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Testing strategies to contain COVID-19 in migrant worker dormitories

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ABSTRACT

Introduction: COVID-19 transmission within overcrowded migrant worker dormitories is an ongoing global issue. Many countries have implemented extensive control measures to prevent the entire migrant worker population from becoming infected. Here, we explore case count outcomes when utilizing lockdown and testing under different testing measures and transmissibility settings.

Methods: We built a mathematical model which estimates transmission across 10 different blocks with 1000 individuals per block under different parameter combinations and testing conditions over the period of 1 month. We vary parameters including differences in block connectivity, underlying recovered proportions at the time of intervention, case importation rates and testing protocols using either PCR or rapid antigen testing.

Results: We estimate that a relatively transmissible environment with fortnightly PCR testing at a relatively low initial recovered proportion of 40%, low connectivity where 10% of contacts occurred outside of the infected individuals' block and a high importation rate of $\frac{1}{100000}$ per day, results in an average of 39 (95%Interval: 9–121) new COVID-19 cases after one month of observation. Similar results were observed for weekly rapid antigen testing at 33 (9–95) cases.

Interpretation: Our findings support the need for either fortnightly PCR testing or weekly rapid antigen testing in high population density environments such as migrant worker dormitories. Repeated mass testing is highly effective, preventing localized site outbreaks and reducing the need for site wide lockdowns or other extensive social distancing measures within and outside of dormitories.

Introduction

An estimated 46 million migrants from Asia-Pacific countries in 2019 migrated to other countries in the region (United Nations 2021), many of whom face heightened risks of overcrowded living conditions, barriers to health-seeking behaviors and high exposure to respiratory infections due to dense living and working conditions (Chen et al., 2016; Fonseca Lima et al., 2016; Kolavic-Gray et al., 2002; Burki, 2013; Viboud et al., 2004). Particularly within urban settings, migrant worker crowding is common due to high land prices and demand for employment (World Health Organisation, 2022), and worker dormitories are particularly susceptible (Lee et al., 2014) due to high staff turnover and the continual arrival of workers who may have acquired undetected infections in their country of origin.

Within large Asian cities such as Hong Kong, Seoul and Singapore, the majority of lower-income migrants are employed in the manual sector such as construction, manufacturing or domestic work (Koh, 2020). Accommodation is consequently typically designed to be affordable with multiple small-sized units. The structure, with shared facilities and close living environment, enhances mixing among workers. As a result, the spread of virus-laden aerosol particles may occur at key points within the buildings such as lifts, lobbies, shared facilities and stairwells (Jayaweera et al., 2020), or between units if ventilation is insufficient. Due to the densely populated site, by the time cases of a respiratory disease are identified, it is likely that multiple individuals have already been infected, complicating control and potentially requiring multiple blocks or large areas to be isolated. When workers from different dormitories are interacting at worksites, transmission can additionally begin at uninfected susceptible dormitories, further facilitating a spatially widespread outbreak.

Consequently, during national outbreaks, these locales are expected to require intensive control measures to prevent spread. This has been especially noteworthy during the COVID-19 pandemic. In Kuwait, targeted intervention measures of zonal quarantining in Alijeeb and Mahboula, where the highest density of migrant workers are located, was successful in temporarily lowering spread and clustering events (Alkhamis et al., 2020).

In Thailand, outbreaks have been recorded among migrants in overcrowded dormitories in the Samut Sakhon province, warranting large scale testing and active case finding efforts (Rajatanavin et al., 2021). In Singapore, migrant worker dormitories became epicenters of infection with over half having been infected by the end of the first wave in August 2020 (Tan et al., 2021), constituting over 90% of Singapore's cases. Extensive control measures were implemented including dormitory lockdown, contact tracing, environmental infection control and testing of the entire worker population (Ministry of Health, Singapore, 2022). As of August 2021, routine rostered testing of dormitory-inhabiting foreign workers has been ongoing for a year, bringing logistical and financial challenges due to the intense organizational efforts required to systematically test all 320,000 such workers residing in Singapore (Ministry of Health, Singapore, 2022). With the arrival of the more transmissible

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B.1.617.2 strain (the delta variant) across Southeast Asia (Planas et al., 2021), testing remains critical as a forefront measure for epidemic control (Vandenberg et al., 2021) to ensure future outbreaks do not occur within these vulnerable locations. Alongside testing, migrant worker site lockdown, which is the localized isolation of blocks upon the detection of cases, also effectively prevents ongoing spread (Mahase, 2020).

