Appendix

This Appendix provides a detailed formulation of the model that generated the results reported in the main text. The within-household model is described in §1, and a key component to embed this household model into the the broader epidemiological model of disease spread described in §2.2 of the main text is formulated in §2. The global infection rate is estimated in §3 and infection control measures are assessed in §4.

1 The Within-Household Model

This section describes the mathematical model of disease spread within a household that contains a single infected person and $n-1$ susceptibles. The viral dynamics are modeled in §1.1, the dose is calculated in §1.2 and the dose-response model is formulated in §1.3. For more information about this model see [1].

1.1 The Viral Dynamics

The infected person becomes infectious at time 0, develops symptoms at time T_p, and stops being infectious at time T_I. This subsection develops an ordinary differential equation model for the time period $[0, T_I]$. We assume the house consists of three compartments: the living quarters (indexed by $j = 1$), the infected's bedroom ($j = 2$), and a susceptible's bedroom ($j = 3$). We do not explicitly model multiple susceptible bedrooms, and hence ignore the small amount of virus that could leak out of the living quarters and into these other bedrooms. There are three state variables: the concentration of virus of diameter x in the air at time t in each of the three compartments ($C_j(x, t)$ for $j = 1, 2, 3$).

There are several differences in the model between the pre-symptomatic phase $[0, T_p]$ and the symptomatic phase $(T_p, T_I]$. The infected person stays in his bedroom throughout the symptomatic period and one of the susceptibles, referred to as the caregiver, spends some time in the infected's bedroom to provide care; during the symptomatic period, the other $n-2$ non-caregiving susceptibles never go into the infected's bedroom. In addition, as described in §3 of [1], some of the parameters take on different values in the pre-symptomatic and symptomatic periods to reflect increased social distancing.

We use the indicator functions $I_{ij}(t)$ to describe the presence or absence of person i ($i=1$ for infected, $i=2$ for non-caregiving susceptible, $i=3$ for caregiver) in compartment j. During the pre-symptomatic period, each person spends Δ_1 hours each day in the living quarters followed by Δ_2 hours in his bedroom, and is out of the house for $24-\Delta_1-\Delta_2$ hours. We assume that these times are perfectly synchronized, so that each family member is in his bedroom, in the living quarters, or out of the house during the same hours. The non-caregiving symptomatics follow this same schedule throughout the symptomatic period, and the caregiver does also, except for spending Δ_3 hours providing care in the infected's bedroom in lieu of being out of the house. The indicator functions (where time is in hours and τ_1 and τ_2 specify the beginning of the living quarters time and the caregiving time, respectively - note also that $T_p=24$ hr and that $x^+=\max\{x,0\}$) are

$$I_{11}(t) = \begin{cases} 1 & \text{for } t \in [(24i+\tau_1)^+, \min\{(24i+\tau_1+\Delta_1)^+, T_p\}) \text{ for } i = -1, \ldots, \frac{T_p}{24} - 1; \\ 0 & \text{otherwise,} \end{cases} \tag{A.1}$$

$$I_{12}(t) = \begin{cases} 1 & \text{for } t \in [\min\{(24i+\tau_1+\Delta_1)^+, T_p\}, \min\{(24i+\tau_1+\Delta_1+\Delta_2)^+, T_p\}) \cup t \in [T_P, T_I] \text{ for } i = -1, \ldots, \frac{T_p}{24} - 1; \\ 0 & \text{otherwise,} \end{cases} \tag{A.2}$$

$$I_{21}(t) = \begin{cases} 1 & \text{for } t \in [(24i+\tau_1)^+, \min\{(24i+\tau_1+\Delta_1)^+, T_I\}) \text{ for } i = -1, \ldots, \frac{T_I}{24} - 1; \\ 0 & \text{otherwise,} \end{cases} \tag{A.3}$$

$$I_{31}(t) = \begin{cases} 1 & \text{for } t \in [(24i+\tau_1)^+, \min\{(24i+\tau_1+\Delta_1)^+, T_I\}) \text{ for } i = -1, \ldots, \frac{T_I}{24} - 1; \\ 0 & \text{if } I_{32}(t) = 1 \\ 0 & \text{otherwise,} \end{cases} \tag{A.4}$$

$$I_{23}(t) = \begin{cases} 1 & \text{for } t \in [\min\{(24i+\tau_1+\Delta_1)^+, T_I\}, \min\{(24i+\tau_1+\Delta_1+\Delta_2)^+, T_I\}) \text{ for } i = -1, \ldots, \frac{T_I}{24} - 1; \\ 0 & \text{otherwise,} \end{cases} \tag{A.5}$$

$$I_{33}(t) = \begin{cases} 1 & \text{for } t \in [\min\{(24i+\tau_1+\Delta_1)^+, T_I\}, \min\{(24i+\tau_1+\Delta_1+\Delta_2)^+, T_I\}) \text{ for } i = -1, \ldots, \frac{T_I}{24} - 1; \\ 0 & \text{if } I_{32}(t) = 1 \\ 0 & \text{otherwise,} \end{cases} \tag{A.6}$$

$$I_{32}(t) = \begin{cases} 1 & \text{for } t \in [24i+\tau_3, 24i+\tau_3+\Delta_3) \text{ for } i = 1, \ldots, \frac{T_I}{24} - 1; \\ 0 & \text{otherwise,} \end{cases} \tag{A.7}$$

and $I_{13}(t) = I_{22}(t) = 0$ for $t \in [0, T_I]$. In the base case, we assume that bedroom doors are closed when the bedroom is occupied and open when the bedroom is unoccupied.

Let $\lambda(t)$ be the rate of viral shedding at time $t \in [0, T_I]$. We assume that the viral shedding rate grows exponentially at rate ν starting from the level Λ_0 during the pre-symptomatic phase and then drops exponentially at rate ω during the symptomatic phase, so that

$$\lambda(t) = \begin{cases} \Lambda_0 e^{\nu t} & \text{for} \quad t \in [0, T_p]; \\ \Lambda_0 e^{\nu T_p - \omega(t - T_p)} & \text{for} \quad t \in [T_p, T_I]. \end{cases} \tag{A.8}$$

Equation (A.8) is consistent with the observation that viral shedding is maximal at the time when symptoms appear, and decreases exponentially thereafter [2, 3]. To capture the interperson heterogeneity in viral shedding, we let Λ_0 be a log-normal random variable with probability density function $h(\lambda)$, where the natural logarithm of the iniital shedding rate has mean m_λ (i.e., the median initial shedding rate is e^{m_λ}) and standard deviation σ_λ (i.e., the dispersion is e^{σ_λ}).

