

Online Supplement to
Modeling Influenza Pandemic and Planning Food
Distribution

1. Literature Review on Modeling Pandemic Influenza and Annual Influenza

Table OS-1 Literature review.

Spread Model	Reference	Main Goal
Random Graphs (Heterogeneous mixing)	Glass et al. (2006)	Study targeted social distancing as an intervention policy within a local community
	Carrat et al. (2006)	Study intervention strategies such as vaccination, neuraminidase inhibitors, quarantine and closure of schools or workplaces
	Carrat et al. (2010)	Predict the spread in the second season of the 2009 pandemic using the model by Carrat et al. (2006)
Difference Equations (Homogeneous mixing)	Rvachev and Longini (1985)	Forecast the global spread of Hong Kong flu based on estimated parameters from Hong Kong
	Grais et al. (2003)	Study the effect of current international airline traffic by simulating the 1968 pandemic with the current volume of airline traffic for 52 global cities
	Flahault et al. (2006)	Study the impact of intervention strategies such as vaccination, isolation, antiviral usage and air traffic reduction on the global spread using the model developed by Rvachev and Longini (1985)
	Larson (2007)	Investigate social distancing as an intervention strategy
Differential Equations (Homogeneous mixing)	Flahault et al. (1994)	Simulate the spread of influenza for 9 cities in Europe considering only the regular air traffic
	Ferguson et al. (2003)	Investigate neuraminidase inhibitor therapy on drug-resistant annual influenza
	Chowell et al. (2006)	Evaluate hypothetical public health measures during the 1918 influenza pandemic in Geneva, Switzerland
	Hansen and Day (2011)	Find the optimal time-varying antiviral usage strategy to minimize IAR
	Fung et al. (2012)	Study the effect of an intervention implemented during the first outbreak of a two-consecutive outbreak scenario
	Lee et al. (2012)	Study the effect of travel restrictions by a multi-city model
Simulation (Heterogeneous mixing)	Longini et al. (2005)	Study intervention strategies such as vaccination, antiviral usage and quarantine for rural Southeast Asia
	Patel et al. (2005)	Find optimal vaccine usage to minimize the number of illnesses given limited quantities of vaccine
	Wu et al. (2006)	Investigate household-based interventions such as quarantine, contact tracing, antiviral usage and isolation for Hong Kong
	Germann et al. (2006)	Study intervention policies such as travel restrictions, vaccine and antiviral usage for the US
	Ferguson et al. (2006)	Study intervention strategies such as vaccine and antiviral usage, isolation, quarantine, school or workplace closure, travel restrictions for Great Britain and the US
	Das et al. (2008)	Study the impact of an influenza pandemic from several perspectives such as infected, dead, healthcare cost and lost wages
	Halloran et al. (2008)	Study intervention strategies such as antiviral treatment, quarantine, school closure, community and workplace social distancing for Chicago
	Lee et al. (2010)	Study the prioritization recommendations for vaccine allocation in Washington, DC, metropolitan region
	Dimitrov et al. (2011)	Study the distribution of national antiviral stockpile across 100 largest metropolitan areas in the US

2. Natural Disease History Parameters

Table OS-2 Natural disease history parameters.

Parameter	Value	References
p_A	0.4 for working adults (19 to 64) and 0.25 for others	Ferguson et al. (2003), Germann et al. (2006), Longini et al. (2005), Wu et al. (2006)
p_H	0.18 for children between 0 and 5, 0.12 for elderly (65+) and 0.06 for others	Longini et al. (2005), Wu et al. (2006)
p_D	0.344 for elderly and children between 0 and 5 and 0.172 for others	Carrat et al. (2006), Wu et al. (2006)
Duration of $E + I_P$	Weibull distribution with mean 1.48 days (including an offset of 0.5 days) and standard deviation 0.47	Ferguson et al. (2005), Wu et al. (2006)
Duration of I_P	0.5 days	Ferguson et al. (2005), Wu et al. (2006)
Duration of I_S	Exponential distribution with mean 2.7313 days	Wu et al. (2006)
Duration of I_A	Exponential distribution with mean 1.63878 days	Wu et al. (2006)
Duration of I_H	Exponential distribution with mean 14 days	Ferguson et al. (2005), Wu et al. (2006)

3. Details of Disease Spread Model

In this section, we explain the parameters used in the disease spread model. Workplace sizes are generated using a Poisson distribution with mean 20 and maximum workplace size assumed to be 1000 (similar to Germann et al. (2006)). The working adults mix with other working adults depending on the tract-to-tract worker flow data (U.S. Census Data, 2000). The average peer group sizes are 14, 20 and 30 for kindergarten, elementary schools and secondary schools, respectively (Georgia Accrediting Commission, 2008).

At the beginning of the simulation, every individual is assumed to be susceptible. We introduce an initial number (30) of infected individuals randomly to the community to represent the entrance of the virus to the population (similar results were obtained for varying numbers of initially infected individuals). The length of the simulation is 365 days which is sufficient to capture the spread of the disease.

The coefficient of transmission (β), relative hazards of an infected individual at presymptomatic and asymptomatic stages (h_P and h_A) to symptomatic stage and relative hazards in peer groups and community (h_G and h_C) to households are used to define different disease settings (Wu et al., 2006). For example, h_P is defined as the hazard of an individual during the presymptomatic stage relative to that during the symptomatic stage. As the base case, we take the hazard of an individual

at symptomatic stage, h_S , and the hazard in households, h_H , as 1. Extending Wu et al. (2006), we make age-based adjustments to the calculation of these parameters. Let r_{XY} be the average number of people infected in Y by an individual who is at stage X where Y is the household (H), peer group (G) or the community (C) and X is the presymptomatic (P), asymptomatic (A) or symptomatic (S) stage. The r_{XY} values are calculated as follows:

$$r_{PH} = \sum_{n=1}^7 p_n(n-1)(1 - \phi_P(\frac{h_P\beta}{2n}))$$

$$r_{AH} = p_A \sum_{n=1}^7 p_n(n-1)\phi_P(\frac{h_P\beta}{2n})(1 - \phi_A(\frac{h_A\beta}{2n}))$$

