A Simple Contact Tracing Model

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Overview

As the exponential growth of the COVID-19 pandemic appears to slowing in the United States, one may cautiously start to speculate about the next phase of managing the disease. As shelter-in-place restrictions are gradually loosened, how can new eruptions of infection be prevented? One technique proposed to mitigate the reemergence of the epidemic is *contact tracing*, where infected people are isolated, and the contacts of those infected people are screened for illness. It may not be possible to identify all contacts, or it may be that testing of contacts results in false negatives. Under what circumstances will imperfect contact tracing allow the disease to gain a new foothold?

In this note I propose a simple model for contact tracing which allows us to analyze the conditions on reproduction number and testing effectiveness which preclude new infections from becoming a new epidemic.

The Hawkes Process

Let's say a sick individual infects new people according to a Poisson process with a time-dependent intensity. We call this intensity the *infectivity*, which is described by $f \in L^1(\mathbb{R}^+)$ with $f \geq 0$. For example, the infectivity of a newly infected individual may start low, rising to a higher level as the viral load increases and with the onset of coughing, finally decreasing to zero as the individual recovers or succumbs to the disease. It will be convenient to set $f(t) = 0$ for $t < 0$, corresponding to the common-sense notion that you can't infect someone before you yourself became infected. Let K be the number of people infected by an individual. While K is stochastic, and the times $\tau_1, \ldots, \tau_K$ of new infection are stochastic, the infectivity f is taken to be deterministic and is assumed to be the same for all individuals. A key parameter is R_0, the expected number of individuals infected, which is given by

$$R_0 = \mathbb{E}(K) = \int_0^\infty f(t)\, dt$$

The epidemic may be modeled as a time-dependent branching process (a recursive cluster process) where the realization of new infections at times τ_i spawn a new Poisson processes with intensities given by $f(t-\tau_i)$. Its common to think of a branching process in terms of offspring, and to use language like "parent" and "child" when referring to branching events. A slight generalization of the model includes infected "immigrants" which migrate from outside the population at a rate μ. Note that in contrast with the SIR model, the infectivity of an individual is not a function of the how many people are currently infected. In this sense, the branching process corresponds to an infinite population with perfect mixing.

An alternate perspective is to consider the total number of infected individuals $N(t)$ as a function of time, so that $dN(t)$ is a point process whose events correspond to the new infections.

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In [2], the branching process described above is shown to be equivalent to a point process with conditional intensity

$$\lambda(t) = \mu + \int_{-\infty}^{t} f(t-u)\, dN(u)$$

The conditional intensity controls the distribution of events in a small interval $(t, t+dt)$

$$\mathbb{P}(dN(t) = 1 \mid N(s), s < t) = \lambda(t)\, dt + o(dt)$$
$$\mathbb{P}(dN(t) > 1 \mid N(s), s < t) = o(dt)$$

By introducing multiple types of events which mutually spawn each other, we may generalize this model further. Let the cumulative count of type i be given by N_i and let its conditional intensity be given by

$$\lambda_i(t) = \mu_i + \sum_j \int_{-\infty}^{t} f_{ij}(t-u)\, dN_j$$

From the perspective of a branching process, each event of type j independently generates child events of type i according to a Poisson process with intensity f_{ij}, and migrants of type i arrive at a rate μ_i. The use of a multi-type Hawkes process as a model for epidemics is suggested in the the first paper on the subject [1].

Here is the main result on the stability of Hawkes processes. First we need a definition. A non-negative matrix is *irreducible* if M^n eventually has all positive entries. This will correspond to a multitype Hawkes process where each type is influenced by every other type, perhaps indirectly.

1. Theorem *Let $M = (\int_0^\infty f_{ij}(u)\, du)_{ij}$ be the matrix of the mean number of offspring of each type, and assume M is irreducible. Let ρ be the spectral radius of M. If $\rho < 1$ then almost surely (dN_i) converges to a stationary distribution with $\mathbb{E}[\lambda_i] < \infty$. If $\rho > 1$ then $\mathbb{E}[\lambda_i] \to \infty$ exponentially as $t \to \infty$.*

Informally, in the subcritical case $\rho < 1$, the number of newly infected individuals in a fixed interval of time does not increase to infinity, whereas in the supercritical case $\rho > 1$ it does. We ignore the knife-edge critical case $\rho = 1$. A heuristic reason for why this is plausible may be seen by ignoring the timing of new infections and instead grouping them by the number of ancestors. If v is a vector representing the number of individuals of each type at time 0, then Mv is the expected number of directly infected by these individuals, and $M^n v$ is the expected number of individuals in the nth generation of infections. The limiting behavior of M^n is controlled by ρ.

