

1 Supplement to SAFE Transport: Wearing Face Masks Significantly Reduces the Spread of COVID-19 on Trains

1.1 *Transit assignment engine*

The transit assignment engine is an agent-based simulation platform allowing to understand the network of contacts between travelling passengers (with whom and how long they stay in contact with) – a key input to model the spread of infection in public transport networks. Its architecture is presented in Figure 1.

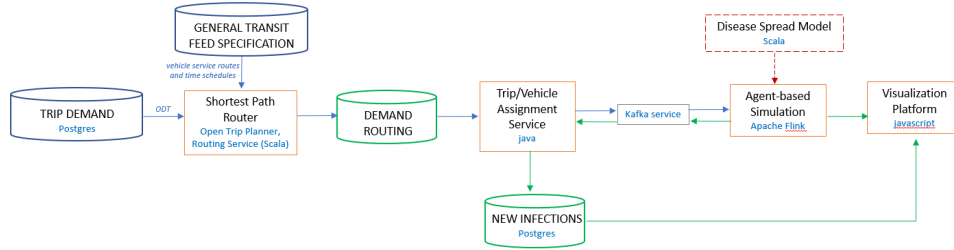


Figure 1: Transit assignment engine

The transit assignment engine requires input data on: (1) trip demand (that is, trip origin, trip destination and start trip time for every passenger), and (2) transit service supply (that is, routes and schedules of public transport services). The total number of trips in Sydney’s train network before COVID-19 was about 8 million per week. To account for the patronage drop during COVID-19 pandemic we assume the demand to be 10% of the total which aligns with the real-life observations [20, 19, 21] (see Table 1 in §1.3). The supply of services before and during COVID-19 was the same and so it was applied without changes.

Simply speaking, the transit assignment engine maps trip demand to transit service supply. In our case study, this means assigning a passenger to a train trip leg. Trip demand captured by smart cards, including tap-on and tap-off data with location GPS coordinates and timestamp, is used by the Shortest Path Router to calculate a set of time-dependent shortest paths for each travelling passenger using classic Dijkstra algorithm. Each path represents one passenger trip which may include multiple legs since passengers may transfer between services. Next, the paths are passed to

the Trip/Vehicle Assignment Service module, where an attempt is made to assign each leg of the trip to a transit service vehicle (for example, a train) with some prior specified capacity and current occupancy. Transit service vehicles operate in accordance with a pre-defined schedule. The start time of the passenger trip needs to be matched with the scheduled transit vehicle operation. If a passenger cannot be assigned to a vehicle at a particular leg due to vehicle capacity constraint, the next shortest path is requested from the Shortest Path Router and the assignment process is repeated until a feasible trip/vehicle assignment is found. Once all the feasible paths are defined and assigned to the vehicles, they are simulated in an agent-based environment. As the simulation is ongoing the passengers are tracked and detailed dynamic outputs are collected for every: agent (that is, a passenger), link (link is the part of the trip between two consecutive stops/stations), stop/station, service vehicle, service line, and the whole network. Every time a passenger finishes their trip a Disease Spread Model is triggered and this passenger is assigned a “status” corresponding with its outcome (that is, during their trip a passenger got infected or not). The information about new infections is sent to the Visualization Platform to be displayed on a dashboard. At the end of every simulated day and at the end of the simulation horizon, the total number of new infections is calculated and reported for final evaluation.

When it comes to the implementation of the transit assignment engine, traditional Java-based services backed by a relational database for transit assignment and trip event processing were developed, and an open source multi-modal trip planning library - Open Trip Planner (OTP) was used for the routing step in transit assignment. OTP was utilised as it includes implementations of both routing approaches (graph and adjacency list). It is robust and performs efficiently. Additionally, a front-end, based on the modern JavaScript framework Vue.js, D3.js and P5.js, was built to visualise network activity at an individual vehicle level and provide aggregate origin-destination information in a dashboard format.

1.2 Transmission model on trains

At its core, the disease spread model on public transport provides a probability of becoming infected for each susceptible passenger, based on the current and past travel of infectious passengers in the same spatial area. The model uses a number of simplifying assumptions, the most important being that we ignore any age-based effects (all agents are identical), and we assume homogeneity of mixing within the spatial area considered. We

assume a half-carriage spatial area for our model, largely due to the upper and lower deck structure of the Sydney train carriages (Waratah design). Subsequently, our homogeneity of mixing assumptions means that passengers are equally divided amongst half-carriages of a train, and any spatial effects within a half-carriage are ignored.

