Optimizing COVID-19 screening for shift-workers to reduce introduction of disease from the community

Methods and results

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5/20/2021

## To-dos

* Detection limit rather than sensitivity
* Add non-compliance with testing regimen
* Make self-isolation a scenario
  + Grid of self-isolation to testing compliance?
* Format for analytic results, followed by grid search of scenarios, followed by sensitivity analyses of optimal scenario subjected to: non-compliance, leaky cohort

## Model framework and parameterization for SARS-CoV2

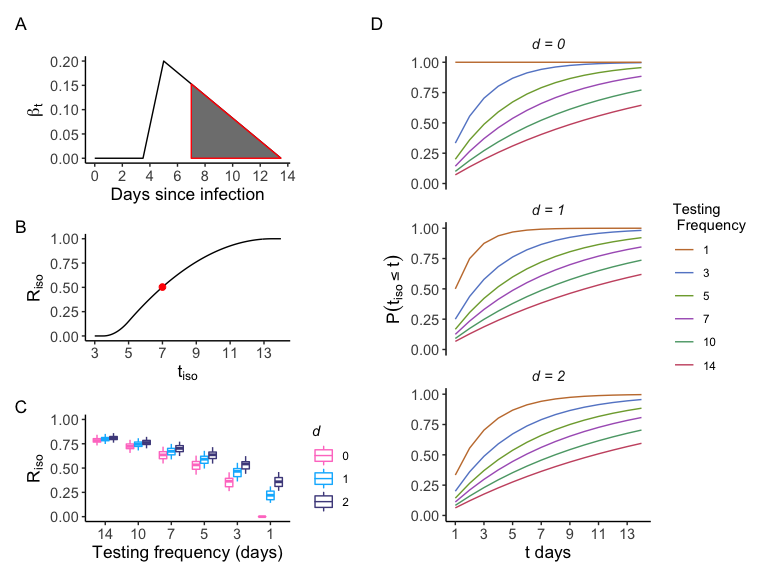
Building on previous work investigating the effects of non-pharmaceutical interventions [ [1](https://doi.org/10.1073/pnas.1616438114) ] and testing [ [2](https://doi.org/10.1126/sciadv.abd5393) ] on the transmission of infectious diseases, we model individual contributions to infection through time from an infectiousness profile, , generated from key biological parameters of the disease that determine the distribution of infectiousness over time. We model using a triangle distribution with infectiousness beginning after the latent period, ending after the duration of the infectious period, and peaking at some point in between (, , , and a<c<b).

The viral dynamics of SARS-CoV2 make control efforts challenging, as asymptomatic and presymptomatic transmission caused by high infectiousness in the absence of symptoms are common [CITE](https://wwwnc.cdc.gov/eid/article/27/4/20-4576_article), [CITE](https://science.sciencemag.org/content/368/6490/489). In terms of the infectiousness profile for SARS-CoV2, this means that peak infectiousness tends to coincide with the onset of symptoms (for cases that are symptomatic), but occurs after completion of the latent period (i.e. >). The expected number of new cases generated at time is thus , where is the effective reproduction number interpreted as the expected number of cases generated by a new case over the duration of the infectious period. The model therefore assumes that new cases are most likely to be generated around the time of peak infectiousness, . Table X lists the distributions of , , and used here.

In the presence of interventions that isolate infectious individuals prior to , e.g. through contact tracing, self-isolation following the onset of symptoms, or testing, the effect of isolation on can be directly estimated from the time to isolation as , where is the time at which isolation occurs. Reducing via improved contact tracing or more frequent testing can thus be envisioned as removing a larger slice from the overall infectiousness triangle by reducing . The size of the slice removed is dependent on the shape of the overall triangle distribution, which is primarily determined by and , and the location of in relation to . For instance, if , then the proportional reduction in can be estimated as . Other interventions that reduce across all levels of infectiousness such as wearing a mask or reducing the contact rate between infectious and susceptible individuals can also be accommodated simply by multiplying by a constant.

