Heterogeneous transmission mixture model protocol

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1 Background

Heterogeneity in disease transmission, where a a small proportion of hosts cause a large proportion of transmission events, arises frequently in epidemics. Individuals can vary in their ability to transmit infectious agents through a variety of mechanisms. For example, infectiousness can differ between individuals, due to variation in pathogen load or shedding rates, or differences in immune response. Variation in outbreak cluster sizes could reflect heterogeneity in contact patterns. For example, in respiratory infections such as SARS-CoV-2, influenza and tuberculosis, household infection clusters tend to arise from direct contact with an infected case within a household. However a small proportion of clusters in a given dataset may be large. These clusters could arise from aerosol exposure, perhaps when the index case is highly infectious and has high viremia, or if transmission to susceptible individuals occurs in a densely populated location, e.g., on poorly ventilated public transport or in shared accommodation.

To model transmission heterogeneity, we consider a variation of the 80:20 rule (Woolhouse et al. 1997), where a small proportion of hosts have high transmission potential, and the remainder of the population have low transmission potential. Individual transmission potential is characterized by individual R_0, the expected number of secondary infections caused by an individual over the course of their infectious period (Lloyd Smith et al. 2005). The small percentage of hosts that have high transmission potential are assumed to contribute substantially to community transmission (i.e., cases arising from unknown sources). For example, these individuals have high numbers of contacts per day including and outside their own household, or they may have high viremia and are therefore highly infectious. Aerosolized transmission of respiratory pathogens is another mechanism for high contribution of a particular individual to community transmission. On the other hand, the majority of hosts in a population do not contribute substantially to community transmission. For example, they are self-isolating and therefore limit their contacts with susceptible individuals, or they have low pathogen load, or in the case of a respiratory infection, they only transmit to close contacts via droplet transmission.

Branching process models are appropriate for modeling transmission at the beginning of an epidemic, when depletion of susceptibles can be ignored, or for spread of rare pathogens, e.g., zoonoses that spillover to humans, producing transmission chains that quickly go extinct (Lloyd Smith et al. 2005, Yan 2008, Nishiura et al. 2012, Blumberg and Lloyd Smith 2013). In heterogeneous settings with small population level R_0, what does the cluster size distribution look like in comparison to that obtained from homogeneous settings, where there is no variation in contact patterns or infectiousness? To address this question, we compare the outputs of a Poisson mixture branching process with a baseline Poisson branching process. A Poisson mixture offspring distribution is a simple and flexible model that can capture both positively skewed and bimodal distributions of the number of secondary infections generated by an infectious individual (Figure 1). Like other commonly used offspring distributions for infectious disease transmission, such as the geometric distribution and the negative binomial distribution, its variance-to-mean ratio is greater than one, but in contrast to these distributions, it has the additional feature of capturing both unimodal and bimodal differences in individual transmission potential. For example, bimodality in transmission potential may be present in core-periphery contact networks found in health care settings, where health-care workers have many contacts and therefore high transmission potential when infected, but patients have low numbers of contacts. Finite Poisson mixture distributions are commonly used in actuarial science to model the number of insurance claims filed by an individual, but to our knowledge, they have not been used to model the sizes of infectious disease outbreaks.

The heterogeneity in transmission potential outlined above could result in two epidemiological outcomes. Epidemics could be more "bursty" or grow more explosively than in the homogeneous setting due to the contribution of community transmission. On the other hand, the contribution of direct contact transmission may override that of community transmission, leading to slower epidemics than those obtained from a homogeneous transmission process with the same R_0.

1.1 Research questions

1. How does the number of secondary infections per index case change as the proportion of community transmission increases? Do we observe "burstiness" in the number of cases and in their summary statistics?
2. How does the number of secondary infections per index case change as community transmission R_0 increases? Do we observe "burstiness" in the number of cases and in their summary statistics? How does it change relative to homogeneous transmission with the same R_0?
3. How does heterogeneity in transmission potential affect the cluster size distribution in the early stages of an epidemic? Do clusters look "bursty" relative to clusters generated from a homogeneous transmission process? (i.e. how does changing R_0^A affect the cluster size distribution?)