$$r_{SH} = (1 - p_A) \sum_{n=1}^7 p_n(n-1)\phi_P(\frac{h_P\beta}{2n})(1 - \phi_S(\frac{\beta}{2n}))$$

$$r_{PG} = (q_1n_1 + q_2n_2 + q_3n_3 + q_4n_4 + q_5n_5)(1 - \phi_P(\frac{h_P h_G \beta}{2}))$$

$$r_{AG} = p_A(q_1n_1 + q_2n_2 + q_3n_3 + q_4n_4 + q_5n_5)\phi_P(\frac{h_P h_G \beta}{2})(1 - \phi_A(\frac{h_A h_G \beta}{2}))$$

$$r_{SG} = (1 - p_A)((q_1n_1 + q_2n_2 + q_3n_3)\phi_P(\frac{h_P h_G \beta}{2})(1 - \phi_S(0)) + (q_4n_4 + q_5n_5)\phi_P(\frac{h_P h_G \beta}{2})(1 - \phi_S(\frac{h_G \beta}{4})))$$

$$r_{PC} = N(1 - \phi_P(\frac{h_P h_C \beta}{N}))$$

$$r_{AC} = p_A N \phi_P(\frac{h_P h_C \beta}{N})(1 - \phi_A(\frac{h_A h_C \beta}{N}))$$

$$r_{SC} = (1 - p_A) N \phi_P(\frac{h_P h_C \beta}{N})(1 - \phi_S(\frac{h_C \beta}{N})),$$

where q_i is the proportion of population in age group i for $i \in \{1, 2, 3, 4, 5\}$, and p_A is the probability that a presymptomatic individual does not develop symptoms. Using the probability of not developing symptoms defined for each age group in Section 2 of the Online Supplement, p_A is calculated as follows:

$$p_A = 0.25q_1 + 0.25q_2 + 0.25q_3 + 0.40q_4 + 0.25q_5.$$

n_i is the average size of peer groups for age group i . In our model, maximum household size is 7 (U.S. Census Data, 2000). p_n is the probability that an individual lives in a household size of n , and N is the total number of people in the considered area. $\phi_P(h)$ is the probability that an infection does not occur between two individuals during the presymptomatic phase for a constant hazard of infection h . $\phi_A(h)$ and $\phi_S(h)$ are the similar probabilities of no infection during the asymptomatic and symptomatic phases for constant hazard of infection h . In addition, $\phi_X(h)$ is defined as follows:

$$\phi_X(h) = E(e^{-hD_X}) = \int_0^{\infty} e^{-ht} f_X(t) dt,$$

where the duration (D_X) of disease stage X for $X \in \{P, A, S\}$ is defined by the probability density function f_X for which values are given in Section 2 of the Online Supplement.

To make it easier to understand, we explain one of the equations above. r_{SH} is the average number of people an infected individual infects in her/his household while s/he is at symptomatic stage. It is calculated by finding the average number of people in her/his household ($p_n(n-1)$) and multiplying this number by the probability that no infection occurs at presymptomatic stage ($\phi_P(\frac{h_P\beta}{2n})$) and infection occurs at symptomatic stage ($1 - \phi_S(\frac{\beta}{2n})$). This term is summed over different household sizes and finally multiplied by the probability of developing symptoms ($1 - p_A$). Here, $\phi_P(\frac{h_P\beta}{2n})$ gives us the probability that no infection occurs at the presymptomatic stage because $\frac{h_P\beta}{2n}$ is the relative hazard of the infected individual for every other individual. The term $h_P\beta$ gives the total relative hazard and this term is divided by n to find the relative hazard for each individual and then divided by 2 since the individuals in the same household interact only for 12 hours out of 24 hours.

The r_{XY} values are used to calculate the following disease parameters. R_0 is the average number of secondary cases generated by an infectious individual in a totally susceptible population. θ is the proportion of transmission that occurs at either presymptomatic or asymptomatic stage. ω is the proportion of infections generated by individuals who are never symptomatic. γ is the proportion of transmission that occurs outside the households. δ is the proportion of transmission outside the home that occurs in the community.

$$R_0 = \sum_{X \in \{P, A, S\}} \sum_{Y \in \{H, G, C\}} r_{XY}$$

$$\theta = \frac{\sum_{X \in \{P, A\}} \sum_{Y \in \{H, G, C\}} r_{XY}}{R_0}$$

$$\omega = \frac{\sum_{Y \in \{H, G, C\}} r_{AY} + p_A r_{PY}}{R_0}$$

$$\gamma = \frac{\sum_{X \in \{P, A, S\}} \sum_{Y \in \{G, C\}} r_{XY}}{R_0}$$

$$\delta = \frac{\sum_{X \in \{P, A, S\}} r_{XC}}{\sum_{X \in \{P, A, S\}} \sum_{Y \in \{G, C\}} r_{XY}}$$

After setting these five parameters, the coefficient of transmission (β) and the relative hazards (h_P, h_A, h_G, h_C) can be calculated. We assumed that θ is 0.3 and ω is 0.15 (Wu et al., 2006) and 70% of the infections occur outside the household and half of these infections occur within the peer groups. These estimates are consistent with the estimates in Ferguson et al. (2006).

In the disease spread model, we simulate the time of next infection and choose the individual that will be infected. The following infection time is generated by calculating the “instantaneous force of infection” for each individual (Wu et al., 2006) using the parameters $(\beta, h_P, h_A, h_G, h_C)$, which are discussed above. We have adjusted the calculation of force of infection for our age-based model using the age-based parameters (see below for the calculation of the force of infection). After the infected individual is selected, the disease progresses according to the natural history with the assumed transition times and probabilities for influenza.

In our model, we assume that the relative infectivity of the children (0 to 18) compared to adults (19+) is 1.5 and the relative susceptibility of the children compared to adults is 1.15 (Carrat et al., 2006). The relative infectivity values are adjusted from the corresponding probability of transmission values between children and adults proposed by Carrat et al. (2006). The susceptibility and infectivity parameters are normalized so that the expected susceptibility of an individual is 1.0, and the expected infectivity of an individual is 1.0, h_P and h_A for symptomatic, presymptomatic and asymptomatic cases, respectively.

