SUPPLEMENTARY INFORMATION

S1 Complete Data Likelihood for SIS-type Contagions

For an SIS-type infectious disease, the complete data likelihood can be derived following the same steps in Section 3.2. Alternatively, one can slightly modify (22) to arrive at the complete likelihood for an SIS-type contagion. Since an individual doesn't acquire immunity upon recovery, it is equivalent to setting $H(t) \equiv S(t)$ at any time t. Thus the complete data likelihood is

$$\begin{aligned}
&\mathcal{L}(\beta, \gamma, \tilde{\alpha}, \tilde{\omega} | \mathcal{G}_0) \\
=&\gamma^{n_R} \beta^{n_E - 1} \alpha_{SS}^{C_{SS}} \alpha_{SI}^{C_{SI}} \alpha_{II}^{C_{II}} \omega_{SS}^{D_{SS}} \omega_{SI}^{D_{SI}} \omega_{II}^{D_{II}} \prod_{j=2}^{n} \left[\tilde{M}(t_j) \left(I_{p_{j1}}(t_j) \right)^{F_j} \right] \\
&\times \exp\left(- \int_0^{T_{\max}} \left[\beta SI(t) + \gamma I(t) + \tilde{\alpha}^T \mathbf{M}_{\max}(t) + (\tilde{\omega} - \tilde{\alpha})^T \mathbf{M}(t) \right] dt \right). \qquad (13)
\end{aligned}$$

S2 Auxiliary Proofs and Derivations

Derivation of complete data likelihood Let i_k be the infection time for individual k ($k = 1, 2, \ldots, n_E$), r'_k be the k'th observed recovery time ($k' = 1, 2, \ldots, n_R$), and without loss of generality, set $i_1 = 0$. Recall that the widely used "random mixing" assumption in classical epidemiological models is equivalent to assuming that the contact network is a complete graph, $\mathcal{K}_N$, and the complete data likelihood under this assumption is

$$\mathcal{L}(\beta, \gamma) = p(\text{epidemic events} | \beta, \gamma) = \gamma^{n_R} \prod_{k=2}^{n_E} \left[\beta I(i_k) \right] \exp\left(- \int_0^{T_{\max}} \left[\beta S(u) I(u) + \gamma I(u) \right] du \right).$$

Instead, assume that the network is an arbitrary *static* network $\mathcal{G}$. Then when writing down the complete data likelihood, we have to explicitly account for the number of infected contacts for each individual at the time of infection as well as the total number of S-I links in the system:

$$\mathcal{L}(\beta, \gamma | \mathcal{G}) = \gamma^{n_R} \prod_{k=2}^{n_E} \left[\beta I_k(i_k) \right] \exp\left(- \int_0^{T_{\max}} \left[\beta SI(u) + \gamma I(u) \right] du \right). \qquad (14)$$

Here $I_k(t)$ denotes the number of infectious individuals that are connected to person k at time t, which remains fixed for a static network.

If the network is not static, but the *entire network process* $\{\mathcal{G}_t : 0 < t < T_{\max}\}$ is given (or all the network changes during the contagion process are fully observed), then the form of the data likelihood remains unchanged, *conditioned* on $\{\mathcal{G}_t\}$. Note that the dynamic nature of the network can be implicitly subsumed into the terms $I_k(i_k)$'s and $SI(u)$.

To incorporate network dynamics, we begin with the simpler *decoupled* process in which the network evolves *independently* of the epidemic process with edge activation rate α and deletion rate ω. Given the initial network $\mathcal{G}_0$, the likelihood of the network process alone can be easily written down as

$$\begin{aligned}
&\mathcal{L}(\alpha,\omega|\mathcal{G}_0) = p(\text{network events}|\alpha,\omega,\mathcal{G}_0)\\
=&\alpha^C\omega^D\prod_{\ell=1}^{n_N}\left[\left(\frac{N(N-1)}{2}-M(s_\ell)\right)^{1-D_\ell}M(s_\ell)^{D_\ell}\right]\\
&\times\exp\left(-\alpha\frac{N(N-1)}{2}T_{\max}+(\alpha-\omega)\int_0^{T_{\max}}M(u)du\right). \qquad (15)
\end{aligned}$$

Here s_ℓ is the time of the ℓth network event, and $D_\ell = 1$ if this event is a *link termination* and otherwise $D_\ell = 0$. Therefore, when the epidemic process and network process are decoupled, the complete data likelihood is simply a product of the terms in (14) and (15):

$$\begin{aligned}
&\mathcal{L}(\beta,\gamma,\alpha,\omega|\mathcal{G}_0) = p(\text{epidemic events}|\beta,\gamma,\mathcal{G}_t)p(\text{network events}|\alpha,\omega,\mathcal{G}_0)\\
=&\beta^{n_E-1}\gamma^{n_R}\alpha^C\omega^D\prod_{k=2}^{n_E}[I_k(i_k)]\prod_{\ell=1}^{n_N}\left[\left(\frac{N(N-1)}{2}-M(s_\ell)\right)^{1-D_\ell}M(s_\ell)^{D_\ell}\right] \qquad (16)\\
&\times\exp\left(-\int_0^{T_{\max}}[\beta SI(u)+\gamma I(u)+(\omega-\alpha)M(u)]\,du-\alpha\frac{N(N-1)}{2}T_{\max}\right).
\end{aligned}$$

Then we consider the coupled process with an *adaptive* network, where link activation and termination are dependent on individual disease status. Define $g(p,t)$ as the indicator function of infectiousness, i.e. $g(p,t) = 1$ if person p is infected at time t and $g(p,t) = 0$ otherwise. We further assume that the S and R populations behave identically in the network process:

$$\alpha_{R\cdot} \equiv \alpha_{S\cdot}, \text{ and } \omega_{R\cdot} \equiv \omega_{S\cdot}.$$

We shall refer to them as the "H" (healthy) population; let $H(t) = R(t) + S(t) = N - I(t)$ denote the number of healthy individuals at time t. Naturally the term "H-H link" represent an S-S link, an S-R link, or an R-R link, and the term "H-I link" represent either an S-I link or an R-I link.

Combine the epidemic events and network events and re-denote all events as $\{e_j = (t_j, p_{j1}, p_{j2})\}_{j=1}^{n}$, $n = n_E + n_R + n_N$. Here t_j is the event time (set $t_1 = 0$, the infection time of the first patient), with $t_1 < t_2 < \ldots < t_n$. If e_j is a network event, p_{j1}, p_{j2} are the two individuals getting connected or disconnected, and if e_j is an epidemic event, let p_{j1} be the person getting infected or recovered and set $p_{j2} = 0$. Furthermore let event type indicators F_j, C_j, D_j take the value 1 only if e_j is an infection, a link activation, and a link deletion, respectively, and otherwise take the value 0.