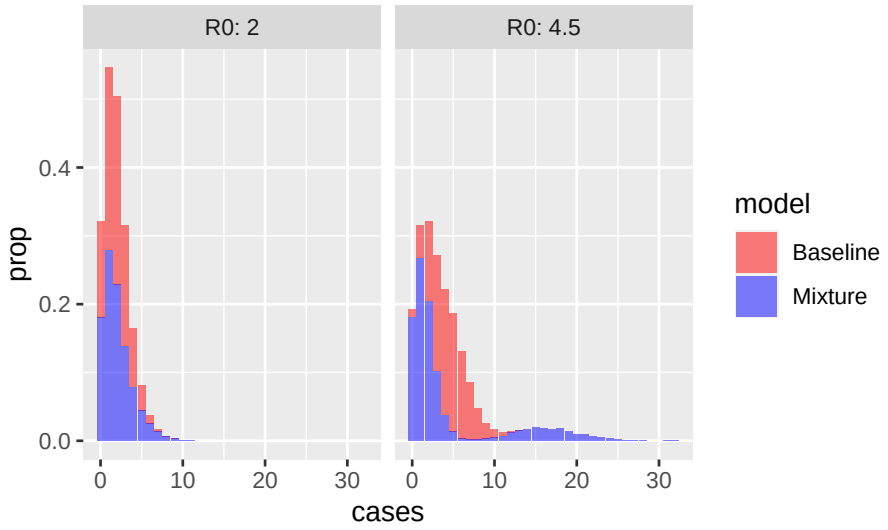

Figure 1: The case distribution generated by a Poisson mixture can be unimodal or bimodal. The Poisson mixture offspring distribution with $p = 0.8$, $R_0^D = 1.5$ and $R_0^A = 4$ (mean $R_0 = 2$) is unimodal, but the mixture offspring distribution with $p = 0.8$, $R_0^D = 1.5$ and $R_0^A = 16.5$ (mean $R_0 = 4.5$) is bimodal. The case distribution generated by the respective baseline Poisson offspring distributions are unimodal. To generate the barplots, we used 10,000 realizations of each offspring distribution.

To motivate examples, we should show an epidemic COVID 19 case studies

4. How does the chain size distribution conditioned on extinction change as the proportion of community transmission increases? Do we observe "burstiness" in cluster sizes?

5. Given heterogeneities in transmission potential, how do interventions affect statistics such as the probability of extinction, probability of a major epidemic, and mean and variance of the cluster sizes conditioned on extinction?

To answer the above questions, we derive key summary statistics of the two branching processes, and the cluster size distributions conditional on extinction arising from them. We will verify the theoretical results by simulation. We also examine the implications for control.

Individual reproduction number $\mathcal{V}$	Probability $P(\mathcal{V} = \nu)$
R_0^D	p
R_0^A	$1-p$

Table 1: Probability distribution of the discrete random variable $\mathcal{V}$ (individual R_0). The population mean is $R_0 = pR_0^D + (1-p)R_0^A$.

2 Study design

2.1 Offspring distribution model

We assume the number of secondary cases caused by an infectious individual is a Poisson random variable with rate R_0, but R_0 varies among infected individuals and is therefore a random variable. Infected individuals can either contribute substantially to community transmission or individuals do not contribute substantially to community transmission and transmission to others occurs via close contact only.

We use a Poisson mixture model to model a combination of close contact transmission and community transmission. We assume p% of cases occur through close contact, and $(1-p)$% of cases occur via community transmission. Community transmission events and close contact events are mutually exclusive in the model. The number of secondary infections per individual is conditional on whether the individual transmits to close contacts only (with rate R_0^D) or to the wider community (with rate R_0^A). Thus R_0 is a random variable with probabilities $P(R_0 = R_0^D) = p$ and $P(R_0 = R_0^A) = 1-p$. Assuming the number of secondary infections per individual X is a Poisson random variable with rate R_0, whose distribution is given in Table 1, the number of cases X is therefore a finite mixture of two Poisson distributions. For example, if 5 cases arise from a single infectious case in the next generation, the probability that the number of cases is five arising from that index case is

$$\begin{aligned} P(5 \text{ cases}) &= P(5 \text{ cases due to close contact}) \text{ or } P(5 \text{ cases due to community}) \\ &= P(\text{close contact})P(5 \text{ cases conditional on close contact}) \\ &\text{or } P(\text{community})P(5 \text{ cases conditional on community}), \end{aligned}$$

or expressed mathematically as,

$$P(X = 5) = pP(X = 5|\text{close contact}) + (1-p)P(X = 5|\text{community}). \quad (1)$$

Given that an infected individual makes few contacts leading to successful transmission, the number of secondary infections generated by that individual follows a Poisson distribution with mean R_0^D. Given that an infected individual makes many contacts or is highly infectious, the number of secondary infections generated by that individual follows a Poisson distribution with mean R_0^A. We assume $R_0^D < R_0^A$, for example, aerosol transmission may give rise to many

more cases than droplet transmission. The offspring distribution of the number of cases X is a finite Poisson mixture,

$$X \sim p\,\text{Poisson}(R_0^D) + (1-p)\text{Poisson}(R_0^A), \quad 0 \le p \le 1. \tag{2}$$

The mean number of cases is $R_0 = pR_0^D + (1-p)R_0^A$.

