Online Supplement

S1 Model diagnostics

We have two sets of diagnostics. First, our primary analysis used the case definition requiring laboratory evidence of infection among those with an ARI. Next, our secondary analysis required both an ARI and laboratory evidence from the index case within a household, but later cases only needed evidence of an ARI.

S1.1 Primary analysis

The posterior probability, up to proportionality, of the Markov chains showed good mixing (Figure S1). The marginal posterior probability distributions of $\beta$ and $\gamma$ were often insufficient to make inference; although, the marginal posterior probability distribution of $\alpha$ and the posterior probability distribution of the ratio $\beta/\gamma$ were informative (Figure S2). So, the data are sufficient to discuss the CAR and the SAR but insufficient to discuss the length of the infectious period or infectiousness of the circulating virus. This paradox is because of the strong correlation between $\beta$ and $\gamma$ (Figure S3).

For influenza A (H1) during 2013-2014, the uniform prior clipped the right tail of the marginal posterior probability distribution of $\gamma$ (Figure S2). Extending the support of the uniform prior to $[10^{-2}, 10^{14}]$3 neither changes our estimates within rounding error nor eliminates the clipping of the marginal posterior probability distribution of $\gamma$. This is because the two apparent secondary cases occurred the day after the household index case. Exponential distributions defined by these large values of $\gamma$ have almost all probability in a small neighborhood of the origin, consistent with the timing of the apparent secondary cases and our model, by (1). The uniform distri-
bution clipped $\beta$ and $\gamma$ for other virus and season combinations, except for influenza A (H3) during 2014–2015 when there were 9 apparent secondary cases (Figure S2). These details suggest that data with few or even no secondary cases may still provide useful results, despite their limitations.

The chains approached their limiting distribution quickly, which is why we do not discard any initial iterates as burn-in. If we had used the first 500,000 iterations as burn-in, then our estimates would change only within rounding error. This is because most of the low probability regions were visited after the chain visited the high probability region (Figure S1). The shape of the sample from the joint posterior distribution in Figure S4 quickly summarizes the diagnostics: more apparent secondary cases results in elliptical posterior distributions.

To assess goodness of fit, we used Monte Carlo methods. First, we permuted the onset dates within the cohort. Next, we sample from our posterior to obtain parameter values. Then, we compute the likelihood of the permuted data given these parameters and compute the same likelihood of the observed data. Finally, we repeat this process for a total of 10,000 trials. We visualize the comparison of the two likelihoods in Figure S5: points below the line indicate a better fit to the observed data than the permuted data. Analyses with no apparent secondary cases had poorer fit. We chose to partially present results from these models for completeness; otherwise, we only present results with higher estimates of SAR. We did not include results from these models in the abstract, the graphical abstract, the main figures, or the discussion.

S1.2 Secondary analysis

Because the secondary analysis used similar data to the primary analysis, the diagnostics lead to similar conclusions. As the case definition was less restrictive, these
data were a superset of the data from the primary analysis. So, the posterior distribution was less flat, and the algorithm performed slightly better. The trace plots showed good mixing (Figure S6). The marginal distribution of $\beta$ and $\gamma$ were typically insufficient to make inference about infectiousness or the length of the infectious period, but the distribution of $\alpha$ and $\beta/\gamma$ were sufficient to make inference about the CAR and the SAR (Figure S7). The correlation between $\beta$ and $\gamma$ is what allowed inference using $\beta/\gamma$ (Figure S8). More apparent secondary cases generally lead to a more elliptical posterior distribution (Figure S9). Because there were more apparent secondary cases, the goodness of fit was better for the secondary analyses (Figure S10).

S2 Simulation study

To validate our model, we simulated household transmission data. In our model, the hazard from the household adds with the hazard from the community. However, we wanted to distinguish community transmission from household transmission. So, we considered household transmission and community transmission as two competing risks. We built a cohort of 2,000 households each with two people for a followup time of 365 days. We chose to use a household size of $n = 2$ for simplicity, because we need to consider if and when the $2^n + n$ possible routes of infection occur within each household. To reduce the censoring of household transmission, cohort members were at risk of community transmission only for the first 350 days.

For each household, we randomly assigned two times to event, $t_C^{(1)}$ and $t_C^{(2)}$, from the exponential distribution with rate $h = 0.0002$. We assumed without loss of generality that $t_C^{(1)} < t_C^{(2)}$. We computed onset dates from these times to event, as follows. If both $t_C^{(1)} > 350$ and $t_C^{(2)} > 350$, then we assigned $\infty$ to both onset dates for that
household. Otherwise, we had $t_C^{(1)} < 350$ and the possibility of further transmission in the household. We assigned the ceiling of $t_C^{(1)}$ as the first person’s onset date, and we recorded this case as community transmission. We drew an infectious period, $t_I$, from the exponential distribution with rate $\gamma = 0.07$, and we drew a time to household transmission, $t_{HH}$, from the exponential distribution with rate $\beta/2 = 0.01$. We continued assigning onset dates to the remaining household members with a case study.

- If $t_{HH} < t_I$ and $t_{HH} + t_C^{(1)} < 365$, then household transmission may have occurred.
  