Contact Tracing

Let's say there are two types of individuals: type 1 are monitored and type 2 are unmonitored. The unmonitored individuals have infectivity given by $f(t)$ with $R_0 = \int_0^\infty f(t)\, dt$ representing the average number of people infected. Monitored individuals are isolated, and have reduced infectivity given by $\kappa f(t)$ for some $\kappa < 1$. Let $S_0 = \int_0^\infty \kappa f(t)\, dt = \kappa R_0$ be the average number of people infected by these isolated, monitored individuals.

Owing to monitoring, any individual infected by a monitored individual is detected with probability α and is also monitored. However, with probability $1 - \alpha$ the new infection is undetected and therefore not monitored. This could be because of a false negative on a test, or because of a failure to identify every contact. Consider now the unmonitored population. Assume an individual infected by an unmonitored individual remains a part of the undetected population with probability β. With probability $1 - \beta$, the individual is detected and isolated. In this model an individual is either

monitored or unmonitored for his entire life. In reality, we might imagine a individual exhibits symptoms partway through the course of their disease, and switches categories from unmonitored to monitored at that point. This is a possible avenue for model refinement at a later time.

In light of theorem 1, consider the mean offspring matrix

$$M = R_0 \begin{pmatrix} \kappa\alpha & 1-\beta \\ \kappa(1-\alpha) & \beta \end{pmatrix}$$

The maximum eigenvalue is given by

$$\rho = \frac{1}{2}R_0(\kappa\alpha + \beta) + \frac{1}{2}R_0\sqrt{(\kappa\alpha+\beta)^2 + 4\kappa(1-\alpha-\beta)}$$

We solve he problem of determining when contact tracing is effective by finding the values of $\kappa, \alpha, \beta, R_0$ where $\rho < 1$. For example, take $R_0 = 2$, but say under isolation this reduces to $\kappa R_0 = 0.5$ (so $\kappa = 0.25$). Assume the contact tracing detection failure rate is 10% among monitored individuals (so $\alpha = 0.9$), and that the unmonitored infection detection rate is 60% (so $\beta = 0.4$). In this case we find $\rho \approx 0.93$ and we conclude contact tracing will work under these circumstances. However if the infected contact detection failure rate is slightly higher, say 25%, or the unmonitored detection rate is slightly lower, say 50%, then contact tracing doesn't work because $\rho > 1$.

Let's find some easy bounds on ρ. Because M has positive entries, ρ is greater than any diagonal element of M. Furthermore, ρ is at least as big as the smallest column-sum which, in our case, is κR_0. Thus we must have $\beta R_0 < 1$ and $\kappa R_0 < 1$ in the subcritical case. The first condition can be seen intuitively by focusing on the unmonitored infections only, and ignoring monitored infections (and any subsequent infections from those infections). This process on its own must not explode. The second condition is also intuitive, since if the reproduction number of both monitored and unmonitored individuals exceeds 1, there's no way for the infection to die out.

The eigenvalues satisfy the characteristic equation of M, so we may set $\lambda = 1$ in the characteristic equation to get an implicit expression for the boundary curve of the subcritical region.

$$1 - (\alpha\kappa + \beta)R_0 + \kappa(\alpha + \beta - 1)R_0^2 = 0 \qquad \Rightarrow \qquad 1 - R_0S_0 = S_0(1-R_0)\alpha + R_0(1-S_0)\beta$$

The last symmetrical expression is in terms of $S_0 = \kappa R_0$, the expected number of infections of a monitored individual.

From this we see that, given R_0 and κ, the subcritical region for the variables α and β has a linear boundary, in addition to the boundaries $0 \leq \alpha, \beta \leq 1$. This boundary line passes through $\alpha = 1, \beta = 1/R_0$ and its slope ranges between 0 and ∞ as κ ranges between 0 and $1/R_0$. In the case considered above, where $R_0 = 2$ and $S_0 = 0.5$, the subcritical region is the given by $\beta < \alpha/2$.

For fixed α and β, the boundary of the subcritical region is a hyperbola passing through $S_0 = 0, R_0 = 1/\beta$ and $S_0 = 1, R_0 = 1$. The region is also bound by $R_0 \geq S_0$ and $0 \leq S_0 \leq 1$. The convexity of the boundary is controlled by whether $\alpha + \beta$ is greater than or less than 1.

