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 β and γ were often insufficient to make inference; although, the marginal posterior probability distribution of α and the posterior probability distibution of the ratio β/γ 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 β and γ (Figure S3).

For influenza A (H1) during 2013–2014, the uniform prior clipped the right tail of the marginal posterior probability distribution of γ (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 γ. This is because the two apparent secondary cases occured the day after the household index case. Exponential distributions defined by these large values of γ 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 β and γ 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 β and γ were typically insufficient to make inference about infectiousness or the length of the infectious period, but the distrubiton of α and β/γ were sufficient to make inference about the CAR and the SAR (Figure) S7). The correlation between β and γ is what allowed inference using β/γ (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 competinig 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 $2\binom{n}{2} + n$ 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 ∞ 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 occured.
  - 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 ∞ 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 ∞.

For each trial, we would expect the CAR to be about $1 - e^{-ht} = 1 - e^{-0.0002 \cdot 350} \approx 0.0676$, and the SAR to be about $\frac{\beta}{2\gamma} = \frac{0.02}{2 \cdot 0.07} = 0.14$. Our simulation of the data never used α, but we applied Jensen's inequality and the law of total probability to approximate

$$\alpha \gtrapprox \frac{\text{CAR}}{\text{CAR} + \text{SAR} \cdot \text{CAR}} = \frac{1}{1 + \text{SAR}} \approx 0.877.$$

Because our rate h is small, we expect our lower bound on α 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 α (Table S1). Our point estimates for β and γ 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).

S4 Supplemental Tables

Table S1: Summary of parameter estimates from the simulation study.

Trial	α (95% CI)	β (95% CI)	γ (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.

			Secondary Attack Rate					
Time	Virus	Location	Syndromic	RIDT	Seroconversion	PCR	Culture	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]
1965–1969	A	Seattle, Washington, USA			27			Hall 1973[7]
1965–1969	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.

			Secondary Attack Rate					
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		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			6		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.

			Secondary Attack Rate					
Time	Virus	Location	Syndromic	RIDT	Seroconversion	PCR	Culture	Reference
2007–2008	A	Pittsburgh, Pennsylvania, USA	31					Azman 2013[30]
2007–2008	B	Pittsburgh, Pennsylvania, USA	25					Azman 2013[30]
2007–2010	A and B	Vietnam			8			Cauchemez 2014[31]
2008	A and B	Australia	24					McCaw 2012[32]
2008	A and B	Nairobi, Kenya	9					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 Gemert 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.

			Secondary Attack Rate					
Time	Virus	Location	Syndromic	RIDT	Seroconversion	PCR	Culture	Reference
2009	pH1N1	Ontaio, 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[37]
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.

			Secondary Attack Rate					
Time	Virus	Location	Syndromic	RIDT	Seroconversion	PCR	Culture	Reference
2009	pH1N1	Kobe, Japan				8		Odaira 2009[66]
2009	pH1N1	Milwaukee, Wisconsin, USA				13		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					Behnaz 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.

			Secondary Attack Rate					
Time	Virus	Location	Syndromic	RIDT	Seroconversion	PCR	Culture	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[87]
2013–2014	pH1N1	South Africa				18		Cohen 2019[87]
2013–2014	A	South Africa				20		Cohen 2019[87]
2013–2014	B	South Africa				28		Cohen 2019[87]
2013–2014	H7N9	China				1		Yang 2015[88]
2014–2015	H3N2	Ann Arbor, Michigan, USA				17		Petrie 2017[89]
2014–2015	B Yamagata	Ann Arbor, Michigan, USA				6		Petrie 2017[89]