  - If $t_{HH} + t_C^{(1)} < t_C^{(2)}$, then the onset date was the ceiling of $t_{HH} + t_C^{(1)}$. We recorded this case as household transmission.
  
  - If $t_{HH} + t_C^{(1)} \geq t_C^{(2)}$ and $t_C^{(2)} < 350$, then the onset date was the ceiling of $t_C^{(2)}$. We recorded this case as community transmission.
  
  - If $t_{HH} + t_C^{(1)} \geq t_C^{(2)}$ and $t_C^{(2)} \geq 350$, then we assigned $\infty$ as the onset date.

- If $t_{HH} \geq t_I$ or $t_{HH} + t_C^{(1)} \geq 365$, then household transmission did not occur.
  
  - If $t_C^{(2)} < 350$, then the onset date was the ceiling of $t_C^{(2)}$. We recorded this case as community transmission.
  
  - If $t_C^{(2)} \geq 350$, then the onset date was $\infty$.

For each trial, we would expect the CAR to be about $1 - e^{-ht} = 1 - e^{-0.002 \cdot 350} \approx 0.0676$, and the SAR to be about $\frac{\beta}{2\gamma} = \frac{0.02}{2 \times 0.07} = 0.14$. Our simulation of the data never used $\alpha$, but we applied Jensen’s inequality and the law of total probability to approximate

$$\alpha \approx \frac{\text{CAR}}{\text{CAR} + \text{SAR} \cdot \text{CAR}} = \frac{1}{1 + \text{SAR}} \approx 0.877.$$
Because our rate $h$ is small, we expect our lower bound on $\alpha$ to be tight compared to sampling error.

The goodness of fit statistic for each of the 5 simulations was less than 0.0001, suggesting that the model explains the transmission dynamics well (Figure S11). The 95% credible intervals all captured the parameter values used to simulate the data as well as our approximation of $\alpha$ (Table S1). Our point estimates for $\beta$ and $\gamma$ were larger than what we used to simulate the data because we used the median for our point estimates. To approximate the conversion of means to medians we divide by $\log 2$, which gives $\beta/\log 2 \approx 0.029$ and $\gamma/\log 2 \approx 0.10$. We are primarily interested in comparing the estimates of the CAR and SAR from the model with the truth in the simulated data (Table S2). The model estimates of the CAR and the SAR were close to the truth without apparent bias, and all the 95% credible intervals covered the truth.

To estimate coverage probabilities of our CIs and mean percent error (MPE), we reduced the number of iterations in our algorithm to $10^5$ and increased the number of simulations to 100. The coverage of our 95% CIs for the CAR and for the SAR were both 1. The MPE of the CAR was 3.2%; the MPE of the SAR was 0.0001%. In conclusion, the model recovered the simulated values of CAR and SAR, even with a discontinuous hazard from the community, right censoring at day 365, and interval censoring when computing the onset dates.

S3 Selected estimates of secondary attack rate

We wanted to tabulate previously published estimates of SARs for comparison to our results. We performed a non-systematic review of the literature and included publications which reported SAR or enough results to calculate SAR. We excluded
publications which reported SAR using the same data as another publication. Published estimates of the SAR of influenza vary widely with the working case definition, time, place, and type or subtype of virus (Table S3).
Table S1: Summary of parameter estimates from the simulation study.

| Trial | $\alpha$ (95% CI) | $\beta$ (95% CI) | $\gamma$ (95% CI) |
|-------|-------------------|------------------|------------------|
| 1     | 0.898 (0.797, 1.01) | 0.0303 (0.0184, 0.0488) | 0.101 (0.0662, 0.155) |
| 2     | 0.915 (0.808, 1.03) | 0.0137 (0.0102, 0.0230) | 0.073 (0.0485, 0.113) |
| 3     | 0.901 (0.795, 1.02) | 0.0254 (0.0147, 0.0426) | 0.100 (0.0644, 0.154) |
| 4     | 0.880 (0.775, 0.99) | 0.0251 (0.0152, 0.0406) | 0.078 (0.0511, 0.120) |
| 5     | 0.906 (0.797, 1.02) | 0.0229 (0.0126, 0.0402) | 0.110 (0.0670, 0.171) |
Table S2: The community attack rate and secondary attack rate from the simulation study and the corresponding estimates from the transmission model.

| Trial | Community Attack Rate | Secondary Attack Rate |
|-------|-----------------------|-----------------------|
|       | Truth | Estimate (95% CI)    | Truth | Estimate (95% CI)    |
| 1     | 284/4000 = 0.0710 | 0.0707 (0.0627, 0.0793) | 37/274 = 0.135 | 0.140 (0.100, 0.187) |
| 2     | 263/4000 = 0.0658 | 0.0653 (0.0576, 0.0736) | 23/259 = 0.089 | 0.092 (0.062, 0.132) |
| 3     | 258/4000 = 0.0645 | 0.0651 (0.0574, 0.0733) | 33/255 = 0.129 | 0.119 (0.082, 0.165) |
| 4     | 253/4000 = 0.0632 | 0.0630 (0.0556, 0.0711) | 37/248 = 0.149 | 0.149 (0.105, 0.200) |
| 5     | 273/4000 = 0.0682 | 0.0619 (0.0544, 0.0699) | 29/244 = 0.119 | 0.100 (0.065, 0.144) |
Table S3: Selected estimates of SARs from the literature by time, virus type, location, evidence for infection, and supporting reference.

