Supporting Information

Nuño *et al.* 10.1073/pnas.0712014105

SI Text

Methods. The objective of this study was to evaluate a range of nonpharmaceutical interventions (NPIs) and their consequences in mitigating influenza pandemics of increasing severity. This supporting information is organized into four sections: (i) *Description of Nonpharmaceutical Interventions*; (*ii*) *Epidemic Models: Nonpharmaceutical Interventions*, which provides a detailed derivation of the model used to evaluate various NPIs discussed in the first section; (*iii*) *Stochastic Poisson Simulation of Disease Progression*, which provides details of the simulation method implemented in this study; and (*iv*) *Calculation of the Basic Reproduction Number,* which outlines the computation of the basic reproduction number ($\mathcal{R}_0$).

Description of Nonpharmaceutical Interventions. Before defining nonpharmaceutical intervention plans, it is necessary to define a baseline scenario. Our scenario assumed visitors maintained their 2-h (per week) visiting periods, staff reported for their 8-h (5-day) work shifts, and no transmission reduction measures were implemented by staff or visitors. This scenario assumed that visits to the facility did not depend on any individual's then-current infectious state, i.e., all individuals reported to work and visiting hours regardless of the infectious state (susceptible, exposed, infected, or recovered) of the staff member, visitor, or resident. We recognize that it is more realistic that a portion of infected and symptomatic individuals would likely be unwilling or physically unable to leave their homes, so the baseline scenario mimics a worst-of-usual possible situation.

The NPIs proposed in this study are control measures that can plausibly be implemented by resident care facility personnel during an influenza pandemic. The first level of control efforts (plan category 1–2) was aimed at reducing contacts that staff and visitors might have with potentially infected individuals outside a facility. Our model assumed that staff and visitors reduced their contacts with potentially infected individuals and thereby reduced their risk of transmission to residents by 50% ($\pi_i = 0.5$). These reductions were to be accomplished by the use of facemasks, gowns, or other protective clothing, and the adoption of social distancing measures in high-density public gatherings such as churches, schools, and theaters. This plan also assumed that staff further reduced their risk of infection during the time spent outside a facility, and covered anticipated absenteeism and reduced the risk of introducing the pandemic virus by 20% (one fewer reentry) by switching their working schedule from an 8-hr to a 12-h-per-day average time spent in a facility.

The second level of control measures (plan category 3–4) simulated in our study augmented the interventions described in category 1–2 by extending staff schedules to four-days-on/4-days-off-site periods. In addition, staff and visitors entering a facility were monitored for elevated temperature and a history was documented and signed. The effectiveness of monitoring was captured by the parameter p_i, where i indexed exposed, asymptomatic, and infected individuals. For exposed individuals, p_E described the probability that exposed but asymptomatic individuals became infectious either before or during work (staff) or visiting periods (visitors). The off-site 4-day portion of the augmented staff schedule included a period of isolation from the community in the company of the staff person's living group that began the evening of the second day off and included the full third and fourth day before reentering a facility (2.3 days of self-isolation). The length of the isolation period was chosen to reduce the probability of a virus introduction on reentry by 75%, and to increase the chances that monitoring would detect infected individuals when entering a facility. The probability that an asymptomatic individual would actually not be infected at the end of the self-isolation period was estimated as follows: The time interval in which an individual was off-site and out in the community was divided into 10 equal subintervals. The likelihood that an individual might be exposed to the pandemic virus was assumed to be equal in each subinterval. We estimated the probability that an individual who was exposed in each subinterval would be asymptomatic at the end of the self-isolation period as the product of the probability of having been infected in each subinterval multiplied by the probability that a person who had been exposed in that subinterval would still remain asymptomatic at the time the person was to report for work. We used crude data on infection with the A(H3N2) pandemic virus (R. Couch and P. Glezen, personal communication) to estimate the distribution of time to first symptoms. For self-isolation periods of 1.3, 2.3, and 3.3 days, this calculation produced estimates that a person with access to the community over the 2.7, 1.7, and 0.7 days preceding the self-isolation period, respectively, and who remained asymptomatic at the end of isolation period would reduce the likelihood of introduction of the pandemic virus by levels of 0.5, 0.75 and 0.86, respectively.

