

Shocks to Hospital Occupancy and Mortality: Evidence from the 2009 H1N1 Pandemic *

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Existing literature suggests that hospital occupancy matters for quality of care as measured by various patient outcomes. However, estimating the causal effect of increased hospital busyness on in-hospital mortality remains an elusive task due to statistical power challenges and the difficulty in separating shocks to occupancy from changes in patient composition. Using data from a large public hospital system in Mexico, we estimate the impact of congestion on in-hospital mortality by exploiting the shock in hospitalizations induced by the 2009 H1N1 pandemic, instrumenting hospital admissions due to acute respiratory infections (ARIs) with measures of ARI cases at nearby healthcare facilities as a proxy for the size of the local outbreak. Our instrumental variables estimates show that a one percent increase in ARI admissions in 2009 led to a 0.25% increase in non-ARI in-hospital mortality. We show that these effects are non-linear in the size of the local outbreak, consistent with the existence of tipping points. We further show that effects are concentrated at hospitals with limited infrastructure, suggesting that supply-side policies that improve patient assignment across hospitals and strategically increase hospital capacity could mitigate some of the negative impacts. We discuss managerial implications, suggesting that up to 25-30% of our estimated deaths at small and non-ICU hospitals could have been averted by reallocating patients to reduce congestion.

Key words: healthcare operations; hospital occupancy; hospital mortality; pandemics; H1N1; health shocks; non-linearities

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1 Introduction

Hospital overcrowding may lead to lower quality of care due to smaller staff-to-patient ratios, fewer material resources per patient, and increased stress among personnel, all of which may be exacerbated in settings with limited infrastructure. By negatively affecting quality of care, congestion may then have an impact on patient outcomes. During large shocks, such as epidemics, understanding the impact of overcrowding on mortality is crucial for the mitigation strategy.

In this paper, we ask whether increased hospital occupancy has a causal impact on in-hospital mortality. We overcome the empirical challenges facing the literature by exploiting a dataset that is comprehensive of a large Mexican hospital system and leveraging arguably exogenous variation in admissions driven by the 2009 H1N1 pandemic for identification.

This setting is informative for several reasons. First, Mexico is a middle-income country with important limitations in healthcare infrastructure ([OECD, 2005](#)), which may be relevant for congestion. Second, the H1N1 pandemic was a quasi-exogenous health shock. Lastly, although our findings may not necessarily be externally valid for Covid-19 due to differences in timing, severity, and other characteristics with respect to H1N1, they may still inform management strategies for dealing with the current pandemic.¹

We follow an instrumental variables (IV) approach to estimate the impact of increased hospital admissions due to acute respiratory infections (ARIs) on non-ARI in-hospital mortality. We instrument ARI hospitalizations with weekly local measures of ARI cases at nearby healthcare centers.² We establish a strong first stage, estimating that a one standard deviation (sd) increase in neighboring ARIs increased ARI hospitalizations by 0.11 sd, equivalent to a 17 to 41% increase relative to previous years. Next, we present some evidence that the exclusion restriction holds, finding no effects of local ARI outbreaks on non-ARI admissions, suggesting that this shock was exogenous to non-ARI hospitalizations and that selection is unlikely to drive our results.

Our main IV estimates show that non-ARI hospital deaths increased by 0.17 sd for a one sd increase in ARI admissions, or equivalently, a 0.25% increase due to a one percent increment in hospitalizations. We show that this effect is non-linear in the size of the local ARI outbreak, with

¹Moreover, the data for the H1N1 pandemic in Mexico has now been revised and vetted by researchers ([Charu et al., 2011](#)), rendering it more reliable than some of the current real-time Covid-19 data.

²Other studies, such as [Lau et al. \(2019\)](#), have analyzed excess ARI admissions due to the H1N1 outbreak in different settings.

effects concentrated at the top quintile. Given the linearity of the first stage, this is consistent with the existence of tipping points.³ Furthermore, our mortality results are driven by smaller hospitals (fewer than 19 beds) and by hospitals without an intensive care unit (ICU). Crucially for interpretation, all coefficients correspond to the marginal effect of a one sd increase in admissions.

Our findings have important practical implications for the management of hospital strain. We discuss three potential policies in our setting: better allocation of patients across hospitals, changes in admission and treatment decisions, and transforming hospital capacity. Given the availability of beds at hospitals within 5 km, we calculate that around 25 to 30% of our estimated mortality effects could have been avoided by reallocating patients to nearby hospitals, but that geographically isolated hospitals had no such low-cost alternative to avoid overcrowding. We conclude that centralizing patient allocation could be a feasible and cost-effective intervention, although constraints in bed availability at nearby facilities may hamper its effectiveness during severe surges in hospital demand.

The causal estimation of overcrowding on patient outcomes – and mortality in particular – is a difficult empirical task requiring exogenous variation in hospital strain and large samples for identifying mortality effects and tipping points.⁴ Most of the literature documents correlations, ignoring potential changes in patient selection, and treating variations in admissions as exogenous (see [Eriksson et al., 2017](#) for a review). A smaller set of papers have exploited high-frequency variation in hospitalizations to claim a causal impact ([Freedman, 2016](#); [Marks and Choi, 2019](#); [Hoe, 2019](#)). We build on them by focusing on in-hospital mortality, identifying non-linearities, and investigating the role of hospital infrastructure.

Within the hospital management literature, most papers have focused on case studies of single hospitals or groups of hospitals ([Freeman et al., 2017](#); [Kc and Terwiesch, 2012](#); [Song et al., 2019](#); [Kim et al., 2015](#)), and some do not have a clear source of exogenous variation in occupancy although various controls are included ([Berry Jaeker and Tucker, 2017](#); [Friebel et al., 2019](#); [Abir et al., 2020](#)). Moreover, the majority focus on outcomes related to length of stay and readmission.

³[Kuntz et al. \(2015\)](#) identifies safety tipping points in hospital utilization among 83 German hospitals, showing that high occupancy beyond the threshold increases in-hospital mortality. However, this estimation does not use an exogenous source of variation in hospital admissions.

⁴Six out of thirty studies reviewed in [Eriksson et al. \(2017\)](#) explore non-linearities, but treat variations in hospital strain as exogenous. One study does consider unexpected changes in strain, but the specification does not allow for non-linearities ([Schwierz et al., 2012](#)). See Table S1 in the online appendix for more details.

We make at least three important contributions to this literature. First, we analyze an entire public hospital system in Mexico corresponding to 50% of all public sector admissions. Second, we exploit spatial and temporal fluctuations in the severity of the 2009 H1N1 pandemic as a source of arguably exogenous variation in hospital admissions due to ARIs to estimate the impact on non-ARI-related mortality.⁵ This allows us to better isolate hospital business from contemporaneous supply and demand factors. Third, we focus on the link between congestion and in-hospital mortality, exploring non-linearities.⁶ Lastly, given our setting, our results also contribute to the current discussions regarding the optimal management strategy for the ongoing coronavirus pandemic.