We compare the mixture (2) with a Poisson offspring distribution with mean R_0 that models the number of cases caused by a single infectious individual assuming no heterogeneity in contact patterns or infectiousness,

$$X \sim \text{Poisson}(R_0). \tag{3}$$

Both equations (2) and (3) represent Galton-Watson branching processes since individuals infect a mean number R_0 of individuals at the end of their infectious period, and the infectious period (generation time) is constant. Note that neither model is appropriate for capturing heterogeneity in infection duration among individuals.

The probability mass function that describes the heterogeneous contact Poisson mixture branching process is given by (Johnson and Hoboken, 2005)

$$P(X=k) = p\frac{(R_0^D)^k}{k!}e^{-R_0^D} + (1-p)\frac{(R_0^A)^k}{k!}e^{-R_0^A}. \tag{4}$$

For example, the probability that an index case does not transmit the disease, which may be obtained from transmission trees (Lloyd Smith et al. 2005), is $P(X=0) = pe^{-R_0^D} + (1-p)e^{-R_0^A}$. Note that if R_0^A gets increasingly large (and consequently R_0 increases), but R_0^D and p do not change, the probability that an index cases does not produce secondary infections approaches $pe^{-R_0^D}$. Increasing the mean number of secondary infections caused by community transmission, which increases R_0, causes the probability of non-transmission by an index case to be increasingly dominated by the $p\%$ of direct contact non-transmission events.

The probability generating function of the heterogeneous mixture branching process is the weighted sum of Poisson generating functions (Johnson and Hoboken, 2005)

$$\begin{aligned} G(z) = \sum_{k=0}^{\infty} P(X=k)z^k &= p\sum_{k=0}^{\infty}\frac{(R_0^D)^k}{k!}e^{-R_0^D}z^k + (1-p)\sum_{k=0}^{\infty}\frac{(R_0^A)^k}{k!}e^{-R_0^A}z^k \\ &= pe^{R_0^D(z-1)} + (1-p)e^{R_0^A(z-1)} \end{aligned} \tag{5}$$

It is easy to check that equation (5) satisfies all the requirements of a generating function. The probability of the branching process going extinct (probability of a minor outbreak) is given by numerically finding the root z_∞ of the equation

$$z = G(z) = pe^{R_0^D(z-1)} + (1-p)e^{R_0^A(z-1)}. \tag{6}$$

If $R_0 < 1$, then $z_\infty = 1$, i.e., the probability of a minor outbreak going extinct is certain. If $R_0 > 1$, then $0 < z_\infty < 1$. The probability of a major outbreak is

$1 - z_\infty$. In the limit of $R_0^A \to \infty$, equation (6) approaches $z = pe^{R_0^D(z-1)}$, and therefore the extinction probability saturates to the solution of this equation as R_0^A increases (Figure 2).

2.2 Summary statistics of the heterogeneous mixture offspring distribution

To obtain succinct expressions for the summary statistics, we write

$$R_0^A = R_0^D + \delta, \quad \delta > 0. \tag{7}$$

Then the expression for the mean of the offspring mixture becomes

$$R_0 = R_0^D + (1-p)\delta, \tag{8}$$

which lies between R_0^D and R_0^A if $0 < p < 1$. Here we will study the effect of two ways of increasing R_0:

a) increasing the proportion of community transmission $1 - p$;

(b) increasing the mean number of secondary infections due to community transmission (i.e., increasing δ in equation (7)).

By increasing $1-p$, R_0 can be at most equal to R_0^A, but there is no upper bound on R_0 if δ is increased.

Using the generating function or taking expectations yields the variance in the number of secondary cases generated by each case in the next generation,

$$V(X) = R_0^D + (1-p)\delta + p(1-p)\delta^2. \tag{9}$$

Clearly, the variance is greater than the mean R_0 provided $0 < p < 1$. Increasing the mean number of secondary infections due to community transmission will increase the variance, but the variance is a non-monotonic function of community transmission. The second derivative test yields $V_{pp}(X) = -2\delta^2 < 0$ for $\delta > 0$ and so the variance has a local maximum. The variance has a local maximum at

$$p = \frac{1}{2}\left(1 - \frac{1}{\delta}\right), \tag{10}$$

which is positive if $\delta > 1$ and approaches $1/2$ as $\delta \to \infty$ (i.e., as R_0^A gets increasingly large.) If $0 < \delta < 1$, then the local maximum occurs for negative p and therefore the variance is a decreasing function of p (i.e., an increasing function of the proportion of aerosol transmission). If $\delta > 1$ then the variance is non-monotonic on the interval $0 < p < 1$. The variance is a concave-up function of δ, with local minimum at $\delta = -1/2p$, and therefore the variance is a increasing function of δ on the interval $0 \le p \le 1$. Therefore the variance is an increasing function of R_0^A. The variance-to-mean ratio is $1+p(1-p)\delta^2/R_0$, which is greater than one in the interval $0 < p < 1$ and thus the mixture offspring distribution