For asymptomatic and infected individuals, p_A and p_I, respectively, represented the probabilities that these individuals passed undetected through the monitoring process. Therefore, $p_I = 0.1$ implied a 10% chance that an infected and infectious individual reported to work and entered undetected by the monitoring process. Asymptomatic individuals represented the highest potential risk to a facility because symptoms would never be apparent and monitoring efforts would be ineffective ($p_A = 1$).

The third level of control measures addressed the scenario of a severe pandemic (plan category 5). This plan assumed complete visitor restrictions and staff social distancing transmission control measures that reduced the risk of transmission to residents by 95%, and self-isolation periods of 3.3 days in a 4-day-on/-day-off staff shift implementation. The simulated implementation of these control measures effectively prevented introduction of a pandemic virus into a facility, but it also placed strong constraints on visitors and staff.

Epidemic Models: Nonpharmaceutical Interventions. We used an extension of a Susceptible–Exposed–Infected–Recovered (SEIR) model within a stochastic framework to study the dynamics of a pandemic virus introduction and its propagation among individuals in a resident care facility that is part of a larger community. We used the average size of nursing home populations in the United States (200 residents), an effective daily staff size of 83 to accommodate reports of 3 h of daily staff contact time with each patient, and a coterie of 40 visitors (only one in five residents are reported to receive visitors). In the absence of a vaccine (assumed to be unavailable early in a pandemic), individuals left the susceptible class only when exposed to the pandemic virus. For seasonal influenza, it is not uncommon for individuals to become infected with the circulating influenza virus and to remain asymptomatic not only for a short period after viral shedding (and therefore infectiousness) has begun, but throughout the course of the infection. However, for pandemic viruses, experience with the A(H3N2) viruses responsible for the last pandemic suggests the following:

1. There will be few, if any, asymptomatic carriers.

2. The time between the onset of infectiousness and the appearance of symptoms will be very short or nonexistent (P. Glezen, R. Couch, and R. Belshe, personal communications).
3. In the event that antiviral agents are available and deployed, it is possible that, in individuals for whom these agents are only partially effective, an asymptomatic (but still infectious) condition could exist.

With the above in mind, we modeled the total population of individuals inside (N_{in}) and outside (N_{out}) a facility by dividing these populations into subpopulations according to their current epidemiological state: Susceptible (S_i), Exposed (E_i), Asymptomatic (A_i), Infected (I_i), Recovered (R_i), and disease-induced Dead (D). The index i was used to denote the resident (R), visitor (VC and VF), staff (SC and SF), and community (C) populations. Visitors and staff were indexed according to their location in a facility (VF, SF) or the community (VC, SC).

Resident Model. Susceptible residents (S_R) acquired infection with a force of infection, λ_{in}, on contact with symptomatic (infectious) individuals ($I_R + I_{VF} + I_{SF}$) or asymptomatic (assumed less infectious) individuals ($A_R + A_{VF} + A_{SF}$) circulating within the facility. We assumed that residents interacted directly with visitors (VF) and staff (SF) during the time spent in a facility. The force of infection for residents in a facility was given by $\lambda_{\text{in}} = [\Sigma_{i=R,SF,VF}\beta_i(\pi_i I_i + \rho_i \eta i A_i]/N_{\text{in}}$, where η_i $(0 < \eta_i < 1)$ accounted for the relative infectiousness of asymptomatic individuals. Similarly, π_i $(0 < \pi_i < 1)$ and ρ_i $(\pi_i < \rho_i < 1)$ represented the reduction in disease transmission produced by the control measures when applied to infected and asymptomatic persons, respectively. A fraction, m, of exposed residents (E_R) progressed to symptomatic infection (I_R) at a rate ϕ_R, whereas the remaining fraction, $1 - m$ (asymptomatic but potentially infectious) moved into A_R at the same rate. Infectious and symptomatic residents recovered at a rate γ_I or succumbed to disease at a rate δ_I. Mortality rate was adjusted according to the case fatality proportion (CFP) such that $\delta_I = ((\text{CFP}/(1 - \text{CFP})))\gamma_I$, where $1/\gamma_I$ was the average time spent infectious before succumbing to disease. Therefore, mortality for each scenario is simply the total number of cases multiplied by the value of the case fatality proportion. Asymptomatic residents recovered at a rate γ_A. Because the time scale of a pandemic is small compared with that of human demographics, we excluded natural mortality but included disease-induced mortality. Based on these assumptions, the resident model was given by the following system of differential equations:

$$\begin{aligned}
\dot{S}_R &= -\lambda_{in} S_R \\
\dot{E}_R &= \lambda_{in} S_R - (m\phi_R + (1-m)\phi_R)E_R \\
\dot{A}_R &= (1-m)\phi_R E_R - \gamma_A A_R \\
\dot{I}_R &= m\phi_R E_R - (\gamma_I + \delta_I) I_R \\
\dot{R}_R &= \gamma_A A_R + \gamma_I I_R \\
\dot{D}_R &= \delta_I I_R
\end{aligned} \qquad [1]$$

Visitor and Staff Model. We modeled the interaction of visitors and staff with residents in a facility and community individuals by dividing these populations into epidemiological classes similar to those assumed in the resident model. Susceptible individuals (S_i) became exposed (E_i) at a rate β_i, a fraction, m, of these individuals progressed to the infectious class (I_i) at a rate ϕ_i. The remaining fraction, $1 - m$, continued into the asymptomatic (A_i) class. Recovery rates were defined to be the same as in the resident model. The model was indexed by i, where $i = VC$, VF, SC, SF denoted visitors and staff inside and outside (in the community) a facility. We expressed the probability that persons who might be infectious or become so while on-site would be undetected by the monitoring process among exposed, asymptomatic, and infected individuals circulating between the community and a facility by p_E, p_A, and p_I, respectively. For simplicity of computation, we assumed that the period that a staff member/visitor spent in the residential facility/community was exponentially distributed, and therefore, that the average time that staff and visitors spent between changes in location was denoted by $1/\xi_i$ (see supporting information (SI) Table S3). The equations describing the dynamics of visitors in the community and in a facility follow:

$$\begin{aligned}
\dot{S}_{VC} &= \xi_{VF} S_{VF} - (\lambda_{out} + \xi_{VC}) S_{VC} \\
\dot{E}_{VC} &= \xi_{VF} E_{VF} + \lambda_{out} S_{VC} - [p_E \xi_{VC} + m(1-p_E)\phi_{VC} \\
&\quad + (1-m)(1-p_E)\phi_{VC}]E_{VC} \\
\dot{A}_{VC} &= \xi_{VF} A_{VF} + (1-m)(1-p_E)\phi_{VC} E_{VC} \\
&\quad - [\gamma_A(1-p_A) + p_A \xi_{VC}]A_{VC} \\
\dot{I}_{VC} &= \xi_{VF} I_{VF} + m(1-p_E)\phi_{VC} E_{VC} - (\gamma_I + p_I \xi_{VC}) I_{VC} \\
\dot{R}_{VC} &= \xi_{VF} R_{VF} + \gamma_I I_{VC} + \gamma_A (1-p_A) A_{VC} - \xi_{VC} R_{VC} \\
\dot{S}_{VF} &= \xi_{VC} S_{VC} - (\lambda_{\text{in}} + \xi_{VF}) S_{VF} \\
\dot{E}_{VF} &= p_E \xi_{VC} E_{VC} + \lambda_{\text{in}} S_{VF} - [m\phi_{VF} + (1-m)\phi_{VF} + \xi_{VF}]E_{VF} \\
\dot{A}_{VF} &= p_A \xi_{VC} A_{VC} + (1-m)\phi_{VF} E_{VF} - (\gamma_A + \xi_{VF}) A_{VF} \\
\dot{I}_{VF} &= p_I \xi_{VC} I_{VC} + m\phi_{VF} E_{VF} - (\gamma_I + \xi_{VF}) I_{VF} \\
\dot{R}_{VF} &= \xi_{VC} R_{VC} + \gamma_I I_{VF} + \gamma_A A_{VF} - \xi_{VF} R_{VF}
\end{aligned} \qquad [2]$$

The force of infection for individuals outside a facility included the contributions of community members (indexed by C), staff in the community (indexed by SC), and visitors in the community (indexed by VC). This rate was denoted by $\lambda_{\text{out}} = [\Sigma_{i=C,SC,VC}\beta_i(\pi_i I_i + \rho_i \eta i A_i]/N_{\text{out}}$. The model equations for the staff population can be derived by replacing indices VC and VF in Eq. **2** with SC and SF, accordingly.