Compartment j has air volume V_j and ventilation (i.e., outdoor air supply) rate Q_j. In addition, Q_{ij} is the air flow rate from compartment i to compartment j, where $Q_{23} = Q_{32} = 0$. We assume that particles smaller than d_c in diameter immediately evaporate in the air and become droplet nuclei with diameter half their original size, and all particles with diameter larger than d_c instantaneously settle on a surface; the actual times until evaporation for sub$-d_c$ particles and settling times for super$-d_c$ particles are orders-of-magnitude smaller than T_p [4, 5]. The diameter of a particle is the aerodynamic diameter and when we refer to a particle of diameter x it is the pre-evaporation size (and thus its size in the air as a droplet nuclei would be of diameter $\frac{x}{2}$). Let $p(x)$ be the probability density function (pdf) of particle sizes (i.e., diameters) emitted by the infected (before evaporation), which represents a weighted average of particle sizes emitted during coughing and sneezing. Because the amount of virus in a particle is roughly proportional to the particle's volume [6], we define $f(x) = \frac{x^3 p(x)}{\int_0^\infty x^3 p(x)\ dx}$, which is the pdf for the proportion of shed virus that is in particles of each size. Let p_r be the penetration factor of the infected's respirator (i.e., the fraction of expelled virus that escapes the respirator and gets into the air), where $p_r = 1$ if the infected person does not wear a respirator. The death rate of virus in the air is μ_a. By Stokes Law, we assume that airborne virus of diameter x deposits on the surfaces at rate proportional to the diameter squared [7], which we denote by κx^2. We also assume that at each point in time and independent of particle size, a fraction p_s of the shed virus ends up on surfaces due to protective measures on the part of the infected (e.g., the virus lands on tissues for a sneeze, or hands for a cough).

The above description implies that for $x \in [0, d_c]$, the state equations are

$$\dot{C}_1(x,t) = \frac{p_r f(x)(1-p_s)\lambda(t) I_{11}(t)}{V_1} + \frac{Q_{21}C_2(x,t) + Q_{31}C_3(x,t)}{V_1} - \Big[\frac{Q_1 + Q_{12} + Q_{13}}{V_1} + \mu_a + \kappa x^2\Big] C_1(x,t), \quad \text{(A.9)}$$

$$\dot{C}_2(x,t) = \frac{p_r f(x)(1-p_s)\lambda(t) I_{12}(t)}{V_2} + \frac{Q_{12}C_1(x,t)}{V_2} - \Big[\frac{Q_2 + Q_{21}}{V_2} + \mu_a + \kappa x^2\Big] C_2(x,t), \quad \text{(A.10)}$$

$$\dot{C}_3(x,t) = \frac{Q_{13}C_1(x,t)}{V_3} - \Big[\frac{Q_3+Q_{31}}{V_3} + \mu_a + \kappa x^2\Big] C_3(x,t). \tag{A.11}$$

Equations (A.9)-(A.11) can be solved numerically for $\{C_j(x,t), t \geq 0\}$ for $j = 1, 2, 3$ and $x \in [0, d_c]$.

1.2 The Dose

Aerosol transmission occurs because a susceptible inhales droplet nuclei that contain virus. In this subsection, we compute the dose received by a susceptible in terms of the solution $C_j(x,t)$ to (A.9)-(A.11). The relationship between this dose and the likelihood of infection is described in §1.3.

Let b be the breathing rate and p_{aj} be the penetration factor (the virus concentration in the air outside of the respirator divided by the virus concentraton of the air inside of the respirator) for a susceptible in compartment j, where $p_{aj} = 1$ if no respirator is being worn. Then the total dose inhaled of size x by a non-caregiving susceptible $(i = 2)$ and the caregiver $(i = 3)$ is

$$D_i(x) = b \int_0^{T_I} \sum_{j=1}^{3} p_{aj} C_j(x,t) I_{ij}(t)\, dt. \tag{A.12}$$

1.3 The Dose-Response Relationship

In this subsection, we derive the probability that a susceptible gets infected in terms of the dose $D_i(x)$ in equation (A.12). Human influenza virus preferentially binds to $\alpha 2$-6-linked sialic acids on receptors of ciliated columnar epithelial cells, leading to viral replication in the respiratory epithelium [8, 9]. Let $g(x)$ be the fraction of inhaled virus of size x that is deposited on the respiratory epithelium (Figure 3 of [1] illustrates $g(x)$). Then the total dose deposited on the respiratory epithelium for non-caregiving susceptibles ($i = 2$) and the caregiver ($i = 3$) is

$$\bar{D}_i = \int_0^{d_c} D_i(x) g(x)\, dx. \tag{A.13}$$

We use the Poisson model to compute the likelihood of infection, which is standard in the literature and which has been shown to model influenza infection and other airborne infections reasonably well [4, 10]. Let ID_{50} denote the median infectious dose for inhaled virus deposited in the respiratory epithelium and define the constant $\alpha = \frac{\ln 2}{\mathrm{ID}_{50}}$. We denote the infection probability as P_{Ii}, where capital I stands for infection and lowercase i distinguishes between the non-caregiving susceptible $(i = 2)$ and the caregiver ($i = 3$). If we write the dose in (A.13) as a function of the initial viral shedding rate Λ_0, which has pdf $h(\lambda)$, then the probability that a non-caregiving susceptible $(i = 2)$ and the caregiver ($i = 3$) become infected, assuming probabilistic independence between the various modes of infection, is

$$P_{Ii} = 1 - \int_0^\infty e^{-\alpha \bar{D}_i(\lambda)} h(\lambda)\, d\lambda. \tag{A.14}$$

2 Calculation of the Total Number of Household Infections

In this section we will derive the expression for $E[C]$, introduced in §2.2.1 of the main text, for $n = 4$ because that is the value used in this study. We can no longer describe susceptibles as either caregiver or noncaregiver, for we need to take into account situations where the caregiver becomes ill and a noncaregiver must take over the role as caregiver and care for all sick individuals in the house. We assume that the original caregiver remains caregiver as long as he remains healthy, regardless of how many other susceptibles become sick. However, the noncaregivers are no longer indistinguishable; each is given a rank based on when they will become the caregiver. A noncaregiver becomes the caregiver only if the original caregiver, as well as all other noncaregivers with higher rank, become sick. In this section, to avoid confusion, we will refer to the members of the house as person 1 (initial infected), person 2 (initial caregiver), person 3 (initial noncaregiver, first alternate caregiver), and person 4 (initial noncaregiver, second alternate caregiver). In order to compute $E[C]$ we need to compute the infection probabilities for person 2, person 3, and person 4.