2 Context

The 2009 H1N1 pandemic began in Mexico in March with unusual increases in influenza-like illnesses (Fraser et al., 2009), evolving into a three-wave pandemic with varying severity across space and time (Chowell et al., 2011).⁷ Figure 1 shows nation-wide trends in ARIs for multiple years. Consistent with the literature (Charu et al., 2011), we focus on data for all ARIs since misdiagnoses and unconfirmed H1N1 cases may misrepresent the full extent of the epidemic. However, the pattern holds when restricting to lab-confirmed H1N1 counts only. The two large increases in ARIs during 2009 in Figure 1 are completely out of line with the usual ARI trends in Mexico. Total excess ARIs, taken as the difference with respect to the 2007-2008 average, amount to 6.98 million cases or a 30% increase.

By December 2009, the H1N1 pandemic was associated with 12.5-16 thousand excess all-cause deaths and 4.4-5.6 thousand pneumonia and influenza deaths (Charu et al., 2011).⁸ Researchers identified early on that delayed access to inpatient treatment was a key risk factor (Echevarría-Zuno et al., 2009), leading to concerns about increased hospital occupancy and congestion, especially given the low hospital supply and lack of human resources in Mexico (OECD, 2005).⁹

⁵Since we focus on the H1N1 pandemic, our work is closely related to the literature exploring the surge in mortality worldwide during this outbreak (see Duggal et al., 2016 for a review).

⁶In this sense, our paper is related to the literature attempting to estimate the value of the marginal hospital admission (see, for example, Currie and Slusky, 2020 and the literature review therein).

⁷Influenza-like illnesses are respiratory diseases characterized by a sudden onset of symptoms commonly seen in influenza cases, such as fever, shivering, chills, cough, body aches, and nausea.

⁸This is an average mortality rate of 4.5 and 12.7 per 100,000, for ARIs and all causes, respectively.

⁹For example, there were 1.6 hospital beds per 1,000 people in Mexico during the study period, compared to 3.1 in the US and 5.9 in the Euro area. Mexico had 4.1 medical personnel (doctors, nurses, and midwives) per 1,000 people, relative to 15 for the US and 11.8 for the Euro area. See Figure S1 in the online appendix.

The public healthcare system in Mexico is heavily fragmented. Formal sector workers are covered by the Mexican Institute of Social Security (IMSS), and government workers by the Institute for Social Security and Services for State Workers (ISSSTE). We focus on a third system of healthcare facilities administered by the Ministry of Health (SSA). The SSA hospital system provides healthcare to informal sector workers and the unemployed. According to the 2012 National Health Survey (ENSANUT), SSA covers 36 and 69% of the population in urban and rural areas, respectively. SSA hospitals are on average somewhat smaller than other public hospitals.¹⁰

3 Data

We use three publicly available datasets. First, we obtain data on all admissions at SSA hospitals. There were a total of 660 SSA hospitals operating in 2009 according to the data, with 50% of all public sector hospitalizations occurring at SSA hospitals.¹¹ We drop 48 hospitals with over 26 weeks of zero hospitalizations, and construct a balanced panel of hospital-weeks by date of admission. We calculate counts of hospitalizations and deaths due to ARIs and all other causes (non-ARIs) from ICD-10 codes for the initial diagnosis recorded by physicians during admission and the reason for discharge (death or improvement).¹²

Second, we use all new cases of ARIs on a weekly basis (from ICD-10 codes in epidemiological surveillance data), gathered from all geocoded healthcare facilities.¹³ We then construct measures of ARI prevalence in the vicinity of each SSA hospital by assigning facilities to SSA hospitals in two ways. First, we consider the 10 healthcare facilities nearest to the SSA hospital, based on Euclidean distance. Second, we include all healthcare facilities located within a 5 km radius of the hospital. For each definition, we count the total ARIs from these neighboring facilities for each week.¹⁴

Lastly, we recover data on hospital-level infrastructure measured in 2013, the earliest year for which such data are available. We focus on the total number of hospital beds and the presence

¹⁰On average, SSA hospitals had 63 beds and 2.4 operating rooms per hospital, compared to 86 beds and 3.1 operating rooms for IMSS and ISSSTE hospitals. Only 54% of SSA hospitals had over 30 beds, relative to 64% in other public hospitals (SSA, 2007).

¹¹According to ENSANUT, 21 and 17% of all hospitalizations occurred at privately-run hospitals in 2006 and 2012, respectively, suggesting our data cover 40-42% of all hospitalizations.

¹²Table S2 in the online appendix shows the top five causes of hospital deaths for ARIs and non-ARIs.

¹³All public hospitals and clinics are required to report by law. Data for the private sector may be less reliable since there is no enforcement in reporting.

¹⁴In a robustness check in the online appendix, we further define neighbors as the five closest healthcare facilities, and as all facilities located within a 1 or 2 km radius, showing that our results hold.

of an ICU, noting that these characteristics are more likely to remain unchanged over time than staffing and equipment. We discuss limitations in Section 8.

Table 1 presents summary statistics for various years of our data. Panel A shows hospitalizations and deaths for the SSA hospitals by diagnosis (ARIs vs non-ARIs). Panel B shows prevalence of ARIs for different measures of neighboring healthcare facilities. Lastly, Panel C shows infrastructure at SSA hospitals. Characterizing hospitals by number of beds, 13% of all small SSA hospitals (bottom quintile of the distribution) are within 5 km of a larger SSA hospital and 18% are more than 50 km away. Moreover, 12% of SSA hospitals without an ICU are within 5 km of an SSA hospital with an ICU, while 42% are more than 50 km away from the nearest one (online appendix Figure S5).

4 Empirical Strategy

We are interested in estimating the effect of increased hospital occupancy on hospital-level mortality outcomes in a model of the following form:

$$outcome_{jw} = \beta hospARI_{jw} + \theta_j + \lambda_w + \nu_{jw} \quad (1)$$

where $outcome_{jw}$ is an outcome of interest for hospital j in week w , ARI_{jw} are ARI admissions in the same hospital-week, θ_j and λ_w are hospital and week fixed effects (FE), respectively, and ν_{jw} is the error term. The inclusion of hospital FE implies that we identify the effect off of changes within each hospital, while the week FE account for overall seasonality.

However, such a regression may not allow for a causal interpretation. Without a clear understanding of what drives the variation in occupancy, it may be difficult to disentangle changes in mortality that are due to hospital strain from those attributable to changes in patient composition. For example, a surge in admissions due to an expansion of healthcare coverage or insurance is likely to affect both busyness and patient composition, biasing the estimates in equation 1.

Therefore, exploiting quasi-exogenous variation in hospitalizations during the H1N1 pandemic in 2009 and distinguishing between ARI admissions and non-ARI deaths, we follow an IV approach:

$$nonARIdeaths_{jw} = \alpha hospARI_{jw} + \theta_j + \lambda_w + \varepsilon_{jw} \quad (2)$$

where $nonARIdeaths_{jw}$ are non-ARI deaths in hospital j during week w , ε_{jw} is the error term, and everything else is as defined above. Our endogenous variable of interest is congestion as measured by ARI admissions, which we instrument with our measures of ARI cases at neighboring healthcare facilities using a two-stage least squares procedure.