In the event of a new outbreak in migrant worker dormitories, several unknowns remain for policymakers in assessing the efficacy of testing and block lockdown measures, in terms of the effects of (1) block connectivity, (2) underlying recovered proportion at the time of intervention, (3) differing case importation rates and (4) regularity of testing with either fortnightly more sensitive PCR or weekly less sensitive rapid antigen testing (Pekosz et al., 2021; Xiao et al., 2020). To address these questions we have built a mathematical model that explores these questions and thereby seeks to establish strategies that mitigate outbreak size in migrant worker dormitories (Fig. 2).

Methods

Residual infectivity

For an infected individual who incubates θ days after infection, the expected number of secondary cases that can arise t days after infection is given by

$$I_t(\theta) = R_0 \cdot \int_t^\infty \tau(x-\theta) dx, \ x \in (-\infty, \ \infty)$$

where R_0 is the basic reproductive number and τ the infectiousness probability density function (PDF).

The average number of secondary cases that can arise from an infected individual after day *t*, defined as the residual infectivity $\mathbb{E}[I_t]$, is expressed as I_t weighted by *p*, the incubation probability mass function (PMF), and defined to be

$$\mathbb{E}\big[I_t\big] = \sum_{\theta} I_t(\theta) p(\theta).$$

Single test residual infectivity

An infected individual who fully incubates on day θ and takes a SARS-CoV-2 test on day *t*, will test positive with a probability of $\sigma(t - \theta)$, where $\sigma(x)$ is the diagnostic test sensitivity of SARS-CoV-2 *x* days after onset. Two types of diagnostic tests were considered for this study, namely the polymerase chain reaction (PCR) and rapid antigen tests. For all simulations, an infectiousness probability density function and incubation PMF were used (He et al., 2020), as well as a PCR and rapid antigen test sensitivity profile (Pekosz et al., 2021; Xiao et al., 2020). When an infected individual tests positive, they are assumed to be removed from the population through strict isolation in a healthcare facility (Dickens et al., 2020) and therefore to have zero residual infectivity. Therefore, the residual infectivity of an individual for a single testing event done on day *t* is

$$\rho_r(t) = \mathbb{E}[I_t] - \mathbb{E}[I_t\sigma_t] = \sum_{\theta} I_t(\theta)p(\theta) - \sum_{\theta} I_t(\theta)\sigma(t-\theta)p(\theta).$$

Multi-test residual infectivity

Multiple tests may be conducted on individuals over time. If two tests are carried out at time t_0 and t_1 , where $t_0 < t_1$, the combined residual infectivity at t_0 and t_1 is

$$\rho_r(t_0, t_1) = \mathbb{E}\Big[I_{t_0}\Big] - \mathbb{E}\Big[I_{t_0}\sigma_{t_0}\Big] - \mathbb{E}\Big[I_{t_1}\Big(1 - \sigma_{t_0}\Big)\sigma_{t_1}\Big]$$

which is the reduced number of secondary infections occurring from the first test and subsequent test.

This can be generalised for *n* tests, conducted on the days $t_0, ..., t_{n-1}$ where $t_0 < ... < t_{n-1}$. The residual infectivity can be reduced with each subsequent test, which is determined by I_{t_k} , *p* and σ , where $k \in$

 $\{0, ..., n - 1\}$ is an index for the test number and $j \in \{0, ..., k - 1\}$ an index for the number of test failures. This can be expressed as