We first analyze person 2, who is infected in the first wave with probability P_{I3}. With probability $(1 - P_{I3})$, person 2 is not directly infected in the first wave, and there are 3 remaining scenarios where person 2 can be infected indirectly. If both person 3 and person 4 get sick in the first wave (with probability P_{I2}^2), then person 2 now cares for all three sick people, however he can only be infected in the second wave by person 3 or person 4. Assuming transmission from each infected is independent, the total probability of person 2 getting sick in the second wave in this scenario is $(1 - P_{I3})P_{I2}^2(1 - (1 - P_{I3})^2)$. In the next scenario, person 3 gets sick in the first wave but person 4 does not (with probability $(1 - P_{I2})P_{I2}$). There are two subcases where person 2 can now get sick. Person 3 can infect person 2 in the second wave (with probability P_{I3}), or person 4 can infect him in the third wave after person 3 infects person 4 in the second wave (which implies person 3 does not infect person 2 in the second wave). The total probability that person 2 gets sick in the second wave in this scenario is $(1 - P_{I3})(1 - P_{I2})P_{I2}P_{I3}$, whereas the total probability that person 2 gets sick in the third wave in this scenario is $(1 - P_{I3})(1 - P_{I2})P_{I2}(1 - P_{I3})P_{I2}P_{I3}$. The final scenario is person 4 gets sick in the first wave but person 3 does not, however this is equivalent to the last scenario. Thus the total probability person 2 (the original caregiver) gets sick is

$$\begin{aligned} P_c = & P_{I3} + \underbrace{(1 - P_{I3})P_{I2}^2}_{\text{1st wave}}\underbrace{(1 - (1 - P_{I3})^2)}_{\text{2nd wave}} + \underbrace{2(1 - P_{I3})(1 - P_{I2})P_{I2}}_{\text{1st wave}}\underbrace{P_{I3}}_{\text{2nd wave}} \\ & + \underbrace{2(1 - P_{I3})(1 - P_{I2})P_{I2}}_{\text{1st wave}}\underbrace{(1 - P_{I3})P_{I2}}_{\text{2nd wave}}\underbrace{P_{I3}}_{\text{3rd wave}} . \end{aligned} \tag{A.15}$$

The derivation for person 3 is similar. If both person 2 and person 4 get sick in the first wave, then person 3 becomes the caregiver. If person 4 gets sick in the first wave but person 2 does not, then person 3 remains a noncaregiver in the second wave (but may become a caregiver in the third wave). Finally if person 2 gets sick in the first wave and person 4 does not, then person 3 becomes the caregiver. Thus the total probability person 3 (original noncaregiver, first alternate caregiver) gets sick is

$$\begin{aligned} P_{nc,1} =& P_{I2} + \underbrace{(1-P_{I2})P_{I2}P_{I3}}_{\text{1st wave}}\underbrace{(1-(1-P_{I3})^2)}_{\text{2nd wave}} + \underbrace{(1-P_{I2})(1-P_{I3})P_{I2}}_{\text{1st wave}}\underbrace{P_{I2}}_{\text{2nd wave}} \\ &+ \underbrace{(1-P_{I2})(1-P_{I3})P_{I2}}_{\text{1st wave}}\underbrace{(1-P_{I2})P_{I3}}_{\text{2nd wave}}\underbrace{P_{I3}}_{\text{3rd wave}} + \underbrace{(1-P_{I2})(1-P_{I2})P_{I3}}_{\text{1st wave}}\underbrace{P_{I3}}_{\text{2nd wave}} \\ &+ \underbrace{(1-P_{I2})(1-P_{I2})P_{I3}}_{\text{1st wave}}\underbrace{(1-P_{I3})P_{I2}}_{\text{2nd wave}}\underbrace{P_{I3}}_{\text{3rd wave}}. \end{aligned} \tag{A.16}$$

Finally, person 4 becomes the caregiver only when both person 2 and person 3 are sick. Using similar calculations, the total probability that person 4 (original noncaregiver, second alternate caregiver) gets sick is

$$\begin{aligned} P_{nc,2} =& P_{I2} + \underbrace{(1-P_{I2})P_{I2}P_{I3}}_{\text{1st wave}}\underbrace{(1-(1-P_{I3})^2)}_{\text{2nd wave}} + \underbrace{(1-P_{I2})(1-P_{I3})P_{I2}}_{\text{1st wave}}\underbrace{P_{I2}}_{\text{2nd wave}} \\ &+ \underbrace{(1-P_{I2})(1-P_{I3})P_{I2}}_{\text{1st wave}}\underbrace{(1-P_{I2})P_{I3}}_{\text{2nd wave}}\underbrace{P_{I3}}_{\text{3rd wave}} + \underbrace{(1-P_{I2})(1-P_{I2})P_{I3}}_{\text{1st wave}}\underbrace{P_{I2}}_{\text{2nd wave}} \\ &+ \underbrace{(1-P_{I2})(1-P_{I2})P_{I3}}_{\text{1st wave}}\underbrace{(1-P_{I2})P_{I3}}_{\text{2nd wave}}\underbrace{P_{I3}}_{\text{3rd wave}}. \end{aligned} \tag{A.17}$$

Putting these values together, we have

$$E[C] = 1 + P_c + P_{nc,1} + P_{nc,2}, \tag{A.18}$$

which allows us to compute the threshold parameter in equation (1) of the main text. While it is certainly possible to extend these computations for a general n, the number of possible scenarios grows exponentially and there is no obvious way to write the general probabilities in a compact form.

We also look at a situation where a susceptible shares a room with the infected (§4.1). To embed this in the global model we assume that person 3 (original noncaregiver, first alternate caregiver) shares the room. We also assume that the room is shared only during the first wave (e.g., if person

4 gets sick in the first wave, then person 3 does not then share a room with person 4). Making these slight adjustments, and defining P_{Ish} to be the probability the susceptible sharing the room is directly infected by the initial infected (P_{I2} and P_{I3} are the same as in the other situations), we have the following probabilities for the roomsharing scenario:

$$\begin{aligned} P_c =& P_{I3} + \underbrace{(1-P_{I3})P_{I2}P_{Ish}}_{\text{1st wave}}\underbrace{(1-(1-P_{I3})^2)}_{\text{2nd wave}} + \underbrace{(1-P_{I3})(1-P_{I2})P_{Ish}}_{\text{1st wave}}\underbrace{P_{I3}}_{\text{2nd wave}} \\ &+ \underbrace{(1-P_{I3})(1-P_{I2})P_{Ish}}_{\text{1st wave}}\underbrace{(1-P_{I3})P_{I2}}_{\text{2nd wave}}\underbrace{P_{I3}}_{\text{3rd wave}} + \underbrace{(1-P_{I3})(1-P_{Ish})P_{I2}}_{\text{1st wave}}\underbrace{P_{I3}}_{\text{2nd wave}} \\ &+ \underbrace{(1-P_{I3})(1-P_{Ish})P_{I2}}_{\text{1st wave}}\underbrace{(1-P_{I3})P_{I2}}_{\text{2nd wave}}\underbrace{P_{I3}}_{\text{3rd wave}}. \end{aligned} \tag{A.19}$$

$$\begin{aligned} P_{nc,1} =& P_{Ish} + \underbrace{(1-P_{Ish})P_{I2}P_{I3}}_{\text{1st wave}}\underbrace{(1-(1-P_{I3})^2)}_{\text{2nd wave}} + \underbrace{(1-P_{Ish})(1-P_{I3})P_{I2}}_{\text{1st wave}}\underbrace{P_{I2}}_{\text{2nd wave}} \\ &+ \underbrace{(1-P_{Ish})(1-P_{I3})P_{I2}}_{\text{1st wave}}\underbrace{(1-P_{I2})P_{I3}}_{\text{2nd wave}}\underbrace{P_{I3}}_{\text{3rd wave}} + \underbrace{(1-P_{Ish})(1-P_{I2})P_{I3}}_{\text{1st wave}}\underbrace{P_{I3}}_{\text{2nd wave}} \\ &+ \underbrace{(1-P_{Ish})(1-P_{I2})P_{I3}}_{\text{1st wave}}\underbrace{(1-P_{I3})P_{I2}}_{\text{2nd wave}}\underbrace{P_{I3}}_{\text{3rd wave}}. \end{aligned} \tag{A.20}$$