Focusing on ARI hospitalizations during the pandemic allows us to leverage shocks to hospital occupancy that were arguably unexpected and uncorrelated with the underlying local demand prior to the shock. However, estimates may still be biased since different hospitals may be attracting different types and quantities of patients (for example, hospitals with highly skilled physicians may receive more and sicker patients). Our IV approach deals with this potential issue.

Since counts of neighboring ARIs may be mechanically correlated with the number of nearby facilities, we take two approaches. First, we fix the number of healthcare facilities at 10, regardless of distance. Second, we fix the distance at 5 km, regardless of the number of neighbors. The IV estimate may be interpreted as causal if the exclusion restriction holds, namely, that neighboring ARIs had no direct impact on non-ARI mortality, except via their effect on ARI admissions.

We validate our IV approach by showing a strong first stage (the effect of neighboring ARIs on ARI hospitalizations) and suggestive evidence that the exclusion restrictions holds (the effect of neighboring ARIs on non-ARI hospitalizations, which should be zero if the pandemic is truly a quasi-random shock). We also present the reduced-form effect of the instrument on non-ARI deaths. These three estimates correspond to the reduced-form model in equation 1, substituting the right-hand-side endogenous variable with ARI cases at neighboring facilities.

5 Main Results

Using our balanced panel of 612 SSA hospitals over 52 weeks in 2009, we present reduced-form estimates of equation 1 with neighboring ARI cases as the independent variable and IV estimates of equation 2 using a two-stage least squares procedure. We cluster our standard errors at the hospital level throughout to allow for serial correlation in the error term. To account for differences in size, patient burden, and the number of neighboring facilities across hospitals, we normalize all

variables by taking the z-score.¹⁵ We show similar estimates for unnormalized variables, both in levels and rates, in the online appendix.

Table 2 displays the results. Panel A assigns weekly ARIs from neighboring facilities as the sum over the 10 nearest healthcare centers. Panel B uses the sum over all facilities within a 5 km radius. Column 1 shows the effect of our (normalized) measure of neighboring ARIs on (normalized) ARI hospitalizations at SSA hospitals. We estimate a strong first stage, indicating that a one sd increase in neighboring ARIs led to a 0.11 sd increase in own ARI admissions. Hence, every nine neighboring ARI cases were associated with one additional hospitalization. Given the average 32% increase in ARIs at neighboring facilities in 2009 relative to the previous years (Table 1), we estimate between 13 and 32 additional admissions per week, equivalent to a 17 to 41% increase.

Column 2 shows an extension of the first stage, by estimating the effect of neighboring ARIs on in-hospital mortality due to ARIs. Given the rise in ARI admissions, this could also lead to increases in ARI deaths. Indeed, a one sd increase in ARIs at nearby healthcare facilities led to a 0.05 sd increment in ARI deaths.

Column 3 presents suggestive evidence that the exclusion restriction holds. If the variation in neighboring ARIs is correlated with local demand conditions, then we would also see effects on non-ARI hospitalizations. However, our estimates for the effect on non-ARI admissions are an order of magnitude smaller, negative, and statistically indistinguishable from zero. This provides reassurance that the increases in ARIs as measured by counts at nearby facilities were likely exogenous to determinants of non-ARI deaths, except for their indirect effect via ARI hospitalizations.

Column 4 shows our main reduced-form result. We find a significant positive coefficient, indicating that a one sd increase in neighboring ARIs led to a 0.02 sd increase in deaths due to non-ARIs, which implies one additional non-ARI hospital death for every 91 neighboring ARI cases.

Columns 5 and 6 correspond to our IV estimates. We first replicate our findings for hospital admissions due to non-ARIs, obtaining again null effects. Further evidence that the exclusion restriction holds is presented in online appendix Figure S6, with IV estimates that show that the age distribution of non-ARI admissions is also unaltered. Taken together, this suggests that the ARI shock did not change the quantity or composition of non-ARI patients.

¹⁵We subtract the hospital-specific mean and divide by the standard deviation, so that the normalized variable has mean zero and a standard deviation of one. We use the mean and standard deviation across all years from 2007 to 2011 for this calculation. Results are very similar to just using 2009 data or limiting to pre-pandemic data.

Column 6 shows a positive and significant IV estimate for the effect of increased ARI hospitalizations on non-ARI in-hospital mortality. We find that a one sd increase in ARI admissions led to a 0.17 sd increase in non-ARI deaths, or equivalently, that a one percent increase in ARI hospitalizations (relative to the mean) increased non-ARI hospital deaths by 0.25%.¹⁶

We interpret our IV estimates as the local average treatment effect (LATE), which is the effect for compliers: SSA hospitals that increased their ARI admissions in response to an increase in nearby outbreaks of ARIs. We cannot speak to the effects for hospitals that would never (or always) increase their ARI patient load, regardless of the number of nearby ARI cases. Nevertheless, this is the causal parameter of interest for policy design.

The results in Table 2 hold under a series of robustness checks, all shown in the online appendix. First, we consider alternative definitions of neighboring ARIs and additional controls in Table S5. Specifically, we add flexible time trends by state and by quintiles of the share of neighboring facilities under SSA management. We also control for the number of patients currently hospitalized. Second, we examine IV log-log regressions where the dependent variable is either non-ARI deaths or the non-ARI mortality rate in Table S6. Lastly, we estimate the effect of increased ARI hospitalizations on non-ARI lengths of stay and the share of patients with an early discharge, defined as a length of stay below the diagnosis-specific median in the 2007-2008 data, in Table S7. We find that a one percent increase in ARI hospitalizations significantly decreased the average length of stay by 0.18%, total hospital days by 0.13%, and increased the share of early discharges by 0.04%, consistent with the effects of congestion on mortality documented above.

6 Non-Linearities and Heterogeneity

6.1 Tipping Points

We now ask whether these results may be non-linear in the size of the local ARI outbreak. Identifying tipping points is important for hospital management. A visual inspection of the raw data in the

¹⁶To see this, first note from Table 1 that a 1% increase in hospitalizations relative to the mean and expressed in sd is equal to $0.01 \times 3.60/5.68$. Then to express the coefficient in levels, multiply by the sd of the outcome $\hat{\beta} \times \text{sd}_y$. Lastly, divide by the mean of the outcome to express as a percentage. Hence, for a one percent increase in ARI hospitalizations, we estimate a $\frac{0.01 \times (3.60/5.68) \times \hat{\beta} \times \text{sd}_y}{\bar{y}}$ percent change in the outcome.

form of a binned scatterplot suggests that tipping points matter and are present in the top quintile of the distribution of the size of the local ARI outbreak (see Figure S7 in the online appendix).

Hence, we are interested in estimating the effect by quintile of our measure of neighboring ARIs. We present reduced-form evidence, since an IV estimation would require a strong instrument for each quintile. We estimate equation 1, substituting the explanatory variable with indicators for each quintile of the normalized measure of neighboring ARIs.