The disease spread model for COVID-19 on trains has two transmission pathways, with quite different modelling approaches. The first pathway is direct (that is, person-to-person) transmission, for which we used the empirical attack rate on trains determined by Hu *et al.* [7] as a function of shared ride time (that is, exposure time). The second pathway is due to fomite (that is, person-to-surface-to-person) transmission, for which we use a more mechanistic approach, calibrated to recover the attack rate reported by Hu *et al.* [7]. These transmission pathways are combined into a single probability of infection for each susceptible passenger.

The overall probability of a susceptible passenger being infected from a single trip ($P(S \rightarrow E)$) is a combination of the probability of transmission (p_i) from each infectious passenger (i), for each of the n infectious passengers the susceptible person has shared a half-carriage with, and from the probability of transmission from the half-carriage surfaces (p_c , which depends on i). The overall probability of a susceptible passenger becoming exposed as a result of their travel is the standard probability

$$P(S \rightarrow E) = 1 - (1 - p_c)\prod_i^n (1 - p_i). \quad (1)$$

Both probabilities (p_i and p_c) are dependent on the mask wearing status of each agent of interest (that is, the susceptible being considered, and each of the infectious passengers).

The direct transmission probability, p_i , is based on the overall attack rate on a train found by found by Hu *et al.* [7]. It takes into account shared travel time (Δt_i for travel time with infectious passenger i) but not relative seat location, giving a probability of

$$p_{i0} = \frac{0.121 + 0.022\Delta t_i^2}{100}, \quad (2)$$

where the regression by Hu *et al.* [7] was for joint travel times of 8 hours or less. Note that Hu *et al.* [7] reported the attack rate as a percentage, which is why the probability has a different denominator here. We modify the probability p_i to take into account a number of factors, including: f_s for the proportion of the virus contributing to fomite transmission (p_s); m_i for the mask wearing status of each infectious passenger ($m_i = 1$ for those

wearing a mask and 0 otherwise); and m_s for the mask wearing status of the susceptible passenger being considered (m_s). Subsequently, the direct transmission probability from infectious passenger p_i is given by

$$p_i(m_s, m_i) = (1 - m_s f_s)(1 - p_{s0}(m_i)) \min(p_{\max}, p_{i0}). \quad (3)$$

The $\min(p_{\max}, p_{i0})$ term is to ensure the probability stays in a sensible range, in case of the remote possibility of passengers sharing an especially long trip.

To calibrate the model we relied on evidence reported in the literature. How mask wearing affects a susceptible passenger's probability of infection, through f_s , was calibrated to obtain the Odds Ratio of 0.22 reported by Wang *et al.*, 2020 [9]. We use $p_{\max} = 0.16$ based on work by Tang *et al.* 2020 [11] and Chu *et al.*, 2020 [12].

Some ($p_s(m_i)$) of the virus shed by an infectious passenger who enters a half-carriage will be deposited on surfaces. How much depends on the mask wearing status of the infectious individual (m_i). We base our model of the amount of deposited virus on the work by Atkinson and Wein, 2008 [5], and the estimates of decay on surfaces by Riddell *et al.* [8]. Due to these processes, the fomite transmission is modelled by considering the aggregated effect of infectious passengers on the half-carriage over time, and then we convert this into a probability of infection (p_c) via a standard dose response model. The overall probability of infection from surfaces (that is, the dose response) is given by

$$p_c(m_s) = p_{\max} \left[1 - \exp \left(-\frac{D_s(m_s)}{k} \right) \right], \quad (4)$$

where k is the response characteristic to an effective dose of COVID-19, which from Zhang and Wang, 2020 [10], is $k = 6.19 \times 10^4$; and the dose ($D_s(m_s)$) is the effect of the aggregated concentration of the virus on surfaces, taking into account the virus decay. The virus decay value comes from fitting an exponential decay model to the observed data reported by Riddell *et al.* [8].