| Time          | Virus | Location                              | Secondary Attack Rate | Reference         |
|---------------|-------|---------------------------------------|-----------------------|------------------|
| 1948–1949     |       | Bowerchalke, Wiltshire, England       | 24                    | Lidwell 1951[1]  |
| 1948–1950     |       | Cleveland, Ohio, USA                  | 31                    | Badger 1953[2]   |
| 1951          | A     | Cirencester, Gloucestershire, England | 78                    | Hope Simpson 1954[3] |
| 1952–1954     |       | London, Ontario, Canada               | 18                    | Buck 1956[4]     |
| 1953          |       | London, England                       | 16                    | Brimblecombe 1958[5] |
| 1957          | H2N2  | Tokyo, Japan                          | 7                     | Nishiura 2007[6] |
| 1957          | H2N2  | Osaka, Japan                          | 9                     | Nishiura 2007[6] |
| 1961          | B     | Osaka, Japan                          | 18                    | Nishiura 2007[6] |
| 1961–1969     | A     | Seattle, Washington, USA              | 27                    | Hall 1973[7]     |
| 1961–1969     | B     | Seattle, Washington, USA              | 32                    | Hall 1973[7]     |
| 1968–1969     | H2N2  | Helena, Montana, USA                  | 27                    | CDC & WHO 1969[8] |
| 1968–1969     | H2N2  | Baltimore, Maryland, USA              | 43                    | CDC & WHO 1969[8] |
| 1968–1969     | H3N2  | England and Wales                     | 17                    | Hope Simpson 1979[9] |
| 1969          | H2N2  | Cirencester, Gloucestershire, England | 17                    | Hope Simpson 1970[10] |
| 1969–1970     | H3N2  | England and Wales                     | 14                    | Hope Simpson 1979[9] |
| 1972–1973     | A     | England and Wales                     | 9                     | RCGP and PHLS 1981[11] |
| 1972–1973     | A     | Seattle, Washington, USA              | 27                    | Foy 1976[12]     |
| 1973          | H3N2  | Port Chambers, Otago, New Zealand     | 58                    | Jennings 1978[13] |
| 1974          | H3N2  | Port Chambers, Otago, New Zealand     | 33                    | Jennings 1978[13] |
| 1975          | H3N2  | Port Chambers, Otago, New Zealand     | 38                    | Jennings 1978[13] |
| 1975–1979     | H3N2  | Seattle, Washington, USA              | 53                    | Fox 1982[14]     |
Table S3: Selected estimates of SARs from the literature by time, virus type, location, evidence for infection, and supporting reference.

| Time        | Virus | Location                        | Syndromic | RIDT | Seroconversion | PCR | Culture | Reference       |
|-------------|-------|---------------------------------|-----------|------|----------------|-----|---------|----------------|
| 1975–1979   | H1N1  | Seattle, Washington, USA        | 44        |      |                |     |         | Fox 1982[14]   |
| 1975–1979   | B     | Seattle, Washington, USA        | 47        |      |                |     |         | Fox 1982[14]   |
| 1975–1976   | B     | Seattle, Washington, USA        | 13        |      |                |     |         | Longini 1982[15]|
| 1976–1980   | B     | Houston, Texas, USA             | 21        | 38   |                |     |         | Frank 1983[16] |
| 1977–1979   | H3N2  | Seattle, Washington, USA        | 21        |      |                |     |         | Longini 1982[15]|
| 1977–1978   | H3N2  | Tecumseh, Michigan, USA         | 15        |      |                |     |         | Longini 1982[15]|
| 1978–1979   | H1N1  | Seattle, Washington, USA        | 31        |      |                |     |         | Longini 1982[15]|
| 1985        | H3N2  | Tecumseh, Michigan, USA         |           | 12   |                |     |         | Longini 1988[17]|
| 1996–1997   | A and B | San Diego, California, USA     | 51        |      |                |     |         | Hurwitz 2000[18]|
| 1997        | H5N1  | Hong Kong                       | 2         | 12   |                |     |         | Katz 1999[19]  |
| 1998–1999   | A     | North America and Europe        |           | 26   |                |     |         | Hayden 2000[20]|
| 1998–1999   | B     | North America and Europe        |           | 34   |                |     |         | Hayden 2000[20]|
| 1998–1999   | A and B | North America and Europe       |           | 13   |                |     |         | Welliver 2001[21]|
| 1999–2000   | A     | France                          |           | 24   |                |     |         | Viboud 2004[22]|
| 2000–2001   | A and B | North America and Europe       |           | 26   |                |     |         | Hayden 2004[23]|
| 2000–2001   | A and B | 11 countries                   |           | 17   |                |     |         | Monto 2002[24] |
| 2000–2001   | A and B | Seattle, Washington, USA        |           | 22   |                |     |         | Neuzil 2002[25]|
| 2001–2002   | A and B | Taiwan                         |           | 9    |                |     |         | Hsu 2014[26]   |
| 2006        | H5N1  | Karo Regency, North Sumatra, Indonesia | | 29   |                |     |         | Yang 2007[27] |
| 2007        | A and B | Hong Kong                      |           | 18   |                |     |         | Cowling 2008[28]|
| 2007–2008   | A and B | Hong Kong                      |           | 8    |                |     |         | Ng 2010[29]    |
Table S3: Selected estimates of SARs from the literature by time, virus type, location, evidence for infection, and supporting reference.