The contribution of all network events to the complete data likelihood is in essence of the same form as (15), except that for every activation or termination event the link type has to be considered. Then the likelihood component of the adaptive network process is

$$\alpha_{SS}^{C_{HH}} \alpha_{SI}^{C_{HI}} \alpha_{II}^{C_{II}} \omega_{SS}^{D_{HH}} \omega_{SI}^{D_{HI}} \omega_{II}^{D_{II}} \prod_{j=2}^{n} \tilde{M}(t_j) \exp\left(- \int_0^{T_{\max}} \left[\tilde{\alpha}^T \mathbf{M}_{\max}(t) + (\tilde{\omega} - \tilde{\alpha})^T \mathbf{M}(t) \right] dt \right),$$

where

$$\begin{aligned}
\tilde{M}(t_j) = & \left[(\alpha_{SS} M_{HH}^d(t_j))^{C_j} (\omega_{SS} M_{HH}(t_j)^{D_j} \right]^{(1-g(p_{j1},t_j))(1-g(p_{j2},t_j))} \\
& \times \left[(\alpha_{SI} M_{HI}^d(t_j))^{C_j} (\omega_{SI} M_{HI}(t_j))^{D_j} \right]^{|g(p_{j1},t_j) - g(p_{j2},t_j)|} \qquad (17) \\
& \times \left[(\alpha_{II} M_{II}^d(t_j))^{C_j} (\omega_{II} M_{II}(t_j))^{D_j} \right]^{g(p_{j1},t_j) g(p_{j2},t_j)} \\
\tilde{\alpha} = & (\alpha_{SS}, \alpha_{SI}, \alpha_{II})^T, \qquad (18) \\
\tilde{\omega} = & (\omega_{SS}, \omega_{SI}, \omega_{II})^T, \qquad (19) \\
\mathbf{M}_{\max}(t) = & \left(\frac{H(t)(H(t)-1)}{2}, H(t)I(t), \frac{I(t)(I(t)-1)}{2} \right)^T, \qquad (20) \\
\mathbf{M}(t) = & (M_{HH}(t_j), M_{HI}(t), M_{II}(t))^T. \qquad (21)
\end{aligned}$$

Therefore, given the initial network structure $\mathcal{G}_0$ and one infected case at time 0, the complete data likelihood can be expressed as

$$\mathcal{L}(\beta, \gamma, \tilde{\alpha}, \tilde{\omega} | \mathcal{G}_0) = p(\text{epidemic events}, \text{network events} | \beta, \gamma, \tilde{\alpha}, \tilde{\omega}, \mathcal{G}_0)$$

$$
\begin{aligned}
=&\gamma^{n_R}\beta^{n_E-1}\alpha_{SS}^{C_{HH}}\alpha_{SI}^{C_{HI}}\alpha_{II}^{C_{II}}\omega_{SS}^{D_{HH}}\omega_{SI}^{D_{HI}}\omega_{II}^{D_{II}}\prod_{j=2}^{n}\left[\tilde{M}(t_j)\left(I_{p_{j1}}(t_j)\right)^{F_j}\right]\\
&\times\exp\left(-\int_0^{T_{\max}}\left[\beta SI(t)+\gamma I(t)+\tilde{\alpha}^T\mathbf{M}_{\max}(t)+(\tilde{\omega}-\tilde{\alpha})^T\mathbf{M}(t)\right]dt\right). \qquad (22)
\end{aligned}
$$

Proof for Theorem 3.1 From (22), we can obtain the log-likelihood:

$$
\begin{aligned}
&\ell(\beta,\gamma,\tilde{\alpha},\tilde{\omega}|\mathcal{G}_0)=\log\mathcal{L}(\beta,\gamma,\tilde{\alpha},\tilde{\omega}|\mathcal{G}_0)\\
=&\sum_{j=2}^{n}\left[\log\tilde{M}(t_j)+F_j\log\left(I_{p_{j1}}(t_j)\right)\right]+n_R\log\gamma+(n_E-1)\log\beta \qquad (23)\\
&+C_{HH}\log\alpha_{SS}+C_{HI}\log\alpha_{SI}+C_{II}\log\alpha_{II}+D_{HH}\log\omega_{SS}+D_{HI}\log\omega_{SI}+D_{II}\log\omega_{II}\\
&-\sum_{j=1}^{n}\left[\beta SI(t_j)+\gamma I(t_j)+\tilde{\alpha}^T(\mathbf{M}_{\max}(t_j)-\mathbf{M}(t_j))+\tilde{\omega}^T\mathbf{M}(t_j)\right](t_j-t_{j-1}).
\end{aligned}
$$

Taking partial derivatives of the right hand side of (23) with respect to the parameters and setting them to zero yields the MLEs

$$
\begin{aligned}
\hat{\beta}&=\frac{n_E-1}{\sum_{j=1}^{n}SI(t_j)(t_j-t_{j-1})}, \quad \hat{\gamma}=\frac{n_R}{\sum_{j=1}^{n}I(t_j)(t_j-t_{j-1})},\\
\hat{\alpha}_{SS}&=\frac{C_{HH}}{\sum_{j=1}^{n}\left[\frac{H(t_j)(H(t_j)-1)}{2}-M_{HH}(t_j)\right](t_j-t_{j-1})}, \quad \hat{\omega}_{SS}=\frac{D_{HH}}{\sum_{j=1}^{n}M_{HH}(t_j)(t_j-t_{j-1})},\\
\hat{\alpha}_{SI}&=\frac{C_{HI}}{\sum_{j=1}^{n}\left[H(t_j)I(t_j)-M_{HI}(t_j)\right](t_j-t_{j-1})}, \quad \hat{\omega}_{SI}=\frac{D_{HI}}{\sum_{j=1}^{n}M_{HI}(t_j)(t_j-t_{j-1})},\\
\hat{\alpha}_{II}&=\frac{C_{II}}{\sum_{j=1}^{n}\left[\frac{I(t_j)(I(t_j)-1)}{2}-M_{II}(t_j)\right](t_j-t_{j-1})}, \quad \hat{\omega}_{II}=\frac{D_{II}}{\sum_{j=1}^{n}M_{II}(t_j)(t_j-t_{j-1})}.
\end{aligned}
$$

Simplifying the joint conditional distribution of missing recovery times

Lemma S2.1. *The left hand side of (10) in Section 4 can be simplified into the following expression:*

$$
p\left(\{r_{\ell,i}\}_{i=1:R_\ell}\mid\gamma^{(s-1)},\{e_j\}_{t_j\in(u_\ell,v_\ell]},\mathcal{Z}_{u_\ell}\right), \qquad (24)
$$

where $\mathcal{Z}_t$ is the state of the process at time t, including the epidemic status of each individual and the social network structure.

