

## Supplementary Materials for

### **Test sensitivity is secondary to frequency and turnaround time for COVID-19 screening**

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#### **This PDF file includes:**

Figs. S1 to S8  
Supplementary Text

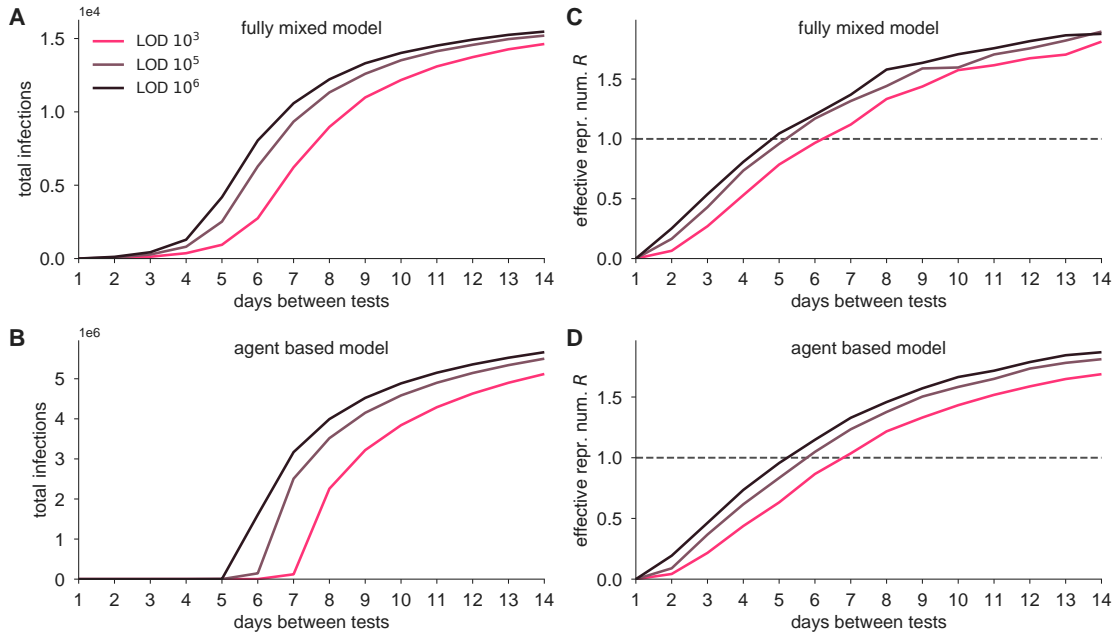


Figure S1: **Epidemiological model outcomes for various test LODs and frequencies.** The fully mixed model (top row) and agent based model (bottom row) were simulated (Methods) with various test frequencies, ranging from daily to once every 14 days, and with LODs of  $10^3$ ,  $10^5$ , and  $10^6$ . Modeling results show mean outcomes from 50 independent simulations at each point, expressed as (A, B) total infections and (C, D) effective reproductive number  $R$ , from a baseline of  $R_0 = 2.5$ . For the fully mixed model, only secondary infections are shown, excluding imported infections. Total population sizes were  $N = 2 \times 10^4$  for the fully mixed model and  $8.4 \times 10^6$  for the agent based model. Dashed lines indicate  $R = 1$  for reference.