Conclusions

This analysis shows that in order for contact tracing to work, some simple constraints must be satisfied. We must be able to detect many of the people infected by unmonitored individuals. To prevent an epidemic, on average fewer than 1 individual infected by an unmonitored individual can remain unmonitored. Further, isolation must reduce the reproduction number of a monitored individual to be less than 1. However, if new cases are detected at a high enough rate, and if isolation results in a low enough rate of new infections, then an epidemic will not result regardless of the effectiveness of contact tracing. In intermediate cases where the reproduction number

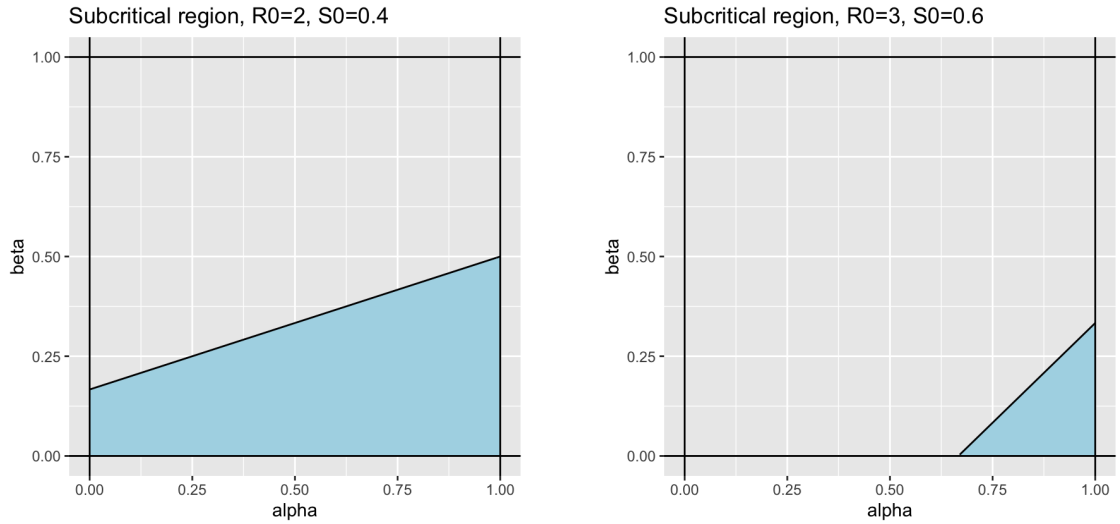

Figure 1: Fixed R_0 and κ

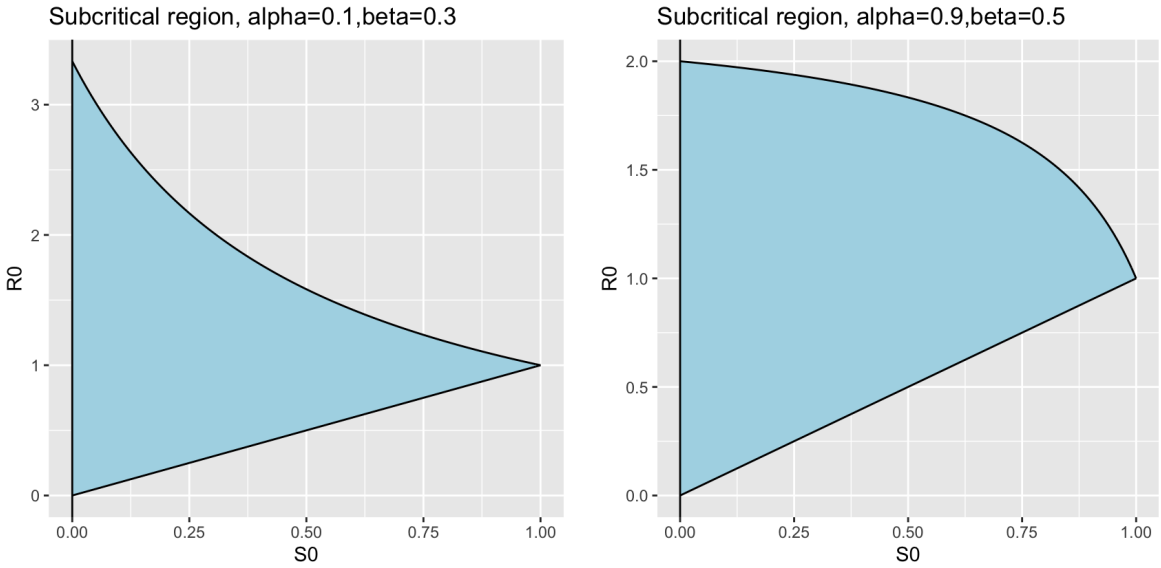

Figure 2: Fixed α and β

under isolation is higher or the unmonitored infection detection rate is lower, then contact tracing becomes important.

References

[1] Alan G Hawkes. Point spectra of some mutually exciting point processes. *Journal of the Royal Statistical Society. Series B (Methodological)*, pages 438–443, 1971.

[2] Alan G Hawkes and David Oakes. A cluster process representation of a self-exciting process. *Journal of Applied Probability*, 11(3):493–503, 1974.