Using these parameters, the force of infection experienced by the i^{th} individual during the day (λ_i^D) and during the night (λ_i^N) are calculated as follows:

$$\lambda_i^D = S_i \sum_{j=1}^N (\delta_{ij}^G m_j \epsilon_j h_G \beta + \delta_{ij}^C \frac{m_j h_C \beta}{N_i}),$$

$$\lambda_i^N = S_i \sum_{j=1}^N (\delta_{ij}^H \frac{m_j \beta}{n_i^{HA}} + \delta_{ij}^C \frac{m_j h_C \beta}{N_i}).$$

In the above equations, S_i is the relative susceptibility of the individual and

$$S_i = \begin{cases} S_C, & \text{if individual } i \text{ is a child} \\ S_A, & \text{otherwise.} \end{cases}$$

Let q_A be the proportion of adults in our population, then S_C and S_A can be calculated using the following equations.

$$(1 - q_A)S_C + q_A S_A = 1.0$$

$$S_C = 1.15 S_A$$

N_i is the number of individuals in the i^{th} individual's community and n_i^{HA} is the active household size of this individual where dead and hospitalized individuals are not counted. δ_{ij}^H , δ_{ij}^G and δ_{ij}^C are the indicator functions defined for households, peer groups and community, respectively.

$$\delta_{ij}^H = \begin{cases} 1, & \text{if individuals } i \text{ and } j \text{ are in the same household} \\ 0, & \text{otherwise} \end{cases}$$

$$\delta_{ij}^G = \begin{cases} 1, & \text{if individuals } i \text{ and } j \text{ are in the same peer group} \\ 0, & \text{otherwise} \end{cases}$$

$$\delta_{ij}^C = \begin{cases} 1, & \text{if individuals } i \text{ and } j \text{ are in the same community} \\ 0, & \text{otherwise} \end{cases}$$

ϵ_j is the indicator variable showing whether j^{th} individual withdraws from work or school.

$$\epsilon_j = \begin{cases} 0, & \text{with probability 1.0 if individual } j \text{ is a symptomatic child} \\ 0, & \text{with probability 0.5 if individual } j \text{ is a symptomatic adult} \\ 1, & \text{otherwise} \end{cases}$$

Finally, m_j (infectivity of individual j) is defined as follows:

$$m_j = \begin{cases} I_X^C, & \text{if } j \text{ is a child at stage } X \\ I_X^A, & \text{if } j \text{ is an adult at stage } X \\ 0, & \text{otherwise} \end{cases}$$

where I_X^C and I_X^A are the infectivity of an infected child and an infected adult at stage X (for $X \in \{P, A, S\}$), respectively. The values of these infectivity parameters are calculated as follows by using the expected relative hazard of an individual.

$$(1 - q_A)I_X^C + q_A I_X^A = \begin{cases} h_P, & \text{if } X = P \\ h_A, & \text{if } X = A \\ 1.0, & \text{if } X = S \end{cases}$$

$$I_X^C = 1.5I_X^A$$

4. Comparison of Our Disease Spread Model and the Models in the Literature

Table OS-3 Comparison of the proposed model with the ones in the literature.

Reference	Natural History	Spatial Component	Age-Based	Night/Day Differentiation
Wu et al. (2006)	Detailed SEIR	No	No	No
Ferguson et al. (2005), Ferguson et al. (2006), Longini et al. (2005), Patel et al. (2005)	SEIR	Yes	Yes	No
Germann et al. (2006)	SEIR	Yes	Yes	Yes
Our Model	Detailed SEIR	Yes	Yes	Yes

5. Model Validation

Table OS-4 Calibrated parameters to achieve the age-specific clinical attack rates of the 1957 pandemic.

Parameter	Calibrated	Original
Workplace sizes	30	20
p_A for elderly	0.5	0.25
Proportion of community infections in total number of outside home infections (θ)	0.2	0.5
Relative susceptibility of children	0 to 5	1.8
	6 to 11	1.7
	12 to 18	1.6

Table OS-5 Comparison of age-specific clinical attack rates.

Age Group	Our Model	1957 Pandemic
1 (0 to 5)	32.62%	32.17%
2 (6 to 11)	35.18%	35.02%
3 (12 to 18)	39.08%	38.44%
4 (19 to 64)	22.07%	22.24%
5 (65+)	10.45%	10.00%
Total	24.72%	24.72%

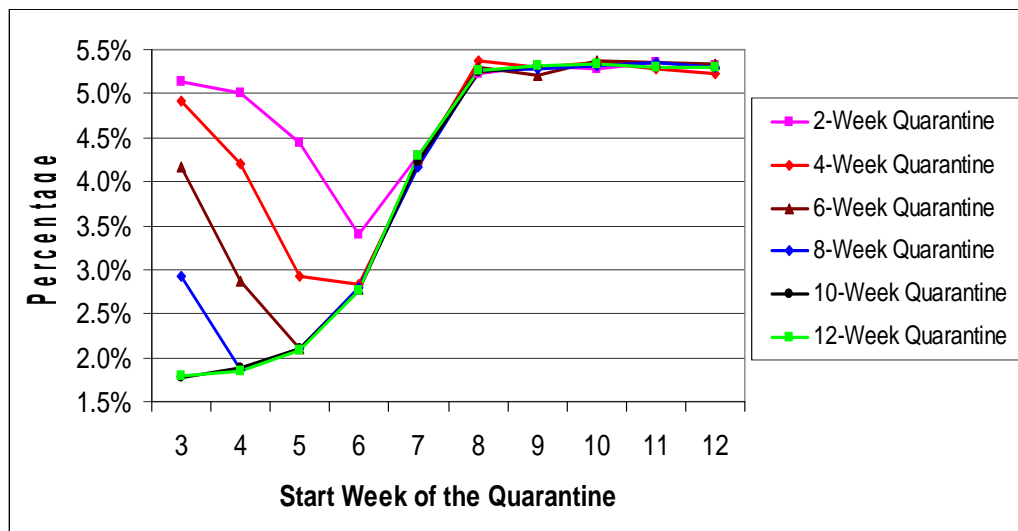
6. Intervention Policy: Voluntary Quarantine

In this section, we investigate the effect of voluntary quarantine for several R_0 values. Under voluntary quarantine, an infected individual and her/his household members are encouraged to stay home until all symptoms disappear from the household members. We assume 50% compliance, that is, individuals in the quarantined households comply with the quarantine independently with probability 0.5. Since quarantined people interact more with other individuals in their households, the risk of getting infection within the households is doubled for quarantined households, consistent with Ferguson et al. (2006), Longini et al. (2005) and Wu et al. (2006). We investigate the impact of voluntary quarantine on the spread of the disease.

