Res*. 2011;39(2):619-628. https://doi.org/10.1177/147323001103900231
98. Pebody RG, Harris R, Kafatos G, et al. Use of antiviral drugs to reduce household transmission of pandemic (H1N1) 2009. *United Kingdom Emerg Infect Dis*. 2011;17(6):990-999. https://doi.org/10.3201/eid/1706.101161

99. Savage R, Whelan M, Johnson I, et al. Assessing secondary attack rates among household contacts at the beginning of the influenza A (H1N1) pandemic in Ontario, Canada, April-June 2009: a prospective, observational study. *BMC Public Health*. 2011;11:234. https://doi.org/10.1186/1471-2458-11-234
100. Simmerman JM, Suntarattiwong P, Levy J, et al. Findings from a household randomized controlled trial of hand washing and face masks to reduce influenza transmission in Bangkok. *Thailand Influenza Other Respir Viruses*. 2011;5(4):256-267. https://doi.org/10.1111/j.1750-2659.2011.00205.x
101. Vargas-Leguas H, Caylà JA, Ballester I, et al. Factores asociados a la transmisión a los convivientes de gripe (H1N1) 2009. *Rev Esp Salud Pública*. 2011;85(1):57-62. https://doi.org/10.1590/S1135-57272011000100007
102. Behnaz F, Mohammadzadeh M, Sadeghian M. Household transmission of 2009 H1N1 influenza virus in Yazd. *Iran J Infect Public Health*. 2012;5(4):275-280. https://doi.org/10.1016/j.jiph.2011.12.006
103. Hirotsu N, Wada K, Oshitani H. Risk factors of household transmission of pandemic (H1N1) 2009 among patients treated with antivirals: a prospective study at a primary clinic in Japan. *PLoS One*. 2012;7(2):e31519. https://doi.org/10.1371/journal.pone.0031519
104. House T, Inglis N, Ross JV, et al. Estimation of outbreak severity and transmissibility: influenza A(H1N1)pdm09 in households. *BMC Med*. 2012;10:117. https://doi.org/10.1186/1741-7015-10-117
105. Janjua NZ, Skowronski DM, Hottes TS, et al. Transmission dynamics and risk factors for pandemic H1N1-related illness: outbreak investigation in a rural community of British Columbia. *Canada Influenza Other Respir Viruses*. 2012;6(3):e54-e62. https://doi.org/10.1111/j.1750-2659.2012.00344.x
106. Kim CY, Breiman RF, Cosmas L, et al. Secondary household transmission of 2009 pandemic influenza A (H1N1) virus among an urban and rural population in Kenya, 2009–2010. *PLoS One*. 2012;7(6):e38166. https://doi.org/10.1371/journal.pone.0038166
107. McCaw JM, Howard PF, Richmond PC, et al. Household transmission of respiratory viruses – assessment of viral, individual and household characteristics in a population study of healthy Australian adults. *BMC Infect Dis*. 2012;12:345. https://doi.org/10.1186/1471-2334-12-345
108. Mohamed AG, BinSaeed AA, Al-Habib H, Al-Saif H. Communicability of H1N1 and seasonal influenza among household contacts of cases in large families. *Influenza Other Respir Viruses*. 2012;6(3):e25-e29. https://doi.org/10.1111/j.1750-2659.2011.00308.x
109. Udompornwattana S, Srajai K, Suwan P, et al. The clinical features, risk of prolonged hospitalization and household infections of hospitalized children for pandemic 2009 influenza A (H1N1) virus infection in Thailand. *J Med Assoc Thail*. 2012;95(3):403-411.