| Time       | Virus | Location                        | Secondary Attack Rate | Reference          |
|------------|-------|---------------------------------|-----------------------|--------------------|
| 2007–2008  | A     | Pittsburgh, Pennsylvania, USA   | 31 Syndromic          | Azman 2013[30]     |
| 2007–2008  | B     | Pittsburgh, Pennsylvania, USA   | 25 RIDT               | Azman 2013[30]     |
| 2007–2010  | A and B | Vietnam                         | 8 Seroconversion      | Cauchemez 2014[31] |
| 2008       | A and B | Australia                        | 24 PCR                | McCaw 2012[32]     |
| 2008       | A and B | Nairobi, Kenya                   | 9 Culture             | Judd 2015[33]      |
| 2008–2009  | A and B | Bangkok, Thailand                | 14 21                 | Simmerman 2011[34] |
| 2008–2010  | A and B | Dhaka, Bangladesh                | 10 4                  | Fry 2015[35]       |
| 2008–2013  | A and B | Hong Kong                        | 10                    | Cheung 2015[36]    |
| 2009       | A      | Hong Kong                        | 19 9                  | Cowling 2010[37]   |
| 2009       | A and B | Nairobi, Kenya                   | 6                     | Judd 2015[33]      |
| 2009       | A and B | Saudi Arabia                     | 14                    | Mohamed 2012[38]   |
| 2009       | pH1N1  | Saudi Arabia                     | 17                    | Mohamed 2012[38]   |
| 2009       | H3N2   | Hong Kong                        | 8                     | Klick 2011[39]     |
| 2009       | H1N1   | Hong Kong                        | 7                     | Klick 2011[39]     |
| 2009       | pH1N1  | Hong Kong                        | 10                    | Klick 2011[39]     |
| 2009       | pH1N1  | Spain                            | 11                    | Vargas-Leguas 2011[40] |
| 2009       | pH1N1  | Thailand                         | 16                    | Udompornwattana 2012[41] |
| 2009       | pH1N1  | Western Australia, Australia     | 15                    | Carcione 2011[42]  |
| 2009       | pH1N1  | Victoria, Australia              | 33                    | Looker 2010[43]    |
| 2009       | pH1N1  | Victoria, Australia              | 15                    | van Genert 2011[44]|
| 2009       | pH1N1  | British Columbia, Canada         | 22                    | Janjua 2012[45]    |
Table S3: Selected estimates of SARs from the literature by time, virus type, location, evidence for infection, and supporting reference.

| Time | Virus | Location                  | Syndrome | RIDT | Seroconversion | PCR | Culture | Reference          |
|------|-------|---------------------------|----------|------|----------------|-----|---------|--------------------|
| 2009 | pH1N1 | Ontario, Canada           | 20       |      |                |     |         | Savage 2011[46]    |
| 2009 | pH1N1 | Edmonton, Alberta, Canada | 30       |      |                |     |         | Sikora 2010[47]    |
| 2009 | pH1N1 | Puerto Montt, Los Lagos, Chile | 35   |      |                |     |         | CTFSPIA 2010[48]   |
| 2009 | pH1N1 | United States             | 27       |      |                |     |         | Yang 2009[49]      |
| 2009 | pH1N1 | United States             | 13       |      |                |     |         | Cauchemez 2009[50] |
| 2009 | pH1N1 | Florida, USA              | 4        |      |                |     |         | Doyle 2011[51]     |
| 2009 | pH1N1 | New York City, New York, USA | 11   |      |                |     |         | France 2010[52]    |
| 2009 | pH1N1 | San Antonio, Texas, USA   | 4        |      |                |     |         | Loustalot 2011[53] |
| 2009 | pH1N1 | New York City, New York, USA | 23 | 19  |                |     |         | Jackson 2011[54]   |
| 2009 | pH1N1 | Hong Kong                 | 26       | 8    |                |     |         | Cowling 2010[57]   |
| 2009 | pH1N1 | San Antonio, Texas, USA   | 13       | 4    |                |     |         | Morgan 2010[55]    |
| 2009 | pH1N1 | Quebec City, Quebec, Canada | 51    | 45   |                |     |         | Papenburg 2010[56] |
| 2009 | pH1N1 | Osaka, Japan              | 3        | 4    |                |     |         | Komiya 2010[57]    |
| 2009 | pH1N1 | United Kingdom            | 22       | 8    |                |     |         | Pebody 2011[58]    |
| 2009 | pH1N1 | United Kingdom            | 52       | 35   |                |     |         | House 2012[59]     |
| 2009 | pH1N1 | Taiwan                    | 27       |      |                |     |         | Chang 2011[60]     |
| 2009 | pH1N1 | Netherlands               | 8        |      |                |     |         | van Boven 2010[61] |
| 2009 | pH1N1 | Germany                   | 26       |      |                |     |         | Suess 2010[62]     |
| 2009 | pH1N1 | United Kingdom            | 17       |      |                |     |         | Calatayud 2010[63] |
| 2009 | pH1N1 | Hong Kong                 | 6        |      |                |     |         | Leung 2011[64]     |
| 2009 | pH1N1 | Ha Nam, Vietnam           | 19       |      |                |     |         | Thai 2014[65]      |
Table S3: Selected estimates of SARs from the literature by time, virus type, location, evidence for infection, and supporting reference.