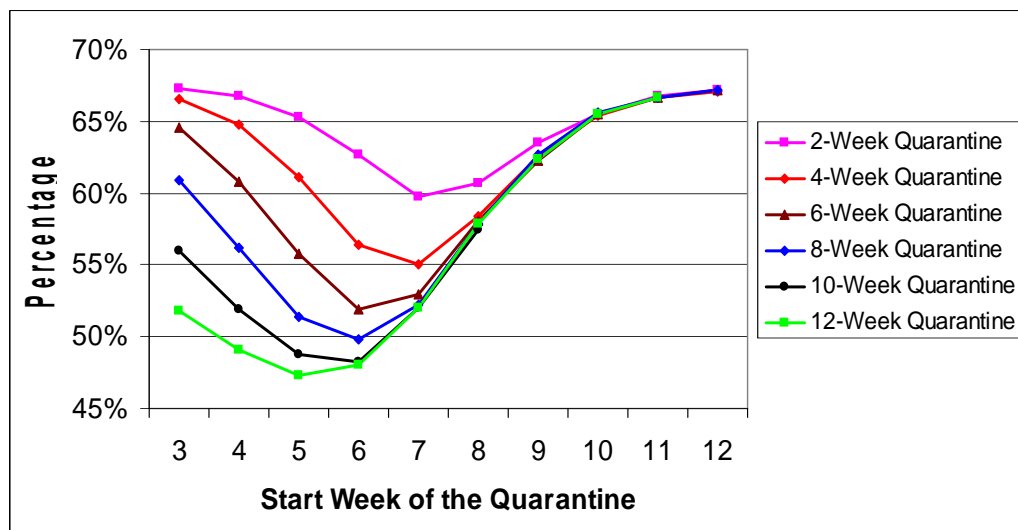
Different from other papers in the literature, we consider the case where voluntary quarantine is active for a limited period of time. An influenza pandemic may last up to one year (Ferguson et al., 2005; Longini et al., 2005; Wu et al., 2006), and the papers in the literature assume that voluntary quarantine remains active during the entire course of the disease. We consider a limited-time voluntary quarantine since prolonged quarantine times may have a negative impact on the public morale

or may be difficult to sustain for the full outbreak (e.g., one year). Social distancing measures such as voluntary quarantine isolate people or limit public gatherings, which are indicators of normal life and help maintain public morale (Northern Ireland Department of Health, Social Services and Public Safety, 2009). According to a survey (Hawryluck et al., 2004) and empirical research (Cava et al., 2005) based on the SARS experience in Toronto, people may develop emotional problems during and after the quarantine. Thus, it is in the public officials' interest to obtain maximum benefit out of voluntary quarantine by keeping the disruption in people's normal lives at a minimum. When voluntary quarantine is encouraged (or promoted via educational materials) for a limited time, the start time and the duration are important decision variables, which have not been studied in the literature. In our analysis, quarantine is active for a limited period of time (2 to 12 weeks), although note that the actual quarantine time for a given household is much shorter, e.g., 1 to 3 weeks, depending on how many members in the household get infected and develop symptoms. Quarantine ends after this period and all the individuals are released regardless of their infection status.

Both the timing and the length of the quarantine are important in order to obtain the maximum benefit. We are particularly interested in the impact of voluntary quarantine on peak prevalence, which is related to the maximum capacity for the resources that governments and NGOs may need to serve the needs of the public, and the IAR, which is related to the total amount of resources required during the course of the disease (Rahmandad and Sterman, 2008). Figure OS-1 shows the effect of the quarantine on the peak prevalence and IAR for $R_0 = 1.8$ as a function of the start time and length. From Figure OS-1(a), for a 2-week quarantine, the peak prevalence is lowest (3.40%) when the quarantine starts at the beginning of the sixth week. On the other hand, for a 12-week quarantine, the best start time in terms of peak prevalence is the beginning of the third week, and in this case the peak prevalence is 1.79%. Even a 12-week quarantine has no effect on the peak prevalence if the timing of the quarantine is not appropriate (e.g., week 7). From Figure OS-1(b), IAR is minimized at 59.71% if a 2-week quarantine is implemented at the beginning of week 7. For a 12-week quarantine, IAR is lowest (47.32%) if it is implemented at the beginning of week 5. *In general, as the length of the quarantine increases, the optimal start time (for minimizing the peak prevalence or IAR) decreases.* Note that the optimal start time of a quarantine is also related to the timing of the peak of the disease, which depends on R_0 . As R_0 increases, it is best to start a limited-time quarantine earlier to reduce peak prevalence. Handel et al. (2007) also provide a similar observation about minimizing the overshoot effect of epidemic dynamics.



(a) Peak prevalence.



(b) Infection attack rate.

Figure OS-1 Effect of timing and length of quarantine on the peak prevalence and infection attack rate.