The Force of Infection. The expression for the forces of infection inside (λ_{in}) and outside (λ_{out}) a facility assumed that transmission reduction measures could be applied with different levels of effectiveness to asymptomatic and symptomatic individuals. In the case in which transmission reduction was equally applicable to symptomatic and asymptomatic persons ($\rho_i = \pi_i$), of course, $\pi_i < \rho_i < 1$. However, some NPIs, such as isolation, are strictly inapplicable to asymptomatic persons, and it is likely that many other NPI forms will not be applied with equal rigor to both obviously infectious and symptom-free individuals. We believe, therefore, that in practice, ρ_i will be closer to 1 than π_i. We have presented the results of simulations for the extreme values for ρ_i. The small difference in the results is consistent with our assumption of a small number and low infectiousness associated with the asymptomatic state for pandemic viruses.

The Community Model. To assess the contributions of staff and visitors to the risk of a virus introduction, we simulated their interactions during the time spent in the community. Although community members did not interact directly with residents, their contacts with visitors and staff outside the facility could indirectly impact (modeled in λ_{out}) the risk of transmission to residents. By using the same epidemiological classes: susceptible (S_C), exposed (E_C), asymptomatic (A_C), infectious (I_C), and recovered (R_C), we derived the following system of equations for the community:

$$\begin{aligned}
\dot{S}_C &= -\lambda_{out} S_C \\
\dot{E}_C &= \lambda_{out} S_C - (m\phi_C + (1-m)\phi_C) E_C \\
\dot{A}_C &= (1-m)\phi_C E_C - \gamma_A A_C \\
\dot{I}_C &= m\phi_C E_C - \gamma_I I_C \\
\dot{R}_C &= \gamma_I I_C + \gamma_A A_C
\end{aligned} \quad [3]$$

Stochastic Poisson Simulation of Disease Progression. Compartmental deterministic models are frequently applied to study the spread of communicable diseases that impact human populations. These models assume homogeneous mixing that arguably may overestimate an outbreak size, and hence, underestimate the effect of NPIs. Such models implicitly assume that the populations under study are sufficiently "large" and, therefore, neglect "small" stochastic event variations. Because the model discussed in this study involved a facility of only 200 residents, 83 staff, and 40 visitors, we implemented a stochastic model and solved it numerically via a Poisson simulation (8).

We illustrate the Poisson simulation approach (8) in a deterministic differential equation Susceptible–Infected–Recovered (SIR) model given by:

$$\begin{aligned}
\frac{dS}{dt} &= -\beta S \frac{I}{N} \\
\frac{dI}{dt} &= \beta S \frac{I}{N} - \gamma I \\
\frac{dR}{dt} &= \gamma I
\end{aligned} \quad [4]$$

where disease transmission coefficient and duration of infectiousness are given by β and $T = 1/\gamma$, respectively. The transition rate from S to I (force of infection) is given by $\beta\, I/N$ and the density of new infections is given by $\beta SI/N$. Applying the Euler algorithm and a Poisson simulation approach we rewrite the deterministic model into a stochastic model with the following form:

$$\begin{aligned}
S(t+\delta_t) &= S(t) - \delta_t\, r_1(t) \\
I(t+\delta_t) &= I(t) + \delta_t\, [r_1(t) - r_2(t)] \\
R(t+\delta_t) &= R(t) + \delta_t\, r_2(t)
\end{aligned} \quad [5]$$

The deterministic mechanism transferring fractions ($\beta S\, I/N$ and γI) of individuals between stages during a time step in model **1** is replaced by a stochastic mechanism based on integer numbers of individuals with fractions given by $r_1(t) = \text{Poisson}\left(\frac{\delta_t\, S(t)\, I(t)\beta}{N(t)}\right) \Big/ \delta_t$ and $r_2(t) = \text{Poisson}\left(\frac{\delta_t\, I(t)\gamma}{N(t)}\right) \Big/ \delta_t$. These transitional events mimic a stochastic mechanism with an underlying Poisson distribution. In this case, events are independent for a short, fixed interval δ_t, and we can assume that the number of transferred cases during t and $t + \delta_t$ are Poisson distributed with expected values $\delta_t\, r_1(t)$ and $\delta_t\, r_2(t)$.