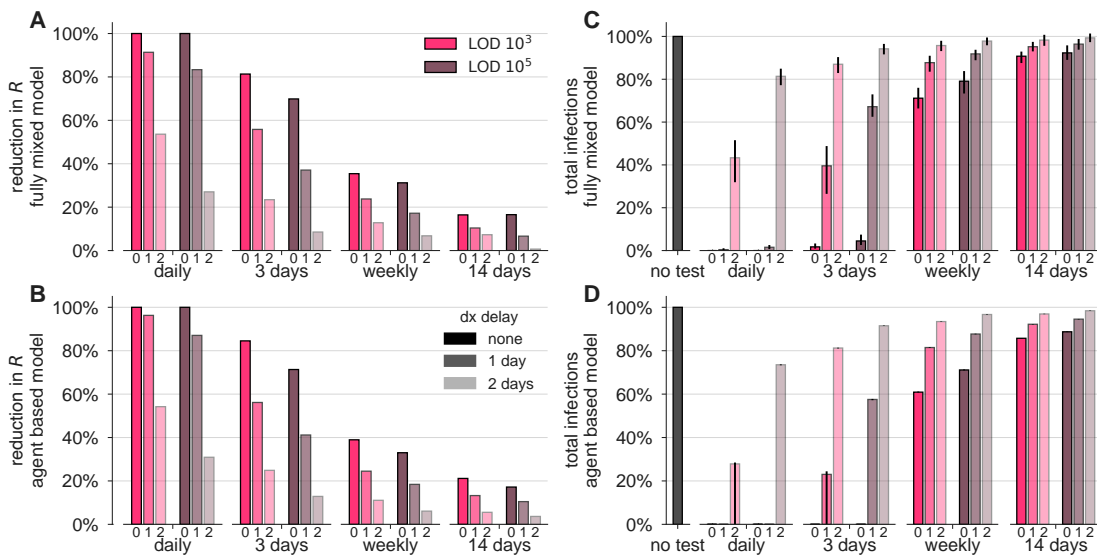


Figure S2: **Delays in reporting decrease the epidemiological impact of screening-driven isolation (proportional infectiousness).** This figure presents results from simulations which were identical to those shown in the main text Figure 5, but in which infectiousness was assumed to be *directly proportional* to viral load. Compare with threshold (binary) infectiousness in Fig. S3 and log-proportional infectiousness in Fig 5. See Methods.

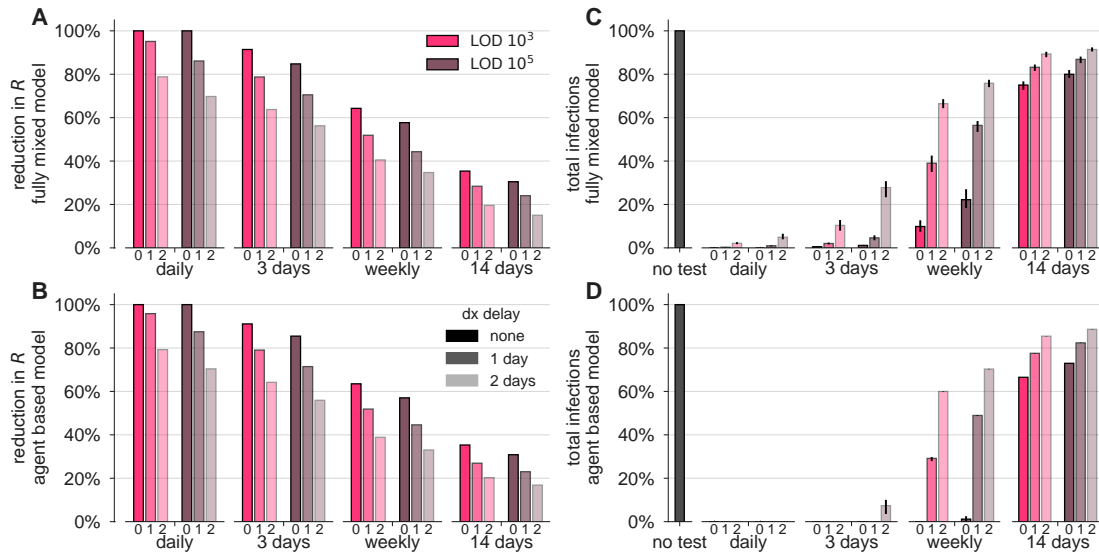


Figure S3: **Delays in reporting decrease the epidemiological impact of screening-driven isolation (threshold infectiousness).** This figure presents results from simulations which were identical to those shown in the main text Figure 5, but in which infectiousness was assumed to be *binary*, i.e. no infectiousness below  $10^6$  and equal infectiousness for any viral load above  $10^6$ . Compare with proportional infectiousness in Fig. S2 and log-proportional infectiousness in Fig 5. See Methods.