| Time     | Virus       | Location                  | Secondary Attack Rate | Syndromic | RIDT | Seroconversion | PCR | Culture | Reference     |
|----------|-------------|---------------------------|-----------------------|-----------|------|----------------|-----|----------|---------------|
| 2009     | pH1N1       | Kobe, Japan               |                       |           |      |                |     |          | Odaira 2009[66] |
| 2009     | pH1N1       | Milwaukee, Wisconsin, USA |                       |           |      |                |     |          | Goldstein 2010[67] |
| 2009-2010| pH1N1       | Germany                   |                       | 10        |      |                |     |          | Remschmidt 2013[68] |
| 2009-2010| pH1N1       | Japan                     |                       | 11        |      |                |     |          | Nishiura 2011[69] |
| 2009-2010| A           | Kawasaki, Kanagawa, Japan |                       | 7         |      |                |     |          | Hirotsu 2012[70] |
| 2009-2010| pH1N1       | Yazad, Iran               |                       | 14        |      |                |     |          | Belmaz 2012[71] |
| 2009-2010| pH1N1       | Lwak, Kenya               |                       | 8         |      |                |     |          | Kim 2012[72] |
| 2009-2010| pH1N1       | Kiberia, Kenya            |                       | 5         |      |                |     |          | Kim 2012[72] |
| 2009-2010| pH1N1       | Navarra, Spain            |                       | 19        |      |                |     |          | Casado 2014[73] |
| 2009-2010| A and B     | Kishoreganj, Bangladesh   |                       | 8         |      |                |     |          | Ram 2015[74] |
| 2009-2010| A and B     | Kishoreganj, Bangladesh   |                       | 8         |      |                |     |          | Weaver 2017[75] |
| 2010     | A and B     | Nairobi, Kenya            |                       | 10        |      |                |     |          | Judd 2015[33] |
| 2010-2011| pH1N1       | Navarra, Spain            |                       | 14        |      |                |     |          | Casado 2014[73] |
| 2010-2011| H3N2        | Baganuur, Ulaanbaatar, Mongolia |                   | 6         |      |                |     |          | Nukiwa-Souma 2012[76] |
| 2010-2011| H3N2        | Ann Arbor, Michigan, USA  |                       | 15        |      |                |     |          | Petrie 2013[77] |
| 2010-2011| pH1N1       | Ann Arbor, Michigan, USA  |                       | 3         |      |                |     |          | Petrie 2013[77] |
| 2010-2011| B           | Ann Arbor, Michigan, USA  |                       | 8         |      |                |     |          | Petrie 2013[77] |
| 2010-2011| A and B     | Ann Arbor, Michigan, USA  |                       | 10        |      |                |     |          | Ohmit 2013[78] |
| 2010-2016| A           | Kawasaki, Kanagawa, Japan |                       | 8         |      |                |     |          | Hirotsu 2019[79] |
| 2010-2016| B           | Kawasaki, Kanagawa, Japan |                       | 5         |      |                |     |          | Hirotsu 2019[79] |
| 2011     | A and B     | Nairobi, Kenya            |                       | 6         |      |                |     |          | Judd 2015[33] |
Table S3: Selected estimates of SARs from the literature by time, virus type, location, evidence for infection, and supporting reference.

| Time       | Virus   | Location                  | Secondary Attack Rate | Reference       |
|------------|---------|---------------------------|-----------------------|-----------------|
| 2011–2012  | A and B | Japan                     | 17                    | Kashiwagi 2013[^80] |
| 2011–2013  | A and B | Bamako, Mali              | 6                     | Buchwald 2019[^81] |
| 2012–2013  | A and B | Managua, Nicaragua        | 17                    | Ng 2016[^82]    |
| 2012–2014  | H1N1    | Managua, Nicaragua        | 13                    | Gordon 2018[^83] |
| 2012–2014  | H3N2    | Managua, Nicaragua        | 14                    | Gordon 2018[^83] |
| 2012–2014  | B       | Managua, Nicaragua        | 20                    | Gordon 2018[^83] |
| 2013       | H7N9    | Shanghai, China           | 0                     | Qiu 2014[^84]   |
| 2013       | H1N1    | South Africa              | 17                    | Iyengar 2015[^85] |
| 2013       | H3N2    | South Africa              | 16                    | Iyengar 2015[^85] |
| 2013       | B       | South Africa              | 21                    | Iyengar 2015[^85] |
| 2013       | A and B | South Africa              | 22                    | Valley-Omar 2018[^86] |
| 2013–2014  | H3N2    | South Africa              | 24                    | Cohen 2019[^57]  |
| 2013–2014  | pH1N1   | South Africa              | 24                    | Cohen 2019[^57]  |
| 2013–2014  | A       | South Africa              | 20                    | Cohen 2019[^57]  |
| 2013–2014  | B       | South Africa              | 28                    | Cohen 2019[^57]  |
| 2013–2014  | H7N9    | China                     | 1                     | Yang 2015[^88]   |
| 2014–2015  | H3N2    | Ann Arbor, Michigan, USA  | 17                    | Petrie 2017[^89] |
| 2014–2015  | B       | Ann Arbor, Michigan, USA  | 6                     | Petrie 2017[^89] |
S5  Supplemental Figures
Figure S1: The joint posterior probability, up to proportionality, of $\alpha$, $\beta$, and $\gamma$ for the primary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015.
Figure S2: The marginal posterior distribution of $\alpha$, $\beta$, and $\gamma$, and posterior distribution of $\beta/\gamma$ from the primary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015.
Figure S3: Kernel smoothed posterior distribution for pairs of $\alpha, \beta,$ and $\gamma$ from the primary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015. Distributions were clipped for clarity. Density estimated using the MASS package in R.[90]