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$$\rho_r(t_0, \cdots, t_n - 1) = \mathbb{E}\Big[I_{t_0}\Big] - \mathbb{E}\left[\sum_{k=0}^{n-1} I_{t_k} \prod_{j=0}^{k-1} \left(1 - \sigma_{t_j}\right) \sigma_{t_k}\right]$$

where an individual's infectivity deteriorates over time and can never be less than zero, hence $\rho > 0$, since, for any $\theta \ge 0$,

$$\begin{split} &\sum_{k=0}^{n-1} I_{t_k}(\theta) \prod_{j=0}^{k-1} \left(1 - \sigma_{t_j}(\theta)\right) \sigma_{t_k}(\theta) < I_{t_0}(\theta) \sum_{k=0}^{n-1} \prod_{j=0}^{k-1} \left(1 - \sigma_{t_j}(\theta)\right) \sigma_{t_k}(\theta) \\ < I_{t_0}(\theta)n \\ \text{where} \\ &\sum_{k=0}^{n-1} \prod_{j=0}^{k-1} \left(1 - \sigma_{t_j}(\theta)\right) \sigma_{t_k}(\theta) < 1. \end{split}$$

Symptomatic profile

We assumed that symptomatic individuals were immediately isolated from the population at a hospital, quarantine facility or *fangcang* hospital (Dickens et al., 2020) to seek medical attention upon symptom onset, which occurs independent of testing. Symptomatic individuals, who make up a proportion, ψ of cases, were therefore assumed to be non-infectious after the onset of symptoms. Therefore, for *n* tests, the residual infectivity for the symptomatic cases is

$$\rho_r^*(t_0,\cdots,t_n-1) = \mathbb{E}\Big[I_{t_0}^*\Big] - \mathbb{E}\left[\sum_{k=0}^{n-1} I_{t_k}^*\prod_{j=0}^{k-1} \left(1-\sigma_{t_j}\right)\sigma_{t_k}\right]$$

where

$$I_t^*(\theta) = \begin{cases} R_0 \cdot \int_t^{\theta} \tau(x-\theta) \, dx & t < \theta \\ 0 & t \ge \theta. \end{cases}$$

Therefore, the sum of the symptomatic and the asymptomatic proportion is the overall residual infectivity, namely

$$\rho_R(t_0, \cdots, t_{n-1}) = (1 - \psi)\rho_r(t_0, \cdots, t_{n-1}) + \psi \rho_r^*(t_0, \cdots, t_n - 1).$$

Infectivity

Given that *n* tests were conducted on days $\{t_0, \dots, t_{n-1}\}$, the infectivity of an individual t_0 days after infection, denoted as ρ_i , is the sum of the number of secondary infections up to t_0 and the residual infectivity $\rho_i(t_0, \dots, t_{n-1})$,

$$\begin{split} \rho_i(t_0, \ \cdots, \ t_{n-1}) &= \left(R_0 - \mathbb{E}\Big[I_{t_0}\Big]\right) + \rho_r(t_0, \cdots, t_{n-1}) \\ &= R_0 - \mathbb{E}\left[\sum_{k=0}^{n-1} I_{t_k} \prod_{j=0}^{k-1} \left(1 - \sigma_{t_j}\right) \sigma_{t_k}\right]. \end{split}$$

By making similar modifications to the symptomatic residual infectivity ρ_r^* , the overall infectivity that includes the symptomatic proportion can be expressed as

$$\rho(t_0, \cdots, t_{n-1}) = (1 - \psi)\rho_i(t_0, \cdots, t_{n-1}) + \psi \rho_i^*(t_0, \cdots, t_{n-1})$$

Simulation in close quarters

We generate the outbreaks in our model using the infectiousness PDF (τ) and incubation PMF (p). As $\vartheta_{j,h}$ is the number of individuals infected on day j with symptom onset on day h, the number of individuals with symptom onset on day h is, therefore,

$$\vartheta_h = \sum_{j=0}^{h-1} \vartheta_{j,h}.$$

If we denote X_h as the number of secondary cases generated from ϑ_h , D_h is a X_h -tuple of independent draws from the infectiousness PDF

(τ), which is a set of new infection occurrences for every $d_i \in D_h$ where a new infection will occur d_i days after day h. Thus, X_h and D_h can be expressed as

$$X_h \sim \sum_{j=0}^{n-1} \text{NegBin} \left(\rho(T-j) s_h \vartheta_{j,h}, k \right)$$

and

$$D_h \sim \tau^{X_h}$$

where *T* is the collection of test days and s_h is the susceptible proportion on day *h* and *k* is the overdispersion parameter for the negative binomial distribution set at 0.1 (Endo et al., 2020) with mean $\rho(T - j)s_h \vartheta_{j,h}$.