The effective viral dose is given by

$$D_s(m_s) = \alpha(m_s) \sum_{\ell} \left[p_{s0} B \hat{G}_{\ell} \tau_{\ell} + \frac{1}{\mu_s} \left(C_{\ell 0} - p_{s0} B \hat{G}_{\ell} \right) (1 - \exp(-\mu_s \tau_{\ell})) \right], \quad (5)$$

where $C_{\ell 0}$ is the concentration on the carriage surfaces at the beginning of a link ℓ , B and $\alpha(m_s)$ are aggregations of constants, $\hat{G}_{\ell}$ is the effective number

of un-masked infectious passengers on the half-carriage during a link ℓ , τ_ℓ is the duration of a link ℓ (note $\Delta t_i = \sum_\ell \tau_\ell$), and $\mu_s = \frac{0.04322}{24}$ is the surface decay rate of the virus from Riddell *et al.*, 2020 [8]. More specifically, $B = 2 \times \frac{10^5}{60\mu_s}$ is the aggregation of constants describing the surface concentration and dose from Zhang and Wang, 2020 [10]. The proportional constant for effective dose of the virus from a surface is given by the assumed functional form

$$\alpha(m_s) = (1 - m_s f_s) \alpha_0, \quad (6)$$

where $\alpha_0 = 0.28$ is the proportional constant for effect dose of virus from a surface in the absence of face masks, calibrated to obtain the attack rate from Hu *et al.*, 2020 [7] (Equation (2)).

Finally, the concentration of virus on the half-carriage surfaces at the end of a link j is given by

$$C_j = p_{s0} B \hat{G}_j \left(C_{j0} - p_{s0} B \hat{G}_\ell \right) \exp(-\mu_s \tau_j), \quad (7)$$

where p_{s0} is the proportion of virus shed by un-masked infectious passengers that ends up on surfaces. The value of p_{s0} is based on evidence that fomite transmission is a relatively unlikely route [26, 27, 13, 28], with Ferretti *et al.* [28] finding a speculative estimate of 10% of transmission being attributable to fomite transmission. We take this to be a rough estimate that $p_{s0} \approx 10\%$.

1.3 Seeding

Our primary disease spread model, as outlined in § 1.2, only considers transmission on the public transport network (and is specifically calibrated to trains). Furthermore, we do not track individuals between trips, so disease progression cannot be explicitly modelled. To overcome these limitations, and to keep the numbers of infectious passengers travelling on trains identical across simulations for comparability, we use a deterministic compartmental model to approximate the transmission dynamics in the general community.

We use the standard susceptible-exposed-infectious-recovered progression structure, with the exposed and infectious compartments repeated to better account for the distribution of time spent in those states (see, for example, [29]). We refer to this as an “SEEIIR” model for short.

As the transmission model is focused on, and calibrated to, transmission that is expected to occur on a train it is necessary to consider community-wide transmission and how that will impact the transmission on a vehicle

space unit (in our case a space unit is a half-carriage). To this end, a simple population level transmission model was used to seed the expected numbers of cases into the train network each day in the simulated time horizon (that is, 7 days). This model, “SEEIIR”, follows a susceptible-exposed-infectious-recovered structure where: S is for susceptible, E for exposed (infected but not yet able to infect others), I for infectious (able to infect others), and R for recovered and immune. To better align with our epidemiological understanding of disease progression, and to account for the distribution of time being spent “exposed” or “infectious” being more like an Erlang rather than an exponential distribution, we repeat those two compartments as shown in Figure 2.

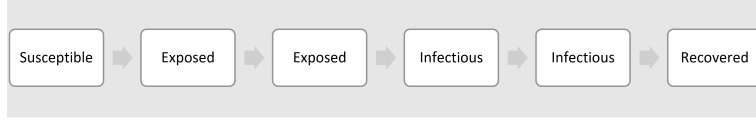


Figure 2: Community-wide “SEEIIR” transmission model, with the common susceptible-exposed-infectious-recovered structure. The repeated compartments better capture disease progression.