Figure S4: Trace plots of the posterior distribution of $\alpha, \beta,$ and $\gamma$ colored by the logarithm of the estimated posterior probability from the primary analysis of influenza A (H1) during influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015. Model parameters are plotted on the log scale for clarity. Every hundredth iterate is plotted for clarity. Images created using the plot3D package in R.[91]
Figure S5: The likelihood of the permuted onset dates versus the likelihood of the observed onset dates from the primary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015. Points below the line indicate higher likelihood of the observed data compared to the permuted data. The Monte Carlo estimate of the probability of a point being above the line is in the top left corner of each plot.
Figure S6: The joint posterior probability, up to proportionality, of $\alpha$, $\beta$, and $\gamma$ for the secondary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015.
Figure S7: Marginal posterior distribution of $\alpha$, $\beta$, and $\gamma$, and posterior distribution of $\beta/\gamma$ from the secondary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015.
Figure S8: Kernel smoothed posterior distribution for pairs of $\alpha, \beta,$ and $\gamma$ from the secondary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015. Distributions were clipped for clarity. Density estimated using the MASS package in R. [90]

Figure S9: Trace plots of the posterior distribution of $\alpha, \beta,$ and $\gamma$ colored by the logarithm of the estimated posterior probability from the secondary analysis of influenza A (H1) during influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015. Model parameters are plotted on the log scale for clarity. Every hundredth iterate is plotted for clarity. Images created using the plot3D package in R. [91]
Figure S10: The likelihood of the permuted onset dates versus the likelihood of the observed onset dates from the secondary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015. Points below the line indicate higher likelihood of the observed data compared to the permuted data. The Monte Carlo estimate of the probability of a point being above the line is in the top left corner of each plot.

Figure S11: The likelihood of the permuted onset dates versus the likelihood of the observed onset dates from the simulation study. Points below the line indicate higher likelihood of the observed data compared to the permuted data. The Monte Carlo estimate of the probability of a point being above the line is in the top left corner of each plot.
References

[1] Lidwell O. M., Sommerville T.. Observations on the incidence and distribution of the common cold in a rural community during 1948 and 1949 J Hyg (Lond). 1951;49:365–381.

[2] Badger G. F., Dingle J. H., Feller A. E., Hodges R. G., Jordan Jr., Rammelkamp Jr.. A study of illness in a group of Cleveland families. IV. The spread of respiratory infections within the home Am J Hyg. 1953;58:174—178.

[3] Hope Simpson R. E., Sutherland I.. Does influenza spread within the household? Lancet. 1954;266:721–726.

[4] Buck C.. Acute upper respiratory infections in families Am J Hyg. 1956;63:1–12.

[5] Brimblecombe F. S. W., Cruickshank R., Masters P. L., Reid D. D., Stewart G. T.. Family studies of respiratory infections Br Med J. 1958;1:119–128.

[6] Nishiura H., Chowell G.. Household and community transmission of the Asian influenza A (H2N2) and influenza B viruses in 1957 and 1961 Southeast Asian J Trop Med Public Health. 2007;38:1075–1083.

[7] Hall C. E., Cooney M. K., Fox J. P.. 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;98:365–380.

[8] 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;18:22–32.

[9] Hope-Simpson R. E.. Epidemic mechanisms of type A influenza J Hyg (Lond). 1979;83:11–26.

[10] Hope-Simpson R. E.. First outbreak of Hong Kong influenza in a general practice population in Great Britain. A field and laboratory study Br Med J. 1970;3:74–77.

[11] The Royal College of General Practitioners, the Public Health Laboratory Service. Long-term study of influenza in families J R Coll Gen Pract. 1981;31:351–356.

[12] Foy H. M., Cooney M. K., Allan I.. Longitudinal studies of types A and B influenza among Seattle schoolchildren and families, 1968–1974 J Infect Dis. 1976;134:362–369.

[13] Jennings L. C., Miles J. A. R.. 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;81:67–75.

[14] Fox J. P., Cooney M. K., Hall C. E., Foy H. M.. 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;116:228–242.