Assuming testing is independent of symptoms, known contacts, and other reasons for explicitly seeking testing, we can estimate the probability of going days without being tested from the testing frequency, , as and the probability that as , assuming isolation occurs immediately after testing. Given substantial turnaround times between testing and isolation, particularly when relying on PCR-based tests, we can also incorporate the delay between testing and isolation, , as: , where is simply the average number of days between tests.

This allows for incorporation of the testing frequency and delay into estimation of :



**Model framework and analytic results.** A) Example infectiousness profile for , , , , with shaded area demonstrating infectiousness slice removed if , leading to . B) as a function of with same parameters as in A and point indicating scenario depicted in A. C) Boxplots showing distributions of as a function of testing frequency, , and test delay, , incorporating uncertainty in , , and by drawing parameter sets for each, with baseline . Boxplots indicate median, interquartile range, and full range of values of . D) Relationship between testing frequency, , test delay, , and probability isolation occurs by day , i.e. , demonstrating that delays in testing substantially reduce the probability of prompt isolation, particularly in more frequent testing scenarios.

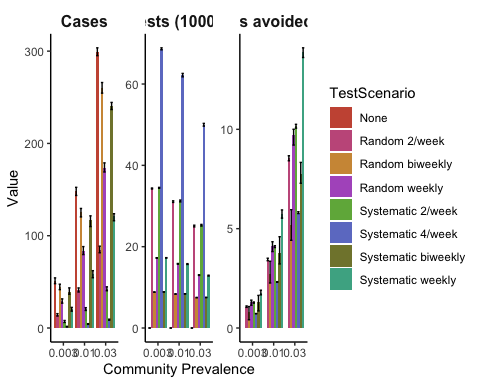
## Individual-based model simulations

* All staff are assigned a work schedule that determines time frames when they are working. is an indicator function for whether staff member is working at the facility on day .
* In addition to their work schedule, all staff are assigned a testing schedule, encoded by function .
* All staff are subject to two sources of infection: community and workplace, represented by force of infection parameters and , respectively. In addition, we introduce a partitioning parameter, , representing the relative probability of acquiring infection in the workplace versus in the community, therefore and . New cases at each time step are then generated by subjecting all susceptible staff to a Bernoulli trial where
* Whenever a new staff infection is generated, parameters for the individual are drawn to determine the latent period (), the incubation period (), and the total infectious period () to generate an infectiousness profile (). Assuming constant across all individuals, the expected number of workplace cases produced on day by individual is
* Main outcome is expected number of cases transmitted by staff over the simulation timeframe:
* Isolated staff are not retested, but recovered staff are tested as normal

### “Grid search” Over frequency, delay, and systematic vs random testing

Basically interested in three variables: pcr vs antigen testing (which basically comes down to turnaround time if we assume antigen tests are equal to pcr in their ability to detect active infection), random vs systematic day of testing, and frequency of testing. We propose the following scenarios encompassing combinations of these variables to explore:

* **S1)** No testing
* **S2)** Random PCR testing once per work week with test report on second day following test
* **S3)** Random antigen testing once per work week with immediate test report
* **S4)** PCR testing on first day of work week with test report on second day following test
* **S5)** Antigen testing on first day of work week with immediate test report
* **S6)** Random PCR testing twice per work week with test report on second day following test
* **S7)** Random antigen testing twice per work week with immediate test report
* **S8)** PCR testing on first and third day of work week with test report on second day following test
* **S9)** Antigen testing on first and third day of work week with immediate test report
* **S10)** PCR testing on all days of work week with test report on second day following test
* **S11)** Antigen testing on all days of work week with immediate test report



This makes it clear that decreasing the delay between test and isolation is more important than increasing frequency. In addition, systematic testing is superior to random testing when infections are only acquired from the community, with systematic testing always resulting in fewer expected cases than random testing. Some systematic testing scenarios even perform similarly to random testing scenarios with higher frequency (e.g. compare S5 to S7). Systematic testing on the first day of the work week with instant turnaround (e.g. antigen testing) also results in the most cases avoided per test conducted.

### Adding workplace transmission

### Adding workplace transmission and leaky cohorts caused by 1 irregular shift per week

### PCR test turnaround time and antigen test sensitivity