S5 Supplemental Figures

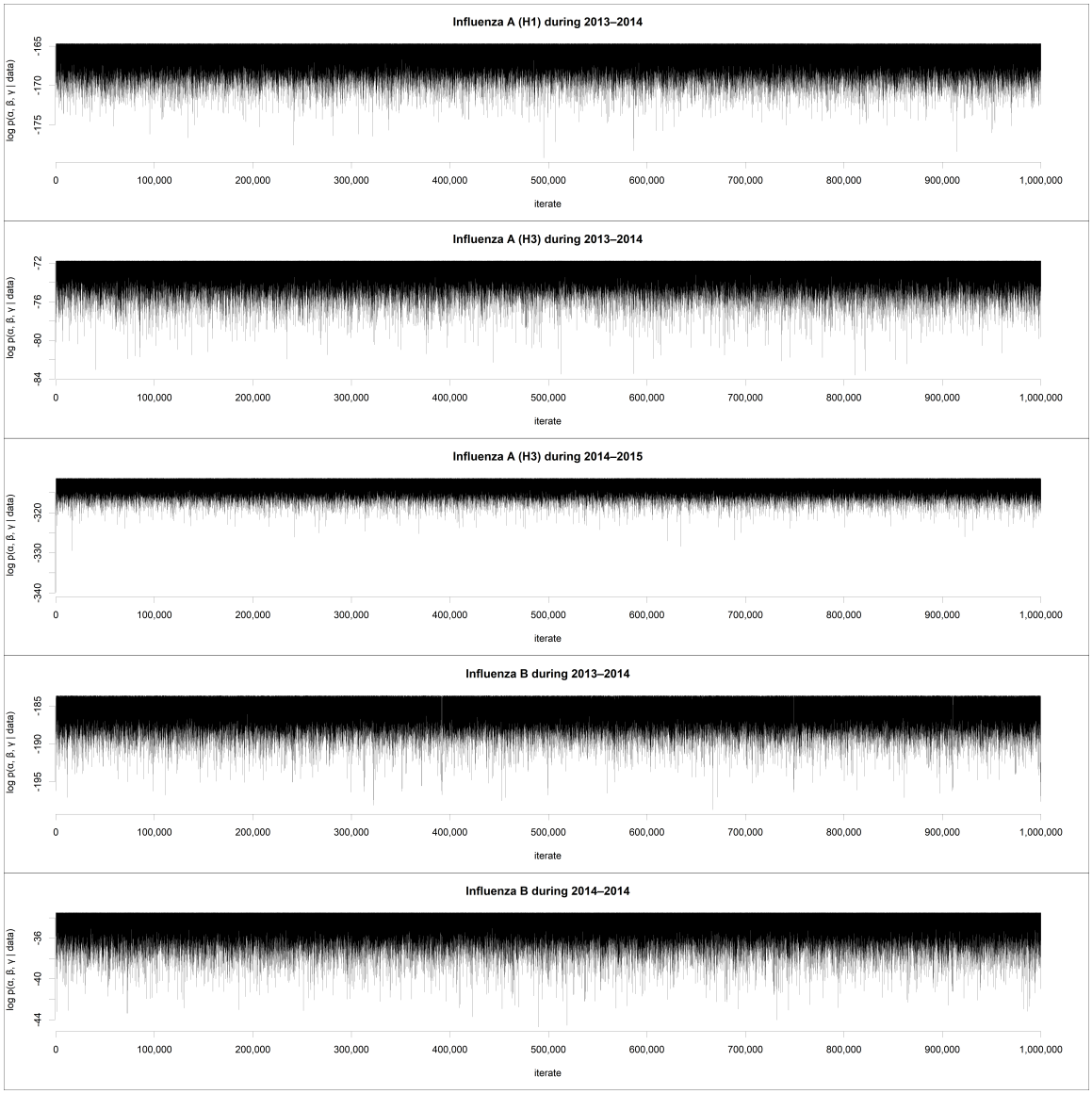

Figure S1: The joint posterior probability, up to proportionality, of α, β, and γ for the primary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015.