During the course of the pandemic, estimating the disease spread parameters accurately, and thus, determining the exact duration and start time of the quarantine can be difficult. Therefore, voluntary quarantine can be announced a few weeks before the estimated optimal timing which can be compensated by extending the length of the quarantine. For example, for $R_0 = 1.8$, if a 6-week voluntary quarantine is started at the beginning of week 3 (instead of the beginning of week 5 to maximize the reduction in peak prevalence), similar peak prevalence (2.08%) can be

achieved at the cost of a 3-week extension of the quarantine. However, delaying the start of the quarantine beyond the optimal timing may have severe consequences. For the previous example, if the quarantine is started 2 weeks late, the peak prevalence cannot be lower than 4.28%. Finally, as the length of the quarantine increases, starting the quarantine early is better than starting it late. *Both the peak prevalence and IAR decrease as the length of the quarantine increases, but there is a diminishing rate of return.* The peak prevalence (in Figure OS-1(a)) in an 8-week quarantine is almost equal to that of a 12-week quarantine. For different R_0 values, Table OS-6 summarizes the results for an 8-week quarantine (with the objective of minimizing the peak prevalence), which is the break point where the diminishing rate of return is clearly observed for the peak prevalence. We observe that for moderate to high R_0 values, i.e, 1.8 and 2.1, an 8-week quarantine has about the same impact on peak prevalence compared to a quarantine that is in effect during the entire course of the disease. Imposing the quarantine during the entire course of the disease versus for only 8 weeks versus no quarantine results in IARs of 42%, 56%, and 67.5%, respectively, for $R_0 = 1.8$ and 58.5%, 63%, and 78%, respectively, for $R_0 = 2.1$. That is, for moderate to high R_0 values, an 8-week quarantine captures most of the benefits in reducing the peak prevalence and IAR. However, for $R_0 = 1.5$, there is a significant reduction in peak prevalence (from 0.80% to 0.30%) and IAR (from 40.5% to 17.4%) if the quarantine is imposed during the entire course of the disease instead of an 8-week time interval. Hence, for low R_0 values (where the epidemic curve is smoother with a smaller peak even under no intervention), a longer quarantine can be more beneficial.

Table OS-6 Summary of results under an 8-week quarantine with 50% compliance.

R_0	Quarantine Start Week	Peak Prevalence	Peak Day	CAR	IAR	Mortality Rate
1.5	7	0.80%	52	26.52%	40.46%	0.47%
1.8	4	1.86%	63	36.82%	56.14%	0.66%
2.1	3	3.97%	49	41.26%	62.87%	0.75%

In a voluntary quarantine, the reduction in the peak prevalence is high when compared to the reduction in other performance measures. For $R_0 = 1.8$, an 8-week quarantine with optimal timing reduces the peak prevalence by 64.71%, CAR by 16.70%, IAR by 16.82% and the mortality rate by 17.22%. Keeping infected individuals at home decreases their interactions with the others outside, but in the long run susceptible individuals may have an interaction with other infected individuals in the community after the quarantine is released. The reductions in the peak prevalence, CAR, IAR and mortality rate for different R_0 values for an 8-week quarantine with the optimal start time are provided in Table OS-7. Depending on the start time and the duration of the quarantine,

we sometimes observe two peaks in prevalence. This occurs because the spread of the disease slows down during the quarantine but speeds up again after the quarantine is released. However, even in these cases, the highest of the two peaks as well as the IAR are lower under quarantine versus no quarantine. In addition, delaying the peak offers tremendous opportunities for better preparedness and response (including the potential development of an appropriate flu vaccine).

Table OS-7 Reductions in the performance measures for an 8-week quarantine.

R_0	Peak Prevalence	CAR	IAR	Mortality Rate
1.5	67.74%	18.40%	18.51%	18.22%
1.8	64.71%	16.70%	16.82%	17.22%
2.1	50.44%	19.52%	19.68%	19.68%

Ferguson et al. (2006), Longini et al. (2005) and Wu et al. (2006) also study voluntary quarantine as an intervention policy. The comparison of the results is available in Table OS-8. Different from our limited-time assumption, all the other papers assume that the quarantine is active during the entire course of the disease, which may not be practical. Our results indicate that as R_0 increases, an 8-week quarantine (if started at the right time) is almost as effective as a quarantine that is imposed during the entire course of the disease.

Table OS-8 Comparison of the quarantine results with the results in the literature.

Reference	R_0	Quarantine		Decrease in	Decrease in
		Start - End	Compliance Rate	Peak Prevalence	IAR
Longini et al. (2005)	1.4 to 1.7	Day 14 - Day 365	70%	Not reported	99%
Ferguson et al. (2006)	1.7 to 2.0	Day 1 - Day 365	50%	25 to 26%	14.7 to 18.5%
Wu et al. (2006)	1.8	Day 1 - Day 365	50%	70%	33%
Our model	1.8	Day 21 - Day 77	50%	64.71%	16.82%

We also ran experiments with other compliance rates (25%, 75%, 100%). We observe that as the compliance rate increases, peak prevalence and IAR decrease. However, for high compliance rates, e.g., 75% and 100%, we may observe two peaks depending on the R_0 value and the duration of the quarantine. After a quarantine with high compliance rate is released, if the virus is still active, the number of infections increases again resulting in a second peak (the maximum of the two peaks is smaller than the peak in the no intervention case). Therefore, higher compliance rates provide a good opportunity to decrease peak prevalence and IAR significantly, and more importantly, they offer more time for preparedness by delaying the (second) peak.

7. Single-Period Add-Drop Heuristic (SPAD)

- 1: **for** $t = 1$ to T **do**
- 2: **for** $l = 1, 2$ **do**
- 3: Let PO^{POD} and PO^{MF} be the set of open PODs and major facilities in period $t - 1$, respectively (These sets are determined by using the decisions made in the previous period which is period $t - 1$). Let FO^{POD} and FO^{MF} be the set of PODs and major facilities that are planned to be open in the future.
- 4: **if** $l == 1$ **then**
- 5: Set the demand of node k (D_k) to $\sum_{j=t+1}^{T-1} \frac{D_{kj}}{2^{j-t}} + \frac{D_{kT}}{2^{T-1-t}}$
- 6: $FO^{POD} = FO^{MF} = \emptyset$
- 7: **else**
- 8: $D_k = D_{kt}$
- 9: **end if**
- 10: **for** $i \in N_2$ **do**
- 11: Set the total serving cost to zero : $TSC_i^{MF} = 0$.
- 12: Calculate the cost of serving a unit of demand : $USC_i^{MF} = c_u^1 \min_{s \in N_1} d_{si}$.
- 13: **end for**
- 14: **for** $j \in N_3$ **do**
- 15: Set the total serving cost to zero : $TSC_j^{POD} = 0$.
- 16: Calculate the cost of serving a unit of demand : $USC_j^{POD} = \min_{i \in PO^{MF}} (c_u^2 d_{ij} + USC_i^{MF})$.
- 17: **end for**
- 18: Define D'_k and C'_j as the remaining demand of demand node k and the remaining capacity of POD j , respectively. Set the initial values: $D'_k = D_k$ for all $k \in N_4$, and $C'_j = C_j$ for all $j \in N_3$.
- 19: Starting from a demand node, assign the demand of this node to the closest POD as the capacity permits. That is, for demand node k , assuming the closest POD is j , assign $\min\{D'_k, C'_j\}$ amount of demand to POD j . Then, update the remaining capacity of POD j and the remaining demand of demand node k . If there is remaining demand, find the next closest POD that has a positive remaining capacity. Otherwise, continue with the next demand node until all the demand is assigned to PODs.
- 20: CO^{POD} : Set of PODs that have been assigned a demand.
- 21: POD_{kj} : Demand amount of demand node k assigned to POD j for all $k \in N_4, j \in N_3$.