At the start of each simulation, we assume that the dormitory is disease free. Therefore, in the initial phase of a simulation, the dormitory residents can only be infected when they are exposed to an external source of infection, for example coming into contact with infected coworkers during their work routine outside the dormitory or with an infected individual within the general population. This external infection of the dormitory residents was represented in our model as the number of imported infections, im_h , is given by a Poisson distribution with mean rS_h ,

$$im_h \sim \text{Pois}(rS_h)$$

where *r* is the importation rate and S_h is the number of susceptible individuals on day *h*. The total number of infections on day *t*, t_t , is the sum of the infections that have occurred within the dormitory and imported infections combined,

$$u_{t} = \sum_{h=0}^{t-1} \left| \left\{ d_{i} : d_{i} \in D_{h}, d_{i} + h = t \right\} \right| + im_{t}$$

where |A| denotes the size of set *A*. We obtain the timings to symptom onset for infected individuals on day *h* by drawing ι_h independent draws from the incubation PMF *p*,

 $E_h \sim p^{(\iota_h)}.$

Hence the number of individuals with symptom onset on day t, ϑ_t , is the sum of individuals infected on day h where $h \in \{1, \dots, t-1\}$,

$$\vartheta_t = \sum_{h=0}^{t-1} \left| \left\{ e_i : e_i \in E_h, e_i + h = t \right\} \right| = \sum_{h=0}^{t-1} \vartheta_{h,t}$$

Interaction between blocks

To model the interaction between blocks, we simulated transmission between migrant workers in M connected blocks. The number of infections is assumed to be proportional to the number of interactions or contacts between blocks. An adjacency matrix, A, representing the interactions between individuals residing in separate blocks, where $a_{ij} \in A$ is the connectivity between block i and j, was used. A value of $a_{ij} = 0$ represents no connectivity between i and j, and $a_{ij} = 1$ representing maximum connectivity, and $\sum a_{ij} = 1$.

We denote the number of individuals in block *m* with symptom onset on day *h* as $\vartheta_{h,m}$ where new infections arise in block *m* on day *h* as

$$X_{h,m} \sim \sum_{j=0}^{h-1} \operatorname{NegBin} \left(\rho(T-j) s_{h,m} \vartheta_{j,h,m}, k \right)$$

and $D_{h,m} \sim \tau^{X_{h,m}}$, the $X_{h,m}$ -tuple of the new $X_{h,m}$ infection timings from block *m* on day *h*. The next step was to determine the distribution of the $X_{h,m}$ infections among the *M* blocks.

First, a $X_{h,m}$ -tuple $U_h = (u_1, ..., u_{h,m}) \in [0, 1]^{X_{h,m}}$ was formed, with each element of U_h drawn from U(0, 1). Using the adjacency matrix A, the characteristic function χ_i determined if the *i*th coordinate of a $X_{h,m}$ -tuple is included in block m',

$$\chi_i(m,m') = \begin{cases} 1, & \sum_{j=1}^{m'} a_{mj} \le u_i < \sum_{k=1}^{m'+1} a_{mj}, \ a_{mj} \in A, \ u_i \in U_h \\ 0, & \text{otherwise.} \end{cases}$$

Table 1

Parameter values used in the simulations.