Figure 2 presents the results. Each coefficient series corresponds to a different definition of neighboring ARIs. The bars show 90 and 95% confidence intervals from robust standard errors clustered at the hospital level. The plot on the left shows the first stage. Estimates are significant and fairly linear, indicating a positive relationship between outbreaks and ARI admissions.

The plot on the right shows the effect on non-ARI deaths. Our coefficients for the first, second, and fourth quintile are all small and statistically indistinguishable from zero (the third quintile is the reference category). The estimate for the fifth quintile is positive and significant, indicating that hospitals in areas with large ARI outbreaks saw increases in non-ARI in-hospital mortality.¹⁷ Given the linearity of the first stage, this pattern is not driven by non-linearities in admissions. This suggests the existence of non-linearities in the effect of increased hospital occupancy on mortality, consistent with the literature identifying tipping points for overcrowding (Kuntz et al., 2015).¹⁸

6.2 Hospital Infrastructure

We explore heterogeneous effects by hospital infrastructure, presenting IV estimates of equation 2 by stratifying the sample. We consider quintiles of the total number of hospital beds and whether the hospital has an ICU. Although hospitals without an ICU also tend to be smaller, there is sufficient variation suggesting that this is an informative result beyond size.¹⁹

Table 3 shows our estimates. Columns 1-5 stratify the sample by quintiles of total hospital beds. Our IV estimate is large and significant for the first quintile, with smaller and insignificant estimates for the remaining four. Hence, for the same increase in ARI hospitalizations (a one sd), smaller hospitals (with 18 or fewer beds) were the ones that saw increases in non-ARI mortality.

¹⁷Figure S8 in the online appendix shows a similar pattern by ventiles instead of quintiles.

¹⁸Note that the medical literature has found overcrowding at hospitals that are at 85% capacity (Madsen et al., 2014). Although this is not a one-to-one mapping, our non-linearities in the top quintile are in line with these findings.

¹⁹Medical staff at hospitals without an ICU may also lack training for providing intensive care necessary during shocks like epidemics (Volkow et al., 2011).

Columns 6 and 7 show large and significant effects for SSA hospitals without an ICU, and smaller and insignificant estimates for those with an ICU, respectively.

Altogether, this suggests that infrastructure plays a key role in the damaging effects of hospital overcrowding. If smaller hospitals are also more likely understaffed and lacking in medical supplies, this finding might also reflect other infrastructure shortcomings.

Given that we only observe infrastructure until 2013, these results should be interpreted with caution. We argue that potentially misclassifying hospitals would lead to attenuation bias. First, we are more likely to misclassify hospitals in contiguous quintiles (i.e., it is unlikely that a hospital would change quintiles drastically from 2009 to 2013). Given our effects in the first quintile, the potential bias is coming from hospitals that became somewhat larger or smaller over time. Second, it is unlikely that a hospital would lose its ICU, so that we capture a weighted average of mostly hospitals without an ICU and some with an ICU. Section 8 further discusses this issue.

7 Implications for Policy and Management

We now discuss the managerial and policy implications of our findings. Three main policy responses could mitigate the mortality impacts. First, managers could better allocate patients across hospitals to avoid overcrowding (Kuntz et al., 2015; Deo et al., 2013). Second, doctors may change admission decisions on the marginal patient and treatment choices of hospitalized patients through early discharges. Lastly, managers may transform hospital capacity.

7.1 Patient Reallocation

Our effects of hospital overcrowding on mortality are driven by small and non-ICU hospitals. Stratifying these hospitals by distance to the nearest larger hospital and by distance to the nearest ICU, we find that the mortality effects are present along the entire distribution of these distances (Figure S12 in the online appendix).²⁰ This suggests that hospitals did not reallocate patients, even when there were larger and ICU hospitals nearby.

To better understand the scope for reallocating patients, we construct weekly measures of available hospital beds at larger and ICU hospitals within 5 km of small and non-ICU hospitals.

²⁰The variance of our estimates for hospitals that are closest to the nearest larger or ICU hospital is sizable. This may reflect that at least some of these hospitals reallocated patients and therefore did not see mortality effects.

We find that there were over 15 available beds at nearby larger and ICU hospitals for around 10% of weeks (online appendix Figure S13). Weighting availability by non-ARI deaths at the small and non-ICU hospitals increases these numbers to 25 and 30%, respectively, suggesting that there was some concentration of deaths at hospitals with available beds nearby. Hence, up to 30% of our estimated deaths may have been averted by better allocating patients to these larger and ICU hospitals.

Under centralized management, this would be a relatively low-cost intervention. However, this policy lever is limited by both the share of geographically isolated hospitals and the number of beds available in nearby hospitals at any given moment. In this particular setting, 42% of small hospitals and 18% of non-ICU hospitals are over 50 km away from the nearest larger hospital or ICU (online appendix Figure S5). It is then unlikely that this hospital system could rely solely on reallocation to mitigate the mortality effects.

7.2 Admission and Treatment Decisions

To understand which conditions concentrate the estimated mortality effects, we stratify the sample by the diagnosis-specific mortality rate at baseline (2007-2008). As expected, we find tight zeros for conditions with a baseline mortality rate of zero. We then find small and weak effects for low-mortality conditions, and large effects for high-mortality diagnostic codes (see online appendix Figure S14). This suggests that our effects are driven mostly by riskier diagnoses that (presumably) require more care. Although hospitals may have changed their marginal admissions, we find insignificant effects for hospitalizations across all three diagnostic groups.

Online appendix Table S7 shows that increased ARI admissions led to shorter non-ARI hospital stays and a larger share of early discharges. This suggests that doctors altered their treatment decisions. Stratifying by mortality rates, it appears that high-mortality diagnostic codes are the ones for which the average length of stay declined significantly, although the share of early discharges also increased for low-mortality codes (online appendix Table S8). Unfortunately, we cannot measure readmission rates or out-of-hospital mortality.

These supplementary estimates suggest that mortality was concentrated in riskier diagnoses, and that hospitals did not turn away patients as a way to avoid congestion, although overcrowded hospitals were more likely to discharge patients early, particularly those with more severe diagnoses.

Given that we cannot observe other patient health measures, we caution against concluding that strategically changing admission decisions might have unambiguously improved patient outcomes.

7.3 Hospital Transformation

Hospital managers could also transform hospital capacity, even just temporarily. Some evidence suggests that hospitals in 2009 implemented triage systems and adapted the existing infrastructure to deal with increased patient flows (Volkow et al., 2011). However, systematic data are unavailable. Instead, we turn then to data from the current Covid-19 outbreak.

Focusing on small and non-ICU hospitals, we find that hospitals that are farther away from the nearest larger hospital or ICU are more likely to have undergone a capacity expansion in 2020, although resources have also been devoted to hospitals with nearby alternatives (online appendix Figure S15). This suggests perhaps that policy-makers learned from the 2009 experience. Nevertheless, managers under strict budgets may be unable to expand capacity. Our results suggest, however, that given the extent to which overcrowding may be avoided through patient reallocation for a large set of hospitals, resources for hospital transformation could be more efficiently allocated if directed more intensively toward geographically isolated facilities.

