1 Supplementary Methods

1.1 Derivation of Residual Degree Distribution

The full derivation for the degree distribution of the residual network model proceeds as follows:

As the extended residual network is defined by the uninfected nodes of the previous epidemic, a proportion \( \alpha \) of the infected nodes of the previous epidemic, and all the edges joining them, we define the degree distribution for the extended residual network as:

\[
p_{\text{res}}(k_r) = \frac{p_{\text{uninfected}}(k_r) + \alpha p_{\text{infected}}(k_r)}{\sum_{k_o} p_{k_o} \eta_{k_o} + \alpha \sum_{k_o} p_{k_o} (1 - \eta_{k_o})}.
\] (1)

We find that the \( p_{\text{uninfected}}(k_r) \) can be found by

\[
p_{\text{uninfected}}(k_r) = \sum_{k_o \geq k_r} p_{k_o} \eta_{k_o} p_{\text{res}}(k_r|k_o)
\] (2)

where \( p_{\text{res}}(k_r|k_o) \) is the probability that a node in the extended residual network will have degree \( k_r \) given that it had a degree of \( k \) in the original network. This condition distribution can be calculated as

\[
d p_{\text{res}}(k_r|k_o) = \binom{k_o}{k_r} (u_1 + (1 - u_1)\alpha)^{k_r} [(1 - u_1)(1 - \alpha)]^{k_o-k_r}
\] (3)

as discussed in the main text. Following Bayes rule, \( p_{\text{uninfected}}(k_r) \) is then the sum of the product of the probabilities that a node in the extended residual network has degree \( k_r \) given that it had original degree \( k_o \), \( p_{\text{res}}(k_r|k_o) \), the probability that the node of original degree \( k_o \) was uninfected in the first epidemic, \( \eta_{k_o} \), and the probability that the node had original degree \( k_o \), \( p_{k_o} \). In a similar way, \( p_{\text{infected}}(k_r) \) can be calculated as:
\[ p_{\text{res}}(k_r) = \sum_{k_o \geq k_r} p_{k_o} (1 - \eta_{k_o}) p_{\text{res}}(k_r | k_o) \]  \hspace{1cm} (4)

Thus, by substituting equations 2 and 4 into equation 1, we have:

\[ p_{\text{res}}(k_r) = \frac{\sum_{k_o \geq k_r} p_{k_o} \eta_{k_o} p_{\text{res}}(k_r | k_o) + \alpha \sum_{k_o \geq k_r} p_{k_o} (1 - \eta_{k_o}) p_{\text{res}}(k_r | k_o)}{\sum_{k_o} p_{k_o} \eta_{k_o} + \alpha \sum_{k_o} p_{k_o} (1 - \eta_{k_o})} \]  \hspace{1cm} (5)

And lastly, by substituting equations 3 into equation 5, we have:

\[ p_{\text{res}}(k_r) = \sum_{k_o \geq k_r} p_{k_o} \eta_{k_o} \binom{k_o}{k_r} (u_1 + (1-u_1)\alpha)^{k_r} (1-u_1)^{k_o-k_r+\alpha} \sum_{k_o \geq k_r} p_{k_o} (1-\eta_{k_o}) \binom{k_o}{k_r} (u_1 + (1-u_1)\alpha)^{k_r} (1-u_1)^{k_o-k_r+\alpha} \frac{\sum_k p_k \eta_k + \alpha p_k (1-\eta_k)}{\sum_k p_k \eta_k + \sum_k \alpha p_k (1-\eta_k)}. \]