The dynamics of transmission are then governed by the following system of ordinary differential equations (ODEs)

$$\frac{dS}{dt} = -\frac{\beta S(t)(I_1(t) + I_2(t))}{N}, \quad (8)$$

$$\frac{dE_1}{dt} = \frac{\beta S(t)I(t)}{N} - 2\sigma E_1(t), \quad (9)$$

$$\frac{dE_2}{dt} = 2\sigma E_1(t) - 2\sigma E_2(t), \quad (10)$$

$$\frac{dI_1}{dt} = 2\sigma E_2(t) - 2\gamma I_1(t), \quad (11)$$

$$\frac{dI_2}{dt} = 2\gamma I_1(t) - 2\gamma I_2(t), \quad (12)$$

$$\frac{dR}{dt} = 2\gamma I_2(t), \quad (13)$$

where the total population $N = S + E_1 + E_2 + I_1 + I_2 + R$ is constant, $\beta = 2.5\gamma$ is the transmission rate (that is, value set to recover the expected $\mathcal{R}_0 = 2.5$) [22, 23], $1/\sigma = 3$ days is the average latent period (time from being infected to being able to infect others) [24, 25], and $1/\gamma = 13$ days is the average infectious period [25]. This model was implemented in Python 3.8, using

the packages “matplotlib 3.3.2”, “scipy 1.5.2”, and “numpy 1.19.2”, and numerical solutions used the “scipy.integrate.solve_ivp” integrator to solve the System of ODEs.

The model makes a number of simplifying assumptions, including: (1) everyone infected ends up with immunity, (2) constant proportion of the infectious passengers travel as per before COVID-19, (3) super-infections are ignored, and (4) any interventions are ignored (at least explicitly). However, many of these limitations are strongly mitigated by the short time periods being considered in the full transport simulation (that is, the 7 day simulation horizon). We also do not explicitly consider asymptomatic infections, but this partially accounts for our assumption ignoring isolation and self-isolation. That is, people are less likely to self-isolate when asymptomatic.

As discussed in the paper, the starting point for the seeding process is 2000 infectious cases. This initial number is used in the SEIIR model, based on the population of Sydney and a basic reproduction number of 2.5. We used estimates of the proportion of the population who commute via public transport (approximately 20%, considering 2016, 2011, and 2006 Census records), and the proportion of commuters who use trains (approximately 50.9%), to arrive at an estimate of 10% of the population using trains in Sydney. This, combined with the estimate of 2000 initial infectious in the Sydney population of 5.73 million in 2019, resulted in the expected number of infectious individuals in the community, determined by running the Python implementation of Equations (8)–(13), and those travelling by train each day as reported in Table 1.

Table 1: Numbers of infectious in the community and estimated seeds for the simulation horizon (that is, 7 days) for the full train simulation modelling, rounded to the nearest integer.

Day	Total infectious in community	Infectious train passengers	Total train passengers
0	2000	200	1340708
1	1999	200	1386593
2	2044	204	1391588
3	2152	215	1409584
4	2312	231	1373788
5	2512	251	675665
6	2743	274	583467

Table 2 provides an overview of all used modelling parameters.

Table 2: Modelling parameters.

Symbol	Description	Value	Source
α_0	Proportional constant for effect dose of virus from a surface, in the absence of masks	0.0655	Calibrated to obtain attack rate from Hu <i>et al.</i> [7]
$\alpha(m_s)$	Proportional constant for effect dose of the virus from a surface	$(1 - m_s f_s) \alpha_0$	Assumed functional form
B	Aggregation of constants describing the surface concentration and dose	$2 \times \frac{10^5}{60\mu_s}$	[10]
f_i	How mask wearing affects infectious individuals' viral shedding	0.1	[6]
f_s	How mask wearing affects susceptible individuals' probability of infection	0.8	Calibrated to obtain the Odds Ratio of 0.22 as reported by Wang <i>et al.</i> [9]
k	Response characteristic to an effective dose of COVID-19	6.19×10^4	[10]
p_{max}	Maximum probability of infection	0.16	[12, 11]
p_{s0}	Proportion of virus shed by infectious individuals that ends up on surfaces when no mask is worn	0.1	Value based on evidence fomite transmission is the relatively unlikely route [26, 27, 13, 28]
μ_s	Decay rate of virus on the surface(s)	$\frac{0.04322}{24}$ per hour	Exponential decay model calibrated to data from Riddell <i>et al.</i> [8]

1.4 Face mask wearing scenarios

To determine the effectiveness of mask wearing, we had to answer two questions: (1) how mask wearing affects infectious passengers shedding, and (2)

how mask wearing affects susceptible passengers' probability of infection. The mask wearing status is then randomly conferred on passengers to recover the desired proportion of adherence to advice, with the effectiveness applied as appropriate and as follows.