We applied this approach to numerically simulate the disease dynamics of residents, visitors, staff, and community individuals. We carried out a sensitivity analysis to determine an appropriate time step ($\delta_t = 0.01$) in the simulations and to monitor for and prevent the occurrence of negative population sizes. Table S1 contains a detailed description of the population initial conditions assumed in these simulations.

Calculation of the Basic Reproduction Number. The basic reproduction number, denoted by $\mathcal{R}_0$, may be taken to be equal to the average number of secondary infections generated by a primary case in a susceptible population (9, 10). The contributions to the epidemiological threshold $\mathcal{R}_0$ involve properties that define the course of infection in an individual, as well as attributes of the host population, such as susceptible pool densities and contact (mixing) rates. In particular, these contributions include the number of susceptibles present for contact with the primary case, the length of time the primary case is infectious to others, and the transmission coefficient (rate of effective mixing). This quantity ($\mathcal{R}_0$) is critical in determining the conditions that may lead to the invasion or extinction of an infectious disease in a population. In general, an infectious agent invades if $\mathcal{R}_0 > 1$ and it dies out for values <1.

Given the relatively high dimensionality of the models presented in this study, we evaluated the expression for the basic reproduction number numerically. First, we applied the next generation-operator approach (9, 10) to study the eigenvalues of the next-generation matrix associated with our deterministic systems of Eqs. **1–3** described in *Epidemic Models: Nonpharmaceutical Interventions*. The method entails finding two matrices F and V, for the new infections and transition terms, respectively, and then computing the spectral radius (dominant eigenvalue) of the next-generation matrix FV^{-1}. The dominant eigenvalue of FV^{-1} gives the basic reproduction number. For the model systems **1–3**, it was not feasible to express the associated basic reproduction number in closed form. Therefore, we numerically evaluated the basic reproduction number for various pandemic severity regimes (Table S3).

1. Gani R, *et al.* (2005) Potential impact of antiviral drug use during influenza pandemic. *Emerg Infect Dis* 11(9):1355–1362.
2. Longini IM, Halloran ME, Nizam A, Yang Y (2004) Containing the pandemic influenza with antiviral agents. *Am J Epidemiol* 159:623–633.
3. Mills CE, Robins JM, Lipsitch M (2004) Transmissibility of 1918 pandemic influenza. *Nature* 432(7019):904–906.
4. Chowell G, Nishiura H, Bettencourt LMA (2007) Comparative estimation of the reproduction number for pandemic influenza from daily case notification data. *J R Soc Interface* 4:155–166.
5. Stiver G (2003) The treatment of influenza with antiviral drugs. *Can Med Assoc J* 168(1):49–56.
6. Thompson WW, *et al.* (2004) Influenza-associated hospitalizations in the United States. *J Am Med Assoc* 292(11):1333–1340.
7. American Health Care Association (2006) *The State Long–Term Health Care Sector 2005: Characteristics, Utilization, and Government*. Available at: http://www.ahca.org/research/index.html. Accessed on September 28, 2007.
8. Gustafsson L (2000) Poisson simulation: A method for generating stochastic variations in continuous system simulation. *Simulation* 74(5):264–274.
9. Diekmann O, Heesterbeek JAP, Metz JAJ (1990) On the definition and the computation of the basic reproduction ratio R0 in models for infectious diseases in heterogeneous populations. *J Math Biol* 28:365–382.
10. van den Driessche P, Watmough J (2002) Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Math Biosci* 180(1–2):29–48.