8 Study Limitations

Observing hospital infrastructure until 2013 is an important data limitation. Using aggregate data on SSA healthcare units, online appendix Figure S16 shows the municipality-level distribution and correlation in 2009 and 2013, showing very few changes in infrastructure over time. We also find no significant correlation between these changes and the municipality-level size of the ARI outbreak.

We complement this information with an estimate of the hospital-level number of beds in our data. We calculate the maximum patient load per hospital during 2007-2008. Evidently, this might be an underestimate of the true hospital capacity. Figure S17 in the online appendix shows the correlation between this measure and our 2013 infrastructure data, suggesting that the latter is not a bad proxy of 2009 data.

As for our methodology, our estimates are internally valid and represent the LATE, as discussed above. However, our local effects may not necessarily be externally valid. We cannot infer the effects

of overcrowding on mortality for hospitals that did not respond to the local outbreak, since IV is uninformative for hospitals that were unaffected by the instrument. Our findings may also not extend to other hospital systems in Mexico that might have responded differently. Nevertheless, our LATE estimates are the relevant measure of how congestion due to health shocks may result in in-hospital mortality increases, and may be informative for the design of mitigating policies from a managerial perspective, as discussed above.

9 Conclusion

This paper revisits the 2009 H1N1 pandemic in Mexico to ask whether shocks in hospital occupancy may increase in-hospital mortality. Leveraging an IV approach to overcome the identification problems, we estimate that a one percent increase in hospital admissions led to a 0.25% increase in non-ARI hospital deaths. We show that this relationship is non-linear in the size of the local ARI outbreak, and that the effects are concentrated at hospitals that are small and lack an ICU.

Supplementary analyses suggest that a combination of patient reallocation for hospitals that are close to larger hospitals and capacity expansion for those that are more geographically isolated would be a relatively cost-effective response to a shock to hospital occupancy. However, capacity constraints may be binding under large surges in hospital admissions – due to, for example, more severe epidemics – which in turn may limit the effectiveness of reallocation policies. Overall, these lessons may provide valuable insights for managing the Covid-19 pandemic.

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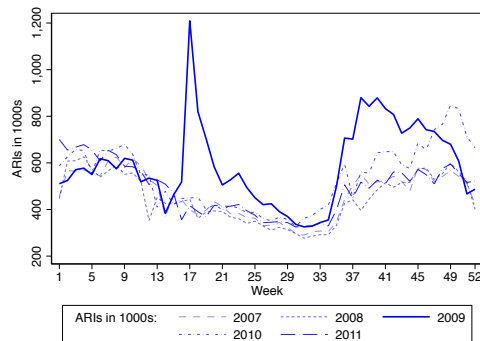
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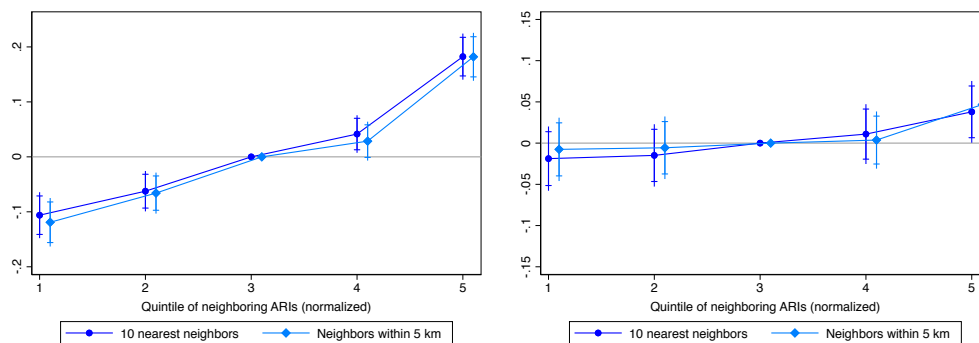
Figures and Tables

Figure 1:
Epidemiological Trends of ARIs



Notes: This graph shows the nation-wide epidemiological trends of ARIs for each year from 2007 to 2011.

Figure 2:
Effect of ARI Outbreaks on Non-ARI Mortality by Size of the
Outbreak



(a) Hospitalizations due to ARIs

(b) Deaths due to Non-ARIs

Notes: These graphs show OLS estimates of (normalized) hospitalizations due to ARIs (left) and (normalized) deaths due to non-ARIs (right) on the (normalized) measure of neighboring ARIs by quintiles. Each coefficient series corresponds to a different definition of neighboring healthcare facilities. Regressions include hospital and week FE. Bars correspond to 90 and 95% confidence intervals, from standard errors clustered at the hospital level.

Table 1:
Summary Statistics

	2009 only	2007-2008	2010-2011	2013
<u>A. Hospital-week outcomes</u>				
Total hospitalizations	80.68 (97.61)	77.41 (86.78)	83.98 (101.23)	
Hospitalizations due to ARIs	3.60 (5.68)	3.08 (4.82)	3.39 (5.70)	
Hospitalizations due to non-ARIs	75.43 (93.96)	67.31 (83.02)	76.59 (97.07)	
Total deaths	1.66 (3.61)	1.52 (3.28)	1.66 (3.53)	
Deaths due to ARIs	0.23 (0.70)	0.17 (0.53)	0.21 (0.63)	
Deaths due to non-ARIs	1.43 (3.22)	1.35 (3.00)	1.45 (3.18)	
<u>B. Clinic-week ARIs of assigned neighbors</u>				
<i>Main definitions:</i>				
ARIs of 10 nearest neighbors	498.89 (626.11)	378.34 (444.46)	428.38 (518.49)	
Neighboring ARIs within 5 km	1143.56 (2000.61)	857.16 (1452.30)	975.15 (1643.84)	
<i>Alternative definitions:</i>				
ARIs of 5 nearest neighbors	299.64 (372.55)	231.84 (272.80)	260.69 (316.77)	
Neighboring ARIs within 1 km	171.69 (278.72)	134.43 (204.46)	150.65 (235.71)	
Neighboring ARIs within 2 km	350.75 (520.04)	269.28 (380.30)	302.50 (438.95)	
<u>C. Hospital-level infrastructure</u>				
Total beds				72.47 (98.57)
Hospital has ICU				0.28 (0.45)
Total beds in ICU				2.68 (6.74)
Observations	31,824	63,648	63,648	612

Notes: This table presents summary statistics for 2009 and other years. Means are shown, with standard deviations in parentheses. Panel A shows hospitalizations and deaths at the hospital-week level. Panel B shows total weekly ARI cases for various definitions of neighboring healthcare facilities. Panel C shows infrastructure at the hospital level (these data are only available for 2013).