2 Supplementary Analysis

2.1 Incidence Data from Influenza Pandemics

Here, we list the confidence intervals for the odds ratio of attack rates in school-age children to adults. We note that the definitions of the school-age children and adult age groups vary across the studies and sometimes slightly differ from our own.
| Year | Strain | Odds Ratio of School-Age Children to Adults [CI] | Location | Source | Data Details |
|------|--------|-----------------------------------------------|----------|--------|--------------|
| 1918 | A (H1N1) | 1.71 [0.89, 3.29] | 10 U.S. cities | Frost et al, 1929 | Data collected by the Public Health Service; influenza case counts per 100 individuals in canvassed group (which included at least 5% of each city's population) by age |
| 1918 | A (H1N1) | 1.71 [0.89, 3.29] | Baltimore | Collins et al, 1957 | cases counts per 100 individuals in canvassed group by age; illnesses counted included influenza, grippe, pneumonia and colds in bed |
| 1918 | A (H1N1) | 1.91 [1.02, 3.57] | U.S. cities | Collins et al, 1931 | cases counts per 100 individuals in canvassed group by age; illnesses counted included influenza, grippe, pneumonia and colds in bed |
| 1957 | A (H2N2) | 2.35 [1.31, 4.22] | Louisiana | Gilbert et al, 1962 | study reports summarized age-specific influenza attack rates by age from 5 high school family survey study |
| 1957 | A (H2N2) | 2.57 [1.38, 4.76] | U.K. | Gani et al, 2005 | age-specific attack rate by age |
| 1957 | A (H2N2) | 2.45 [1.35, 4.47] | Missouri | Davis et al, 1970 | study reports age-specific attack rates of ILI in families of students who attended a high school in Kansas City, MO |
| 1968 | A (H3N2) | 1.0 [0.57, 1.76] | Missouri | Davis et al, 1970 | study reports age-specific attack rates of ILI in families of students who attended a high school in Kansas City, MO |
| 2009 | A (H1N1) | 1.75 | Mexico | Secretaria de Salud | reported case counts; we normalized case counts by age group size in Mexico |
| 2009 | A (H1N1) | 5.06 | USA | CDC | reported case counts as of May 19, 2009; we normalized the case counts by age group size in the US |
| 2009 | A (H1N1) | 15.63 | Japan | Nishiura et al, 2009 | reported confirmed case counts as of June 1, 2009; we normalized case counts by age group size in Japan. |
| 1919 | A (H1N1) | 0.68 [0.25, 1.86] | Baltimore | Collins et al, 1957 | cases counts per 100 individuals in canvassed group by age; illnesses counted included influenza, grippe, pneumonia and colds in bed |
| 1959 | A (H2N2) | 0.9 [0.5, 1.64] | Louisiana | Gilbert et al, 1962 | study reports summarized age-specific influenza attack rates by age from 5 high school family survey study |
| 1959 | A (H2N1) | 0.71 [0.39, 1.31] | Ohio | CDC Report, 1962 | based on questionnaire survey of OH Dept. of Health personnel and their families in Jan 1960; reported percent ill “which includes those personals with a serious cold” |
| 1969 | A (H3N2) | 0.14 [0.02, 0.89] | unknown | Taylor, 1971 | reported rate per 100 at risk by age for influenza or ILI; based on interview by general practitioner; we note that children in this study were 5-14 year olds, and adults were 15-64 |

Tab. 1: Details on attack rate data used for Figure 3
2.2 Data on Immunity to Influenza

Here we review empirical data from literature on effects of immunity due to previous influenza infection.

| Sample Size | Subtype/Strain | Year of study | Reinfection Rate | Timescale | Source |
|-------------|----------------|---------------|------------------|-----------|--------|
| 94          | A H3N2         | 1972          | 9%               | 1 year    | Noble et al, 1974 |
| 43          | A H3N2         | 1973          | 16%              | 1 year    | Foy et al, 1976   |
| 100,000     | various        | 1984-2002     | 7.4%             | 1 year    | Finkenstadt et al, 2005 |
| 138         | A H3N2         | 1977          | 5%               | 1 year    | Pease, 1987       |
| ~600-30,000 | A H1N1         | 1918          | 6.51%            | < 1 year  | Barry et al., 2008 |

Tab. 2: Empirical data on effects of immunity due to previous influenza infection

2.3 Invasion Threshold with Immunity

When invading a population which has previously been (partially) infected by a similar strain, a pathogen faces an epidemic threshold for the a subsequent outbreak, which is a critical value of transmissibility above which a second epidemic is possible in the the same population. This critical transmissibility threshold is a function of the transmissibility of the first season pathogen, $T_1$, and the loss of immunity, $\alpha$, and can be calculated as:

$$T_{c2} = \frac{\langle k_r \rangle}{(k_r \langle k_r - 1 \rangle)}$$

where, $\langle k_r \rangle$ is defined by $\sum_k k p_{res}(k)$ and $p_{res}(k)$ is the residual network degree distribution for our urban network model. $T_2 = T_{c2}$ is equivalent to $R_e = 1$. In a naive population ($\alpha = 1$), the transmissibility of an invading pathogen can be quite low for successful invasion. As you consider scenarios with increasing immunity (i.e. as $\alpha$ decreases), the epidemic threshold for an invading pathogen is increasingly higher. In particular, Figure 1, shows the decreasing value of $T_{c2}$ with increasing $\alpha$ for a first-season epidemic of $R_0 = 1.1, 1.6, 2.1$. 
2.4 Sensitivity to Contact Patterns