Table S1. Confidence intervals (95%) for the probabilities *p*(Intro), *p*(Outbreak,Intro), and *p*(Outbreak|Intro)

	Baseline			Category 1–2		
$\mathcal{R}_0$	p(Intro)	p(Outbreak,Intro)	p(Outbreak|Intro)	p(Intro)	p(Outbreak,Intro)	p(Outbreak|Intro)
1.4	(0.89, 0.93, 0.97)	(0.85, 0.90, 0.95)	(0.69, 0.97, 1)	(0.37, 0.45, 0.53)	(0.02, 0.06, 0.10)	(0, 0.13, 0.51)
1.6	(0.90, 0.94, 0.98)	(0.89, 0.93, 0.97)	(0.83, 0.99, 1)	(0.53, 0.61, 0.69)	(0.04, 0.08, 0.12)	(0, 0.13, 0.56)
1.8	(0.91, 0.95, 0.99)	(0.91, 0.95, 0.99)	(1, 1, 1)	(0.46, 0.54, 0.62)	(0.13, 0.19, 0.25)	(0, 0.35, 0.93)
2	(0.97, 0.99, 1)	(0.96, 0.98, 1)	(0.83, 0.99, 1)	(0.53, 0.61, 0.69)	(0.11, 0.17, 0.23)	(0, 0.28, 0.85)
2.2	(0.97, 0.99, 1)	(0.97, 0.99, 1)	(1, 1, 1)	(0.62, 0.70, 0.78)	(0.16, 0.23, 0.30)	(0, 0.33, 0.98)
2.4	(1, 1, 1)	(1, 1, 1)	(1, 1, 1)	(0.75, 0.81, 0.87)	(0.40, 0.48, 0.56)	(0, 0.59, 1.00)
2.6	(0.97, 0.99, 1)	(0.96, 0.98, 1)	(0.83, 0.99, 1)	(0.83, 0.88, 0.93)	(0.66, 0.73, 0.80)	(0.25, 0.83, 1)
2.8	(1, 1, 1)	(1, 1, 1)	(1, 1, 1)	(0.94, 0.97, 1)	(0.86, 0.91, 0.96)	(0.55, 0.94, 1)
	Category 3–4			**Category 5**		
1.4	(0.07, 0.13, 0.19)	(0, 0.01, 0.03)	(0, 0.08, 0.24)	(0.04, 0.08, 0.12)	(0, 0, 0)	(0, 0, 0)
1.6	(0.10, 0.16, 0.22)	(0, 0.01, 0.03)	(0, 0.06, 0.22)	(0.01, 0.04, 0.07)	(0, 0, 0)	(0, 0, 0)
1.8	(0.08, 0.14, 0.20)	(0, 0.01, 0.03)	(0, 0.07, 0.23)	(0.01, 0.04, 0.07)	(0, 0, 0)	(0, 0, 0)
2	(0.13, 0.19, 0.25)	(0, 0.01, 0.03)	(0, 0.05, 0.21)	(0.02, 0.06, 0.10)	(0, 0, 0)	(0, 0, 0)
2.2	(0.23, 0.31, 0.39)	(0.03, 0.07, 0.11)	(0, 0.23, 0.61)	(0.01, 0.05, 0.09)	(0, 0, 0)	(0, 0, 0)
2.4	(0.43, 0.51, 0.59)	(0.04, 0.08, 0.12)	(0, 0.16, 0.58)	(0.01, 0.05, 0.09)	(0, 0, 0)	(0, 0, 0)
2.6	(0.69, 0.76, 0.83)	(0.21, 0.28, 0.35)	(0, 0.37, 1)	(0.01, 0.05, 0.09)	(0, 0, 0)	(0, 0, 0)
2.8	(0.80, 0.86, 0.92)	(0.47, 0.55, 0.63)	(0, 0.64, 1)	(0.02, 0.06, 0.10)	(0, 0, 0)	(0, 0, 0)

Lower-bound (LB), Mean (M), and upper-bound values (UB) are denoted. These simulations assumed that $p_i = 1$.