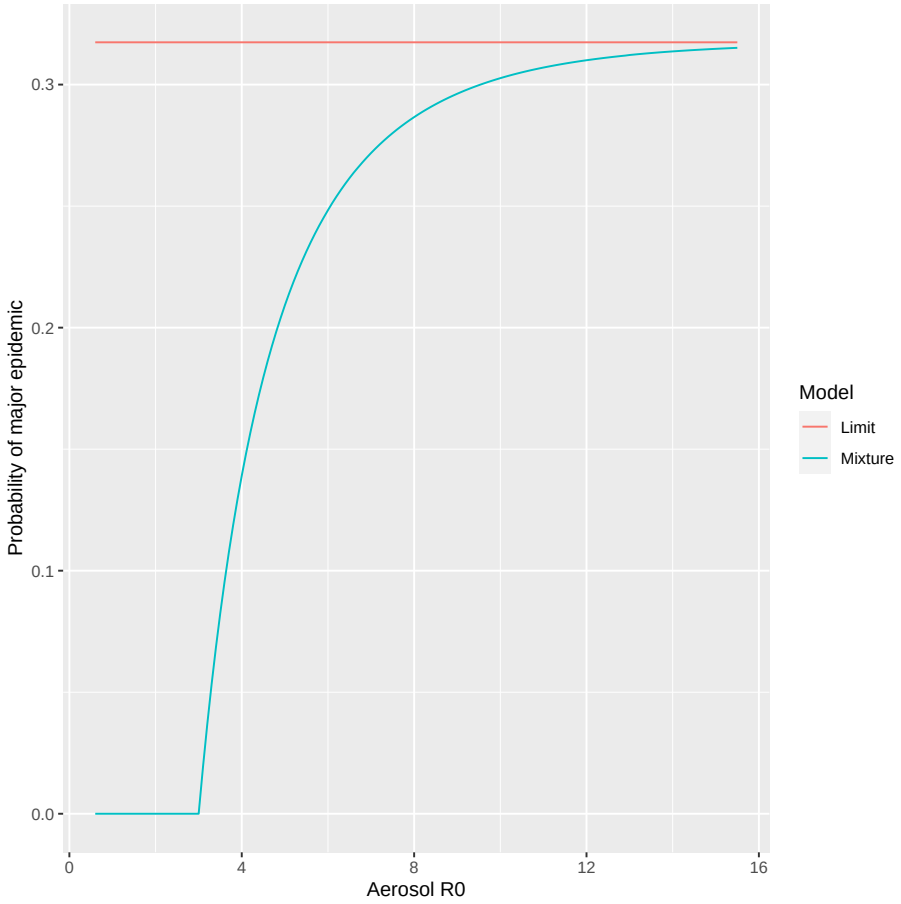

Figure 2: The probability of a major epidemic is an increasing function of R_0^A provided $R_0 > 1$. As R_0^A increases, the probability of a major epidemic increases, and saturates to the solution of $z = pe^{R_0^D(z-1)}$ (horizontal line). Here $p = 0.8$, $R_0^D = 0.5$ and the limiting solution is $z_\infty = 0.6826$. At $R_0^A = 3$, $R_0 = 1$, and for values of $R_0^A < 3$, all outbreaks go extinct.

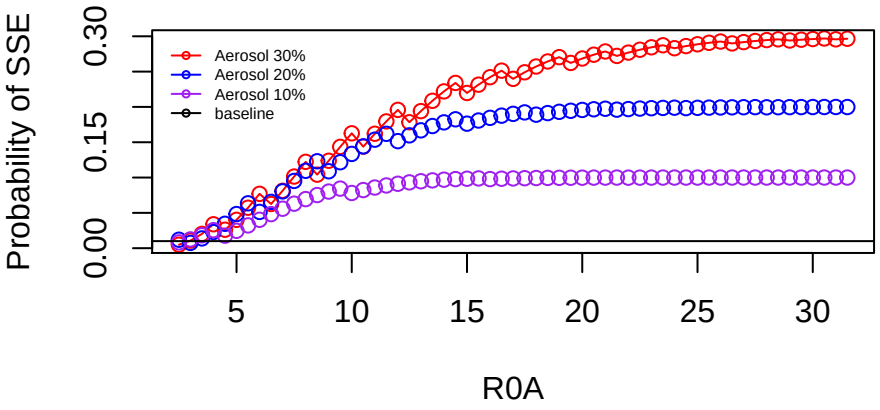

Figure 3: The probability of a superspreading event approaches $1-p$ as R_0^A increases.