22: Update the total serving cost of each POD that has a positive demand assigned to it :

$$TSC_j^{POD} = F_j + \sum_{k \in N_4} POD_{kj} (d_{jk} c_u^3 + USC_j^{POD})$$

23: **repeat**

24: **for** $j \in CO^{POD}$ **do**

25: **if** $j \in PO^{POD} \cup FO^{POD}$ **then**

26: $CS_j^{POD} = TSC_j^{POD}$

27: **else**

28: $CS_j^{POD} = TSC_j^{POD} + f_j + g_j$

29: **end if**

30: **end for**

31: **for** $j \in CO^{POD}$ **do**

32: **repeat**

33: Find the closest POD, say j' , with an assigned demand.

34: Assign the demand of POD j to POD j' as the capacity permits. Let $POD_{kj}^{j'}$ be the demand of demand node k that has been reassigned to POD j' instead of POD j .

35: Update the cost saving : $CS_j^{POD} = CS_j^{POD} - \sum_{k \in N_4} POD_{kj}^{j'} (d_{jk} c_u^3 + USC_{j'}^{POD})$

36: **until** All the demand of POD j can be assigned to other open PODs

37: **if** All the demand of POD j cannot be assigned to other open PODs **then**

38: Set $CS_j^{POD} = 0$.

39: **end if**

40: **end for**

41: Determine the POD to be closed : $j^* = \operatorname{argmax}_{j \in CO^{POD}} CS_j^{POD}$.

42: **if** $CS_{j^*}^{POD} > 0$ **then**

43: Close POD j^* and assign its demand to the PODs determined while calculating the cost saving.

44: Update the set of open PODs (CO^{POD}).

45: **end if**

46: **until** $CS_{j^*}^{POD} \leq 0$

47: **if** $l == 1$ **then**

48: $FO^{POD} = CO^{POD}$

49: **end if**

50: Let D_j^{POD} denote the amount of demand assigned to POD j for all $j \in CO^{POD}$.

51: Assign all the demand of the PODs to the major facilities as the capacity permits (similar to Step 19).

52: CO^{MF} : Set of major facilities that have been assigned a demand.

53: MF_{ji} : Demand amount of POD j assigned to major facility i for all $i \in N_2, j \in CO^{POD}$.

54: Update the total serving cost of each major facility that has a demand assigned to it :

$$TSC_i^{MF} = F_i + \sum_{j \in CO^{POD}} MF_{ji}(d_{ij}c_u^2 + USC_i^{MF})$$

55: **repeat**

56: **for** $i \in CO^{MF}$ **do**

57: **if** $i \in PO^{MF} \cup FO^{MF}$ **then**

58: $CS_i^{MF} = TSC_i^{MF}$

59: **else**

60: $CS_i^{MF} = TSC_i^{MF} + f_i + g_i$

61: **end if**

62: **end for**

63: **for** $i \in CO^{MF}$ **do**

64: **repeat**

65: Find the closest major facility, say i' , with an assigned demand.

66: Assign the demand of major facility i to major facility i' as the capacity permits.
Let MF_{ji}' be the demand of POD j that has been reassigned to major facility i' instead of major facility i .

67: Update the cost saving : $CS_i^{MF} = CS_i^{MF} - \sum_{j \in CO^{POD}} MF_{ji}'(d_{ij}c_u^2 + USC_{i'}^{MF})$

68: **until** All the demand of major facility i can be assigned to other open major facilities

69: **if** All the demand of major facility i cannot be assigned to other open major facilities
then

70: Set $CS_i^{MF} = 0$.

71: **end if**

72: **end for**

73: Determine the major facility to be closed : $i^* = \operatorname{argmax}_{i \in CO^{MF}} CS_i^{MF}$.

74: **if** $CS_{i^*}^{MF} > 0$ **then**

75: Close major facility i^* and assign its demand to the major facilities determined while calculating the cost saving.

76: Update set of open major facilities (CO^{MF}).

77: **end if**

78: **until** $CS_{i^*}^{MF} \leq 0$

79: **if** $l == 1$ **then**

80: $FO^{MF} = CO^{MF}$

81: **end if**

82: **end for**

83: CO^{MF} and CO^{POD} are the set of major facilities and PODs that will be open in period t .

84: **end for**

8. Map of the Metropolitan Atlanta Area



Figure OS-2 Counties in the metropolitan Atlanta area by population.

9. Additional Results on the Performances of the Heuristics

Table OS-9 Results of the additional experiments to test the performances of the heuristics with CPU time in seconds and gap compared to the best lower bound. DN: Demand Node, POD: Point-of-Delivery, MF: Major Facility, SP: Supply Point.