$$\begin{aligned} P_{nc,2} =& P_{I2} + \underbrace{(1-P_{I2})P_{Ish}P_{I3}}_{\text{first wave}}\underbrace{(1-(1-P_{I3})^2)}_{\text{2nd wave}} + \underbrace{(1-P_{I2})(1-P_{I3})P_{Ish}}_{\text{1st wave}}\underbrace{P_{I2}}_{\text{2nd wave}} \\ &+ \underbrace{(1-P_{I2})(1-P_{I3})P_{Ish}}_{\text{1st wave}}\underbrace{(1-P_{I2})P_{I3}}_{\text{2nd wave}}\underbrace{P_{I3}}_{\text{3rd wave}} + \underbrace{(1-P_{I2})(1-P_{Ish})P_{I3}}_{\text{1st wave}}\underbrace{P_{I2}}_{\text{2nd wave}} \\ &+ \underbrace{(1-P_{I2})(1-P_{Ish})P_{I3}}_{\text{1st wave}}\underbrace{(1-P_{I2})P_{I3}}_{\text{2nd wave}}\underbrace{P_{I3}}_{\text{3rd wave}}. \end{aligned} \tag{A.21}$$

3 Estimating the Global Infection Rate λ_G

The values of all of the parameters except for the global infection rate λ_G were estimated in [1], and for convenience are listed in Table A.1. In this section, we estimate λ_G. The basic reproductive number R_0 for the 1918 influenza has been estimated to be $\approx 2-3$, which incorporates both social distancing (e.g., limited school and public gatherings, and crude masks often worn in public, sometimes by decree [28, 29]) and partial immunity from the first wave of disease in the spring of 1918 [30]; if the pandemic hit during the first wave and there was no population immunity, then the reproductive number would be $\approx 2.9-3.9$. More recently, the generation time was estimated to be 2.6 days rather than ≈ 4 days, which revises the pandemic influenza value downward to $R_0 \approx 1.8$ [14]. Because R_* and R_0 have different interpretations – R_* is the basic reproductive ratio of within-house infectious clumps, i.e., infected people within a house arising from a single infected household member [31] – we estimate the global infection rate, λ_G, so that equation (4) in the main text is satisfied with z replaced by the fraction of the population that gets infected.

Although our main goal is to estimate λ_G for pandemic influenza, we also estimate it for interpandemic influenza. Typical z values are 0.15-0.25 for interpandemic influenza [32] and 0.4 for pandemic influenza (Fig. S17 in [14]). For interpandemic influenza, we let the residence times in the bedroom and living quarters be $\Delta_1 = 4$ hr and $\Delta_2 = 8$ hr, respectively. The probability of infection in our model varies with the time of day that the household members gather in the living quarters (τ_1) and the time of day that the caregiver initiates care (τ_3), and we choose the base-case values $\tau_1 = 13$ hr and $\tau_3 = 11$ hr (Fig. 5a in [1]), which achieve typical infection probabilities; e.g., we can think of the infection beginning at 5 am, people mixing in the living quarters during 6-10 pm, and the caregiver providing care during 4-5 pm. In [1], we constrain the secondary attack rate (SAR) within the household, which is the weighted average infection probability of susceptible household members, $\frac{2P_{I2}+P_{I3}}{3}$, to be $\frac{1}{6}$ for interpandemic influenza (which agrees with [33]). This yields the infection probabilities for the non-caregiving susceptibles and the caregiver to be $P_{I2} = .109$ and $P_{I3} = .282$.

Inserting these values for P_{I2} and P_{I3} into the analysis in §2 gives $E[C]$ (and hence $E[S]$). Solving equation (4) in the main text with $z = 0.2$ yields $\lambda_G = 5.63 \times 10^{-3}$/hr and, by equation (1) in the main text, $R_* = 1.15$. These values are very similar to those ($R_* = 1.13$, $z = 0.18$) derived in [34], which uses the households epidemic model and interpandemic influenza data from [31].

We assume less time outside the home in the pandemic setting [28, 29, 35] and let $\Delta_1 = 8$ hr and $\Delta_2 = 12$ hr. We again let $\tau_1 = 13$ hr and $\tau_3 = 11$ hr, which also achieve typical infection probabilities for the pandemic case. Running our model in §1 with the pandemic parameters yields $P_{I2} = 0.176$ and $P_{I3} = 0.296$. Solving Solving equation (4) in the main text with $z = 0.4$ yields $\lambda_G = 5.93 \times 10^{-3}$/hr and $R_* = 1.38$ for pandemic influenza. Because R_0 and R_* have different interpretations, we expect the R_* derived here to be less than the $R_0 = 1.8$ value derived in [30]. Indeed, $R_0 - 1 \approx rT_g \approx 0.8$, where $r \approx 0.3$ is the two-week average gradient in the excess mortality curves and $T_g \approx 2.6$ days is the mean generation time [14]. In our model, the role of the net growth rate $R_0 - 1$ is played by $(R_* - 1)E[C] = 0.73$, and so our parameter values are not inconsistent with the gradients of the excess mortality curves in [30, 14].

We conclude this section by comparing the interpandemic and pandemic values of two other measures. First is the secondary attack rate, which is $\frac{1}{6}$ for interpandemic influenza and 0.216 in the pandemic case. The second measure is the fraction of transmissions that occur outside of the home, or $\frac{1}{E[C]}$, which is 0.586 for interpandemic influenza and 0.516 for pandemic influenza, both of which are lower than the $\frac{2}{3}$ estimate in the interpandemic literature [14].

4 Results

In §4.1 we use the model to assess various infection control measures and in §4.2 we include larger particles in the analysis and determine how the effectiveness of several of the control measures will change under this scenario.

4.1 Infection Control Measures

We analyze six control measures in this section: respirators, humidifiers, ventilation, social distancing, workplace interventions, and surgical masks. We also assess the impact of opening doors and sharing a bedroom.