is overdispersed, in contrast to the Poisson offspring distribution. The skewness of the offspring distribution is

$$S(X) = \frac{R_0^D + (1-p)\delta + 3p(1-p)\delta^2 + p(1-p)(2p-1)\delta^3}{V(X)^{3/2}} \tag{11}$$

Lloyd Smith et al. 2005 defined a superspreading event as any case that infects more than ζ_n others, where ζ_n is the n^{th} percentile of the Poisson(R_0) distribution. For example, a 99th percentile superspreading event is defined as any case that causes more secondary infections than would arise in 99% of offsprings taken from a homogeneous population with the same R_0. The mixture offspring distribution can be used to find the probability of the number of cases exceeding the n^{th} percentile of the Poisson offspring distribution,

$$\begin{aligned}\psi_n = P(X > \zeta_n) &= 1 - P(X \leq \zeta_n) \\ &= 1 - \left(p \sum_{k=0}^{\zeta_n} \frac{(R_0^D)^k}{k!} e^{-R_0^D} + (1-p) \sum_{k=0}^{\zeta_n} \frac{(R_0^A)^k}{k!} e^{-R_0^A} \right). \end{aligned} \tag{12}$$

Holding R_0^D and p fixed, recall that as $R_0^A \to \infty$, $R_0 \to \infty$ and therefore $\zeta_n \to \infty$. Consequently the probability of a superspreading event ψ_n approaches $1-p$ (Figure 3).

2.3 Summary statistics for the chain size distribution conditioned on extinction

We define a chain that goes extinct at time t by

$$Y = \sum_{i=0}^{t-1} x_i$$

with x_i denoting the cumulative number of offspring in the i^{th} generation, and $x_0 = 1$. The final size Y upon extinction is a random variable with probability distribution $P(Y = y)$, $y = 1, 2, ...$ We need to distinguish between when $R_0 < 1$ and $R_0 > 1$. From Yan (2008) we note that

$$\sum_{y=1}^{\infty} P(Y = y) = z_\infty,$$

$$z_\infty = \begin{cases} = 1 \text{ if } R_0 \leq 1 \\ < 1 \text{ if } R_0 > 1 \end{cases}$$

I don't follow this. Also, is this the same z_∞ defined around eqn. 6?

This means that the chain size distribution conditional on extinction sums up to the probability of a minor outbreak if $R_0 > 1$, which means that the probability of larger clusters are determined by the distribution that sums to the probability of a major outbreak, $1 - z_\infty$. Following Yan (2008) let $Q_Y(z) = \sum_{y=1}^{\infty} z^y P(Y = y)$. Finding the first and second derivatives of this function allows us to compute the mean and variance of the final size conditioned on extinction (chain size conditioned on a minor outbreak).

Using $Q_Y(z) = zG(Q_Y(z))$ (Mode and Sleeman 2000), the first and second derivatives of $Q_Y(z)$ are (Yan 2008)

$$Q'_Y(z) = G(Q_Y(z)) + zG'(Q_Y(z))Q'_Y(z), \tag{13}$$

$$Q''_Y(z) = 2G'(Q_Y(z))Q'_Y(z) + z(G''(Q_Y(z))(Q'_Y(z))^2 + G'(Q_Y(z))Q''_Y(z)). \tag{14}$$

If $R_0 < 1$, then

$$E(Y) = Q'_Y(1) = \frac{1}{1 - R_0}, \tag{15}$$

which is valid for any distribution of X. To compute the variance assuming $R_0 < 1$, we use $V(Y) = Q''_Y(1) + Q'_Y(1) - (Q'_Y(1))^2$,

$$V(Y) = \frac{V(X)}{(1 - R_0)^3} \tag{16}$$

If $R_0 > 1$, then $0 < z_\infty < 1$ and we compute

$$R_0^* = G'(z_\infty) < 1, \tag{17}$$

i.e., the basic reproduction number conditioned on extinction of the chain. Then we can calculate the mean and variance of the chain size conditioned on a minor outbreak occurring (Yan 2008),

$$E(Y|\text{minor outbreak}) = \frac{1}{1-R_0^*} \tag{18}$$

$$V(Y|\text{minor outbreak}) = \frac{z_\infty Q_Y''(z_\infty) + R_0^*(1-R_0^*)}{(1-R_0^*)^3} \tag{19}$$

did I miss a definition?

As R_0 increases, the probability of a minor outbreak z_∞ declines. Therefore R_0^* declines with R_0, and so the mean chain size will also decline (Figure 4). For example, for the baseline Poisson model, $R_0^* = G'(z_\infty) = R_0 e^{(R_0(z_\infty - 1))}$, which decreases as z_∞ decreases.