Parameters	Parameter values
Number of blocks	5, 10, 20
Connectivity	0.0-1.0
Recovered proportion	Low (40%), high (70%)
SARS-CoV-2 Test used	PCR (fortnightly), Rapid Antigen (weekly)
Importation rates	$\frac{1}{100,000}$ per day to $\frac{1}{10,000}$ per day
R	6, 8, 10

Hence $D_{h,m\to m'}$ is the sub-tuple of $D_{h,m}$, which are infections from block *m* to block *m'*,

$$D_{h, m \to m'} = \mathbf{X}_{m, m'} \cdot D_{h,}$$

and

$$\chi_{m,m'} = \Big(\chi_1(m,m'),\ldots,\chi_{X_{h,m}}(m,m')\Big).$$

The number of new infections in block m on day t is therefore the sum of the infectious potential from t days before, across the M blocks, and the natural importation rate

$$u_{t,m} = \sum_{m'=1}^{M} \sum_{h=0}^{t-1} \left| \left\{ d_i : d_i \in D_{h,m' \to m}, d_i + h = t \right\} \right| + im_{t,m}.$$

We assumed block lockdown where upon the detection of a symptomatic case, the identified block that the case resides in goes immediately into lockdown, with comprehensive contact tracing and quarantining of contacts, ensuring no further transmission occurs within or outside the block. Furthermore, we assumed that the symptomatic individuals would report their symptoms, upon symptom onset, to the dormitory management. Infected individuals who were identified were removed and stayed in specific isolation units within the block, and therefore do not go on to infect others within the same block.

Simulations

Outbreak simulations in close quarters were carried out assuming a fixed population of n = 10000, uniformly distributed across block configurations of 5, 10, 20 blocks, with corresponding 2000, 1000, 500 individuals per block (Table 1), across a range of connectivity parameters (adjacency matrices), existing SARS-CoV-2 recovered proportions pre-intervention and importation rates. The recovered proportion refers to the proportion of dormitory residents that have recently recovered from SARS-CoV-2, and are assumed to be temporally immune to the virus and its variants within the one-month time scope of this study, and are further uniformly distributed across the different blocks. Two testing regimes, fortnightly PCR and weekly rapid antigen, were explored. R_0 of 6, 8 and 10 were used to simulate a close contact environment within the living quarters and a highly transmissible variant such as delta or omicron. We performed 1000 simulations for each parameter configuration.

Results

We present changes in infectivity at an individual level as a proportion of the R_0 . In a single PCR testing event on the same day of infection, assuming that an average individual is infected at *t* and fully asymptomatic, infectivity is $0.73R_0$ (Fig. 1a). When the test was delayed to four days post infection, infectivity decreased to a minimum of $0.56R_0$ due to maximal test sensitivity at this time. When testing is conducted after four days, infectivity increases to $0.9R_0$ at day 12, as these individuals can infect other susceptible individuals before being tested and identified. With a 60% symptomatic rate, respective infectivity is $0.5R_0$ with testing on the same day of infection, $0.44R_0$ at four days post and $0.62R_0$ at day 12. For a weekly testing scenario using the rapid antigen test (Fig. 1b), when tests are performed on the day of infection and again



Fig. 1. Infectivity relative to initial test day and symptomatic proportion. (a) Single test on day t at the time of infection. (b) Two tests on day t at the time of infection and t + 7 one week later.



Fig. 2. The number of new infections after one month according to the initial importation rate per hundred thousand per day and connectivity for a low recovered proportion (40%; a–c) and high recovered proportion (70%; d–f).

one week later, infectivity is $0.50R_0$. This decreased to a minimum infectivity of $0.45R_0$ when an individual is initially tested four days after infection, subsequently increasing back to R_0 with further delays in testing. At a 60% symptomatic rate, respective infectivity is $0.39R_0$ on the day of infection, with a minimum of $0.36R_0$ 2 days post increasing up to $0.62R_0$ at 12 days.