Proof. Consider the joint density of the complete data given parameter values $\Theta^{(s-1)}$.

$$
\begin{aligned}
& p\left(\mathbf{x}, \{r_{\ell,i}\}_{i=1:R_\ell, \ell=1:L} | \Theta^{(s-1)}\right) \\
= & \prod_{\ell=1:L} \left[p\left(\{e_j\}_{t_j \in (u_\ell, u_{\ell+1}]}, \{r_{\ell,i}\}_{i=1:R_\ell} | \mathcal{Z}_{u_\ell}, \Theta^{(s-1)}\right)\right] \times p\left(\{e_j\}_{t_j \le u_1 \text{ or } t_j > v_L} | \mathcal{Z}_0, \mathcal{Z}_{v_L}, \Theta^{(s-1)}\right) \\
= & \prod_{\ell=1:L} \left[p\left(\{e_j\}_{t_j \in (u_\ell, v_\ell]} | \{r_{\ell,i}\}_{i=1:R_\ell}, \mathcal{Z}_{u_\ell}, \Theta^{(s-1)}\right) p\left(\{r_{\ell,i}\}_{i=1:R_\ell} | \mathcal{Z}_{u_\ell}, \gamma^{(s-1)}\right)\right] \\
& \times \left[\prod_{\ell=1:L} p\left(\{e_j\}_{t_j \in (v_\ell, u_{\ell+1}]} | \mathcal{Z}_{v_\ell}, \Theta^{(s-1)}\right)\right] p\left(\{e_j\}_{t_j \le u_1 \text{ or } t_j > v_L} | \mathcal{Z}_0, \mathcal{Z}_{v_L}, \Theta^{(s-1)}\right).
\end{aligned}
$$

Examining all terms concerning $\{r_{\ell,i}\}_{i=1:R_\ell}$ for each ℓ leads to

$$
p\left(\{r_{\ell,i}\}_{i=1:R_\ell} | \Theta^{(s-1)}, \mathbf{x}, \{r_{\ell',i}\}_{i=1:R_{\ell'}, \ell' \neq \ell}\right) = p\left(\{r_{\ell,i}\}_{i=1:R_\ell} | \gamma^{(s-1)}, \{e_j\}_{t_j \in (u_\ell, v_\ell]}, \mathcal{Z}_{u_\ell}\right).
$$

□

The lemma above suggests that imputation of missing recovery times inside an interval $(u, v]$ only depends on the events that occur in $(u, v]$, the state of the process at the start of the interval, $\mathcal{Z}_u$, and the value of recovery rate γ.

S3 Relaxing the Closed Population Assumption

Suppose the observed population is not fully closed, but is a subset of a larger yet unobserved population. Then it is possible for an individual to get infected by an outsider. Let ξ be the "external infection" rate, the rate for any susceptible individual to be infected by any external infectious source, then the complete data likelihood is

$$
\begin{aligned}
& \mathcal{L}(\beta, \xi, \gamma, \tilde{\alpha}, \tilde{\omega} | \mathcal{G}_0) = p(\text{epidemic events}, \text{network events} | \beta, \xi, \gamma, \alpha, \omega, \mathcal{G}_0) \\
= & \gamma^{n_R} \alpha_{SS}^{C_{HH}} \alpha_{SI}^{C_{HI}} \alpha_{II}^{C_{II}} \omega_{SS}^{D_{HH}} \omega_{SI}^{D_{HI}} \omega_{II}^{D_{II}} \prod_{j=2}^{n} \left[\tilde{M}(t_j) \left(\beta I_{p_{j1}}(t_j) + \xi\right)^{F_j}\right] \\
& \times \exp\left(-\int_0^{T_{\max}} \left[\beta S I(t) + \xi S(t) + \gamma I(t) + \tilde{\alpha}^T \mathbf{M}_{\max}(t) + (\tilde{\omega} - \tilde{\alpha})^T \mathbf{M}(t)\right] dt\right). \qquad (25)
\end{aligned}
$$

MLEs for $\{\gamma, \tilde{\alpha}, \tilde{\omega}\}$ remain unchanged, but estimating β and ξ is less straightforward. Let $\ell(\beta, \xi, \gamma, \tilde{\alpha}, \tilde{\omega}|\mathcal{G}_0)$ be the log-likelihood, then the partial derivatives of the log-likelihood w.r.t. β and ξ are

$$\frac{\partial \ell}{\partial \beta} = \sum_{j=2}^{n} \frac{F_j I_{p_{j1}}(t_j)}{\beta I_{p_{j1}}(t_j) + \xi} - \sum_{j=1}^{n} SI(t_j)(t_j - t_{j-1}),$$
$$\frac{\partial \ell}{\partial \xi} = \sum_{j=2}^{n} \frac{F_j}{\beta I_{p_{j1}}(t_j) + \xi} - \sum_{j=1}^{n} S(t_j)(t_j - t_{j-1}),$$

which do not directly lead to closed-form solutions.

Reparameterizing by $\xi = \kappa\beta$ leads to the following partially derivatives

$$\frac{\partial \ell}{\partial \beta} = \frac{n_E - 1}{\beta} - \sum_{j=1}^{n} [SI(t_j) + \kappa S(t_j)](t_j - t_{j-1}), \tag{26}$$
$$\frac{\partial \ell}{\partial \kappa} = \sum_{j=2}^{n} \frac{F_j}{I_{p_{j1}}(t_j) + \kappa} - \beta \sum_{j=1}^{n} S(t_j)(t_j - t_{j-1}), \tag{27}$$

which are slightly more straightforward in form, and can be solved numerically to obtain the MLEs.

If, somehow, we have information on which infection cases are caused by internal sources and which are caused by external sources, then we can directly obtain the MLEs and Bayesian posterior distributions for all the parameters. For an infection event e_j (with $F_j = 1$), let $\text{Int}_j = 1$ if it is "internal" and let $\text{Int}_j = 0$ otherwise. Then the complete data likelihood can be re-written as

$$\begin{aligned}
&\mathcal{L}(\beta, \xi, \gamma, \tilde{\alpha}, \tilde{\omega}|\mathcal{G}_0) \\
=&\beta^{\left(n_E^{\text{int}} - \text{Int}_1\right)} \xi^{\left(n_E^{\text{ext}} - 1 + \text{Int}_1\right)} \gamma^{n_R} \alpha_{SS}^{C_{HH}} \alpha_{SI}^{C_{HI}} \alpha_{II}^{C_{II}} \omega_{SS}^{D_{HH}} \omega_{SI}^{D_{HI}} \omega_{II}^{D_{II}} \prod_{j=2}^{n} \left[\tilde{M}(t_j) I_{p_{j1}}(t_j)^{F_j \text{Int}_j}\right] \\
&\times \exp\left(-\int_0^{T_{\max}} \left[\beta SI(t) + \xi S(t) + \gamma I(t) + \tilde{\alpha}^T \mathbf{M}_{\max}(t) + (\tilde{\omega} - \tilde{\alpha})^T \mathbf{M}(t)\right] dt\right),
\end{aligned} \tag{28}$$

where n_E^{int} and n_E^{ext} are the total numbers of internal and external infection events, respectively.