Table 2:
Effect of ARI Outbreaks on Hospitalizations and Mortality

	OLS				IV	
	Hosp. ARIs (1)	Deaths ARIs (2)	Hosp. Non-ARIs (3)	Deaths Non-ARIs (4)	Hosp. Non-ARIs (5)	Deaths Non-ARIs (6)
A. 10 nearest neighbors						
Neighboring ARIs	0.112*** (0.009)	0.049*** (0.008)	-0.001 (0.008)	0.019*** (0.006)		
Hospitalizations due to ARIs					-0.007 (0.068)	0.172*** (0.059)
Observations	31,824	31,824	31,824	31,824	31,824	31,824
R-squared	0.167	0.040	0.393	0.055		
Kleibergen-Paap F statistic					158.66	158.66
B. Neighbors within 5 km						
Neighboring ARIs	0.113*** (0.009)	0.051*** (0.008)	-0.005 (0.008)	0.020*** (0.006)		
Hospitalizations due to ARIs					-0.043 (0.068)	0.179*** (0.059)
Observations	31,824	31,824	31,824	31,824	31,824	31,824
R-squared	0.167	0.040	0.393	0.055		
Kleibergen-Paap F statistic					157.09	157.09
Mean dependent variable	3.60	0.23	75.43	1.43	75.43	1.43
SD dependent variable	5.68	0.70	93.96	3.22	93.96	3.22

Notes: This table presents estimates of the effect of ARI outbreaks. Panel A assigns neighboring ARIs based on the 10 nearest healthcare facilities. Panel B uses all healthcare facilities within a 5 km radius. Columns 1-4 show OLS estimates of the (normalized) outcome variable on the (normalized) measure of neighboring ARIs. Columns 5-6 present IV estimates, instrumenting the hospital's own (normalized) ARI admissions with the (normalized) measure of neighboring ARIs. Regressions include hospital and week FE. Standard errors are clustered at the hospital level. The mean and standard deviation of the non-normalized dependent variable are shown in the last two rows.

*** p<0.01, ** p<0.05, * p<0.1

Table 3:
Effect of ARI Outbreaks on Non-ARI Mortality by Hospital
Infrastructure

	Quintiles of number of beds					Has ICU	
	q1 (1)	q2 (2)	q3 (3)	q4 (4)	q5 (5)	No (6)	Yes (7)
<u>A. 10 nearest neighbors</u>							
Hospitalizations due to ARIs	0.560** (0.271)	0.049 (0.147)	0.180 (0.124)	0.171 (0.105)	0.182 (0.145)	0.242*** (0.079)	0.107 (0.101)
Observations	6,500	6,240	6,552	6,188	6,344	22,984	8,840
Kleibergen-Paap F statistic	11.6	25.2	44.3	41.5	32.2	97.8	53.1
<u>B. Neighbors within 5 km</u>							
Hospitalizations due to ARIs	0.541* (0.277)	0.124 (0.154)	0.183 (0.130)	0.164 (0.105)	0.134 (0.137)	0.250*** (0.082)	0.107 (0.098)
Observations	6,500	6,240	6,552	6,188	6,344	22,984	8,840
Kleibergen-Paap F statistic	11.3	21.4	44.7	44.1	29.9	92.1	51.4
Mean dependent variable	0.34	0.11	0.51	1.35	4.87	0.50	3.83
SD dependent variable	0.98	0.39	0.90	1.56	5.64	2.00	4.33

Notes: This table presents IV estimates of the effect of ARI outbreaks, stratifying the main sample by measures of hospital infrastructure. Panel A assigns neighboring ARIs based on the 10 nearest healthcare facilities. Panel B uses all healthcare facilities within a 5 km radius. Columns 1-5 show results for each quintile of the distribution of hospitals by total number of beds. Columns 6-7 stratify hospitals by whether they have an ICU. All variables are normalized as before. Regressions include hospital and week FE. Standard errors are clustered at the hospital level. The mean and standard deviation of the non-normalized dependent variable are shown in the last two rows.

*** p<0.01, ** p<0.05, * p<0.1

Supplementary Online Appendix

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Supplementary Tables and Figures

Table S1:
Existing Literature of the Effects of Hospital Strain on Mortality

Paper	Analyzes Mortality Effects	Instruments for Strain or Occupancy	Explores Non-Linearities
Ball et al. (2006)	Yes	No	No
Bekmezian and Chung (2012)	No	No	No
Chalfin et al. (2007)	Yes	No	No
Clark and Normile (2007)	Yes	No	No
Clark and Normile (2012)	Yes	No	No
Derose et al. (2014)	No	No	No
Evans and Kim (2006)	No	No	Yes
Gabler et al. (2013)	Yes	No	No
Gattinoni et al. (2004)	Yes	No	No
Gilligan et al. (2008)	Yes	No	No
Intas et al. (2012)	Yes	No	No
Iwashyna et al. (2009)	No	No	Yes
Jenkins et al. (2015)	Yes	No	Yes
Madsen et al. (2014)	Yes	No	No
Marcin and Romano (2004)	No	No	No
O'Callaghan et al. (2012)	No	No	No
Pascual et al. (2014)	Yes	No	No
Plunkett et al. (2011)	Yes	No	No
Robert et al. (2012)	Yes	No	No
Rubinson et al. (2013)	Yes	No	No
Schilling et al. (2010)	Yes	No	No
Schwierz et al. (2012)	No	No*	No
Serafini et al. (2015)	Yes	No	No
Singer et al. (2011)	Yes	No	Yes
Sprivulis et al. (2006)	Yes	No	Yes
Stowell et al. (2013)	Yes	No	No
Tarnow-Mordi et al. (2000)	Yes	No	No
UK Neonatal Staffing Study Group (2002)	Yes	No	No
Wagner et al. (2013)	No	No	No
Yergens et al. (2015)	Yes	No	Yes

Notes: This table lists all papers relating measures of hospital strain and mortality reviewed by [Eriksson et al. \(2017\)](#), indicating whether they find an effect on mortality, whether they use an instrumental variable approach to deal with potential endogeneity in admissions, and whether they explore if there are non-linearities in the impacts of strain on mortality. * [Schwierz et al. \(2012\)](#) uses a measure of unexpected shocks to occupancy as the explanatory variable.

Table S2:
Most Common Causes of In-Hospital Mortality

ICD-10 code	Description of diagnosis
<u>2007-2008 data</u>	
ARIs:	
J18	Pneumonia, organism unspecified
J22	Unspecified acute lower respiratory infection
J44	Other chronic obstructive pulmonary disease
J81	Pulmonary edema
J96	Respiratory failure, not elsewhere classified
Non-ARIs:	
A41	Other sepsis
E11	Type II diabetes mellitus
N18	Chronic kidney failure
P07	Disorders related to short gestation and low birth weight, not classified elsewhere
S06	Intracranial injury
<u>2009 data</u>	
ARIs:	
J15	Bacterial pneumonia, not elsewhere classified
J18	Pneumonia, organism unspecified
J44	Other chronic obstructive pulmonary disease
J81	Pulmonary edema
J96	Respiratory failure, not elsewhere classified
Non-ARIs:	
A41	Other sepsis
E11	Type II diabetes mellitus
N18	Chronic kidney failure
P07	Disorders related to short gestation and low birth weight, not classified elsewhere
S06	Intracranial injury

Notes: This table shows the five most common causes of in-hospital mortality using data from before 2009 and from 2009 only. We distinguish between ARIs and non-ARIs. We show the three-digit ICD-10 codes, along with the description of each diagnosis.