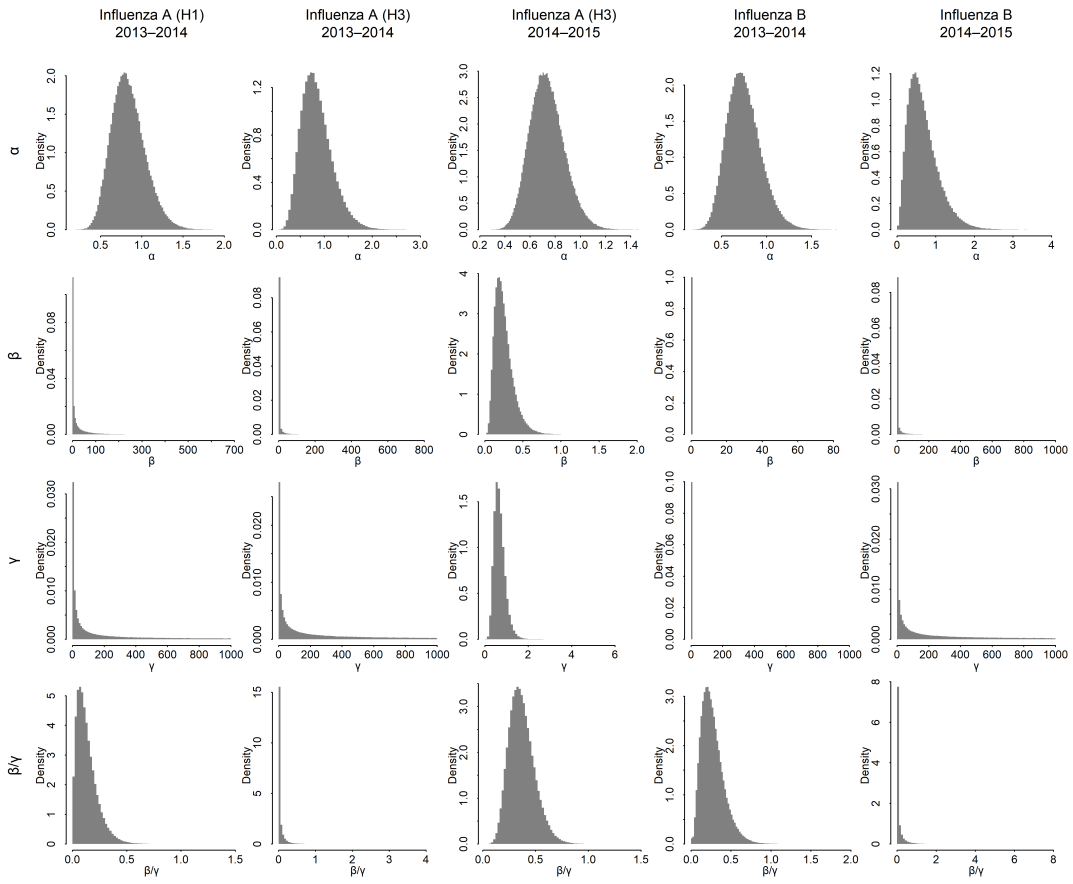

Figure S2: The marginal posterior distribution of α, β, and γ, and posterior distribution of β/γ from the primary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015.

