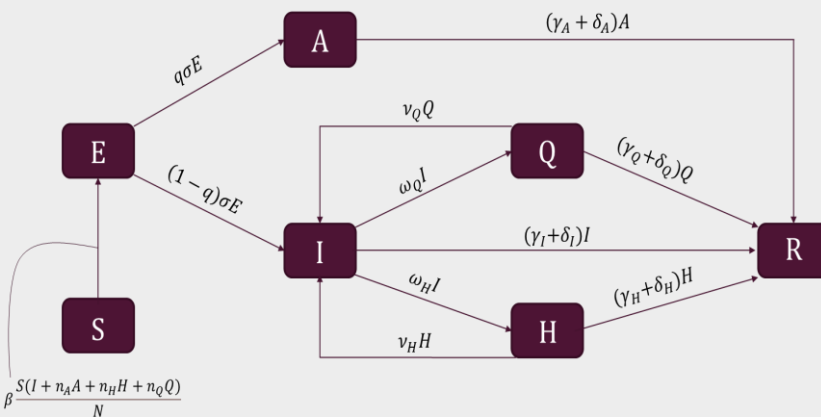


Deterministic Model¹

- ❖ Incorporates heterogeneities in transmission from and recovery of asymptomatic versus symptomatic individuals, as well as hospitalization, quarantine, hospital discharge, and quarantine violation
- ❖ System of deterministic equations corresponding to the following flow diagram:



Stochastic Model^{2,3}

- ❖ Assume early outbreak scenario with a large, completely susceptible population, so that:

$$\frac{S}{N} \approx 1 \Rightarrow \beta \frac{S(I + n_A A + n_H H + n_Q Q)}{N} \approx \beta(I + n_A A + n_H H + n_Q Q)$$

- ❖ Set of events and probabilities based on deterministic movement patterns and rates:

$E_{SE}: S \rightarrow S - 1, E \rightarrow E + 1$	$P(E_{SE}) = \beta(I + n_A A + n_H H + n_Q Q)\Omega^{-1}$
$E_{EA}: E \rightarrow E - 1, A \rightarrow A + 1$	$P(E_{EA}) = q\sigma E\Omega^{-1}$
$E_{AR}: A \rightarrow A - 1, R \rightarrow R + 1$	$P(E_{AR}) = (\gamma_A + \delta_A)A\Omega^{-1}$
$E_{EI}: E \rightarrow E - 1, I \rightarrow I + 1$	$P(E_{EI}) = (1-q)\sigma E\Omega^{-1}$
$E_{IQ}: I \rightarrow I - 1, Q \rightarrow Q + 1$	$P(E_{IQ}) = \omega_Q I\Omega^{-1}$
$E_{IR}: I \rightarrow I - 1, R \rightarrow R + 1$	$P(E_{IR}) = (\gamma_I + \delta_I)I\Omega^{-1}$
$E_{IH}: I \rightarrow I - 1, H \rightarrow H + 1$	$P(E_{IH}) = \omega_H I\Omega^{-1}$
$E_{QI}: Q \rightarrow Q - 1, I \rightarrow I + 1$	$P(E_{QI}) = v_Q Q\Omega^{-1}$
$E_{QR}: Q \rightarrow Q - 1, R \rightarrow R + 1$	$P(E_{QR}) = (\gamma_Q + \delta_Q)Q\Omega^{-1}$
$E_{HI}: H \rightarrow H - 1, I \rightarrow I + 1$	$P(E_{HI}) = v_H H\Omega^{-1}$
$E_{HR}: H \rightarrow H - 1, R \rightarrow R + 1$	$P(E_{HR}) = (\gamma_H + \delta_H)H\Omega^{-1}$

$\Omega = \text{Sum of movement rates}$

Overview

The novel coronavirus SARS-CoV-2 emerged in 2019 and subsequently spread throughout the world. Superspreading – both individual- and event-based – spurs SARS-CoV-2 spread. While individual-based superspreading involves individuals who cause disproportionately more infections over their infectious lifetime, event-based superspreading involves public events and/or social gatherings that result in multiple infections. Here, we adopt an event-based framework. Superspreading events (SSEs) are incorporated into a continuous-time Markov chain model in such a way that their influence on outbreak dynamics may be investigated relative to that of non-SSEs. We explore three versions of this model – two with hospitalization and quarantine and one without – by varying the SSE-related and non-SSE-related infection rates. Limited resources and human behavior are accounted for via discharge of and quarantine violate by infectious individuals. Results indicate that SSE-dominated outbreaks are more variable than non-SSE-dominated outbreaks, and minimizing premature hospital discharge and quarantine violation are crucial for controlling SARS-CoV-2 spread.