To answer the first question, we use the filtration efficacy of a two-layer cloth mask described in Howard *et al.*, 2020 [6] found to provide an 88-94% reduction in infections particle load, and 80-90% filtration efficacy. In our modelling we use 90% as it is within both of these bounds, and reduction in infection particle load is arguably the most important aspect. Due to our one minus effect model structure, this gives us $f_i = 0.1$.

To answer the second question, we use the work by Wang *et al.*, 2020 [9] that identified the odds ratio of 0.22 for wearing a mask, compared to not wearing it. Its relationship to our effectiveness parameter (f_s) is not immediately apparent, and so we calibrated the value to recover the odds ratio from our model,

$$OR = \frac{\frac{\text{Number of infections wearing a mask}}{\text{not infected wearing a mask}}}{\frac{\text{infections not wearing a mask}}{\text{not infected not wearing a mask}}}. \quad (14)$$

We know that the expected number of cases will be

$$\mathbb{E} = P(S \rightarrow E)S_0. \quad (15)$$

Let the subscript m denote mask wearing and n no-masks, then odds ratio equation becomes

$$OR = \frac{\frac{\mathbb{E}_m S_0}{S_0 - \mathbb{E}_m S_0}}{\frac{\mathbb{E}_n}{S_0 - \mathbb{E}_n S_0}} \quad (16)$$

$$= S_0 \frac{\frac{P(S \rightarrow E)_m}{1 - P(S \rightarrow E)_m}}{\frac{P(S \rightarrow E)_n}{1 - P(S \rightarrow E)_n}}. \quad (17)$$

The RHS is implicitly a function of f_s , and hence we can numerically find when the LHS=RHS. We used the `scipy.optimize.fsolve` and a Python 3.8 implementation of our model to determine how f_s varies with different p_{s0} values and different numbers of infectious passengers I_0 , shown in Figure 3. Given the tight range of $f_s \in [0.78, 0.84]$, we have chosen $f_s = 0.8$.

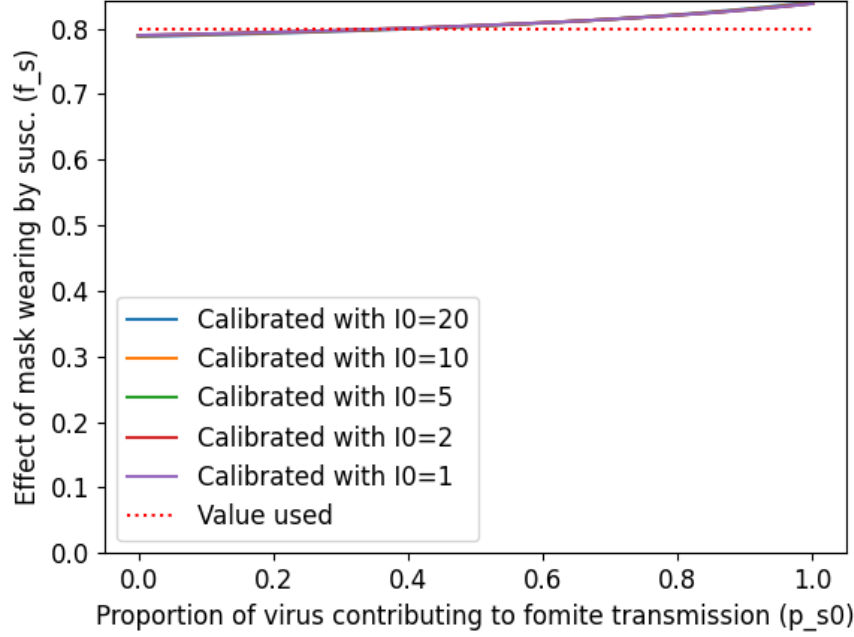


Figure 3: Sensitivity of the calibration of the effect of mask wearing by susceptible individuals, f_s , to the proportion of virus shed contributing to fomite transmission, p_{s0} , for different numbers of infectious passengers, I_0 . Note that this calibration is insensitive to the number of infectious passengers on the half-carriage, I_0 , with the lines completely overlapping.

2 Sensitivity analysis

Sensitivity analyses are key to understanding how assumptions affect outcomes and recommendations. In the main paper, we covered the sensitivity analysis of how our results are affected by the proportion of virus shed that contributes to fomite transmission (p_{s0}) versus direct transmission ($1 - p_{s0}$). Here, we explore how sensitive the calibration of parameter values are to variables of interest, and determine the number of repeats required for consistent variance given the highly stochastic nature of the processes we are simulating.