If $R_0^A \to \infty$, then z_∞ approaches the limit of the solution to $z = pe^{R_0^D(z-1)}$ (e.g., Figure 2) and therefore R_0^* saturates to a limiting value. Therefore the mean and variance of the chain size distribution conditioned on extinction will also saturate as R_0^A and R_0 become increasingly large (Figure 4).

2.4 The chain size distribution conditioned on extinction

We will compare the chain size distribution for the Poisson mixture with the chain size distribution obtained from a Poisson offspring distribution. Chain sizes arising from a Poisson branching process follow the Borel-Tanner distribution (Mode and Sleeman 2000) conditioned on a minor outbreak (Yan 2008),

$$P(Y = y) = \frac{1}{z_\infty} \frac{e^{-R_0 y}(R_0 y)^{y-1}}{y!} \tag{20}$$

where z_∞ is the probability of extinction of a Poisson branching process, which is not equal to one when $R_0 > 1$.

To derive the chain size distribution for the Poisson mixture, we use the result from Blumberg and Lloyd-Smith (2014) and therefore require the derivatives of powers of the generating function (5). Let

$$T_y(z) = \frac{1}{y}(Q_Y(z))^y, \quad y = 1, 2, \ldots$$

Then the probability of a cluster having size y is

$$P(Y = y) = \frac{1}{(y-1)!} T_y^{(y-1)}\Big|_{z=0} \tag{21}$$

To evaluate the derivatives of

$$(Q_Y(z))^y = (pe^{R_0^D(z-1)} + (1-p)e^{R_0^A(z-1)})^y, \tag{22}$$

i.e., the yth power of the generating function (5), we need to apply the chain rule $y-1$ times. The kth derivative of the inner function of equation (22) evaluated at $z = 0$ is

$$g_k = p(R_0^D)^k e^{-R_0^D} + (1-p)(R_0^A)^k e^{-R_0^A}, \quad k = 1, 2, \ldots, y-1. \tag{23}$$

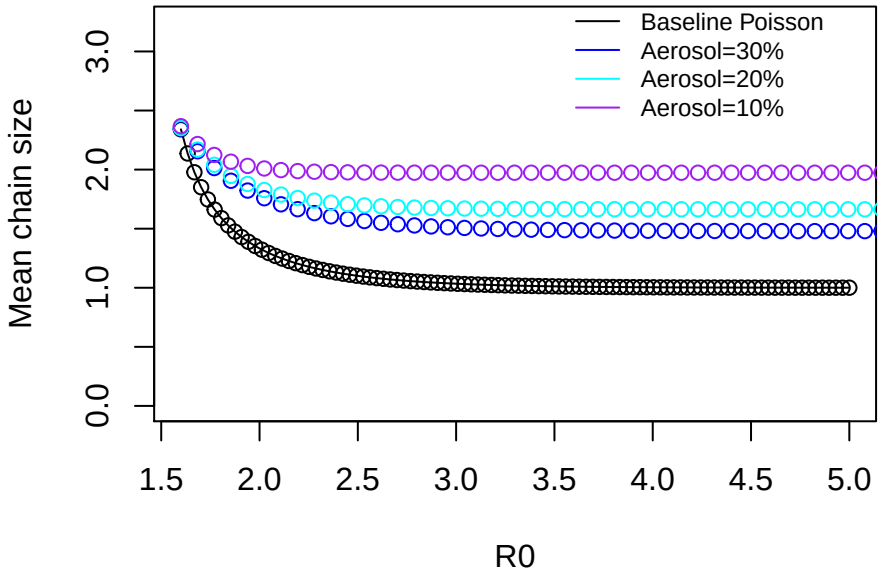

Figure 4: The mean chain size decreases with increasing R_0 (caused by increasing R_0^A) because the probability of a major epidemic increases with R_0 (less likely to have a transmission chain that does not result in a major epidemic). For each value of $1-p$, the mean cluster size approaches a limit because R_0^* saturates with increasing R_0^A.

The kth derivative of the outer function of equation (22) evaluated at $z = 0$ is

$$f_k = \frac{y!}{(y-k)!}\left(pe^{-R_0^D} + (1-p)e^{-R_0^A}\right)^{y-k}, \quad k = 1, 2, \ldots, y-1. \tag{24}$$

The generalized chain rule or Faa di Bruno's formula (citation) yields

$$T_y^{(y-1)}\Big|_{z=0} = \sum_{k=1}^{y-1} f_k B_{y,k}(g_1, g_2, \ldots, g_{y-1-k}) \tag{25}$$

where $B_{y,k}$ are Bell polynomials of the g_k. See Figure 5 for an example of the chain size distribution conditioned on extinction.

2.5 Simulation of cluster sizes

To investigate if Poisson mixture epidemics that do not go extinct within a few generations will grow more slowly or more explosively relative to Poisson epidemics, we will examine the distribution of the first generation to have 50 cases (following Lloyd Smith et al. 2005).