Table S2. Confidence intervals (95%) for the probabilities *p*(Intro), *p*(Outbreak,Intro), and *p*(Outbreak|Intro)

	Category 1–2			Category 3–4		
R_0	p(Intro)	p(Outbreak,Intro)	p(Outbreak |Intro)	p(Intro)	p(Outbreak,Intro)	p(Outbreak |Intro)
1.4	(0.38, 0.46, 0.54)	(0.01, 0.04, 0.07)	(0, 0.09, 0.40)	(0.12, 0.18, 0.24)	(0, 0, 0)	(0, 0, 0)
1.6	(0.50, 0.58, 0.66)	(0.03, 0.07, 0.11)	(0, 0.12, 0.53)	(0.07, 0.14, 0.17)	(0, 0, 0)	(0, 0, 0)
1.8	(0.54, 0.62, 0.70)	(0.07, 0.12, 0.17)	(0, 0.19, 0.71)	(0.09, 0.15, 0.21)	(0, 0, 0)	(0, 0, 0)
2	(0.55, 0.63, 0.71)	(0.07, 0.12, 0.17)	(0, 0.19, 0.70)	(0.13, 0.20, 0.27)	(0, 0.02, 0.04)	(0, 0.10, 0.32)
2.2	(0.62, 0.70, 0.78)	(0.21, 0.28, 0.35)	(0, 0.40, 1)	(0.12, 0.18, 0.24)	(0, 0.03, 0.06)	(0, 0.17, 0.43)
2.4	(0.70, 0.77, 0.84)	(0.34, 0.42, 0.50)	(0, 0.55, 1)	(0.35, 0.43, 0.51)	(0.01, 0.04, 0.07)	(0, 0.09, 0.41)
2.6	(0.89, 0.93, 0.97)	(0.69, 0.76, 0.83)	(0.20, 0.82, 1)	(0.59, 0.67, 0.75)	(0.22, 0.29, 0.36)	(0, 0.43, 1)
2.8	(0.86, 0.91, 0.96)	(0.81, 0.87, 0.93)	(0.63, 0.96, 1)	(0.78, 0.84, 0.90)	(0.51, 0.59, 0.67)	(0.01, 0.70, 1)

Lower-bound (LB), Mean (M), and upper-bound values (UB) are denoted. These results pertain to plan category 1–2 and plan category 3–4 assuming that $p_i = \pi_i$.

Table S3. Variables, parameter definitions, values, and initial conditions assumed in the numerical simulation of a resident facility

Variables	Parameters	Values	Reference
$\mathcal{R}_0$	Basic reproduction number	1.4–2.8	[2,3,4]
m	Fraction of exposed that progress to infection	0.667	[1]
$1/\phi_i$†	Average latency period (days)	1.9	[2,3]
$1/\gamma_A$	Average recovery period for asymptomatic (days)	5	[2,5]
$1/\gamma_I$	Average recovery period for infected (days)	5	[2,5]
CFP	Case Fatality Proportion	0.03-0.15	[6]
δ_I	Resident mortality rate, $\delta_I = \frac{CFP}{1-CFP}\gamma_I$ (days^{-1})	0.0062-0.035	[1]
π_i†	Transmission reduction parameter when applied to infected	0.05–1	[2]
ρ_i†	Transmission reduction parameter when applied to asymptomatic	0.05–1	[2]
η_i†	Infectiousness of asymptomatics relative to symptomatics	0.02	[1]
$1/\xi_{VF}$; $1/\xi_{VC}$	Average time spent between locations by visitors (hours; days)	0-2; 7	[7]
$1/\xi_{SF}$; $1/\xi_{SC}$	Average time spent between locations by staff (hours; hours)	8-12; 12-16	[7]
p_A	Probability of having Asymptomatics escape monitoring efforts	1	Estimated
p_E	Probability of having Exposed escape monitoring efforts	0.14-1	Estimated
p_I	Probability of having Infecteds escape monitoring efforts	0.1-1	Estimated

†$i = R, SF, VF, SC, VC$.

Table S4. Initial conditions for community members (indexed by *C*), facility residents (indexed by *R*), visitors (indexed by *VC*, *VF*), and staff (indexed by *SC*, *SF*)

Epidemiological Classes	Population Size	Reference
S_C; S_R; S_{VC}; S_{VF}; S_{SC}; S_{SF}	50,000; 200; 27; 13; 8; 75	[7]
E_C; E_R; E_{VC}; E_{VF}; E_{SC}; E_{SF}	1; 0; 1; 0; 1; 0	[7]
I_C; I_R; I_{VC}; I_{VF}; I_{SC}; I_{SF}	1; 0; 1; 0; 1; 0	[7]
A_i†; R_i†; D_i†	0; 0; 0	[7]

†i = *C*, *R*, *VC*, *VF*, *SC*, *SF*.