Superspreading Events⁴

- ❖ Assume that SSEs involve a single infected individual and otherwise susceptible individuals so that may incorporate as follows:

ψ : SSE Rate
 K : Poisson r.v. w/ param. ϕ
 ϕ : Expected # infections
 k : Some val. of K

$$E_{SSE}: S \rightarrow S - k, E \rightarrow E + k$$

$$P(E_{SSE}) = \frac{\psi(n_{AA} + I)}{\Omega}$$

Constancy Condition⁴

- ❖ Require that the expected number of infections (SSE and non-SSE combined) following the first change in state be the same as when $\beta^* = \beta$ (no SSEs) for different values of β^*
- ❖ Hold ϕ constant for different values of β^* and use the above requirement to obtain ψ as a function of $\beta, \beta^*, \phi, I_1, I_2$, and Ω' :

$$\psi = \frac{I_1(\beta - \beta^*)\Omega'}{I_2(\phi\Omega' + \beta I_1(\phi - 1))}$$

$$I_1 = n_A A + I + n_Q Q + n_H H$$

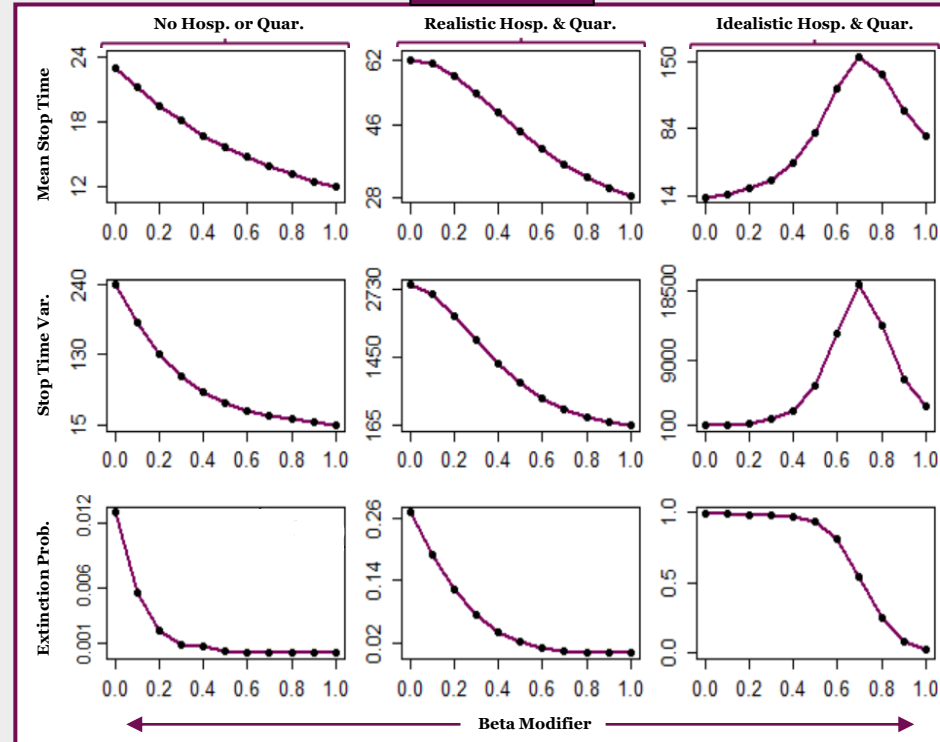
$$I_2 = n_A A + I$$

$$\Omega' = \sigma E + (\gamma_A + \delta_A)A + (w_Q + \omega_H + \gamma_I + \delta_I)I + (v_Q + \gamma_Q + \delta_Q)Q + (v_H + \gamma_H + \delta_H)H$$

Simulation

- ❖ Model simulated model using Gillespie's direct algorithm
- ❖ Simulations ended once 50 current infections or extinction occurs

Results



Conclusions

- ❖ The greater the incidence of SSEs, the greater the variability in outbreak profiles
- ❖ The greater the incidence of SSEs, the longer it takes on average to attain a given number of current infections, but it may still happen more quickly than when there is lower incidence of SSEs
- ❖ The disease easily establishes itself in NHQ but struggles more in RHQ and IHQ
- ❖ The disease much more easily establishes itself in RHQ compared to scenario IHQ

References

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