- Fix $R_0^D = 1.5$ and $R_0^A = 5$. Vary proportion of aerosol transmission $1 - p$ between 10% and 50%. Study its effects on the time it takes to generate outbreaks greater than 50 cases by doing 1000 simulations of each branching process, conditional on non-extinction, and computing the first generation it takes to have a cluster greater than 50 cases. Compare with obtained from Poisson offspring process with equivalent R_0.
- Fix $R_0^D = 1.5$ and $p = 0.8$. Vary R_0 between 1.6 and 4 by increasing R_0^A. Study its effects on the time it takes to generate outbreaks greater than 50 cases by doing 1000 simulations of each branching process, conditional on non-extinction, and computing the first generation it takes to have a cluster greater than 50 cases. Compare with obtained from Poisson offspring process with equivalent R_0. See Figure 6 for preliminary results.

2.6 Control scenarios

Interventions could affect the reproduction number by reducing either of R_0^D or R_0^A, or both simultaneously. For example, reduction of R_0^A may imply that a new HVAC system has been installed, or widespread usage of face coverings could reduce R_0^D. We will compare three interventions: reduce R_0 by a factor q (i.e., reducing both R_0^D and R_0^A), reduce R_0^A by a factor q or reduce R_0^D by a factor q).

TO DO: FLESH OUT. ARE THESE THE INTERVENTIONS WE WISH TO FOCUS ON?

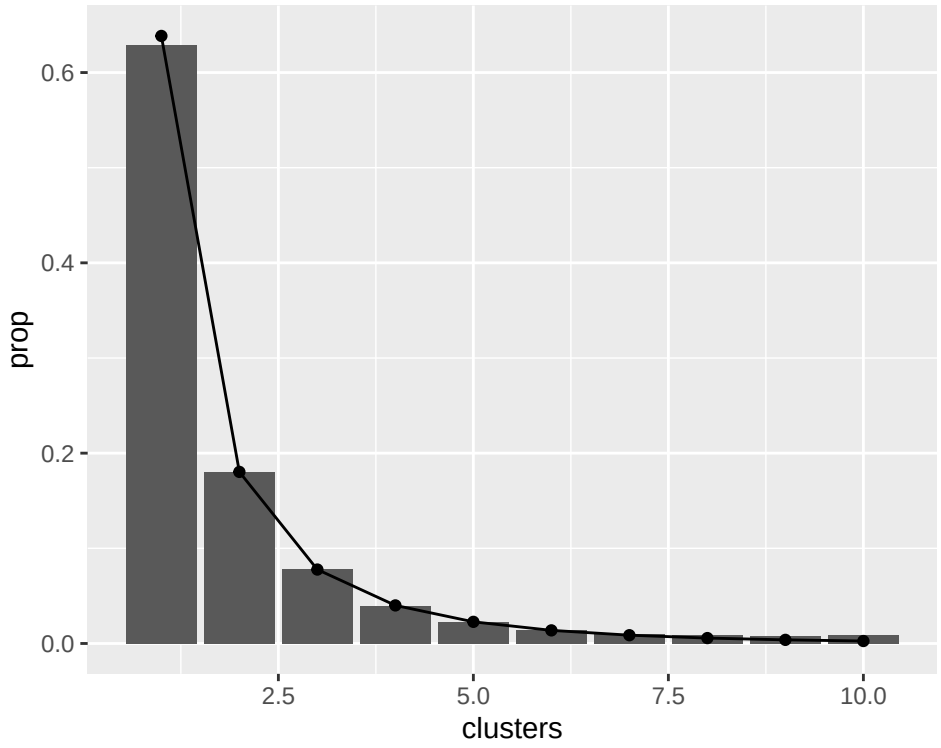

Figure 5: Cluster size distribution obtained from simulation (gray bars) compared to the the theoretical prediction (points). We generated 100000 simulations of a mixture branching process with $p = 0.8$, $R_0^D = 1.5$ and $R_0^A = 4$, retaining those that went extinct within 6 generations ($n = 28625$). Points are the theoretical predictions for the probability of observing a cluster of size $y = 1, 2, \ldots, 10$ generated using equations (21) and (25) conditioned on the probability of extinction, $z_\infty = 0.285$.

