A dynamical Reed-Frost model for Covid-19 household contamination

May 23, 2020

Model description

We consider a population divided into *households*, with individuals interacting both within and outside of their respective households. We will focus on modeling the spread within the household, while taking the possibility of community-acquired infection into account.

For a given household with m individuals, consider at each time t the set S_t of susceptible individuals and the set I_t of infectious individuals. The number of such individuals will be noted s_t and i_t respectively. Given the long incubation time of Covid-19 and the possibility of presymptomatic transmission, we will also consider the set E_t of *exposed* individuals, who have yet to develop symptoms. They are not infectious in their current state (pre-infectious incubation). In accordance with recent evidence about viral shedding in Covid-19, we will divide the set of infectious individuals into pre-symptomatic and post-symptomatic individuals:

$$I_t = I_t^p \cup I_t^s.$$

The level of infectiousness of an infectious individual j will be noted $h_j(t)$. We will assume that infectiousness is constant during the presymptomatic phase $[t_p, t_s)$, then decreases with time in a geometric fashion during the symptomatic phase $[t_s, \infty)$, such that, if j is infectious,

$$h_j(t) = \begin{cases} h_0 & \text{if } t_p \leq t < t_s, \\ h_0 \gamma^{t-t_s} & \text{if } t \geq t_s. \end{cases} \tag{1}$$

Within the household, individuals are mixing completely. Hence, at time t, a susceptible individual gets exposed to all infectious individuals. The collective force of infection is then the sum of the contributions of both presymptomatic and post-symptomatic infectious individuals:

$$H_t = H_t^p + H_t^s = h_0 |I_t^p| + \sum_{j \in I_t^s} h_j(t), \tag{2}$$

and a susceptible individual becomes exposed (is infected with the virus) with probability

$$1 - Q_h(t) = 1 - \exp(-\beta H_t). \tag{3}$$

The term $Q_h(t)$ is sometimes dubbed *household escape probability*.

In addition to infections within the household, each susceptible individual can also be infected in the community. If $1 - A$ is the probability of that event, which is assumed to be independent from within-household infection, then total infection probability at time t is

$$P(t) = 1 - Q(t) = 1 - A \exp(-\beta H_t).$$

This process of infections happens at each timestep, independently for each susceptible individual, so that at time $t+1$, the number of new infections is binomially distributed with parameters s_t and $P(t)$. We will study the Markovian process

$$X_t = (s_t, e_t, i_t^p, i_t^s, H_t^s), \tag{4}$$

whose transitions can be summed up as follow:

$$\begin{aligned} e_{t+1} &= e_t + \Delta E_t - \Delta I_t^p, \\ i_{t+1}^p &= i_t^p + \Delta I_t^p - \Delta I_t^s, \\ i_{t+1}^s &= i_t^s + \Delta I_t^s, \\ H_{t+1}^s &= \gamma H_t^s + h_0 \Delta I_t^s, \\ s_{t+1} &= s_t - \Delta E_t, \end{aligned}$$

where

$$\begin{aligned} \Delta E_t &\sim \mathsf{Bin}(s_t, P(t)), \\ \Delta I_t^p &\sim \mathsf{Bin}(e_t, p_I), \\ \Delta I_t^s &\sim \mathsf{Bin}(i_t^p, p_S) \end{aligned}$$

are the number of newly exposed, newly presymptomatic and symptomatic individuals, respectively.

Observation model

We assume that the data contain the number s_0 of individuals in each household, along with symptom onset times of symptomatic individuals. In our context, we will assume that symptomatic infectious individuals are detected with probability F (thus aggregating subclinical or entirely asymptomatic disease and biases due to self-reporting) and we will write

$$Y_t \sim \mathsf{Bin}(\Delta I_t^s, F)$$

for the observed number of newly-infected, symptomatic individuals.

This, and the Markov property of the hidden process X makes inference through classical HMM algorithms theoretically possible, such as particle MCMC [2], or iterated filtering [4]. As a sanity check, it should also be possible to fit the classical Reed-Frost model to the final size data (s_0, i_∞) as in [1] to estimate some of the same parameters as in this model.

Parameters

- Rate of decrease of infectiousness $\gamma > 0$.

- Initial infectiousness $h_0 > 0$.
- Transmission parameter $\beta > 0$.
- Community escape probability A.
- Rate of asymptomatic infection F.
- Probability of an exposed individual becoming infectious per time unit p_I.

Possible extensions

There are a number of important aspects of Covid-19 infection that we would like to address with these data. This requires refining the model to take individual covariates into account, such as age (which should be modelled as a categorical variable), severity of disease or the frequency of outside contacts.

For now, we assume that infectious individuals have the same transmission characteristics, but this could very well not be the case. The initial infectiousness h_0 could be drawn from a common distribution, such as a Gamma distribution, which allows for varying levels of heterogeneity (see [3]). Also, we did not include the age structure of households, which will have to be done at some point since transmissibility between age groups is a crucial knowledge gap at this time.

The impact of asymptomatic infections should also be modelled carefully, since it is not known at this time whether they can be infectious or not. Following [3], we could distinguish between uninfectious and infectious asymptomatic disease.

A very important factor is the presence of non-Covid-19 infections in the community, which can lead to symptoms and outbreaks similar to Covid-19. This represents a major bias in the data. This can be resolved in several ways: the simplest one is to independently estimate the proportion of influenza-like illnesses who are in fact due to Covid-19 disease (for instance, by using the rate of positive PCR tests as a proxy), and adjust the parameters accordingly. A more sophisticated approach would be to finely distinguish between symptoms, according to their predictive value for Covid-19.

References

[1] Simon Cauchemez, Neil M. Ferguson, Annette Fox, Le Quynh Mai, Le Thi Thanh, Pham Quang Thai, Dang Dinh Thoang, Tran Nhu Duong, Le Nguyen Minh Hoa, Nguyen Tran Hien, and Peter Horby. Determinants of Influenza Transmission in South East Asia: Insights from a Household Cohort Study in Vietnam. *PLoS Pathogens*, 10(8):2–9, 2014.

[2] Akira Endo, Edwin van Leeuwen, and Marc Baguelin. Introduction to particle Markov-chain Monte Carlo for disease dynamics modellers. *Epidemics*, 29(February):100363, 2019.

[3] Christophe Fraser, Derek A.T. Cummings, Don Klinkenberg, Donald S. Burke, and Neil M. Ferguson. Influenza transmission in households during the 1918 pandemic. *American Journal of Epidemiology*, 174(5):505–514, 2011.

[4] Edward L. Ionides, Dao Nguyen, Yves Atchadé, Stilian Stoev, and Aaron A. King. Inference for dynamic and latent variable models via iterated, perturbed Bayes maps. *Proceedings of the National Academy of Sciences of the United States of America*, 112(3):719–24, 2015.