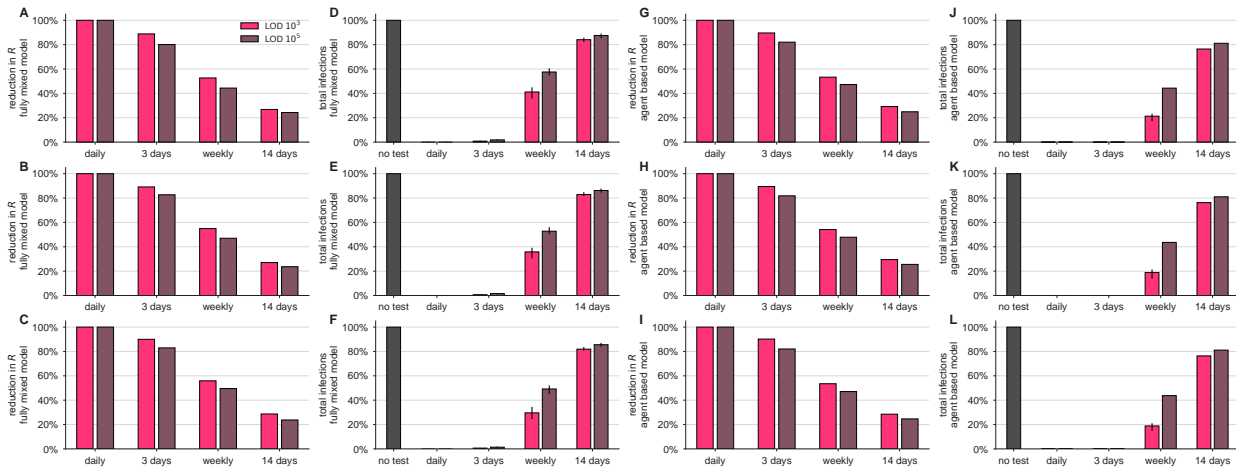


Figure S4: **Robustness of screening regimen effectiveness to the fraction of individuals who are symptomatic.** (A-F) Results from fully-mixed simulations and (G-L) agent-based simulations using an asymptomatic rate of 50% (top row), 65% (middle row; identical to main text Fig 2), and 80% (bottom row).