Estimation for all parameters remains unchanged except for β and ξ. Their MLEs are

$$\hat{\beta} = \frac{n_E^{\text{int}} - \text{Int}_1}{\sum_{j=1}^{n} SI(t_j)(t_j - t_{j-1})}, \quad \hat{\xi} = \frac{n_E^{\text{ext}} - 1 + \text{Int}_1}{\sum_{j=1}^{n} S(t_j)(t_j - t_{j-1})}, \tag{29}$$

and with Gamma priors $\beta \sim Ga(a_\beta, b_\beta)$ and $\xi \sim Ga(a_\xi, b_\xi)$, their posterior distributions are

$$\beta|\{e_j\} \sim Ga\left(a_\beta + (n_E^{\text{int}} - \text{Int}_1), b_\beta + \sum_{j=1}^{n} SI(t_j)(t_j - t_{j-1})\right), \tag{30}$$

$$\xi|\{e_j\} \sim Ga\left(a_\xi + (n_E^{\text{ext}} - 1 + \text{Int}_1), b_\xi + \sum_{j=1}^{n} S(t_j)(t_j - t_{j-1})\right). \tag{31}$$

When there is missingness in recovery times, the Bayesian inference procedure described in Section 4 can still be carried out, with two slight modifications. First, in the data augmentation step, when drawing missing recovery times in an interval $(u, v]$, the DARCI algorithm (Prop. 4.1) inspects $\mathcal{I}_p$ only for each $p \in \mathcal{P}^{\text{int}}$, where $\mathcal{P}^{\text{int}}$ is the group of individuals who get *internally* infected during $(u, v]$. Second, in each iteration, parameter values are drawn from the posterior distributions specified in (9) except for β and ξ, for which the posteriors are stated in (30) and (31), respectively.

S4 More Simulation Details and Results

S4.1 Complete data experiments

Data simulation procedure The steps of the simulation procedure are detailed as follows:

1. **Initialization.** Randomly select $I(0)$ individuals to be the infected/infectious (then the rest of the population are all susceptible). Set $t_{\text{cur}} = 0$.
2. **Iterative update.** While $t_{\text{cur}} < T_{\max}$, do:
   (a) **Bookkeeping.** Summarize the following statistics at t_{cur}: 1) $SI(t_{\text{cur}})$, the number of S-I links in the population; 2) $\mathbf{M}_{\max}(t_{\text{cur}})$, the possible number of links of each type defined in (20); 3) $\mathbf{M}(t_{\text{cur}})$, the number of existing links of each type defined in (21). Then set $\mathbf{M}^d(t_{\text{cur}}) = \mathbf{M}_{\max}(t_{\text{cur}}) - \mathbf{M}(t_{\text{cur}})$.

(b) **Next event time.** Compute the instantaneous rate of the occurrence of any event, $\Lambda(t_{\text{cur}}) = \beta SI(t_{\text{cur}}) + \gamma I(t_{\text{cur}}) + \tilde{\alpha}^T \mathbf{M}^d(t_{\text{cur}}) + \tilde{\omega}^T \mathbf{M}(t_{\text{cur}})$, and draw $\Delta t \sim \text{Exponential}(\Lambda(t_{\text{cur}}))$.

(c) **Next event type.** Sample $Z \sim \text{Multinomial}(\tilde{\lambda}(t_{\text{cur}}))$, where

$$\tilde{\lambda}(t_{\text{cur}}) = \left(\frac{\beta SI(t_{\text{cur}})}{\Lambda(t_{\text{cur}})}, \frac{\gamma I(t_{\text{cur}})}{\Lambda(t_{\text{cur}})}, \frac{\tilde{\alpha}^T \mathbf{M}^d(t_{\text{cur}})}{\Lambda(t_{\text{cur}})}, \frac{\tilde{\omega}^T \mathbf{M}(t_{\text{cur}})}{\Lambda(t_{\text{cur}})} \right)^T.$$

Then do one of the following based on the value of Z:

If $Z = 1$ (infection), uniformly pick one S-I link and infect the S individual in this link.

If $Z = 2$ (recovery), uniformly pick one I individual to recover.

If $Z = 3$ (link activation), randomly select $Y \in \{\text{H-H}, \text{H-I}, \text{I-I}\}$ with probabilities proportional to $\tilde{\alpha} \circ \mathbf{M}^d(t_{\text{cur}})$, and uniformly pick one de-activated "Y link" to activate.

If $Z = 4$ (link termination), randomly select $Y \in \{\text{H-H}, \text{H-I}, \text{I-I}\}$ with probabilities proportional to $\tilde{\omega} \circ \mathbf{M}(t_{\text{cur}})$, and uniformly pick one existing "Y link" to terminate.

(d) Replace t_{cur} by $t_{\text{cur}} + \Delta t$, record relevant information about the sampled event, and repeat from (a).

In Step 2 (c), "$\circ$" refers to the Hadamard product (entrywise product) for two vectors.

Supplement for "inference from complete event data" Figure S1 and S2 complement Figure 2 and 3 in the main text, showing inference results for all the parameters in the corresponding experiments.

Experiments on larger networks Figure S3 shows MLEs and 95% confidence bands for parameters with complete data generated on a network with $N = 500$ individuals. Other experimental settings are the same as those in Section 5.1. With a larger population, there tends to be more events available for inference, so the accuracy is in fact improved.

Experiments on different initial network configurations Still set population size $N = 100$, but instead of a random Erdős–Rényi graph as $\mathcal{G}_0$, the initial network is a "hubnet": one individual (the "hub") is connected to everyone else in the population while the others

form an $ER(N-1,p)$ random graph, with edge probability $p = 0.1$. Figure S4 summarizes results of Bayesian inference carried out on complete event data generated in this setting.

Supplement for "Assessing model flexibility" Estimate parameters Θ of the full model on datasets generated from 1) the decoupled temporal network epidemic process with type-independent edge rates, and 2) the static network epidemic process where the network remains unchanged. For both simpler models, fix $\beta = 0.03$ and $\gamma = 0.12$, and for the former model, let link activation rate $\alpha = 0.005$ and termination rate $\omega = 0.05$. Still, set population size $N = 100$ and let the initial network be a random Erdős–Rényi graph with edge probability $p = 0.1$.