[15] Longini Jr., Koopman J. S., Monto A. S., Fox J. P.. Estimating household and community transmission parameters for influenza Am J Epidemiol. 1982;115:736–751.
[16] Frank A. L., Taber L. H., Glezen W. P., Geyer E. A., 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;118:313–325.

[17] Longini Jr., Monto A. S.. Efficacy of virucidal nasal tissues in interrupting familial transmission of respiratory agents. A field trial in Tecumseh, Michigan Am J Epidemiol. 1988;128:639–644.

[18] Hurwitz E. S., Haber M., Chang A., et al. Effectiveness of influenza vaccination of day care children in reducing influenza-related morbidity among household contacts JAMA. 2000;284:1677–1682.

[19] Katz J. M., Lim W., Bridges C. B., 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;180:1763–1770.

[20] Hayden F. G., Gubareva L. V., Monto A. S., et al. Inhaled zanamivir for the prevention of influenza in families. N Engl J Med. 2000;343:1282–1289.

[21] Welliver R., Monto A. S., Carewicz O., et al. Effectiveness of oseltamivir in preventing influenza in household contacts: a randomized controlled trial JAMA. 2001;285:748–754.

[22] Viboud C., Boëlle P.-Y., Cauchemez S., et al. Risk factors of influenza transmission in households Br J Gen Pract. 2004;54:684–689.

[23] Hayden F. G., 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;189:440–449.
[24] Monto A. S., Pichichero M. E., Blanckenberg S. J., et al. Zanamivir prophylaxis: an effective strategy for the prevention of influenza types A and B within households *J Infect Dis.* 2002;186:1582–1588.

[25] Neuzil K. M., 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;156:986–991.

[26] Hsu C.-Y., Yen A. M.-F., Chen L.-S., Chen H.-H.. Surveillance of influenza from household to community in Taiwan *Trans R Soc Trop Med Hyg.* 2014;108:213–220.

[27] Yang Y., Halloran M. E., Sugimoto J. D., Longini Jr.. Detecting human-to-human transmission of avian influenza A (H5N1) *Emerg Infect Dis.* 2007;13:1348–1353.

[28] Cowling B. J., Fung R. O. P., Cheng C. K. Y., et al. Preliminary findings of a randomized trial of non-pharmaceutical interventions to prevent influenza transmission in households *PLoS One.* 2008;3:e2101.

[29] Ng S., Cowling B. J., Fang V. J., et al. Effects of oseltamivir treatment on duration of clinical illness and viral shedding and household transmission of influenza virus *Clin Infect Dis.* 2010;50:707–714.

[30] Azman A. S., Stark J. H., Althouse B. M., et al. Household transmission of influenza A and B in a school-based study of non-pharmaceutical interventions *Epidemics.* 2013;5:181–186.
[31] Cauchemez S., Ferguson N. M., Fox A., et al. Determinants of influenza transmission in South East Asia: insights from a household cohort study in Vietnam PLoS Pathog. 2014;10:e1004310.

[32] McCaw J. M., Howard P. F., Richmond P. C., 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.

[33] Judd M. C., Emukule G. O., 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;212:740–744.

[34] Simmerman J. M., 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;5:256–267.

[35] Fry A. M., 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;15:654–662.

[36] Cheung D. H., Tsang T. K., Fang V. J., et al. Association of oseltamivir treatment with virus shedding, illness, and household transmission of influenza viruses J Infect Dis. 2015;212:391–396.

[37] Cowling B. J., Chan K. H., Fang V. J., et al. Comparative epidemiology of pandemic and seasonal influenza A in households N Engl J Med. 2010;362:2175–2184.
[38] Mohamed A. G., BinSaeed A. A., 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:e25–e29.

[39] Klick B., Nishiura H., Ng S., et al. Transmissibility of seasonal and pandemic influenza in a cohort of households in Hong Kong in 2009 Epidemioloy. 2011;22:793–796.

[40] Vargas-Leguas H., Cayl`a J. A., Ballester I., et al. Factores asociados a la transmisión a los convivientes de gripe (H1N1) 2009 Rev Esp Salud Pública. 2011;85:57–62.

[41] 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 Thai. 2012;95:403–411.

[42] Carcione D., Giele C. M., Goggin L. S., et al. Secondary attack rate of pandemic influenza A(H1N1) 2009 in Western Australian households, 29 May–7 August 2009 Euro Surveill. 2011;16.

[43] 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:e13702.

[44] Gemert C., Hellard M., McBryde E. S., et al. Intrahousehold transmission of pandemic (H1N1) 2009 virus, Victoria, Australia Emerg Infect Dis. 2011;17:1599–1607.
[45] Janjua N. Z., Skowronski D. M., Hottes T. S., 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:e54–e62.

[46] 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.

[47] Sikora C., Fan S., Golonka R., et al. Transmission of pandemic influenza A (H1N1) 2009 within households: Edmonton, Canada J Clin Virol. 2010;49:90–93.

[48] Chilean Task Force for study of Pandemic Influenza A (H1N1) , Pedroni E., García M., et al. Outbreak of 2009 pandemic influenza A(H1N1), Los Lagos, Chile, April-June 2009 Euro Surveill. 2010;15.

[49] Yang Y., Sugimoto J. D., Halloran M. E., et al. The transmissibility and control of pandemic influenza A (H1N1) virus Science. 2009;326:729–733.