Respirators. An estimate of the average penetration factor (i.e., the fraction of virus that passes through the respirator) for a N95 filtering-facepiece respirator is 0.1 [36, 37], where nearly all of the inefficiency occurs due to face-seal leakage. We consider penetration values of 0.1, 0.3, and 0.8, the latter two values incorporate suboptimal fit and intermittent use [28, 29]. The penetration values of 0.3 and 0.8 correspond to a 0.1 penetration factor when the respirator is worn due to face-seal leakage as well as accounting for individuals only wearing the respirators approximately 75% (penetration value of 0.3) or 25% (penetration value of 0.8) of the time they are suppose to wear the respirators. If an individual only wears the respirator 75% of the time, the average penetration factor will be $0.1 \times 0.75 + 1 \times 0.25 = .325$, and rounding down yields a penetration factor of 0.3. A similar calculation yields a 0.8 penetration factor when the respirator is worn only 25% of the time. We look at five possiblities: (i) respirators are only worn by the caregiver in the infected's bedroom during the symptomatic period; (ii) respirators are worn by all susceptibles during the symptomatic period; (iii) respirators are worn by the caregiver when he is providing care during the symptomatic period and by everyone (including the infected) when they congregate in the living quarters during the pre-symptomatic period; (iv) respirators are worn by everyone during the pre-symptomatic period and by all susceptibles during the symptomatic period; and (v) same as (iv) except the infected also wears a respirator with penetration factor 0.5 during the symptomatic period (in the 0.8 penetration factor scenario, the penetration factor of the infected's respirator is also 0.8). We do not change the protection factor p_s because the infected may still attempt to protect his coughs and sneezes with his hand or a tissue. The results appear in Table I of the main text and Tables A.2 and A.3 for penetration factors 0.3, 0.1, and 0.8, respectively.

Humidifiers. We assume that a humidifier increases μ_a from 0.36/hr to 6.0/hr, which is the loss rate when the relative humidity is 65% [21]. We consider five possibilities for humidifier use: (i) in the infected's bedroom during the symptomatic period; (ii) in the living quarters during the symptomatic period; (iii) in the entire house during the symptomatic period; (iv) in the infected's bedroom during the symptomatic period and in the living quarters when people congregate there during the pre-symptomatic period; and (v) in the entire house during the entire infectious period. The results appear in Table A.4.

Ventilation. A portable indoor air cleaner with a fan operated at a flow rate of 404 m^3/hr, which is representative of commercial air cleaners, can achieve an air exchange rate of 3.0/hr in a closed room the size of our infected's bedroom [38]. The outdoor air supply rates range from 0.3 to 2.9 air exchanges per hour in various office buildings, with higher values associated with schools [39]. One wide open window in a house can increase the air exchange rate by approximately 1/hr even without fans [40]. We change the ventilation rates in our model to be five times the room volume in locations where additional ventilation is attempted, e.g., by using fans and wide open windows. We consider the same five possibilities as for the humidifiers. The results appear in Table A.5.

Bedroom doors. The air flow rate between two rooms has been measured to be 60 m^3/hr if the door is open and 1.0 m^3/hr if the door is closed [18]. In the base case, bedroom doors are closed when occupied and open when unoccupied. We continue to keep bedroom doors open when unoccupied and explore four other possibilities for opening bedroom doors when occupied: (i) all bedroom doors are open during the pre-symptomatic period; (ii) all bedroom doors are open during the pre-symptomatic period and susceptible bedroom doors are also open during the symptomatic period; (iii) and all bedroom doors are open during the pre-symptomatic period and the infected's bedroom door is also open during the symptomatic period; and (iv) all bedroom doors are open throughout the infectious period. The results appear in Table A.6.

Shared bedroom. Here, we consider the possibility that the infected and a non-caregiving susceptible share the same bedroom. We consider four different scenarios: (i) they share during the pre-symptomatic period with their bedroom door open when occupied; (ii) they share during the pre-symptomatic period with their bedroom door closed when occupied; (iii) they share during the entire period with their bedroom door open when occupied; and (iv) they share during the entire period with their bedroom door closed when occupied. The other susceptibles' bedroom doors are as in the base case: closed when occupied and open when unoccupied. Similarly, in scenarios (i) and (ii) above, the infected's bedroom door is closed throughout the symptomatic period, as in the base case. The results appear in Table A.7. See section §2 for how the value of $E[C]$ changes in this scenario.

Social distancing. Social distancing within the house is achieved in our model by simultaneously reducing the amount of time household members spend together in the living quarters and increasing the time they spend alone in their bedrooms with the door closed. Fig. 4 in the main text presents the values of R^*, SAR, and the proportion of susceptibles that get infected as a function of the number of hours per day spent together in the living quarters.

Interventions in the workplace. A careful analysis of interventions in the workplace would require a generalization of the households model to the overlapping groups model [41], in which the population is partitioned into households and workplaces. Because it is far more difficult to obtain explicit results for the latter model [41], we obtain a rough assessment of workplace interventions by reducing the global infection rate λ_G in the households model (Fig. A.1). We assume that out-of-house transmissions are evenly divided between the community and the workplace [14]. Recall that each household member spends 4 hr out of the house during the 24-hr pre-symptomatic period. Let us redistribute these 16 hr so that 1 of the 4 household members is the worker who spends 8 hr at work and the other 3 members each spend $2\frac{2}{3}$ hr in the community. We conservatively assume that no interventions are used in the community. We further assume that the percentage reduction in the workplace infection rate from the respirators-humidifiers-ventilation combination is equal to the percentage reduction from the combination intervention in the pre-symptomatic SAR in the home, the rationale being that people spend 8 hr/day in the living quarters during the 24-hr pre-symptomatic period and this is also how long the infected worker spends with his fellow workers during the pre-symptomatic period. To aid in the construction of Fig. A.1, we compute the pre-symptomatic SAR in the home (assuming all household members spend 8 hr together in the living quarters) versus the proportion of households complying to the respirators-humidifiers-ventilation combination (Fig. A.2).

To review how Fig. A.1 is constructed, we derive the global infection rate λ_G that corresponds to 70% of the workplaces complying to the respirators-humidifiers-ventilation combination. By Fig. A.2, the pre-symptomatic SAR in the home is reduced from 0.176 to 0.089 if there is 70% compliance in the home. The base-case global infection rate is $\lambda_G = 5.93 \times 10^{-3}$/hr (Table A.1). Hence, because the reduction only impacts half of the global infections (the half transmitted in the workplace), the global infection rate that corresponds to 70% workplace compliance is $0.5(5.93 \times 10^{-3}) + 0.5\left(\frac{0.089}{0.176}\right)(5.93 \times 10^{-3}) = 4.46 \times 10^{-3}$/hr, which agrees with the two horizontal axes in Fig. A.1.