110. Azman AS, Stark JH, Althouse BM, et al. Household transmission of influenza A and B in a school-based study of non-pharmaceutical interventions. *Epidemics*. 2013;5(4):181-186. https://doi.org/10.1016/j.epidem.2013.09.001
111. Kashiwagi S, Watanabe A, Ikematsu H, et al. Laninamivir octanoate for post-exposure prophylaxis of influenza in household contacts: a randomized double blind placebo controlled trial. *J Infect Chemother*. 2013;19(4):740-749. https://doi.org/10.1007/s10156-013-0622-9
112. Remschmidt C, Stöcker P, de Heiden AM, et al. Preventable and non-preventable risk factors for influenza transmission and hygiene behavior in German influenza households, pandemic season (H1N1) 2009/2010. *Influenza Other Respir Viruses*. 2013;7(3):418-425. https://doi.org/10.1111/j.1750-2659.2012.00407.x
113. Casado I, Martínez-Baz I, Burgui R, et al. Household transmission of influenza A(H1N1)pdm09 in the pandemic and post-pandemic seasons. *PLoS One*. 2014;9(9):e108485. https://doi.org/10.1371/journal.pone.0108485
114. Qiu C, Yuan S, Tian D, et al. Epidemiologic report and serologic findings for household contacts of three cases of influenza A (H7N9) virus infection. *J Clin Virol*. 2014;59(2):129-131. https://doi.org/10.1016/j.jcv.2013.12.004
115. Cheung DH, Tsang TK, Fang VJ, et al. Association of oseltamivir treatment with virus shedding, illness, and household transmission of influenza viruses. *J Infect Dis*. 2015;212(3):391-396. https://doi.org/10.1093/infdis/jiv058
116. Iyengar P, Van Mollendorf C, Tempia S, et al. Case-ascertained study of household transmission of seasonal influenza — South Africa. *J Infect*. 2015;71(5):578-586. https://doi.org/10.1016/j.jinf.2015.09.001
117. Ram PK, DiVita MA, Jannat KEK, et al. Impact of intensive handwashing promotion on secondary household influenza-like illness in rural Bangladesh: findings from a randomized controlled trial. *PLoS One*. 2015;10(6):e0125200. https://doi.org/10.1371/journal.pone.0125200
118. Yang Y, Zhang Y, Fang L, et al. Household transmissibility of avian influenza A (H7N9) virus, China, February to May 2013 and October 2013 to March 2014. *Euro Surveill*. 2015;20(10):21056. https://doi.org/10.2807/1560-7917.es2015.20.10.21056
119. Ng S, Lopez R, Kuan G, et al. The timeline of influenza virus shedding in children and adults in a household transmission study of influenza in Managua. *Nicaragua Pediatr Infect Dis J*. 2016;35(5):583-586. https://doi.org/10.1097/INF.0000000000001083
120. Weaver AM, Khatun-e-Jannat K, Cercone E, et al. Household-level risk factors for secondary influenza-like illness in a rural area of Bangladesh. *Tropical Med Int Health*. 2017;22(2):187-195. https://doi.org/10.1111/tmi.12820
121. Gordon A, Tsang TK, Cowling BJ, et al. Influenza transmission dynamics in urban households, Managua, Nicaragua, 2012–2014. *Emerg Infect Dis*. 2018;24(10):1882-1888. https://doi.org/10.3201/eid2410.161258
122. Valley-Omar Z, Iyengar P, von Mollendorf C, et al. Intra-host and intra-household diversity of influenza A viruses during household transmissions in the 2013 season in 2 peri-urban communities of South Africa. *PLoS One*. 2018;13(5):e0198101. https://doi.org/10.1371/journal.pone.0198101
123. Cohen C, Tshangela A, Valley-Omar Z, et al. Household transmission of seasonal influenza from HIV-infected and HIV-uninfected individuals in South Africa, 2013–2014. *J Infect Dis*. 2019;219(10):1605-1615. https://doi.org/10.1093/infdis/jiy702
124. Hirotsu N, Saisho Y, Hasegawa T. The effect of neuraminidase inhibitors on household transmission in Japanese patients with influenza A and B infection: a prospective, observational study. *Influenza Other Respir Viruses*. 2019;13(2):123-132. https://doi.org/10.1111/irv.12590

125. Cauchemez S, Carrat F, Viboud C, Valleron AJ, Boelle PY. A Bayesian MCMC approach to study transmission of influenza: application to household longitudinal data. *Stat Med*. 2004;23(22):3469-3487. https://doi.org/10.1002/sim.1912
126. Halloran ME, Hayden FG, Yang Y, Longini J, Monto AS. Antiviral effects on influenza viral transmission and pathogenicity: observations from household-based trials. *Am J Epidemiol*. 2006;165(2):212-221. https://doi.org/10.1093/aje/kwj362%J
127. Donnelly CA, Finelli L, Cauchemez S, et al. Serial intervals and the temporal distribution of secondary infections within households of 2009 pandemic influenza A (H1N1): implications for influenza control recommendations. *Clin Infect Dis*. 2011;52(Suppl 1):S123-S130. https://doi.org/10.1093/cid/ciq028