[50] Cauchemez S., Donnelly C. A., Reed C., et al. Household transmission of 2009 pandemic influenza A (H1N1) virus in the United States N Engl J Med. 2009;361:2619–2627.

[51] Doyle T. J., Hopkins R. S., 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;139:45–51.
[52] France A. M., 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;201:984–992.

[53] Loustalot F., Silk B. J., Gaither A., et al. Household transmission of 2009 pandemic influenza A (H1N1) and nonpharmaceutical interventions among households of high school students in San Antonio, Texas Clin Infect Dis. 2011;52 Suppl 1:S146–S153.

[54] Jackson M. L., France A. M., 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;53:455–462.

[55] Morgan O. W., Parks S., Shim T., et al. Household transmission of pandemic (H1N1) 2009, San Antonio, Texas, USA, April–May 2009 Emerg Infect Dis. 2010;16:631–637.

[56] Papenburg J., Baz M., Hamelin M.-È., 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;51:1033–1041.

[57] 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;61:284–288.

[58] Pebody R. G., Harris R., Kafatos G., et al. Use of antiviral drugs to reduce household transmission of pandemic (H1N1) 2009, United Kingdom Emerg Infect Dis. 2011;17:990–999.
[59] House T., Inglis N., Ross J. V., et al. Estimation of outbreak severity and transmissibility: Influenza A(H1N1)pdm09 in households *BMC Med.* 2012;10:117.

[60] Chang L.-Y., Chen W.-H., Lu C.-Y., et al. Household transmission of pandemic (H1N1) 2009 virus, Taiwan *Emerg Infect Dis.* 2011;17:1928–1931.

[61] Boven M., Donker T., Lubben M., et al. Transmission of novel influenza A(H1N1) in households with post-exposure antiviral prophylaxis *PLoS One.* 2010;5:e11442.

[62] 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;171:1157–1164.

[63] Calatayud L., Kurkela S., Neave P. E., et al. Pandemic (H1N1) 2009 virus outbreak in a school in London, April-May 2009: an observational study *Epidemiol Infect.* 2010;138:183–191.

[64] Leung Y. H., Li M. P., Chuang S. K.. A school outbreak of pandemic (H1N1) 2009 infection: assessment of secondary household transmission and the protective role of oseltamivir *Epidemiol Infect.* 2011;139:41–44.

[65] Thai P. Q., Mai L. Q., Welkers M. R. A., et al. Pandemic H1N1 virus transmission and shedding dynamics in index case households of a prospective Vietnamese cohort *J Infect.* 2014;68:581–590.

[66] 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 *Euro Surveill.* 2009;14.
[67] Goldstein E., Cowling B. J., O’Hagan J. J., et al. Oseltamivir for treatment and prevention of pandemic influenza A/H1N1 virus infection in households, Milwaukee, 2009 *BMC Infect Dis.* 2010;10:211.

[68] Remschmidt C., Stöcker P., Heiden M., 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;7:418–425.

[69] 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;39:619–628.

[70] 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:e31519.

[71] Behnaz F., Mohammadzadeh M., Sadeghian M.. Household transmission of 2009 H1N1 influenza virus in Yazd, Iran *J Infect Public Health.* 2012;5:275–280.

[72] Kim C. Y., Breiman R. F., 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:e38166.

[73] 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:e108485.
[74] Ram P. K., DiVita M. A., Jannat K., 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:e0125200.

[75] Weaver A. M., Jannat K., Cercone E., et al. Household-level risk factors for secondary influenza-like illness in a rural area of Bangladesh *Trop Med Int Health.* 2017;22:187–195.

[76] 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:e33046.

[77] Petrie J. G., Ohmit S. E., Cowling B. J., et al. Influenza transmission in a cohort of households with children: 2010-2011 *PLoS One.* 2013;8:e75339.

[78] Ohmit S. E., Petrie J. G., Malosh R. E., et al. Influenza vaccine effectiveness in the community and the household *Clin Infect Dis.* 2013;56:1363–1369.

[79] 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;13:123–132.

[80] 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;19:740–749.

[81] Buchwald A. G., Tamboura B., Haidara F. C., 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;100:159–164.
[82] 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;35:583–586.

[83] Gordon A., Tsang T. K., Cowling B. J., et al. Influenza transmission dynamics in urban households, Managua, Nicaragua, 2012–2014 *Emerg Infect Dis.* 2018;24:1882–1888.

[84] 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;59:129–131.

[85] Iyengar P., Mollendorf C., Tempia S., et al. Case-ascertained study of household transmission of seasonal influenza — South Africa, 2013 *J Infect.* 2015;71:578–586.

[86] Valley-Omar Z., Iyengar P., 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:e0198101.

[87] 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;219:1605–1615.

[88] 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;20:21056.
[89] Petrie J. G., Malosh R. E., Cheng C. K., 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;65:1644–1651.

[90] Venables W. N., Ripley B. D.. Modern Applied Statistics with S. New York: Springerfourth ed. 2002. ISBN 0-387-95457-0.

[91] Soetaert Karline. plot3D: Plotting Multi-Dimensional Data 2019. R package version 1.3.