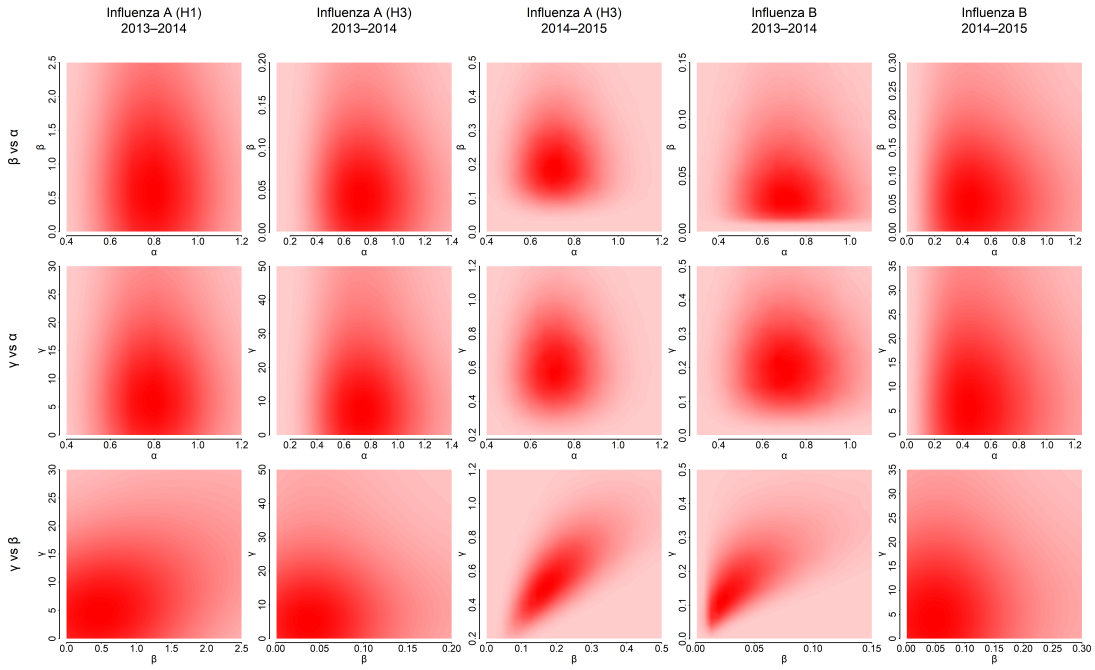

Figure S3: Kernel smoothed posterior distribution for pairs of α, β, and γ from the primary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015. Distributions were clipped for clarity. Density estimated using the MASS package in R.[90]

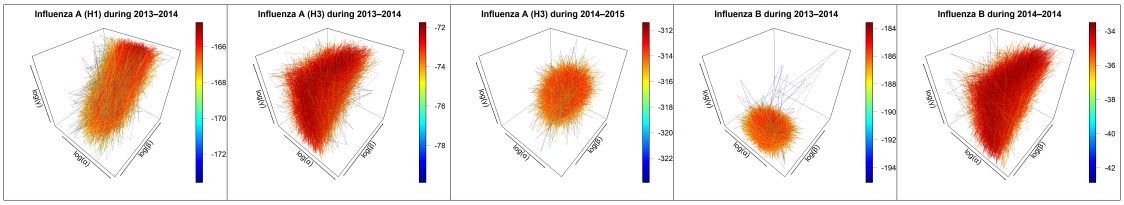

Figure S4: Trace plots of the posterior distribution of α, β, and γ colored by the logarithm of the estimated posterior probability from the primary analysis of influenza A (H1) during influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B 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]

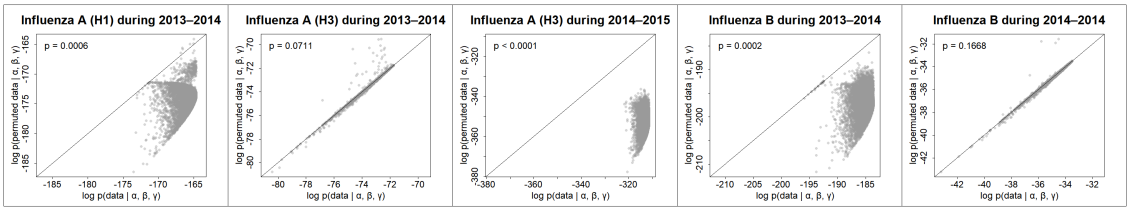

Figure S5: The likelihood of the permuted onset dates versus the likelihood of the observed onset dates from the primary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015. 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.