2.1 Dose response calibration

As discussed in §1.2, we have assumed that the Hu *et al.* [7] attack rate is the ground truth, and have calibrated the fomite model accordingly. Here we show how sensitive those calibrations are to the proportion of virus shed contributing to fomite transmission (p_{s0}), the number of infectious passengers (I_0), and duration of the mutual link (τ_ℓ).

Figure 4 depicts the sum of squared error between a simple simulation of 50 susceptible passengers on a half-carriage for times of $\Delta t_i = [0.1, 0.2, 0.4, 0.6, 0.7, 0.95, 1.4]$ and the Hu *et al.* [7] attack rate, as a function of the dose response constant α_0 . The travel times used reflect the distribution of the available data for the Sydney network. The minimum sum of squared error for α_0 for each of the different numbers of infectious passengers are provided in Table 3. Subsequently, we use $\alpha_0 = 0.28$ for all simulations.

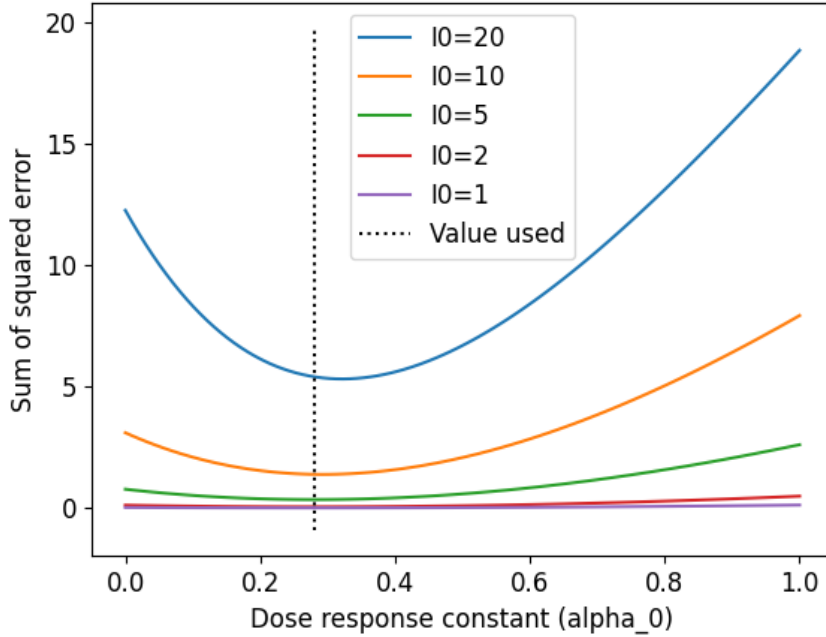


Figure 4: Sensitivity of the calibration of the dose response constant, α_0 , to the proportion of virus shed contributing to fomite transmission, p_{s0} , for different numbers of infectious passengers, I_0 . The dashed horizontal line is the value of α_0 used in all simulations presented in the paper.

Table 3: Optimal value of the dose response constant, α_0 , to minimise the sum of squared error between number infected and the Hu *et al.* [7] attach rate for different numbers of infectious passengers on the half-carriage with 50 susceptible passengers.

Total infectious passengers	Optimal α_0
20	0.32
10	0.29
5	0.28
2	0.27
1	0.27

Figure 5 depicts how the calibration of the dose response constant, α_0 , is affected by the proportion of virus shed contributing to fomite transmission, p_{s0} . It shows that, with the exception of $p_{s0} = 0.0$ (that is, all virus shed contributes to direct transmission), the calibration is within a small range, with $\alpha_0 \in [0.27, 0.33]$. The $\alpha_0 = 0.28$ value used throughout is depicted by the horizontal dashed line, per the minimisation depicted in Figure 4. This aligns well with the smaller values of p_{s0} expected to reflect actual COVID-19 transmission.