We present, in Figure S5, the results of Bayesian inference on datasets generated from the decoupled process model. Across four different realizations, it can be observed that, the posterior samples of link activation rates $(\alpha_{SS}, \alpha_{SI}, \alpha_{II})$ concentrate around the same mean, and uncertainty is reduced with more events available for inference. Same can be said about the link termination rates, $\omega_{SS}, \omega_{SI}, \omega_{II}$. This verifies that the proposed model is indeed a generalization of the aforementioned two simpler processes, and the inference method is able to recover the truth under mild model misspecification.

S4.2 Partial observations experiments

The "DARCI" algorithm is compared with these two baseline sampling methods:

1. **Rejection sampling**: Carry out Step 1 of the inference scheme via rejection sampling. For $\ell = 1 : L$, keep proposing recovery times $\{r^*_{\ell,i}\}_{i=1:R_\ell} \overset{iid}{\sim} \text{TEXP}(\gamma^{(s-1)}, u_\ell, v_\ell)$ until the proposed $\{r^*_{\ell,i}\}_{i=1:R_\ell}$ are compatible with the observed event data in $(u_\ell, v_\ell]$. We label this method by "**Reject**".

2. **Metropolis-Hastings**: Modify Step 1 of the inference scheme into a Metropolis-Hastings step. For $\ell = 1 : L$, propose recovery times $\{r^*_{\ell,i}\}_{i=1:R_\ell} \overset{iid}{\sim} \text{TEXP}(\gamma^{(s-1)}, u_\ell, v_\ell)$, and accept them as $\{r^{(s)}_{\ell,i}\}_{i=1:R_\ell}$ with probability

$$\min\left(1, \frac{p\left(\mathbf{x}, \{r^*_{\ell,i}\}_{i=1:R_\ell}, \{r^{(s-1)}_{\ell',i}\}_{i=1:R_{\ell'},\ell'\neq\ell} | \Theta^{(s-1)}\right) \text{pTEXP}\left(\{r^{(s-1)}_{\ell,i}\}_{i=1:R_\ell}; \gamma^{(s-1)}, u_\ell, v_\ell\right)}{p\left(\mathbf{x}, \{r^{(s-1)}_{\ell,i}\}_{i=1:R_\ell,\ell=1:L} | \Theta^{(s-1)}\right) \text{pTEXP}\left(\{r^*_{\ell,i}\}_{i=1:R_\ell}; \gamma^{(s-1)}, u_\ell, v_\ell\right)}\right),$$

which equals to 1 when the proposed $\{r^*_{\ell,i}\}_{i=1:R_\ell}$ are consistent with the observed event data in $(u_\ell, v_\ell]$ and 0 otherwise. If the proposal is not accepted, then set $\{r^{(s)}_{\ell,i}\}_{i=1:R_\ell} = \{r^{(s-1)}_{\ell,i}\}_{i=1:R_\ell}$. We label this method by "**MH**".

S5 Real Data Experiments

S5.1 Data Pre-processing

All infection events and weekly health statuses of all $N = 103$ individuals are extracted from the weekly surveys. In every survey, study participants were asked if they ever felt ill at all in the past week, if they ever experienced certain symptoms, and, if there were symptoms, when the approximate illness onset time was. We take an "infection" as a positive ILI (influenza-like illness) case, which, following the protocol in [1], is defined as a cough plus at least one of the following symptoms: fever or feverishness, chills, or body aches. We further examine each ILI case and only accept one as a positive infection if the individual also indicated that they "felt ill" in the past week, thus eliminating a small number of reoccurring ILI cases for the same participants [7]. Moreover, since an individual may start exhibiting symptoms at most 3 days *after* getting infected and becoming infectious, for each infection event, we set the "real" infection time as the reported onset time minus a random "delay time" uniformly sampled between 0 and 3 days.

Social link activation and termination events are obtained from the iEpi Bluetooth contact records. Each time two study devices were paired, the iEpi application recorded the unique identifiers of the devices, a timestamp, and a received signal strength indicator (RSSI). Since Bluetooth detection can be activated whenever two devices are within a few meters of each other while the two users may not actually be in contact, we only keep those Bluetooth records with relatively strong signals (high values of RSSIs) [8]. If two consecutive Bluetooth records for one pair of devices are no more than 7.5 minutes apart in time [9], then the two

[7] One particular individual had positive ILI cases and felt ill in week 2, 3, and 5, but not in week 4. We therefore treat his/her illness as an extended one, starting in week 2 and lasting till week 5.

[8] The RSSIs range from -109 to 6, and we set the threshold as -90, so only those records with RSSIs larger than -90 are kept.

[9] We choose 7.5 minutes as a threshold instead of 5 minutes to accommodate potential lapses in Bluetooth detection.

records are considered to belong to one single continuous contact; a social link between two individuals is activated at the time of the first Bluetooth detection record in a series of consecutive records that belong to a single contact, and the link is terminated at a random time point between 1 and 6 minutes after the last Bluetooth detection of a continuous contact.

The resulting processed data contain 24 infection events in total, with 14 before the spring break week and 10 after, as well as 45,760 social link activation and termination events. The weekly disease status (healthy or ill) of every participant can be acquired from the weekly surveys, so we know, for example, if an individual recovered sometime after day 7 and before day 14, but the exact times of all recoveries are unknown.

S5.2 Maximum Likelihood Estimation

Instead of assuming the knowledge of which infection cases are internal and which are external, we directly estimate all the parameters based on the likelihood function in (25), solving (26) and (27) for the MLEs of β and ξ.

However, the real data are incomplete, with the exact times of all the recoveries unobserved. We resolve this issue using a naive imputation method—for each recovery, an event time is randomly sampled from a uniform distribution between the time of infection and the earliest time point the individual no longer felt ill (in response to the weekly surveys). Such imputation, of course, is subject to a considerable level of uncertainty, so we randomly generate 10 differently imputed datasets , obtain the MLEs from every dataset, and then report the averages over the 10 runs (see Table S1).

We can see that the MLEs acquired in this manner generally agree with the Bayesian estimates in Section 6.