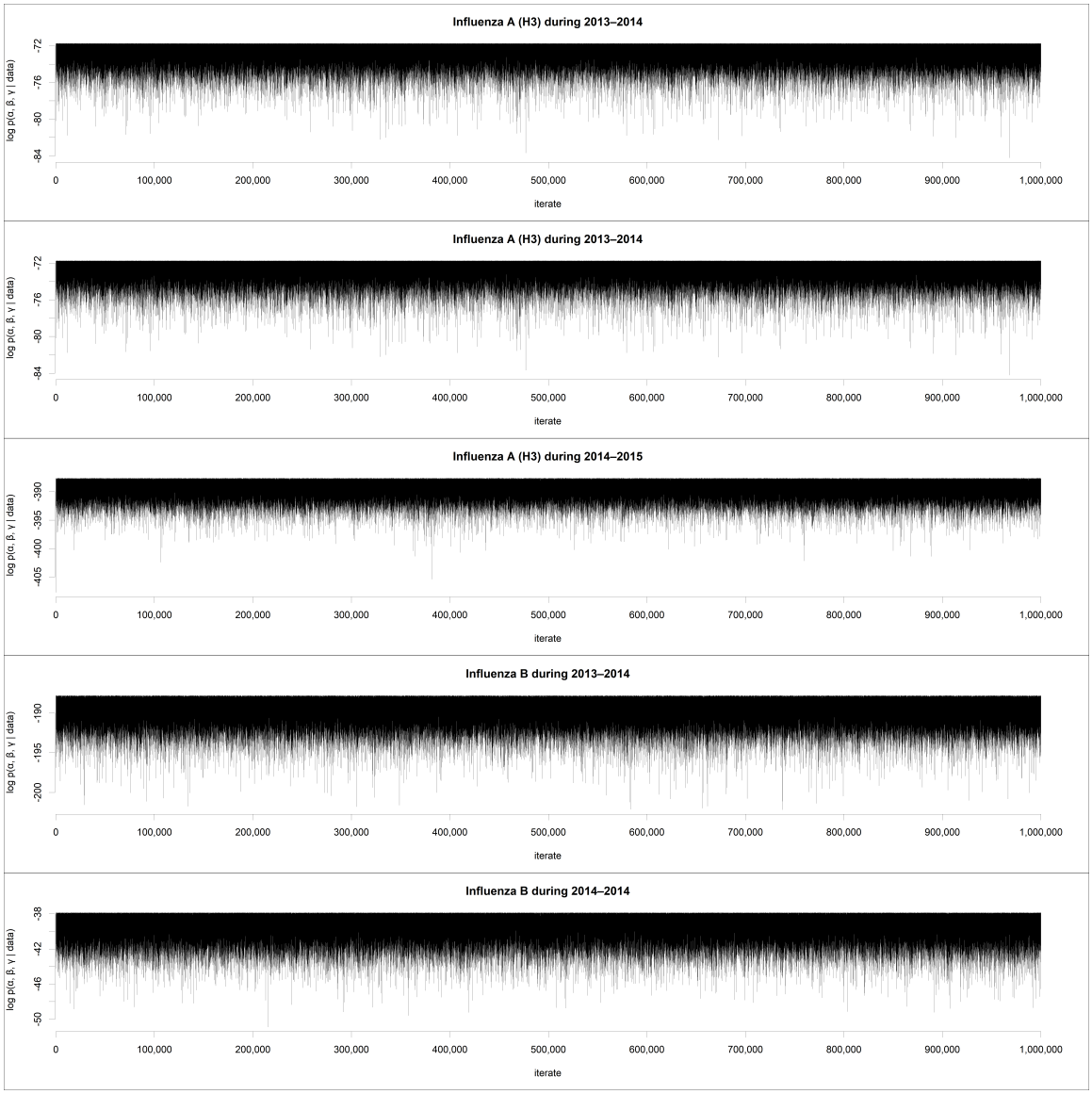

Figure S6: The joint posterior probability, up to proportionality, of α, β, and γ for the secondary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015.