Table S3:
Descriptives of Assigned Neighbors

	<u>Main definitions</u>		<u>Alternative definitions for robustness</u>		
	10 nearest neighbors	Neighbors within 5 km	5 nearest neighbors	Neighbors within 2 km	Neighbors within 1 km
Total neighbors assigned	6.58 (3.18)	12.67 (14.72)	4.25 (1.21)	4.49 (3.56)	2.57 (2.10)
Average distance to neighbors	1.86 (0.94)	2.25 (1.01)	1.53 (0.92)	1.00 (0.43)	0.54 (0.24)
Share SSA neighbors	0.55 (0.27)	0.55 (0.25)	0.54 (0.29)	0.52 (0.33)	0.50 (0.40)
Share IMSS neighbors	0.28 (0.22)	0.28 (0.21)	0.29 (0.24)	0.30 (0.29)	0.29 (0.35)
Share ISSSTE neighbors	0.11 (0.13)	0.11 (0.13)	0.12 (0.15)	0.12 (0.18)	0.15 (0.26)
Share private healthcare neighbors	0.03 (0.07)	0.02 (0.06)	0.02 (0.09)	0.03 (0.10)	0.03 (0.12)
Share neighbors other public institutions	0.04 (0.09)	0.03 (0.08)	0.03 (0.10)	0.03 (0.09)	0.04 (0.14)
Observations	612	612	612	573	455

Notes: This table presents summary statistics of the neighbors assigned to hospitals under each of the two main definitions used in the text, and the three alternative definitions presented as robustness checks. Means are shown, with standard deviations in parentheses. SSA are healthcare facilities run by the Ministry of Health (same as the hospitals in our sample). IMSS and ISSSTE are government healthcare systems for formal workers and government workers, respectively.

Table S4:
Correlation between ARI Hospitalizations and Outcomes

	Deaths ARIs (1)	Hosp. Non-ARIs (2)	Deaths Non-ARIs (3)
Hospitalizations due to ARIs	0.226*** (0.014)	0.042*** (0.008)	0.019*** (0.007)
Observations	31,824	31,824	31,824
R-squared	0.090	0.394	0.055
Mean dependent variable	0.23	75.43	1.43
SD dependent variable	0.70	93.96	3.22

Notes: This table presents the correlation between (normalized) ARI hospitalizations and other (normalized) outcomes from an OLS regression with hospital and week fixed effects. Standard errors are clustered at the hospital level. The mean and standard deviation of the non-normalized dependent variable are shown in the last two rows.

*** p<0.01, ** p<0.05, * p<0.1

Table S5:
Robustness Checks: Alternative Definitions of Neighbors and
Additional Controls

I. Alternative definitions of neighbors						
	Hosp. Non-ARIs			Deaths Non-ARIs		
	5 nearest neighbors (1)	Neighbors within 1 km (2)	Neighbors within 2 km (3)	5 nearest neighbors (4)	Neighbors within 1 km (5)	Neighbors within 2 km (6)
Hospitalizations due to ARIs	0.005 (0.077)	-0.065 (0.113)	-0.027 (0.079)	0.144** (0.061)	0.249** (0.100)	0.122* (0.067)
Observations	31,824	23,660	29,796	31,824	23,660	29,796
Kleibergen-Paap F statistic	127.4	49.9	107.3	127.4	49.9	107.3
Mean dependent variable	75.43	77.60	77.27	1.43	1.51	1.47
SD dependent variable	93.96	97.01	95.51	3.22	3.42	3.28
II. Additional controls						
	Hosp. Non-ARIs			Deaths Non-ARIs		
	State × week FE (7)	SSA share × week FE (8)	Patient load (9)	State × week FE (10)	SSA share × week FE (11)	Patient load (12)
A. 10 nearest neighbors						
Hospitalizations due to ARIs	-0.0195 (0.114)	-0.0158 (0.070)	-0.0280 (0.061)	0.207** (0.100)	0.169*** (0.059)	0.166*** (0.059)
Observations	31,824	31,824	31,824	31,824	31,824	31,824
Kleibergen-Paap F statistic	67.41	150.75	158.62	67.41	150.75	158.62
B. Neighbors within 5 km						
Hospitalizations due to ARIs	-0.0818 (0.121)	-0.0205 (0.069)	-0.0612 (0.061)	0.224** (0.106)	0.180*** (0.061)	0.174*** (0.059)
Observations	31,824	31,824	31,824	31,824	31,824	31,824
Kleibergen-Paap F statistic	59.2	145.5	156.9	59.2	145.5	156.9
Mean dependent variable	75.43	75.43	75.43	1.43	1.43	1.43
SD dependent variable	93.96	93.96	93.96	3.22	3.22	3.22

Notes: This table presents a series of robustness checks on the main results. The first part of the table shows IV estimates under different definitions of neighbors, for non-ARI hospitalizations (columns 1-3) and deaths (columns 4-6), instrumenting (normalized) hospitalizations due to ARIs with the (normalized) measure of neighboring ARIs. The second part of the table returns to the baseline definitions of neighbors (10 nearest neighbors in panel A, and all neighbors within 5 km in panel B). Columns 7 and 10 include additional controls in the form of indicators for each week interacted with indicators for each state. Columns 8 and 11 include indicators for each week interacted with indicators for quintiles of the share of neighboring healthcare facilities that are managed by SSA. Columns 9 and 12 include controls for the patient load in each hospital-week. Regressions include hospital and week FE. Standard errors are clustered at the hospital level. The mean and standard deviation of the non-normalized dependent variable are shown in the last two rows of each table section.

*** p<0.01, ** p<0.05, * p<0.1

Table S6:
Robustness Checks: Alternative Specification of Variables

	IHS non-ARI deaths		IHS non-ARI mortality rate	
	(1)	(2)	(3)	(4)
IHS Hospitalizations due to ARIs	0.0996* (0.059)	0.0959* (0.054)	0.123* (0.065)	0.135** (0.064)
Observations	31,824	31,824	31,824	31,824
Kleibergen-Paap F statistic	150.6	146.0	150.6	146.0
Measure of neighbors:				
10 nearest neighbors	X		X	
Neighbors within 5 km		X		X
Mean dependent variable	1.43	1.43	1.22	1.22
SD dependent variable	3.22	3.22	2.57	2.57

Notes: This table presents IV estimates of the effect of ARI outbreaks using alternative specifications for our main variables. Columns 1 and 2 consider the inverse hyperbolic sine (IHS) of non-ARI deaths as the dependent variable. Columns 3 and 4 consider the IHS of the hospital mortality rate due to non-ARIs. We instrument the endogenous variable, IHS of hospitalizations due to ARIs, with normalized measures of neighboring ARIs. Regressions include hospital and week FE. Standard errors are clustered at the hospital level. The mean and standard deviation of the dependent variable (before the IHS transformation) are shown.