Surgical masks. Fig. 2 of [42] shows the penetration factor of the filter media of 8 surgical masks, as a function of particle diameter. These eight curves naturally group into three types: Three of these curves (the top 3 in the figure) are very similar and are clearly dominated by the other five, in that they have higher penetration factors at all diameters. Three other curves are very similar and have higher penetration factors than the other two curves (which are similar to each other) at diameters < 2 μm and lower penetration factors at diameters > 2 μm. We compute how much protection these latter two sets of curves offer assuming there is no face-seal leakage and the masks are worn throughout the infectious period. We refer to the two curves with lowest penetration factors at diameters < 2 μm as the “lower” curves and refer to the three non-dominated curves as the “middle” curves. We now view the penetration factor as a function of particle diameter x, and refer to it as $p_a(x)$. We fit the lower curves to the function

$$p_a(x) = \begin{cases} 0.061x^{-0.602} & \text{for} \quad x < 3.0\ \mu\text{m}; \\ 0.939x^{-3.09} & \text{for} \quad x \in [3.0, 4.0)\ \mu\text{m}; \\ 0.013 & \text{for} \quad x \geq 4.0\ \mu\text{m}. \end{cases} \tag{A.22}$$

and the middle curves to the function

$$p_a(x) = \begin{cases} 0.4 & \text{for} \quad x < 0.4\ \mu\text{m}; \\ 0.158x^{-1.01} & \text{for} \quad x \in [0.4, 1.1)\ \mu\text{m}; \\ 0.182x^{-2.46} & \text{for} \quad x \in [1.1, 4.0)\ \mu\text{m}; \\ 0.006 & \text{for} \quad x \geq 4.0\ \mu\text{m}, \end{cases} \tag{A.23}$$

The appropriate measure of protection is the ratio of the amount of virus (over the course of the infectious period) that is deposited in the respiratory epithelium with the mask on divided by the amount of virus that is deposited in the respiratory epithelium with the mask off. This quantity differs for the caregiver ($i = 3$) and the non-caregiving susceptibles ($i = 2$) because they are exposed to different doses. By equations (A.12) and (A.13) and noting that the particles shrink to half their size while airborne, these ratios are given by

$$\frac{\int_0^{d_c} b[\int_0^{T_I} \sum_{j=1}^3 p_a(\frac{x}{2}) C_j(x,t) I_{ij}(t) dt] g(x)\ dx}{\int_0^{d_c} b[\int_0^{T_I} \sum_{j=1}^3 C_j(x,t) I_{ij}(t) dt] g(x)\ dx}. \tag{A.24}$$

For the lower curves defined by equation (A.22), the ratio in equation (A.24) is 0.0167 for the caregiver and 0.0166 for the non-caregivers, and for the middle curves in equation (A.23), we get 0.0095 for the caregiver and 0.0093 for the non-caregivers.

Because the 1918 and 1957 influenza strains and the current H5N1 strain all bind to receptors on the respiratory epithelium and the alveoli, we re-assess the penetration factor for the surgical mask filters by adding the alveoli deposition function to $g(x)$ (Fig. 3 of [1]). For the lower curves, the penetration factor is now 0.0179 and 0.0177 for the caregiver and non-caregivers; for the middle curves, the factor is 0.0111 for the caregiver and 0.0108 for the non-caregivers.

4.2 Large Particle Transmission

In our analysis we assume that aerosol transmission occurs only with particles that are smaller than $d_c = 20$ μm in diameter. We assume that particles < 20 μm immediately shrink to half of the original diameter [5] and become droplet nuclei, and that particles > 20 μm immediately settle on surfaces. In a companion paper [1] we analyze aerosol transmission of larger particles only during an expiatory event (such as a cough or sneeze) directly in front of a susceptible's face. Because particles > 20 μm can stay airborne for several minutes, in this subsection we repeat some of the analysis assuming that larger particles can also contribute to aerosol transmission. If larger particles are a part of aerosol transmission than that would strengthen the finding in [1] that aerosol transmission is the dominant route of transmission.

The removal rate (per minute) of a particle of diameter d from gravitational settling is $\frac{0.0018d^2(1+\frac{0.166}{d})}{H}$ [5], where H is the height of room; we set $H = 2.44$m (8 ft). For particles of size 10 μm (the maximum diameter in the air in our model after they have shrunk to half the original diameter) this removal rate is 0.075/min, and for particles of size 50 μm the rate is 1.85/min. Thus after 5 minutes nearly 70% of the particles of size 10 μm will still be airborne, but essentially all of the particles of size 50 μm will have settled on surfaces. Thus we set $d_c = 100$ μm (which corresponds to 50 μm in the air after evaporation and shrinking to half the original diameter) as the upper bound for the diameter of airborne particles in this analysis.

To perform this analysis we first have to recompute several values. The initial median viral shedding rate, 2.88×10^7 TCID$_{50}$/day (Table A.1), was estimated under the assumption that $d_c = 20$ μm [1]. Assuming that d_c is now 100 μm yields an estimate of the initial median viral shedding rate of 5.79×10^6 TCID$_{50}$/day (see §3.5 in [1] for details on this estimation procedure). In the interpandemic case the infection probability is 0.281 for the caregiver and 0.110 for a non-caregiving susceptible. For the pandemic case the values increase to 0.177 and 0.295 respectively. As in §3 we solve equation (4) in the main text with $z = 0.4$ to determine $\lambda_G = 5.93 \times 10^{-3}$/hr. The value of

$R_* = \lambda_G T_I E[C]$ in this scenario is the same as it was in the case where $d_c = 20$ μm ($R_* = 1.38$).

Table A.8 illustrates how effective the three primary interventions (N95 respirators, humidifiers, and ventilation) are when we include these larger particles in the analysis. We only look at the situation where humidifiers and ventilation are implemented in the living quarters when people congregate there during the pre-symptomatic period and in the infected's bedroom during the symptomatic period. Everyone wears a respirator in the living quarters during the pre-symptomatic period, and during the symptomatic period only the caregiver wears a respirator while tending to the infected. We only analyze these scenarios because as Table I of the main text and Tables A.2-A.5 illustrate, most of the potential benefits from the interventions are realized for these implementations.

The relevant comparisons between rows 2, 3, and 4 of Table A.8 are row 4 of Table I in the main text, row 5 of Table A.4, and row 5 of Table A.5, respectively. The respirators are as effective in this scenario as they are for $d_c = 20$ μm because the penetration factor is independent of the particle size. The humidifiers and ventilation are less effective when we include particles with diameter greater than 20 μm. The reason for this is that the larger particles play an important role in the the spreading of influenza because there is a much greater quantity of virus on larger particles. The removal rate of the virus on the larger particles is dominated by settling (the κx^2 term in equations (A.9)-(A.11)) and thus the ventilation and humidity terms in the removal rate do not have as much of an impact.

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Figure Legends

Fig. A.1. The impact of a reduction in the global infection rate λ_G. We assume that out-of-house transmissions are evenly divided between the community (where no interventions are used) and the workplace [14], and that the percentage reduction in the workplace infection rate from the combination intervention is equal to the percentage reduction from the combination intervention in the pre-symptomatic SAR (Fig. A.2) in the home (people spend 8 hr/day in the living quarters in the 24-hr pre-symptomatic period, which corresponds to the pre-symptomatic period falling on a typical work day). We consider no interventions in the home (—) and 70% compliance of the respirators-humidifiers-ventilation intervention in the home (- - -). The right vertical axis is color-coded with the two curves.

Fig. A.2. Pre-symptomatic secondary attack rate (SAR) vs. the proportion of households complying to the respirators-humidifiers-ventilation combination.