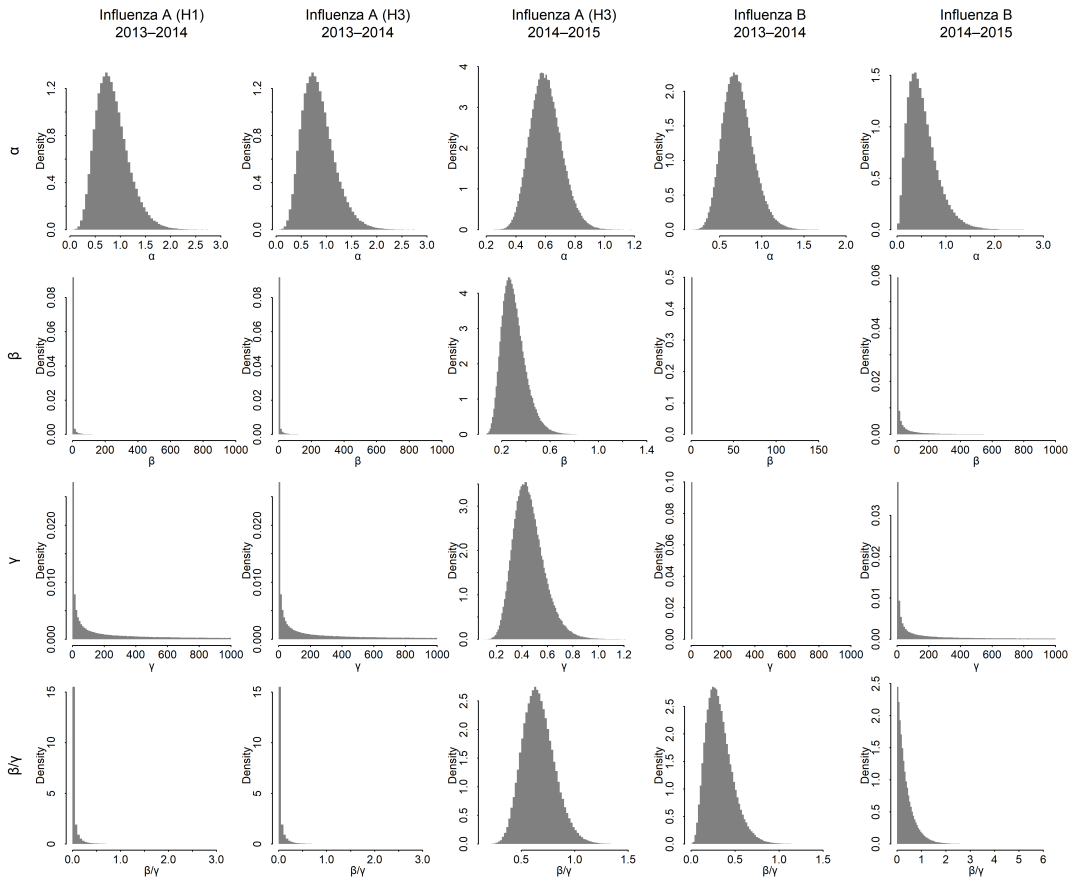

Figure S7: Marginal posterior distribution of α, β, and γ, and posterior distribution of β/γ from the secondary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015.