Instance	Algorithm	Shipment Cost					
		Low		Medium		High	
		CPU	GAP	CPU	GAP	CPU	GAP
71 DN, 41 POD, 5 MF, 10 SP	MH	0.2	5.36%	0.2	6.77%	0.2	7.14%
	SPAD	0.3	5.04%	0.2	4.58%	0.2	4.58%
	SPO	15.3	1.48%	7.9	2.10%	7.1	3.27%
	SPH	4.5	2.61%	2.9	2.68%	2.8	3.38%
	Best Int. Sol.	4089.9	0.00%	256.5	0.00%	111.3	0.00%
71 DN, 36 POD, 5 MF, 15 SP	MH	0.1	3.35%	0.1	4.77%	0.1	6.32%
	SPAD	0.2	2.80%	0.2	2.92%	0.2	2.45%
	SPO	10.2	2.17%	6.1	2.02%	5.2	1.57%
	SPH	3.2	1.91%	2.5	2.26%	2.0	1.51%
	Best Int. Sol.	2232.9	0.00%	184.9	0.00%	54.7	0.00%
71 DN, 36 POD, 10 MF, 10 SP	MH	0.2	7.38%	0.2	8.18%	0.2	11.81%
	SPAD	0.2	7.44%	0.2	5.90%	0.2	7.39%
	SPO	11.8	2.47%	7.8	2.41%	7.5	4.67%
	SPH	3.8	3.97%	3.0	3.75%	3.3	4.42%
	Best Int. Sol.	15542.5	0.53%	2509.8	0.00%	633.7	0.00%
167 DN, 41 POD, 5 MF, 10 SP	MH	0.5	5.36%	0.5	4.23%	0.5	3.73%
	SPAD	0.5	2.84%	0.5	1.85%	0.5	1.81%
	SPO	46.0	4.42%	23.1	1.47%	14.1	0.55%
	SPH	10.0	2.54%	7.0	1.29%	5.2	0.79%
	Best Int. Sol.	16707.6	0.74%	824.8	0.00%	192.5	0.00%
167 DN, 36 POD, 5 MF, 15 SP	MH	0.5	2.69%	0.5	1.93%	0.5	1.67%
	SPAD	0.5	3.03%	0.5	0.97%	0.5	1.49%
	SPO	25.1	0.85%	14.6	0.65%	10.6	1.24%
	SPH	7.6	1.81%	5.4	0.60%	4.3	1.07%
	Best Int. Sol.	12418.6	0.11%	927.2	0.00%	163.8	0.00%
167 DN, 36 POD, 10 MF, 10 SP	MH	0.4	9.56%	0.4	7.41%	0.4	6.14%
	SPAD	0.5	7.56%	0.5	3.19%	0.5	2.75%
	SPO	28.0	3.80%	20.1	1.94%	13.5	2.05%
	SPH	8.7	5.59%	5.9	2.11%	4.8	1.67%
	Best Int. Sol.	26347.6	2.28%	5313.4	0.00%	1652.9	0.00%

10. Details of Robustness Analysis

In the univariate sensitivity analysis, we test the effect of uncertainty in the values of the parameters listed in Table OS-10.

Table OS-10 Parameters to be tested for the robustness of STAT, DYN(1), DYN(2), and STAT-2.

Index	Parameter	Explanation
1	p_A	Probability of not developing symptoms
2	p_H	Probability of hospitalization
3	p_D	Probability of death
4	Duration of I_E	Duration of exposed stage
5	Duration of I_P	Duration of presymptomatic stage
6	Duration of I_S	Duration of symptomatic stage
7	Duration Ratio	Ratio of symptomatic duration to asymptomatic duration
8	θ	Proportion of transmission that occurs at presymptomatic or asymptomatic stage
9	ω	Proportion of infections generated by individuals who are never symptomatic
10	γ	Proportion of transmission that occurs outside the households
11	δ	Proportion of transmission outside the home that occurs in the community
12	Infectivity	Relative infectivity of children
13	Susceptibility	Relative susceptibility of children
14	R_0	Reproductive number

In Table OS-11, we present the parameter values used in the benchmark case (representing the actual disease parameters in the simulation) under column “Base Value” and other estimates from the literature which represent the inaccurate estimates in the simulation under column “Estimated Values.”

The univariate sensitivity analysis results for STAT, DYN(1), DYN(2), and STAT-2 are presented in Table OS-12 where the result in each cell is an average of 20 replications. The first column represents the index of the setting (the parameter for which the actual and estimated values are different, see Table OS-11). For each parameter, we tested two values (one lower and one higher than the actual value, denoted by a and b, respectively). Since the estimated and actual values of the parameters are different, some portion of the demand may not be satisfied and/or the total cost may deviate from the optimal solution as represented in columns 3 to 11 as a percentage of the cost and demand in the “Perfect Solution.”

Table OS-11 Parameter values for robustness experiments.

Index	Base Value	References	Estimated Values	References
1	0.4 for working adults 0.25 for others	Ferguson et al. (2003), Germann et al. (2006), Longini et al. (2005), Wu et al. (2006)	a. 0.25 b. 0.5	Ferguson et al. (2005), Ferguson et al. (2006)
2	0.18 for children (0 to 5), 0.12 for elderly, 0.06 for others	Longini et al. (2005), Wu et al. (2006)	a. 0.06 b. 0.18	Longini et al. (2005), Wu et al. (2006)
3	0.344 for elderly and children (0 to 5), 0.172 for others	Carrat et al. (2006), Wu et al. (2006)	a. 0.172 b. 0.344	Carrat et al. (2006), Wu et al. (2006)
4	Weibull with mean 1.48 days and std. dev. 0.47	Ferguson et al. (2005), Wu et al. (2006)	a. Mean 1 b. Mean 2	Lee et al. (2008), Yang et al. (2009)
5	0.5	Ferguson et al. (2005), Wu et al. (2006)	a. 0.25 b. 1	Atkinson and Wein (2008)
6	Exponential(2.7313)	Wu et al. (2006)	a. Exponential(1.6) b. Exponential(4.1)	Germann et al. (2006), Lee et al. (2008), Longini et al. (2005), Longini et al. (2004), Mylius et al. (2008)
7	5/3	Wu et al. (2006)	a. 1 b. 2	Ferguson et al. (2003), Yang et al. (2009)
8	0.3	Wu et al. (2006)	a. 0.1 b. 0.5	Wu et al. (2006)
9	0.15	Wu et al. (2006)	a. 0.33 of θ b. 0.8 of θ	Wu et al. (2006)
10	0.7	Ferguson et al. (2006)	a. 0.45 b. 0.8	Wu et al. (2006)
11	0.5	Ferguson et al. (2006)	a. 0.19 b. 0.57	Ferguson et al. (2006), Glass et al. (2006)
12	1.5	Carrat et al. (2006)	a. 1 b. 2	Ferguson et al. (2006), Longini et al. (2005),
13	1.15	Carrat et al. (2006)	a. 1 b. 2	Riley et al. (2003), Wu et al. (2006)
14	1.8	Ferguson et al. (2005), Wu et al. (2006)	a. 1.5 b. 2.1	Ferguson et al. (2005), Longini et al. (2005)