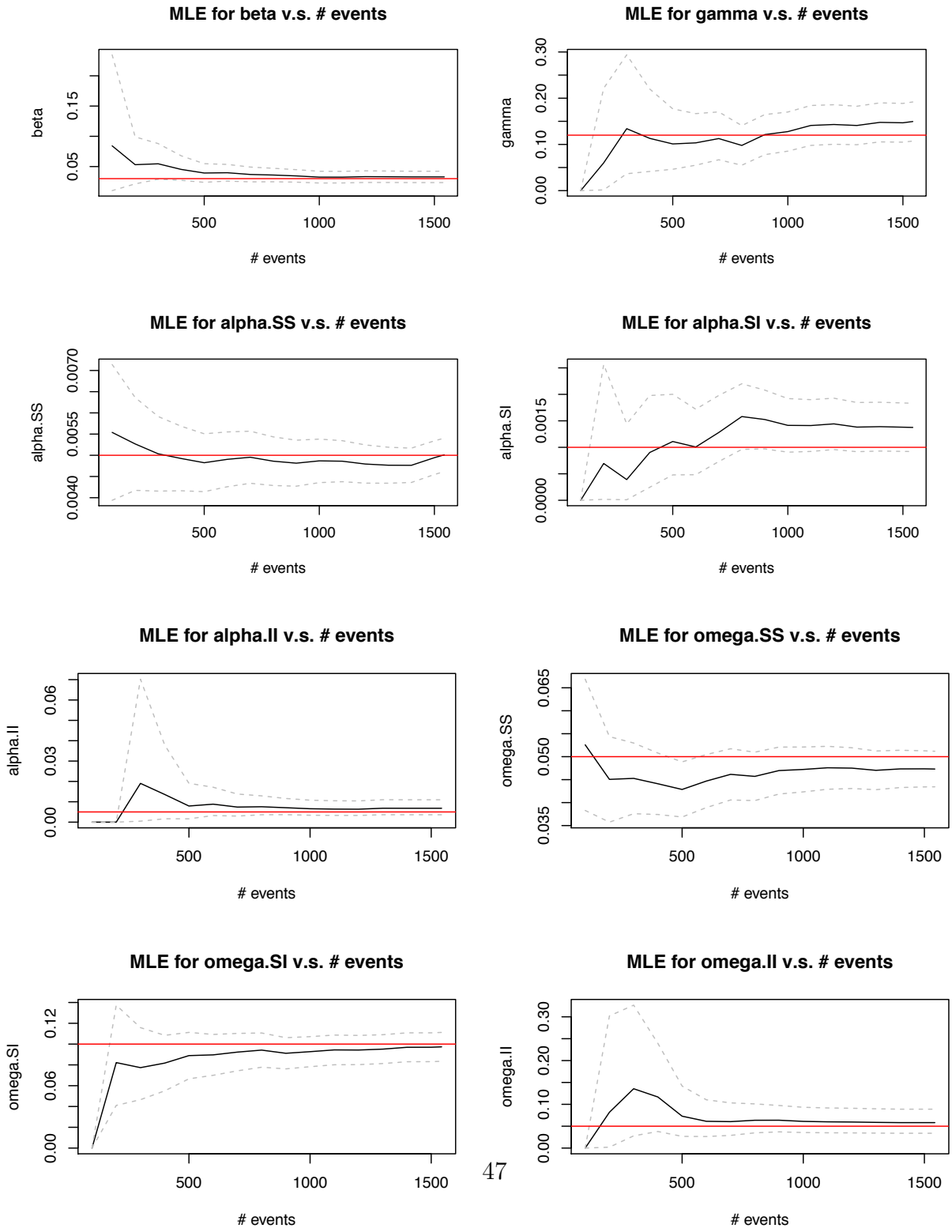

Figure S1: MLEs versus number of events used for inference. Dashed gray lines show the lower and upper bounds for 95% frequentist confidence intervals, and red lines mark the true parameter values.

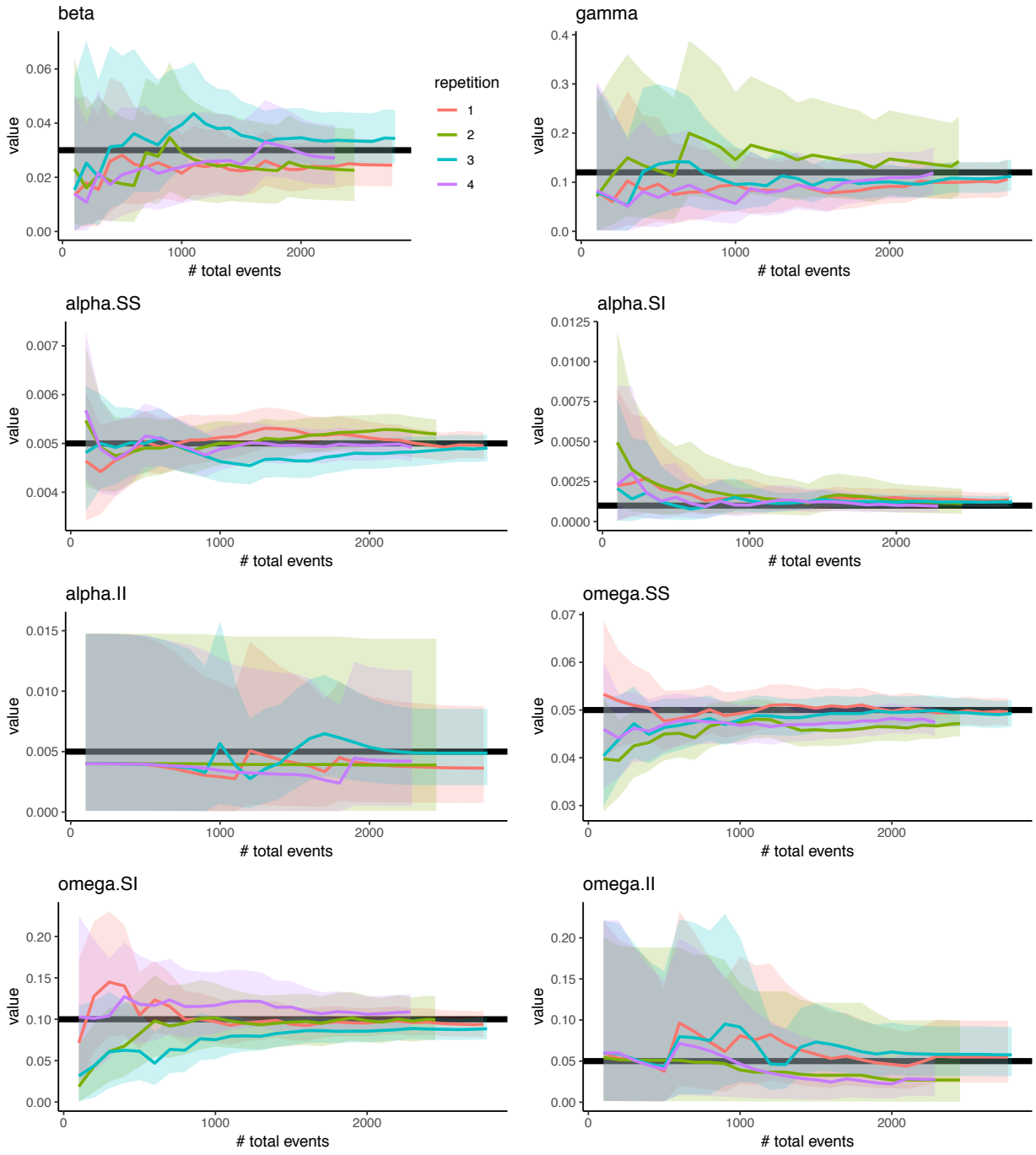

Figure S2: Posterior sample means v.s. number of total events used for inference. True parameter values are marked by **bold dark** horizontal lines, along with 95% credible bands. Results are presented for 4 different complete datasets.