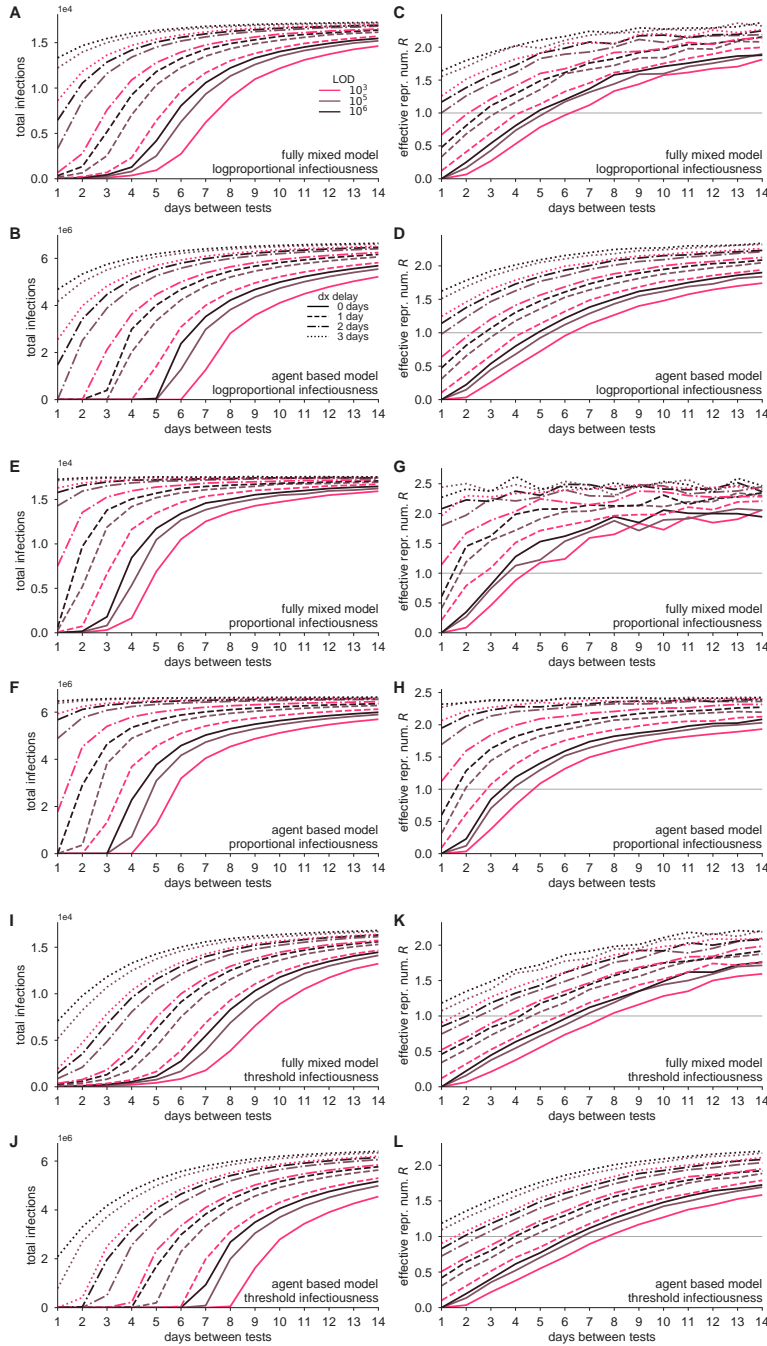


Figure S5: **Epidemiological model outcomes for various test LODs, frequencies, infectiousness functions, and with reporting delays.** The fully mixed model and agent based model were simulated (Methods) with various test frequencies, ranging from daily to once every 14 days, with LODs of  $10^3$ ,  $10^5$ , and  $10^6$ , and with delays of 0, 1, 2, or 3 days, for log-proportional, proportional, and threshold infectiousness functions (see Methods). Legends in panels A and B indicate LODs and delays, and in-plot annotations describe various conditions. Modeling results show mean outcomes from 50 independent simulations at each point, expressed as total infections and effective reproductive number  $R$ , from a baseline of  $R_0 = 2.5$ . For the fully mixed model, only secondary infections are shown, excluding imported infections. Total population sizes were  $N = 2 \times 10^4$  for the fully mixed model and  $8.4 \times 10^6$  for the agent based model. A horizontal line indicates  $R = 1$  for reference.

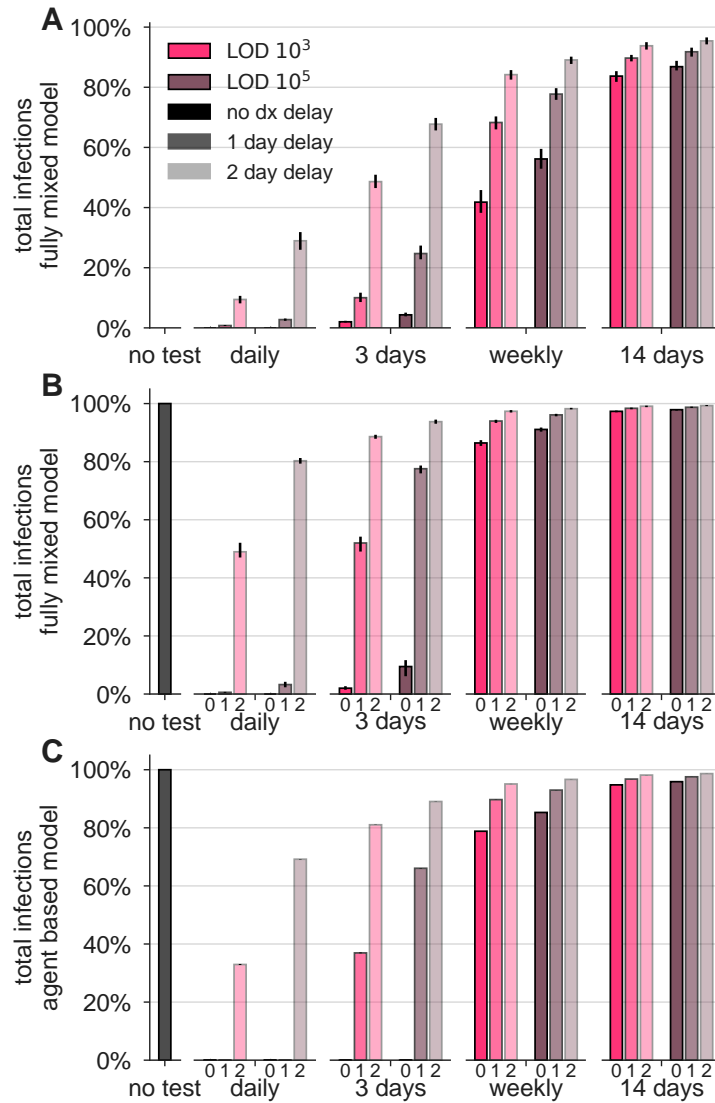


Figure S6: **Robustness of screening regimen effectiveness to epidemiological model parameters.** (A) Results from the fully-mixed simulation with a tripled rate of external infection, i.e.  $3/N$  per person per day. (B) Results from the fully mixed simulation with  $R_0$  doubled, i.e.  $R_0 = 5$ . (C) Results from the agent-based simulation with  $R_0$  doubled, i.e.  $R_0 = 5$ .