2.2 Number of repeats required to capture stochastic variance

Given the number of stochastic processes captured in our simulation, it is important to determine the minimum number of repeat simulations required to consistently capture the variance. Figure 6 depicts the average and 95% confidence intervals from different numbers of repeat simulations (that is, the whiskers represent 95% of the simulations). Here the leftmost whisker plot incorporates the simulations from the runs represented in the rightmost whisker plot. So the “20” repeats include all of the simulations from the “10”, the “30” include all of the simulations from the “20”, and so forth. We conclude from this that 60 repeats are sufficient to capture the stochastic variation. Therefore the 100 repeats we have used throughout sufficient to capture the stochastic variation.

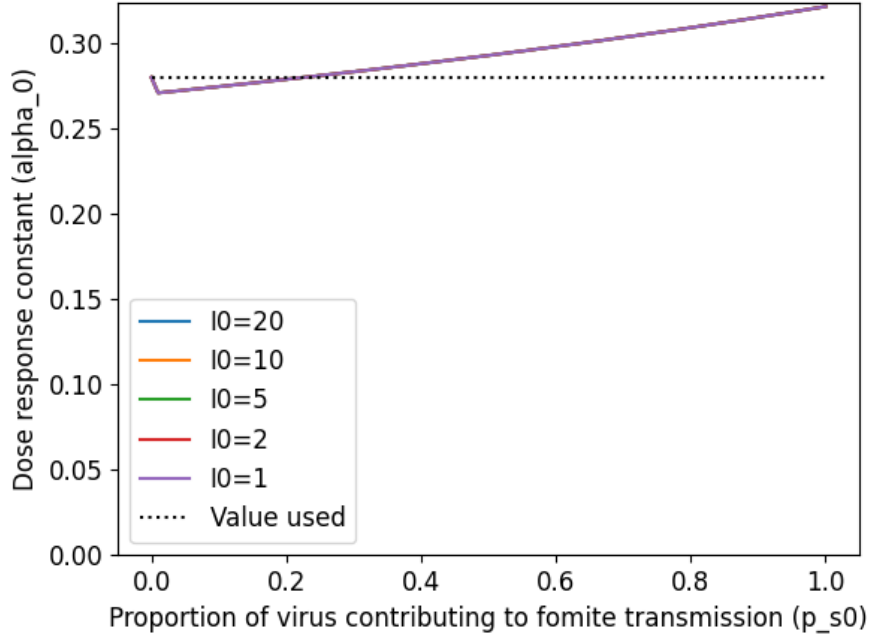
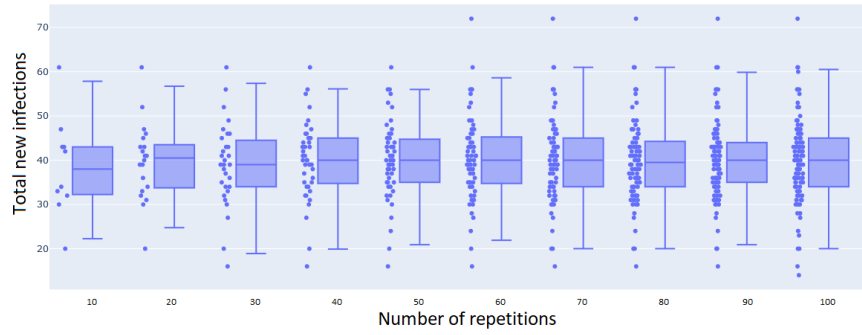
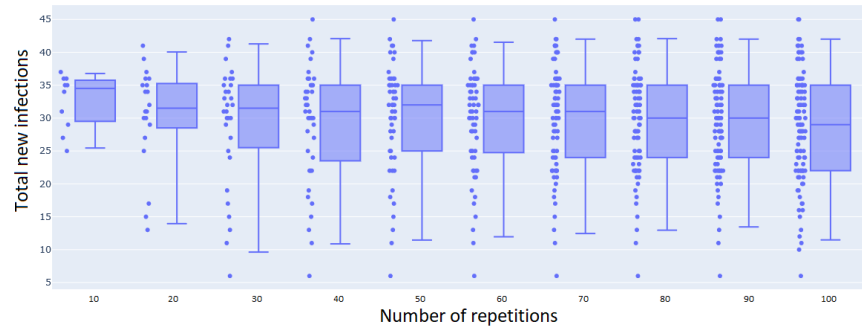
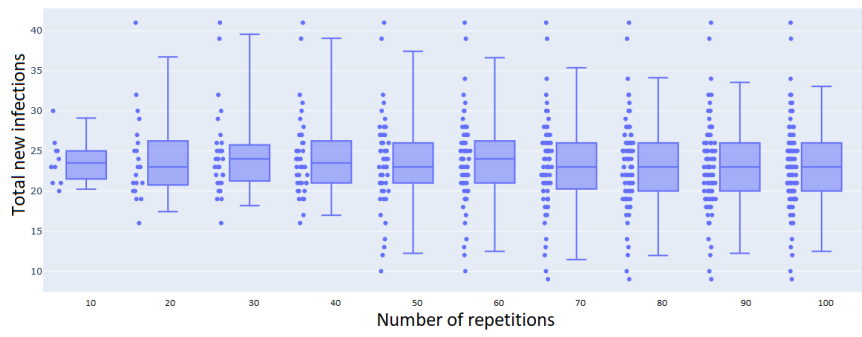