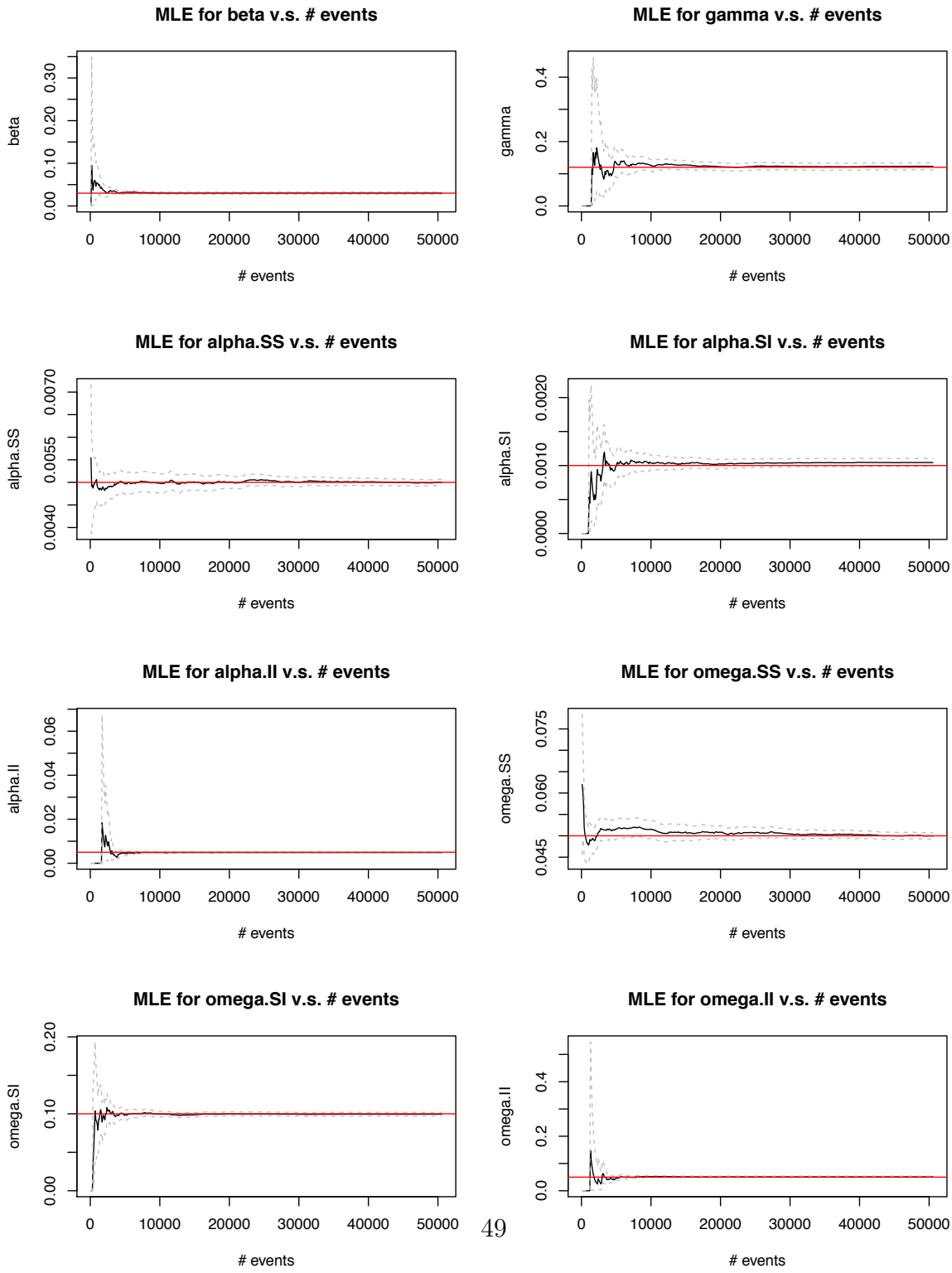

Figure S3: MLEs versus number of total events, on a larger population with $N = 500$. Dashed gray lines show the lower and upper bounds for 95% confidence intervals, and red lines mark the true parameter values. With a larger population size, there tends to be more events, which in fact facilitates estimation.