The contact patterns of the populations in the pandemics of the last century (1918, 1957 and 1968) may have differed from those of today. Here, we explore sensitivity to changes in population structure of the shift that occurs in the risk of infection from an initial pandemic season to a subsequent season. We consider two probability distributions of contacts (degree distributions) different than that of our urban network model: a) the Mossong et al study [4] is an empirical survey which reports daily conversational and physical contacts of individuals in European countries, and b) the Eubank et al study [1] is a synthetic population simulated based on data from the city of Portland, Oregon that reports daily contacts. In addition to differences in contact structure, these studies incorporate differences in the demographic structure of the population. Specifically, the Eubank et al study is made up of 20% children and 51% adults, while the Mossong et al study is made up of 28% children and 48% adults.
compared with 23% children and 61% adults in our population model.

Figure 2 below shows that both populations still exhibit the shift in infection risk from high contact individuals to moderate contact individuals. Thus given higher contact rates for school-age children than adults, a shift in age-specific incidence can be expected from the prior age group to the latter.

Fig. 2: We calculate risk of infection in first and second season epidemics for populations with varying contact patterns. (A) Degree distributions from our urban network model (based on [3], the Mossong et al study [4], and the Eubank et al study [1]. (B) The degree-based risk of infection for a naive population (no prior immunity) for each network for $R_0 = 1.6$ (C) The degree-based risk of infection for a subsequent season (of $R_e = 1.05$), following a first season epidemic (of $R_0 = 1.6$) with partial immunity ($\alpha = 0.05$) for each network. The shift in risk of infection from high contact individuals to moderate contact individuals in a partially immune population is still evident.
2.5 Sensitivity to the Reproductive Number

The reproductive number for the H1N1/09 pandemic has been estimated to be between 1.22 and 2.3 [2, 6, 5]. Figure 3 shows that our results on the shift in risk of infection are robust to varying $R_0$ values.

![Figure 3: Risk of Infection for a range of initial reproductive number ($R_0$) and effective reproductive number ($R_e$) for a second season. The shift in risk of infection is still evident.](image)

2.6 Sensitivity to Demographic Changes

Here, we consider disease consequences in a population with partial immunity from a previous year as well as demographic changes. We ignore births and deaths as they occur at either end of the population age scale and do not impact contact patterns to a significant degree. Instead, we focus on aging in the population and the impact of this on population contact patterns. Specifically, we allow aging of individuals at the boundaries of age (and thus contact) classes: 4 year olds age to 5 year olds (and move from the low-contact class of toddlers to the high-contact class of school-aged children), 18 year olds age to be 19 years old (and move from the high-contact class of school-aged children to the moderate-contact class of adults), and adults of age 64 age to 65 years of age (and move from the moderate-contact class of adults to the low-contact class of elderly.) In Figure 4, we see the differences in the degree distribution of the contact networks with and without these demographic changes in populations.
with full, partial and no immunity ($\alpha = 0, 0.5, 1$). As the left panel shows, aging only impacts the degree distribution in a small way. To quantify the disease impact of these changes, we calculate the size of the epidemic in the populations with and without aging for various levels of immunity and also find that the impact is not dramatic.

Fig. 4: Impact of the demographic process of aging on the contact network structure with varying values of immunity (left panel) and on the disease consequences in the population (right panel.)

### 2.7 Comparing Stochastic Simulations to Percolation Model

Finally, we compare the predictions from our percolation-based model to stochastic simulations. The standard percolation theory framework is based solely on the degree distribution of the population contact network. Comparison to stochastic simulations allow us to quantify the impact of higher order structural properties (such as clustering in edges, degree correlations, etc.) Below, we compare the predictions from our percolation model to that of stochastic simulations for the risk of infection in the second season by degree and find congruent results.
Fig. 5: Comparison of stochastic simulations and analytical percolation model predictions for risk of infection in second season based on individual degree.

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