Figure 5: Sensitivity of the calibration of the dose response constant, α_0 , to the proportion of virus shed contributing to fomite transmission, p_{s0} , for different numbers of infectious passengers, I_0 . The dashed horizontal line is the value of α_0 used in all simulations presented in the paper.

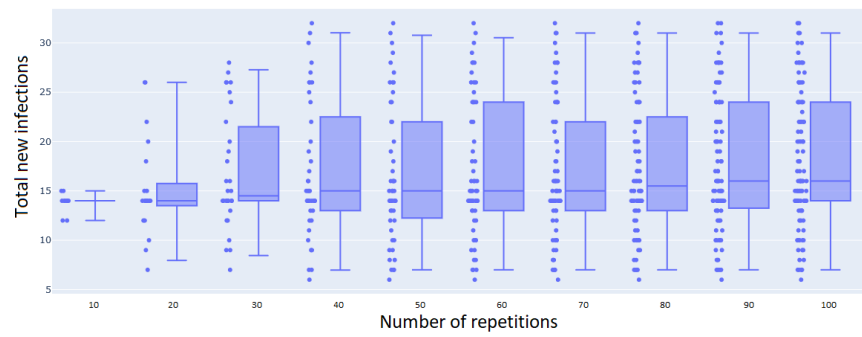




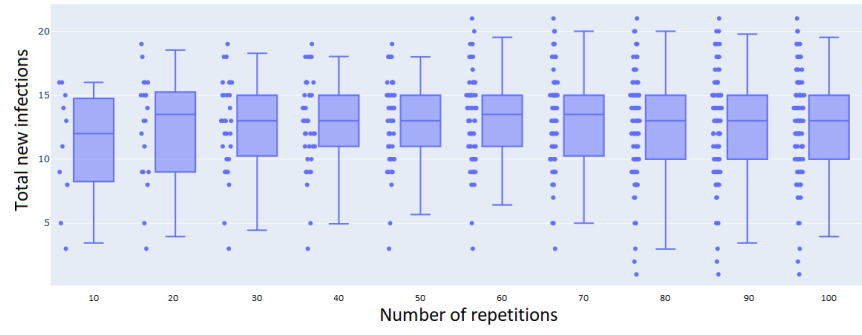
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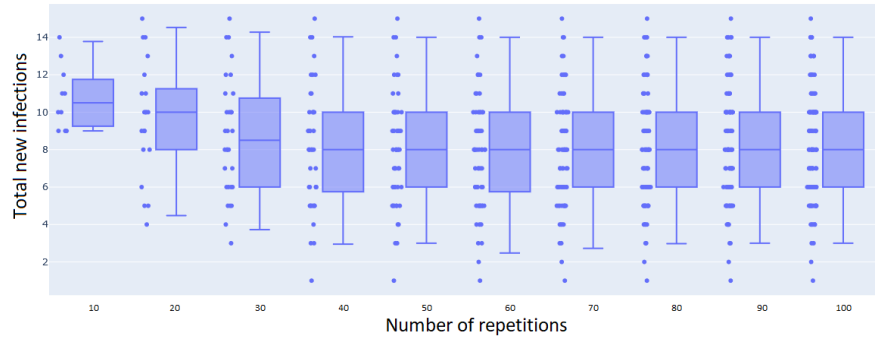
[c]



[d]



[e]



[f]

Figure 6: Effect of number or repeated simulations on stochastic variance for [a] baseline, [b] Mask_25, [c] Mask_50, [d] Mask_75, [e] Mask_80, and [f] Mask_100.

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