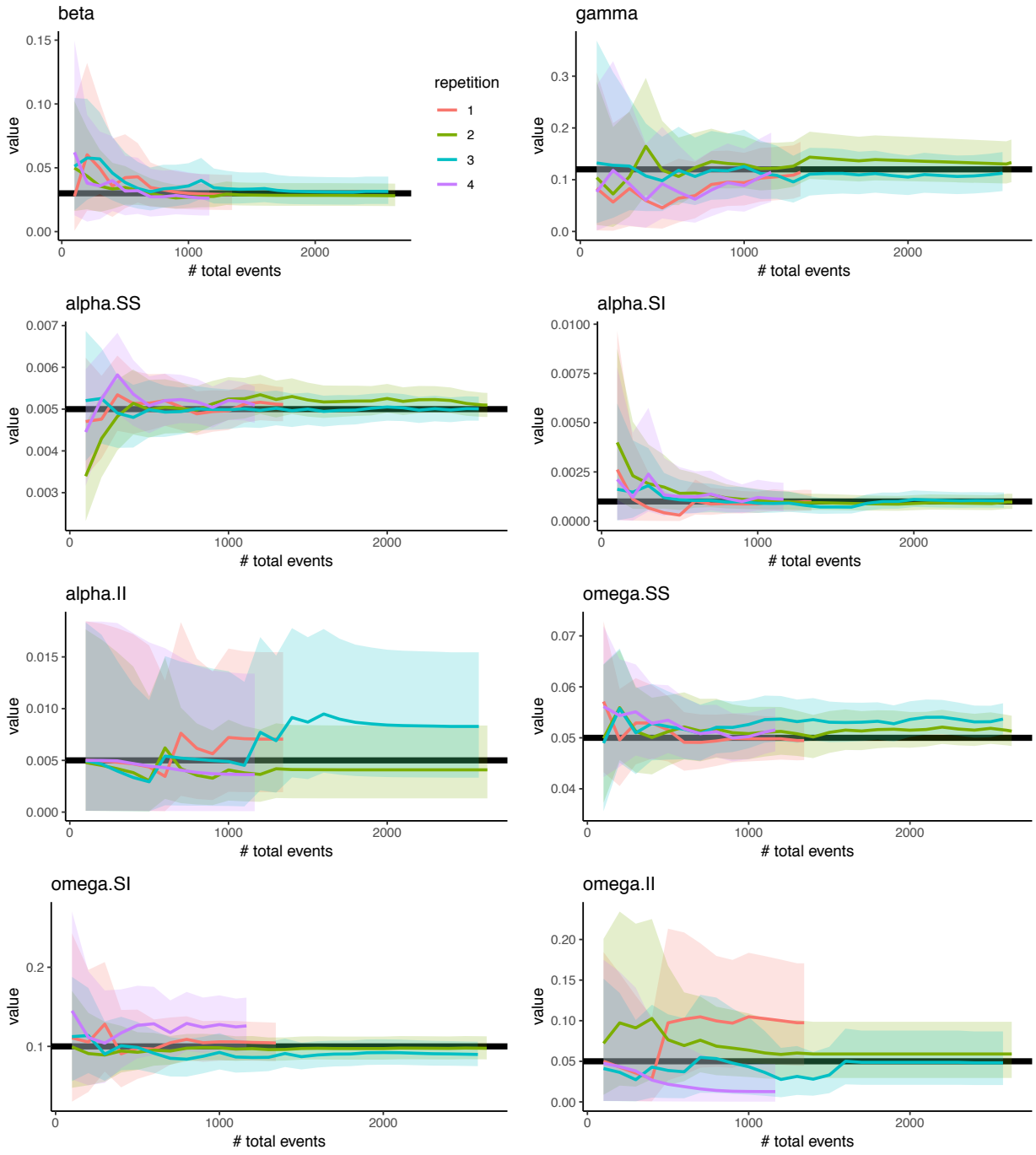

Figure S4: Posterior sample means v.s. number of total events, with $\mathcal{G}_0$ as a $N = 100$-node "hubnet". True parameter values are marked by **bold dark** horizontal lines, along with 95% credible bands. Results are presented for 4 different complete datasets.

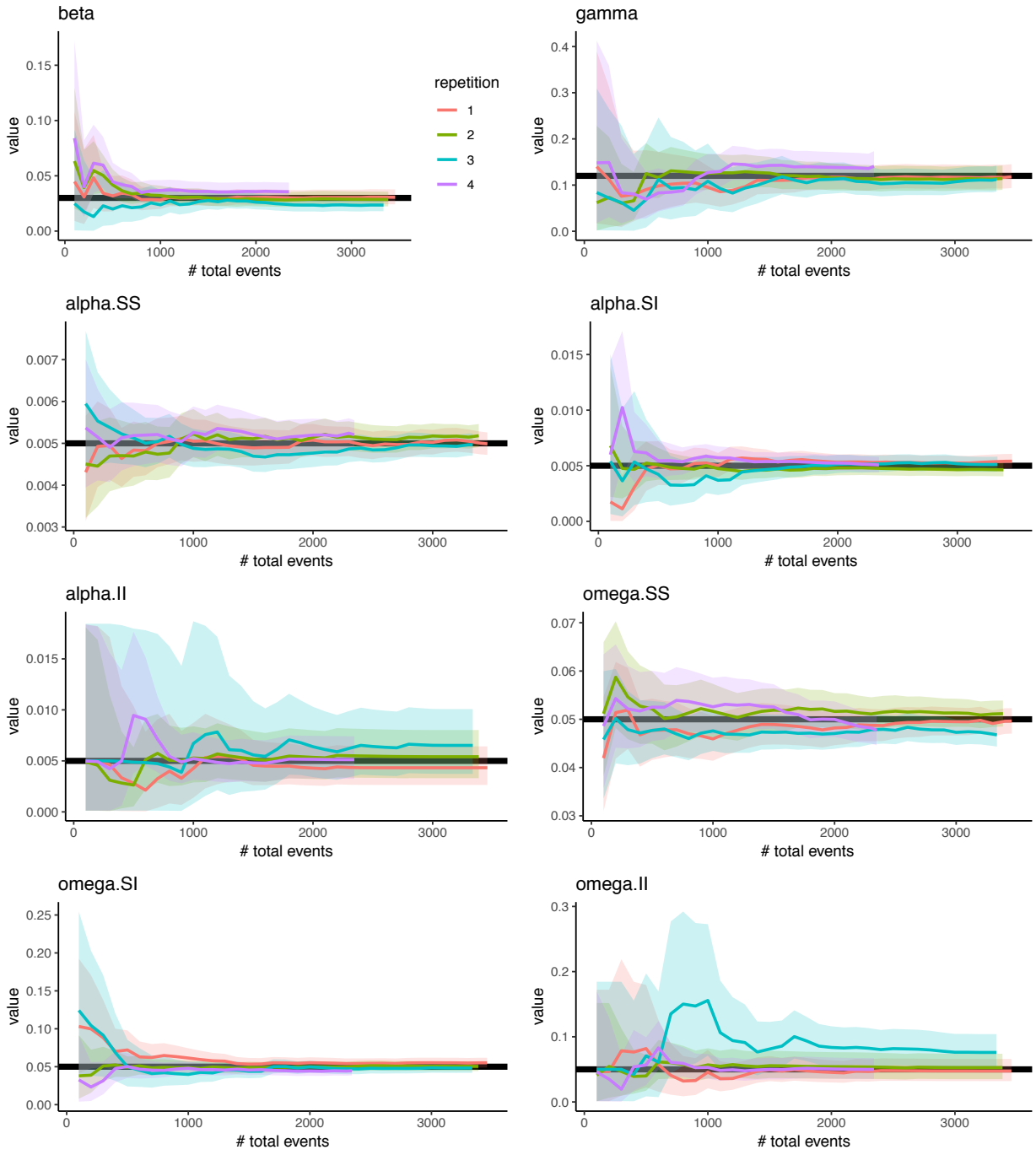

Figure S5: Posterior sample means versus number of total events, estimated using datasets generated by the decoupled process model. True parameter values are marked by **bold dark** horizontal lines, along with 95% credible bands. Results are presented for 4 different complete datasets.

Table S1: MLEs for model parameters using imputed data with all recovery times randomly sampled. The table presents average estimates as well as the standard deviations of estimates over 10 different, randomly imputed datasets. Results generally agree with those acquired using the proposed Bayesian data augmentation inference method.

Parameter	Avg. estimate	Std. deviation
β (internal infection)	0.0676	0.0092
ξ (external infection)	0.00320	1.11×10^{-6}
γ (recovery)	0.236	0.012
α_{SS} (S-S link activation)	0.0530	0.0001
ω_{SS} (S-S link termination)	42.15	0.105
α_{SI} (S-I link activation)	0.0704	0.0028
ω_{SI} (S-I link termination)	52.21	3.83