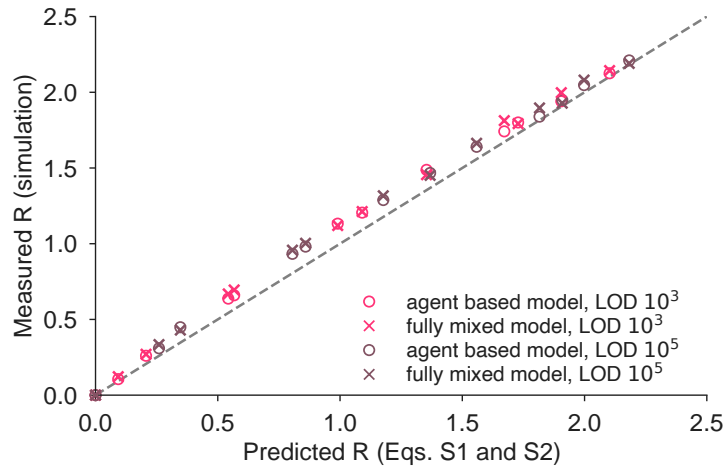


Figure S7: **Predicted and simulated impact of repeated population screening on the reproductive number  $R$ .** Mathematical predictions of the reproductive number  $R$  (see Equation (S1) in Supplemental Text S1) are scattered against their empirical measurements for the simulations shown in the main text (Figs. 2 and 5). Pearson's  $r = 0.998$ ,  $p < 10^{-6}$ .

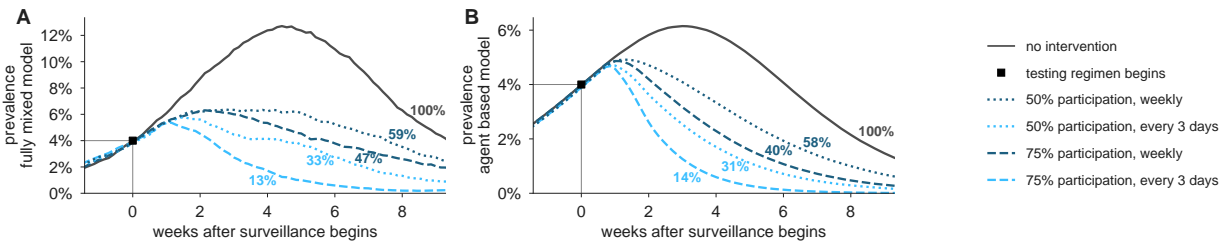


Figure S8: **Repeated population screening suppresses an ongoing epidemic using a test with LOD  $10^6$ .** Widespread testing and isolation of infected individuals drives prevalence downward for both (A) the fully-mixed compartmental model and (B) the agent based model. Time-series of prevalence, measured as the total number of infectious individuals, are shown for no intervention (solid) and population screening scenarios (various dashed; see legend) for individual stochastic simulations. Screening began only when prevalence reached 4% (box), and time series are shifted such that testing begins at  $t = 0$ . Scenarios show the impact of a test with LOD  $10^6$ , no delay in results, and with 10% of samples assumed to be incorrectly collected (and therefore negative) to reflect decreased sensitivity incurred at sample collection in a mass testing scenario. Annotations show total number of post-intervention infections, as a percentage of the no-intervention scenario, labeled as 100%. See Fig. 6 for identical simulations using a test with LOD  $10^5$ .

## Supplemental Text

### S1 Predicting the impact of repeated population screening testing on $R$

The impact of repeated population screening on the reproductive number can be estimated by considering the ratio of population infectiousness with a screening regimen to population infectiousness with no screening. However, note that the impact of a population screening policy may depend on two additional factors.

First, not all individuals may wish to participate in a testing program. Let the fraction of individuals who participate be given by  $\phi$ .

Second, a test may produce a false negative result *unrelated* to its limit of detection—for instance due to an improperly collected sample. Let  $se$  be the test sensitivity, in the particular sense of the probability of correctly diagnosing an individual as positive when that person’s viral load should, in principle, have provided a sufficiently high RNA concentration to be detectable.

Let  $f_0$  be the total infectiousness removed with no testing policy, i.e. due to symptom-driven self isolation. Let  $f_{\text{test}}(se)$  be the fraction of total infectiousness removed with a chosen testing policy, inclusive of symptom-driven self isolation, as well as the test sensitivity  $se$  introduced above.

Both  $f_0$  and  $f_{\text{test}}(se)$  can be estimated rapidly via Monte Carlo by drawing trajectories and applying a population screening regimen to them in which a fraction  $1 - se$  positive tests are discarded uniformly at random. In the main text, we found that estimating these values using 10,000 randomly drawn trajectories was sufficient to produce stable estimates.

Under the assumption of statistical independence between an individual’s participation or refusal, viral load, and  $se$ , we can approximate the reproductive number as

$$R \approx \left[ \phi \frac{1 - f_{\text{test}}(se)}{1 - f_0} + 1 - \phi \right] R_0, \quad (\text{S1})$$

which simply expresses a weighted combination of removed infectiousness via screening regimen participation and no test. Intuitively, note that if there is complete refusal to participate ( $\phi = 0$ ) or an entirely ineffective test ( $f_{\text{test}}(se) = f_0$ ), then  $R \approx R_0$ , as expected.