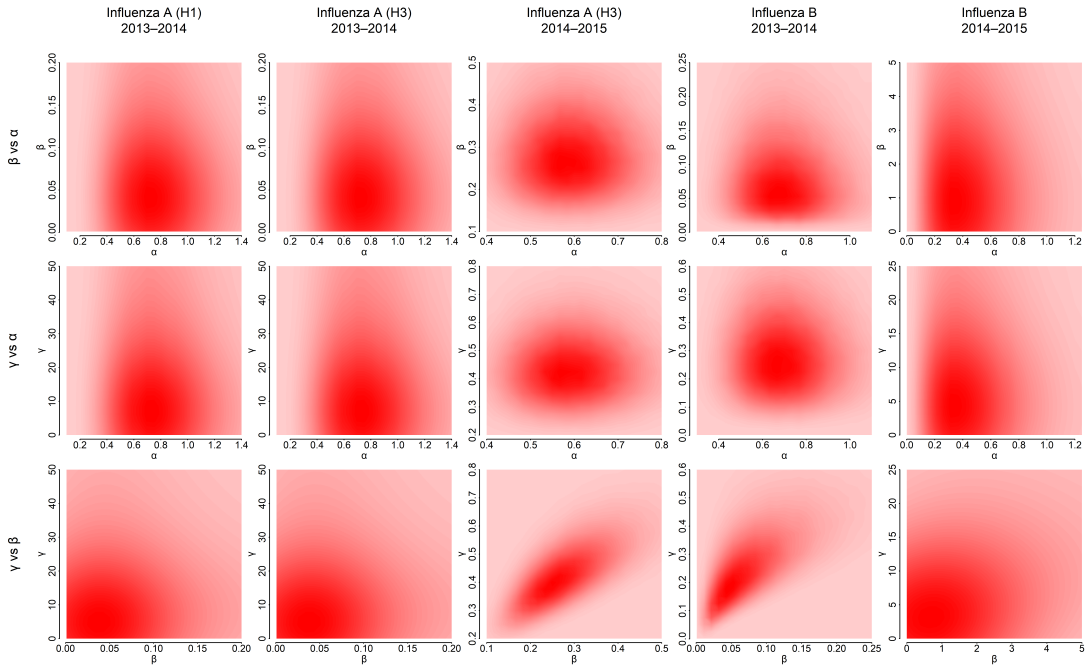

Figure S8: Kernel smoothed posterior distribution for pairs of α, β, and γ from the secondary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015. Distributions were clipped for clarity. Density estimated using the MASS package in R.[90]

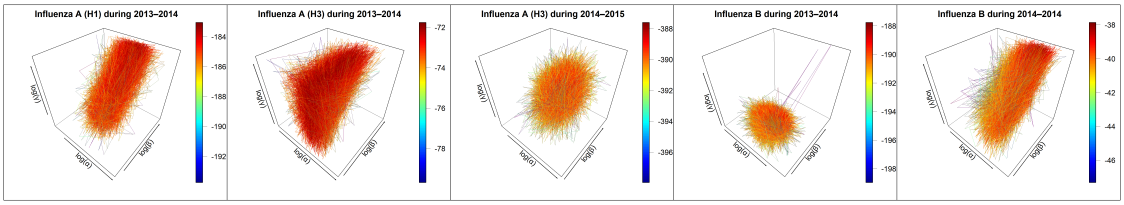

Figure S9: Trace plots of the posterior distribution of α, β, and γ colored by the logarithm of the estimated posterior probability from the secondary analysis of influenza A (H1) during influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza 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]

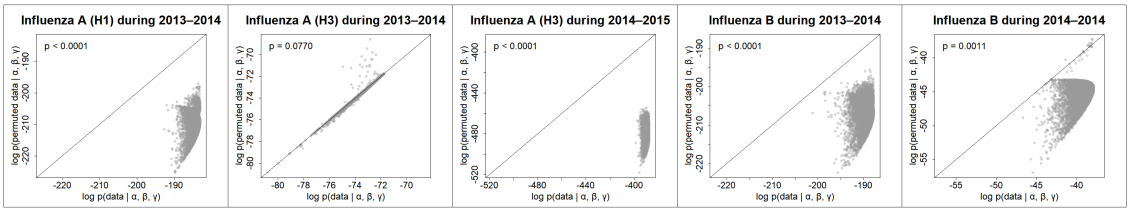

Figure S10: The likelihood of the permuted onset dates versus the likelihood of the observed onset dates from the secondary analysis of influenza A (H1) during 2013–2014, influenza A (H3) during 2013–2014, influenza A (H3) during 2014–2015, influenza B during 2013–2014, and influenza B during 2014–2015. 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.

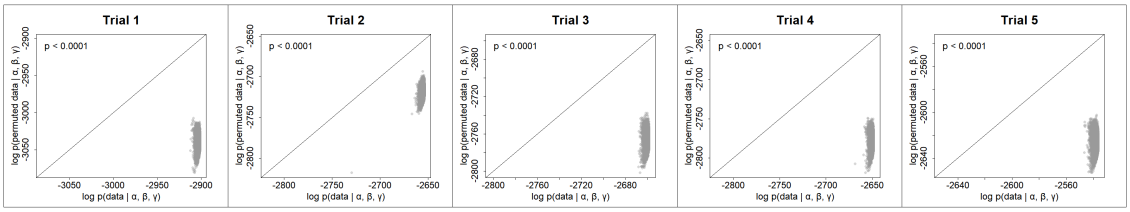

Figure S11: The likelihood of the permuted onset dates versus the likelihood of the observed onset dates from the simulation study. Points below the line indicate higher likelihood of the observed data compared to the permuted data. The Monte Carlo estimate of the probability of a point being above the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

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