For close quarter outbreak simulations, we explored the results for each parameter configuration. We present the results for a dormitory configuration of 10 blocks and R_0 of 8. Summary of the simulations of the other parameter configurations can be found in the Supplementary Material. Assuming fortnightly PCR testing at a relatively low initial recovered proportion (40%) and low connectivity where 10% of contacts occurred outside of the infected individuals' block with a uniform distribution, when a low importation rate of $\frac{1}{100\ 000}$ is applied per day, the number of new COVID-19 infections observed was on average 7 after one month of observation (95% Interval [95%I]: 0–47). When the importation rate was increased to $\frac{1}{20\ 000}$ per day, the number of new

infections increased correspondingly to 25 (4–94). At the highest importation rate of $\frac{1}{10\,000}$ per day, the number of new infections was 39 (9–121). When weekly rapid antigen testing is utilised, we observed new infections of 5 (0–35), 22 (4–81) and 33 (9–95), for importation rates of $\frac{1}{10\,000}$, $\frac{1}{20\,000}$ and $\frac{1}{10\,000}$ per day respectively. Despite having lower sensitivity, the higher frequency of testing reduces the number of new infections observed across different importation rates. The differences are relatively minor, which is also observed at a higher recovered proportion of 70% where for fortnightly PCR testing, the number of new infections observed after one month is 2 (0–14), 9 (1–37) and 17 (4–52), and the equivalent for weekly rapid antigen testing is 2 (0–9), 8 (1–30) and 14 (4–46).

When fortnightly PCR testing is implemented at a low recovered proportion and low connectivity with a low importation rate of $\frac{1}{100\ 000}$ per day, the median time interval between the block lockdowns was 5.0 days (0.2–19.0 days). Lockdown time intervals decreased for both mid (0.5) to high connectivity (0.9) at 3.0 days (0–18.0 days) and 2.0 days

(0-17.0 days). The proportion of blocks that underwent lockdown were 0.2 (0-0.7), 0.3 (0-1.0) and 0.3 (0-1.0), for low, mid and high connectivity respectively.

With fortnightly PCR testing at a low recovered proportion, low connectivity and a higher importation rate of $\frac{1}{10\ 000}$ per day, the lockdown time interval was 2.6 days (1.1-4.6 days), which decreased to 2.0 days (0.6-5 days) and 1.7 days (0.3-4.7 days), for mid and high connectivity. We also note that by increasing the importation rate, more infections were observed at low connectivity than high. For example, at a higher importation rate of $\frac{1}{10\ 000}$ per day, 39 (9–121) infections occur at low connectivity (10%), as compared to the lower infection counts of 27 (7–86) for high connectivity (90%). In both settings, all the blocks went into lockdown, but at different rates, with the low connectivity (10%) setting having block lockdowns at intervals of 2.6 days versus 1.7 days at high connectivity (90%). Differences in connectivity contribute to the initial spread of infection where lower levels allow for greater mixing over the long-term and more cases. This in contrast to higher connectivity which substantially shortens lockdown intervals, causing the rapid lockdown of multiple blocks which can successfully mitigate further spread.

Similar observations and trends were seen for rapid antigen testing although with smaller differences when compared to PCR testing. The number of infections under rapid antigen testing was 33 (9–95) with the time between lockdowns at 2.7 days (1.3–4.8 days) in the low connectivity setting. In contrast, at the high connectivity setting, shorter lockdown intervals of 1.8 days (0.4–5days) were observed and fewer total infections recorded where 24 (7–69) were infected at the end of one month.

Discussion

Our findings estimate the impact of regular COVID-19 testing for migrant populations living in dormitory-type settings. In a population of 10,000 workers, similar to the larger dormitories in Singapore, the expected number of infections ranges from 0-121 per month, and the proportion of blocks undergoing lockdown within one month 20%-100%, depending on the importation rate and connectivity between blocks. The worst-case scenario of a low connected set of residential blocks (10%) and high importation rate $(\frac{1}{10\ 000})$ at a low recovered proportion (40%) incurs 39 (9-121) cases per month in comparison to 2 (0-9) for the inverse scenario. Based on recent observations (Yi et al., 2020), migrant worker dormitories are highly connected and will experience high infection importation rates due to substantial mixing with the local community when not under lockdown. Therefore, estimates of 27 (7-86) for a highly connected area with high importation across a dormitory recovered proportion of 40–70% are expected. These findings indicate the protective effect of mass, frequent testing, which has been illustrated elsewhere to be effective (Mina et al., 2020; Larremore et al., 2021). This is further supported by the fact that since the implementation of fortnightly testing for all dormitory residents, there have been few sizable outbreaks. It also suggests that, as countries reopen, relaxing other social restrictions may be feasible whilst continuing regular testing to maintain low infection spread rates, especially within crowded accommodation sites.