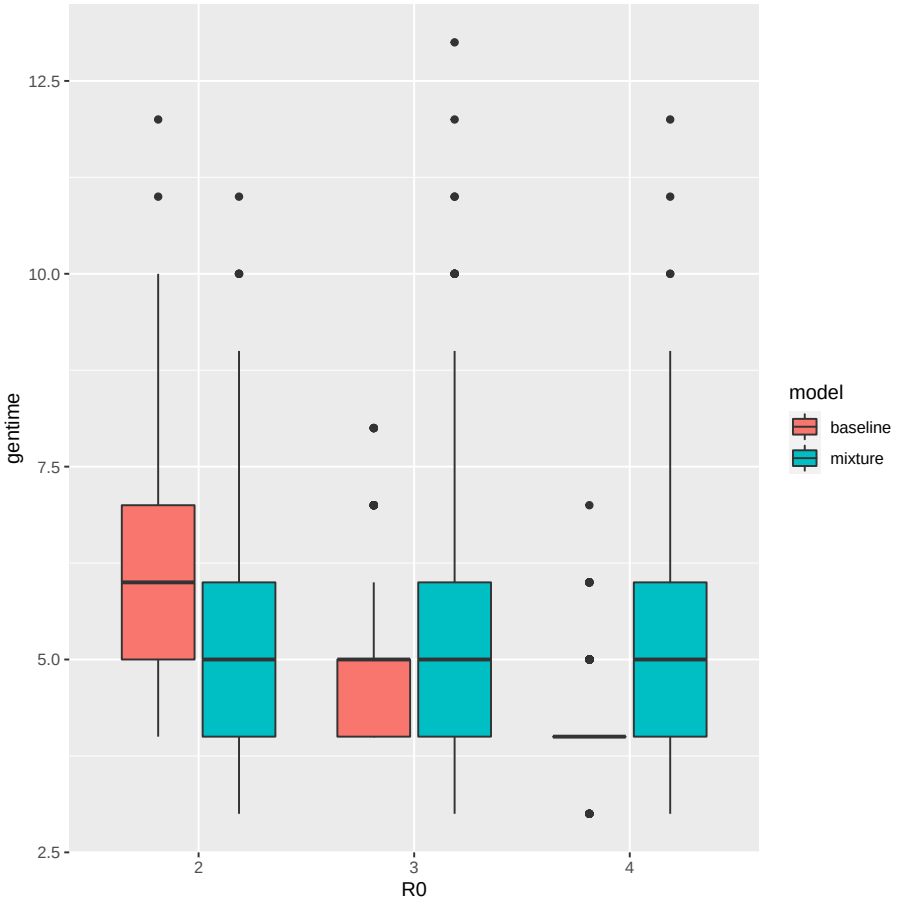

Figure 6: As R_0 increases (resulting from increasing R_0^A), the median first generation to have 50 cases declines for the baseline model, but it does not change in the mixture model. This is due to domination of the direct contact component of transmission leading to saturation effects in outbreak dynamics as R_0^A increases.

3 Data sources

None

4 Analysis

DO WE INCLUDE ALL OF THESE FIGURES?

To address questions 1 and 2, we will examine how the following statistics vary with increasing proportion of community transmission and increasing R_0^A:

- Probability that an index case does not result in secondary infections (theoretical result; 1 figure showing effect of changing $1-p$ and R_0^A simultaneously)
- Non-monotonic variance to mean ratio of the offspring distributions (theoretical result; 2 figures, one each for $1-p$ and R_0)
- Skewness of the offspring distribution? (theoretical result; one figure for $1-p$)
- Probability of superspreading event (simulation; one figure.) Since the cumulative distribution function of a Poisson offspring function is the generalized gamma function, the probability of a superspreading event will saturate to $1-p$. Suggests that this definition of superspreading may not be a very useful one.

To address questions 3 and 4, we will examine how the following statistics vary with increasing proportion of community transmission and increasing R_0^A:

- Mean cluster size (theoretical result; one figure with R_0 on x-axis; different values of $1-p$)
- Variance to mean ratio of the cluster size distributions (theoretical result showing the non-monotonic behavior of the variance-to-mean ratio of the offspring distribution is not retained in the corresponding cluster size distributions conditioned on extinction; one figure with R_0 on x-axis; different values of $1-p$)
- Shape of the cluster size distribution conditioned on extinction (theoretical result and simulation; one figure showing that mixture is fatter tailed than baseline model for same R_0)
- Probability of a major epidemic (theoretical result; 2 figures, one each for $1-p$ and R_0)
- The first generation to have 50 or more cases (simulation; 2 figures, one each for $1-p$ and R_0, see Figure 6 for an example)

Note that many of these statistics generated using the mixture model saturate to a limiting value as R_0 and R_0^A increase.

To address question 5, we will need to make at least one figure to show the effect of interventions.

5 Checklist

- ~~Expressions for summary statistics such as variance and skewness~~
- ~~Derive the chain size distribution conditioned on extinction when $R_0 > 1$ and $R_0 < 1$~~
- Decide which intervention scenarios to examine
- ~~write R code for simulations of branching processes~~
- write R code for branching process postprocessing/summary/visualization using ggplot
- final report