We present the parameter setting used in *Slow Estimated Spread* (SES) and *Fast Estimated Spread* (FES) scenarios in Table OS-13. In this table, “-” means the value of the corresponding parameter is the same as in Table OS-11, “Base Value” column due to insignificant effect in the univariate sensitivity analysis.

Table OS-13 Parameter settings for the two extreme cases for multivariate sensitivity analysis.

Setting	Parameter													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
SES	a	-	-	b	b	a	-	a	b	a	a	-	-	a
FES	b	-	-	a	a	b	-	b	a	b	b	-	-	b

11. Effect of Quarantine on the Food Distribution Supply Chain

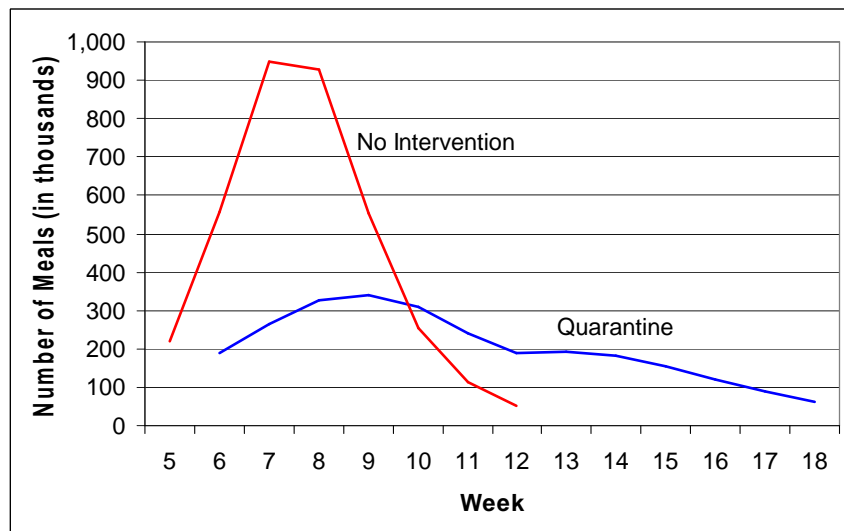
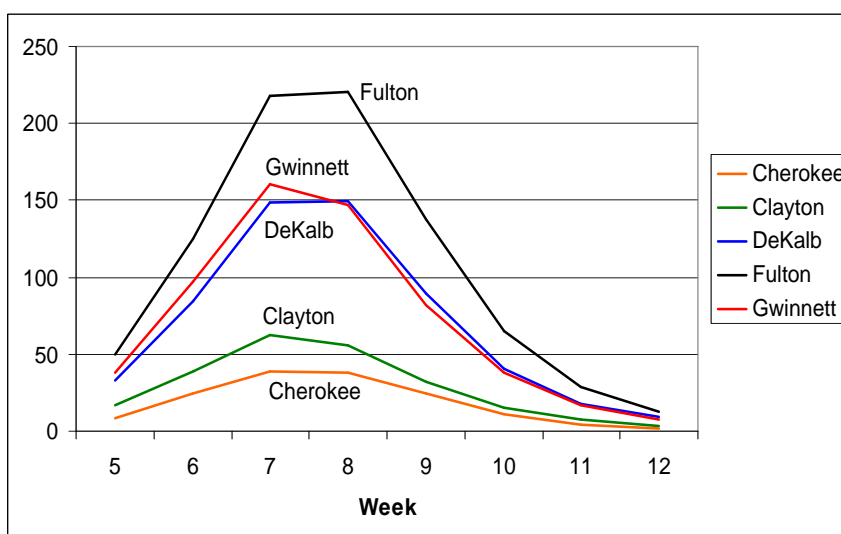
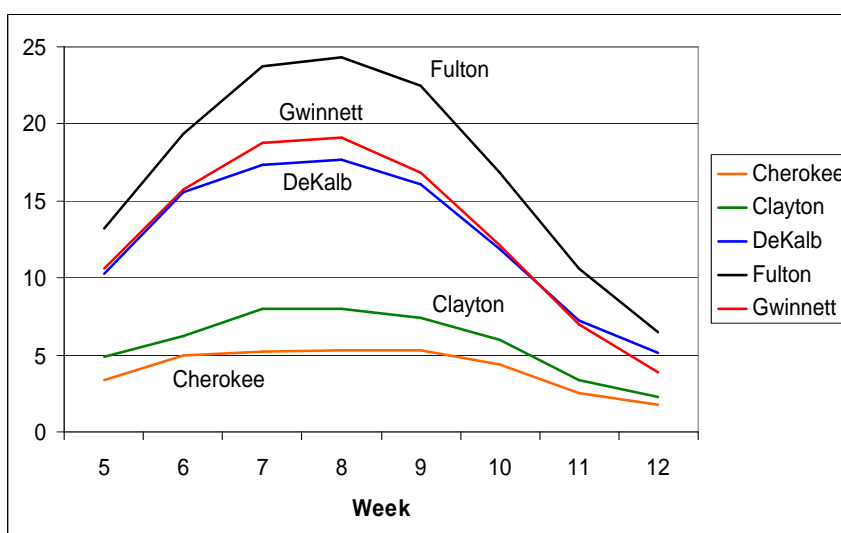


Figure OS-3 The effect of quarantine on food demand.



(a) Number of meals needed (in thousands) over time.



(b) Number of open PODs over time.

Figure OS-4 Number of meals and open PODs over time under the no intervention case.

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