Parameter	Description	Value	References
T_I	Total infectious period	120 hr	[11, 12]
T_p	Pre-symptomatic infectious period	24 hr	[13, 14]
$\Delta_1, \Delta_2, \Delta_3$	Time Durations	8, 12, 1 hr	§3.1 in [1]
τ_1, τ_3	Time Schedules	13, 11 hr	§3.4, §3.5 in [1]
ν	Pre-symptomatic viral shedding parameter	4.94/day	[12]
ω	Symptomatic viral shedding parameter	1.70/day	[12]
e^{m_λ}	Median initial viral shedding rate	2.88×10^7 TCID$_{50}$/day	[5, 12, 15, 16]
e^{σ_λ}	Dispersal of initial viral shedding rate	40	[3, 10, 17]
V_1, V_2, V_3	Volume	228.3, 32.6, 32.6 m^3	§3.1 in [1]
Q_1, Q_2, Q_3	Ventilation rate	228.3, 32.6, 32.6 m^3/hr	[7]
Q_{13}, Q_{31}	Internal air flow rate (door open)	60, 60 m^3/hr	[18]
Q_{12}, Q_{21}	Pre-symptomatic internal air flow rate (door open)	60, 60 m^3/hr	[18]
$\tilde{Q}_{12}, \tilde{Q}_{21}$	Symptomatic internal air flow rate (door closed)	1, 1 m^3/hr	[18]
d_c	Critical diameter for droplet nuclei	20 μm	[5]
$p(x)$	pdf of pre-evaporation particle diameter	equation (22) in [1]	[5, 19, 20]
p_r	Respirator penetration factor for infected	1.0	§3.1 in [1]
μ_a	Death rate of virus in air	0.36/hr	[21]
κ	Deposition parameter	0.18/hr·μm^2	[22, 23]
p_s	Pre-symptomatic fraction of virus to protective surfaces	0.75	§3.1 in [1]
$\tilde{p}_s$	Symptomatic fraction of virus to protective surfaces	0.5	§3.1 in [1]
b	Breathing rate	20 m^3/day	[24]
p_{a1}, p_{a2}, p_{a3}	Respirator penetration factor for susceptibles	1.0, 1.0, 1.0	§3.1 in [1]
$g(x)$	Deposition fraction to respiratory epithelium	Fig. 3 of [1]	[25, 26]
ID$_{50}$	ID$_{50}$ in respiratory epithelium	0.671 TCID$_{50}$	[27]
α	$\ln 2/\text{ID}_{50}$	1.033 TCID$_{50}^{-1}$	
n	Household size	4	§3.1 in [1]
λ_G	Global infection rate	5.93×10^{-3}/hr	§3

Table A.1 Base-case parameters values From [1]. The subscripts 1, 2 and 3 represent the three compartments in our model. Tildes represent values during the symptomatic period.

Respirators					
Who	When	P_{I2}	P_{I3}	R_*	z
no one	never	0.176	0.296	1.38	0.400
caregiver	symptomatic period	0.176	0.197	1.24	0.285
all susceptibles	symptomatic period	0.175	0.197	1.24	0.284
caregiver everyone	when providing care in pre-symptomatic living quarters	0.024	0.121	0.85	—
all but symptomatic infected	infectious period	0.019	0.120	0.84	—
everyone	infectious period	0.019	0.090	0.81	—

Table A.2 Efficacy of N95 respirators with penetration factor 0.1. In Tables A.2-A.8 P_{I2}, P_{I3}, R^* and z are the infection probability for a non-caregiving susceptible, the infection probability for the caregiver, the threshold parameter for an epidemic, and the proportion of susceptibles who get infected if there is an epidemic (which can only occur if $R_* > 1$).

Respirators					
Who	When	P_{I2}	P_{I3}	R_*	z
no one	never	0.176	0.296	1.38	0.400
caregiver	symptomatic period	0.176	0.281	1.36	0.384
all susceptibles	symptomatic period	0.176	0.281	1.36	0.384
caregiver everyone	when providing care in pre-symptomatic living quarters	0.149	0.273	1.29	0.328
all but symptomatic infected	infectious period	0.148	0.273	1.29	0.328
everyone	infectious period	0.148	0.258	1.26	0.309

Table A.3 Efficacy of N95 respirators with penetration factor 0.8.

Humidifiers					
Where	When	P_{I2}	P_{I3}	R_*	z
nowhere	never	0.176	0.296	1.38	0.400
infected bedroom	symptomatic period	0.176	0.279	1.35	0.382
living quarters	symptomatic period	0.176	0.296	1.38	0.400
entire house	symptomatic period	0.176	0.279	1.35	0.382
infected bedroom living quarters	symptomatic period in pre-symptomatic living quarters	0.161	0.275	1.32	0.352
entire house	infectious period	0.161	0.275	1.32	0.352

Table A.4 Efficacy of humidifiers (65% humidity).

Ventilation					
Where	When	P_{I2}	P_{I3}	R_*	z
nowhere	never	0.176	0.296	1.38	0.400
infected bedroom	symptomatic period	0.176	0.283	1.36	0.386
living quarters	symptomatic period	0.176	0.296	1.38	0.400
entire house	symptomatic period	0.176	0.283	1.36	0.386
infected bedroom living quarters	symptomatic period in pre-symptomatic living quarters	0.165	0.280	1.33	0.364
entire house	infectious period	0.165	0.279	1.33	0.364

Table A.5 Efficacy of ventilation (5 outside air exchanges per hr).

Open Bedroom Doors When Occupied					
Which	When	P_{I2}	P_{I3}	R_*	z
none	never	0.176	0.296	1.38	0.400
all	pre-symptomatic period	0.177	0.296	1.38	0.402
all susceptibles	pre-symptomatic period symptomatic period	0.177	0.296	1.38	0.403
all infected	pre-symptomatic period symptomatic period	0.192	0.294	1.41	0.424
all	infectious period	0.198	0.297	1.43	0.435

Table A.6 Impact of status of bedroom doors when occupied (air flow rate = 1 m^3/hr if closed and 60 m^3/hr if open). Bedroom doors are open when unoccupied.

Status of Shared Bedroom Door When Occupied	When Shared	P_{Ish}	P_{I2}	P_{I3}	R_*	z
—	never	—	0.176	0.296	1.38	0.400
open	pre-symptomatic period	0.348	0.176	0.296	1.54	0.501
closed	pre-symptomatic period	0.356	0.176	0.296	1.54	0.505
open	infectious period	0.552	0.191	0.294	1.74	0.604
closed	infectious period	0.561	0.176	0.296	1.73	0.601

Table A.7 Impact of the infected and a non-caregiving susceptible sharing a bedroom. P_{Ish} is the probability of infection for the susceptible who shares the infected's bedroom.