These estimates support ongoing global concerns on migrant worker welfare (World Health Organisation, 2021; Yip et al., 2021), which include the high population density of their accommodation (United Nations, 2021), reduced healthcare seeking behavior (Lee et al., 2014; Goh et al., 2020) and mental wellbeing (Chan and Kuan, 2020). During the COVID-19 pandemic, these concerns have only increased due to occasionally harsh lockdown measures (Yi et al., 2020). Crowding at sites such as worker dormitories is known to increase the risk of respiratory infection through the passing of droplets or aerosols in illventilated settings, or contact between shared surfaces (Cardoso et al., 2004). The sharing of enclosed spaces, high room occupancy and large number of interaction events in common areas are known risk factors (Killingley et al., 2012; Lakdawala and Subbarao, 2012) where migrant workers are often exposed to all of these. Such high connectivity levels will likely result in the rapid implementation of lockdown to prevent a large areawide outbreak in contrast to lower connectivity levels which results in longer delays to lockdown and a slower ramp up in case load.

Efforts are thus underway in Singapore to improve migrant worker conditions by reducing their housing's conductivity for respiratory infection spread (Koh, 2020; Yip et al., 2021) and importantly, the need for repeated and long lockdown periods from case importation events. Under the low recovered proportion and expected high importation settings for new variants such as delta, which is more transmissible and for which an increased risk of complications has been observed (Sheikh et al., 2021), reducing crowding is likely to reduce outbreak spread and size. Similar observations have been made elsewhere. For COVID-19, cities in Brazil with high indices of household overcrowding have been associated with significantly higher incidence at an excess of 461 per 100,000, and the number of households with greater than 3 residents per room was observed to significantly increase risk (Villela, 2021). In China and Italy, the degree to which cases were compressed in terms of the peakedness of the localized epidemic was strongly shaped by population aggregation and heterogeneity down to 1 km (Chen et al., 2016) small spatial scales (Rader et al., 2020) Such temporal clustering of cases has also occurred among the 323,000 migrant workers in Singapore who reside within the 43 licensed, purpose-built dormitories, which accommodate an average of 10 workers per room (Yi et al., 2020).

The vaccination of migrant workers therefore remains a critical intervention. Although approximately 95% of migrant workers in Singapore have now been vaccinated with the mRNA vaccines BNT162b2 (Pfizer-BioNTech) or mRNA-1273 (Moderna) in Singapore, millions of workers elsewhere have yet to be vaccinated (Armocida et al., 2021; Han et al., 2021), and even when they are, the vaccines do not provide full immunity (Lopez Bernal et al., 2021). Depending on vaccination policies, these young migrant populations may be among the last to be vaccinated alongside children and be residing in high density conditions whilst waiting. The risk of outbreak will likely remain high in these settings, necessitating the need for interventions or infrastructure in place to immediately protect individuals living within such facilities from future respiratory outbreaks. In the longer term, this risk also calls for longer term measures to improve the living conditions within these sites.

Limitations of the analysis include the static assumption of R_0 , incubation period and testing times. No delays in result notification from testing time were also assumed, which is valid for rapid antigen tests which generate relatively immediate results, but the speed of PCR test turnaround will depend on laboratory infrastructure. We have however explored how the infectivity profile would change when there are test delays (Supplementary Material). Inter and intra block mixing, which was assumed to be constant across the time-period explored outside of block lockdown events, may not occur with wider movement restrictions in response to individual block outbreaks. Depending on the layout of high-density accommodation, homogeneous mixing across a site may not occur with localized gathering areas or more movement observed in high transit areas. Furthermore, block lockdown may not be fully isolating with non-compliance or contact transfer infections occurring from personnel working onsite. The frequencies of PCR and rapid antigen testing were only taken at two and one weekly intervals based on current test practices, supplies and processing times. Batch testing or the staggering of testing across time may result in more infections being missed. Such strategies could be assessed however through straightforward modifications to the model presented herein.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:.

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