128. Poon LLM, Chan KH, Chu DKW, et al. Viral genetic sequence variations in pandemic H1N1/2009 and seasonal H3N2 influenza viruses within an individual, a household and a community. *J Clin Virol*. 2011;52(2):146-150. https://doi.org/10.1016/j.jcv.2011.06.022
129. Zhang D, Liu W, Yang P, et al. Factors associated with household transmission of pandemic (H1N1) 2009 among self-quarantined patients in Beijing. *China PLoS One*. 2013;8(10):e77873. https://doi.org/10.1371/journal.pone.0077873
130. Tsang TK, Cauchemez S, Perera RAPM, et al. Association between antibody titers and protection against influenza virus infection within households. *J Infect Dis*. 2014;210(5):684-692. https://doi.org/10.1093/infdis/jiu186%J
131. Lau MS, Cowling BJ, Cook AR, Riley S. Inferring influenza dynamics and control in households. *Proc Natl Acad Sci*. 2015;112(29):9094-9099. https://doi.org/10.1073/pnas.1423339112
132. Tsang TK, Cowling BJ, Fang VJ, et al. Influenza a virus shedding and infectivity in households. *J Infect Dis*. 2015;212(9):1420-1428. https://doi.org/10.1093/infdis/jiv225
133. Tsang TK, Fang VJ, Chan KH, et al. Individual correlates of infectivity of influenza a virus infections in households. *PLoS One*. 2016;11(5):1-11. https://doi.org/10.1371/journal.pone.0154418
134. Armstrong C, Hopkins R. An epidemiological study of the 1920 epidemic of influenza in an isolated rural community. *Public Health Rep*. 1921;36(29):1671-1702. https://doi.org/10.2307/4576063
135. Frost WH, Gover M. The incidence and time distribution of common colds in several groups kept under continuous observation. *Public Health Rep*. 1932;47(36):1815-1841. https://doi.org/10.2307/4580544
136. Monto AS, Davenport FM, Napier JA, Francis J. Modification of an outbreak of influenza in Tecumseh, Michigan by vaccination of schoolchildren. *J Infect Dis*. 1970;122(1–2):16-25. https://doi.org/10.1093/infdis/122.1-2.16
137. Fairchok MP, Martin ET, Chambers S, et al. Epidemiology of viral respiratory tract infections in a prospective cohort of infants and toddlers attending daycare. *J Clin Virol*. 2010;49(1):16-20. https://doi.org/10.1016/j.jcv.2010.06.013
138. Johnson S, Ihekweazu C, Hardelid P, et al. Seroepidemiologic study of pandemic (H1N1) 2009 during outbreak in boarding school. *Eng Emerg Infect Dis*. 2011;17(9):1670. https://doi.org/10.3201/eid1709.100761
139. Martin ET, Fairchok MP, Stednick ZJ, Kuypers J, Englund JA. Epidemiology of multiple respiratory viruses in childcare attendees. *J Infect Dis*. 2013;207(6):982-989. https://doi.org/10.1093/infdis/jis934
140. Poritz MA, Blaschke AJ, Byington CL, et al. FilmArray, an automated nested multiplex PCR system for multi-pathogen detection: development and application to respiratory tract infection. *PLoS One*. 2011;6(10):e26047. https://doi.org/10.1371/journal.pone.0026047
141. Wilson EB. Probable inference, the law of succession, and statistical inference. *J Am Stat Assoc*. 1927;22(158):209-212. https://doi.org/10.1080/01621459.1927.10502953
142. O'Neill PD, Balding DJ, Becker NG, Eerola M, Mollison D. Analyses of infectious disease data from household outbreaks by Markov chain Monte Carlo methods. *J Royal Stat Soc Ser C (Appl Stat)*. 2000;49(4):517-542. https://doi.org/10.1111/1467-9876.00210
143. Kaplan EL, Meier P. Nonparametric estimation from incomplete observations. *J Am Stat Assoc*. 1958;53(282):457-481. https://doi.org/10.1080/01621459.1958.10501452
144. Cleveland WS, Grosse E, Shyu WM. *Local Regression Models*. Pacific Grove, CA: Wadsworth and Brooks/Cole; 1992.
145. Metropolis N, Rosenbluth AW, Rosenbluth MN, Teller AH, Teller E. Equation of state calculations by fast computing machines. *J Chem Phys*. 1953;21(6):1087-1092. https://doi.org/10.1063/1.1699114
146. Eddelbuettel D, François R. Rcpp: seamless C and C++ integration. *J Stat Softw*. 2011;40(8):1-18. https://doi.org/10.18637/jss.v040.i08
147. Yang Y, Sugimoto JD, Halloran ME, et al. The transmissibility and control of pandemic influenza A (H1N1) virus. *Science*. 2009;326(5953):729-733. https://doi.org/10.1126/science.1177373
148. Flannery B, Zimmerman RK, Gubareva LV, et al. Enhanced genetic characterization of influenza A(H3N2) viruses and vaccine effectiveness by genetic group, 2014–2015. *J Infect Dis*. 2016;214(7):1010-1019. https://doi.org/10.1093/infdis/jiw181

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