Interventions	P_{I2}	P_{I3}	R_*	z
base case (no interventions)	0.177	0.295	1.38	0.400
N95 respirators (0.3 penetration factor)	0.063	0.188	1.00	—
humidifiers	0.173	0.290	1.36	0.389
ventilation	0.174	0.291	1.37	0.391
N95 respirators, humidifiers, and ventilation	0.060	0.182	0.98	—

Table A.8 Results assuming that particles < 100 μm in diameter contribute to aerosol transmission. All interventions are implemented in the living quarters while people congregate there during the pre-symptomatic period and in the infected's bedroom during the symptomatic period

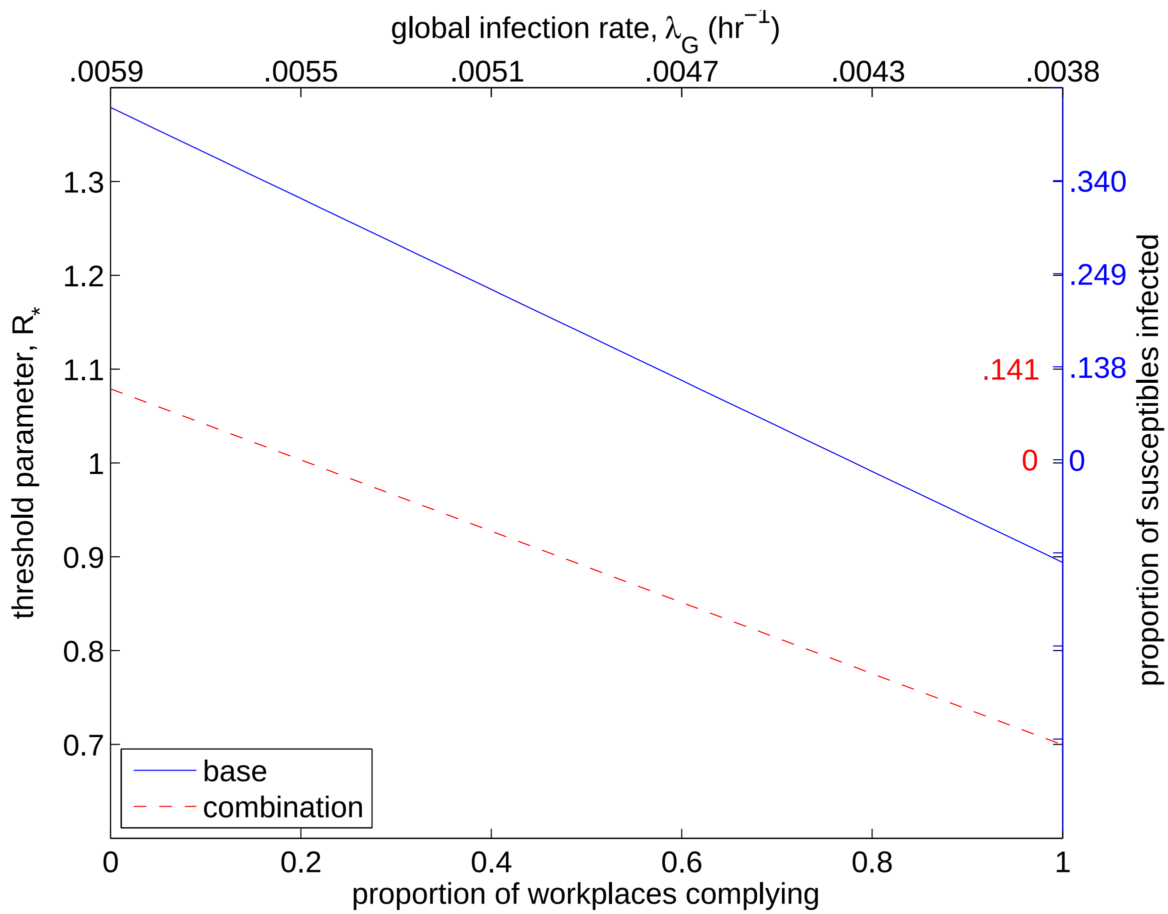
global infection rate, λG (hr−1)
.0059
.0055
.0051
.0047
.0043
.0038
1.3
1.2
1.1
1
0.9
0.8
0.7
threshold parameter, R*
.340
.249
.141
.138
0
0
proportion of susceptibles infected
base
combination
0
0.2
0.4
0.6
0.8
1
proportion of workplaces complying

Figure A.1

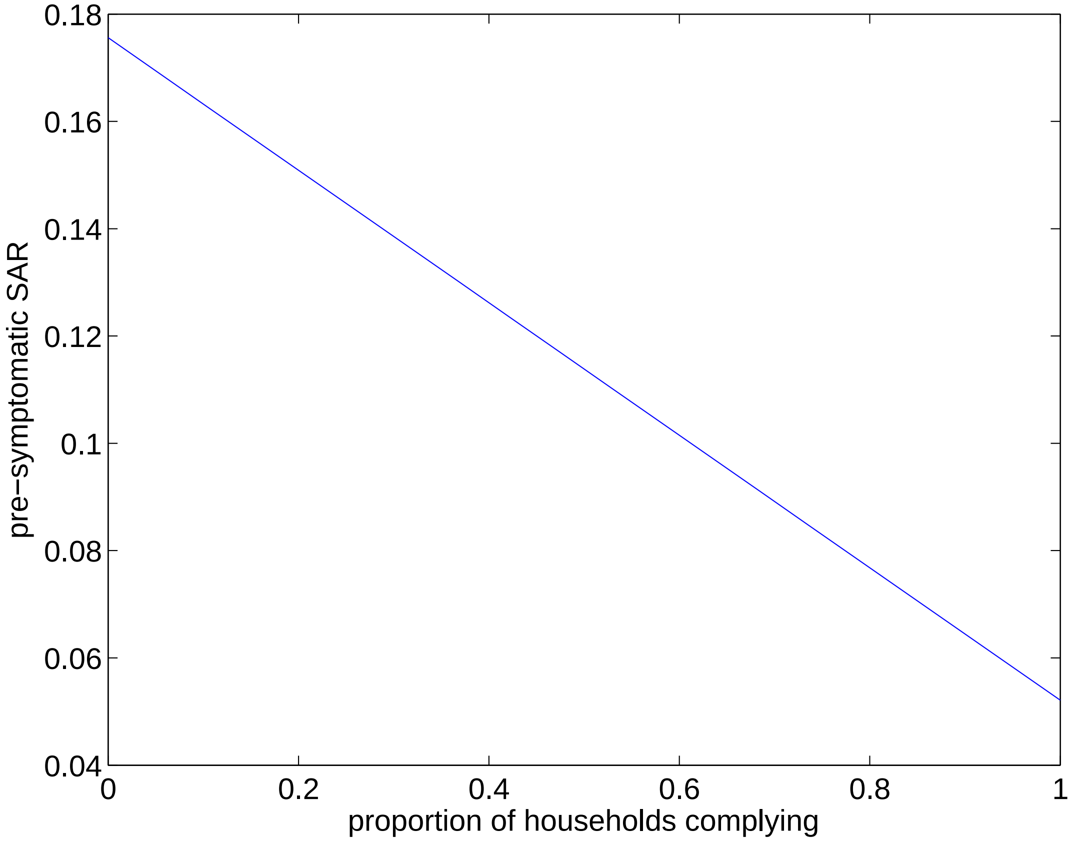
0.18
0.16
0.14
0.12
0.1
0.08
0.06
0.04
pre−symptomatic SAR
0
0.2
0.4
0.6
0.8
1
proportion of households complying

Figure A.2