*** p<0.01, ** p<0.05, * p<0.1

Table S7:
Effect of ARI Outbreaks on Non-ARI Hospitalization Lengths of Stay

	Average length of stay		Total hospital days		Share early discharge	
	(1)	(2)	(3)	(4)	(5)	(6)
Hospitalizations due to ARIs	-0.161*** (0.055)	-0.141*** (0.054)	-0.122** (0.059)	-0.128** (0.061)	0.0247** (0.011)	0.0205* (0.011)
Observations	31,824	31,824	31,824	31,824	31,824	31,824
Kleibergen-Paap F statistic	158.7	157.1	158.7	157.1	158.7	157.1
Measure of neighbors:						
10 nearest neighbors	X		X		X	
Neighbors within 5 km		X		X		X
Mean dependent variable	3.12	3.12	211.6	211.6	0.33	0.33
SD dependent variable	5.87	5.87	352.1	352.1	0.20	0.20

Notes: This table presents IV estimates on (normalized) measures of hospital stay lengths for non-ARIs, instrumenting (normalized) hospitalizations due to ARIs with the (normalized) measure of neighboring ARIs. Columns 1 and 2 consider the (normalized) average of hospital stay lengths. Columns 3 and 4 consider the (normalized) total hospital days (average length \times hospitalizations). Columns 5 and 6 show the share of early discharges defined as below the median diagnosis-specific length of stay observed in 2007 and 2008. Regressions include hospital and week FE. Standard errors are clustered at the hospital level. The mean and standard deviation of the non-normalized dependent variable are shown in the last two rows.

*** p<0.01, ** p<0.05, * p<0.1

Table S8:
Effect of ARI Outbreaks on Non-ARI Hospitalization Lengths of Stay
by Diagnosis-Level Mortality Rates

	Zero mortality		Low Mortality		High mortality	
	(1)	(2)	(3)	(4)	(5)	(6)
<u>A. Average length of stay</u>						
Hospitalizations due to ARIs	-0.0137 (0.055)	0.0002 (0.058)	0.0206 (0.060)	0.0192 (0.061)	-0.181*** (0.057)	-0.181*** (0.056)
Observations	31,824	31,824	31,824	31,824	31,824	31,824
Mean dependent variable	0.18	0.18	0.78	0.78	2.22	2.22
SD dependent variable	1.06	1.06	1.82	1.82	5.06	5.06
<u>B. Share early discharges</u>						
Hospitalizations due to ARIs	0.0034 (0.003)	0.0032 (0.003)	0.0142** (0.007)	0.0136** (0.007)	0.0071 (0.009)	0.0037 (0.010)
Observations	31,824	31,824	31,824	31,824	31,824	31,824
Mean dependent variable	0.02	0.02	0.13	0.13	0.18	0.18
SD dependent variable	0.05	0.05	0.13	0.13	0.16	0.16
Measure of neighbors:						
10 nearest neighbors	X		X		X	
Neighbors within 5 km		X		X		X

Notes: This table presents IV estimates on (normalized) measures of hospital stay lengths for non-ARIs, instrumenting (normalized) hospitalizations due to ARIs with the (normalized) measure of neighboring ARIs, and distinguishing between diagnoses based on their pre-2009 mortality rate. Zero mortality refers to conditions for which we do not observe any deaths in 2007-2008. Low and high mortality correspond to below and above the median death rate, respectively. Panel A considers the (normalized) average of hospital stay lengths. Panel B shows the share of early discharges defined as below the median diagnosis-specific length of stay observed in 2007 and 2008. Regressions include hospital and week FE. Standard errors are clustered at the hospital level. The mean and standard deviation of the non-normalized dependent variable are shown.

*** p<0.01, ** p<0.05, * p<0.1

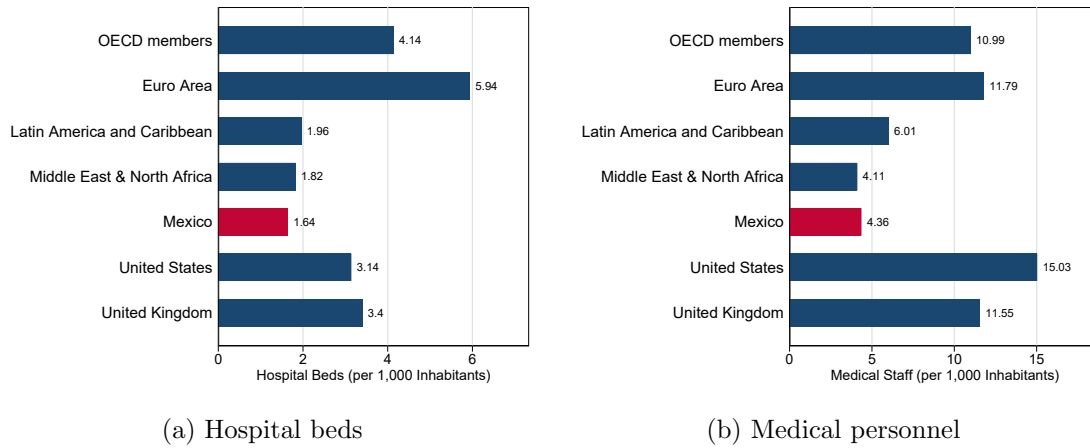
Table S9:
Effect of ARI Outbreaks on Non-ARI Hospitalizations by Hospital
Infrastructure

	Quintiles of number of beds					Has ICU	
	q1 (1)	q2 (2)	q3 (3)	q4 (4)	q5 (5)	No (6)	Yes (7)
<u>A. 10 nearest neighbors</u>							
Hospitalizations due to ARIs	0.239 (0.222)	-0.096 (0.176)	0.176 (0.129)	-0.112 (0.144)	-0.024 (0.173)	0.056 (0.086)	-0.045 (0.127)
Observations	6,500	6,240	6,552	6,188	6,344	22,984	8,840
Kleibergen-Paap F statistic	11.6	25.2	44.3	41.5	32.2	97.8	53.1
<u>B. Neighbors within 5 km</u>							
Hospitalizations due to ARIs	0.171 (0.220)	-0.148 (0.197)	0.174 (0.131)	-0.105 (0.141)	-0.115 (0.166)	0.026 (0.089)	-0.074 (0.120)
Observations	6,500	6,240	6,552	6,188	6,344	22,984	8,840
Kleibergen-Paap F statistic	11.3	21.4	44.7	44.1	29.9	92.1	51.4
Mean dependent variable	30.03	21.69	51.27	88.78	186.73	42.09	162.11
SD dependent variable	40.04	16.12	31.00	54.60	142.74	52.10	119.68

Notes: This table presents IV estimates of the effect of ARI outbreaks on non-ARI hospitalizations, stratifying the main sample by measures of hospital infrastructure. Panel A assigns neighboring ARIs based on the 10 nearest healthcare facilities. Panel B uses all healthcare facilities within a 5 km radius. Columns 1-5 show results for each quintile of the distribution of hospitals by total number of beds. Columns 6-7 stratify hospitals by whether they have an ICU. All variables are normalized as before. Regressions include hospital and week FE. Standard errors are clustered at the hospital level. The mean and standard deviation of the non-normalized dependent variable are shown in the last two rows.

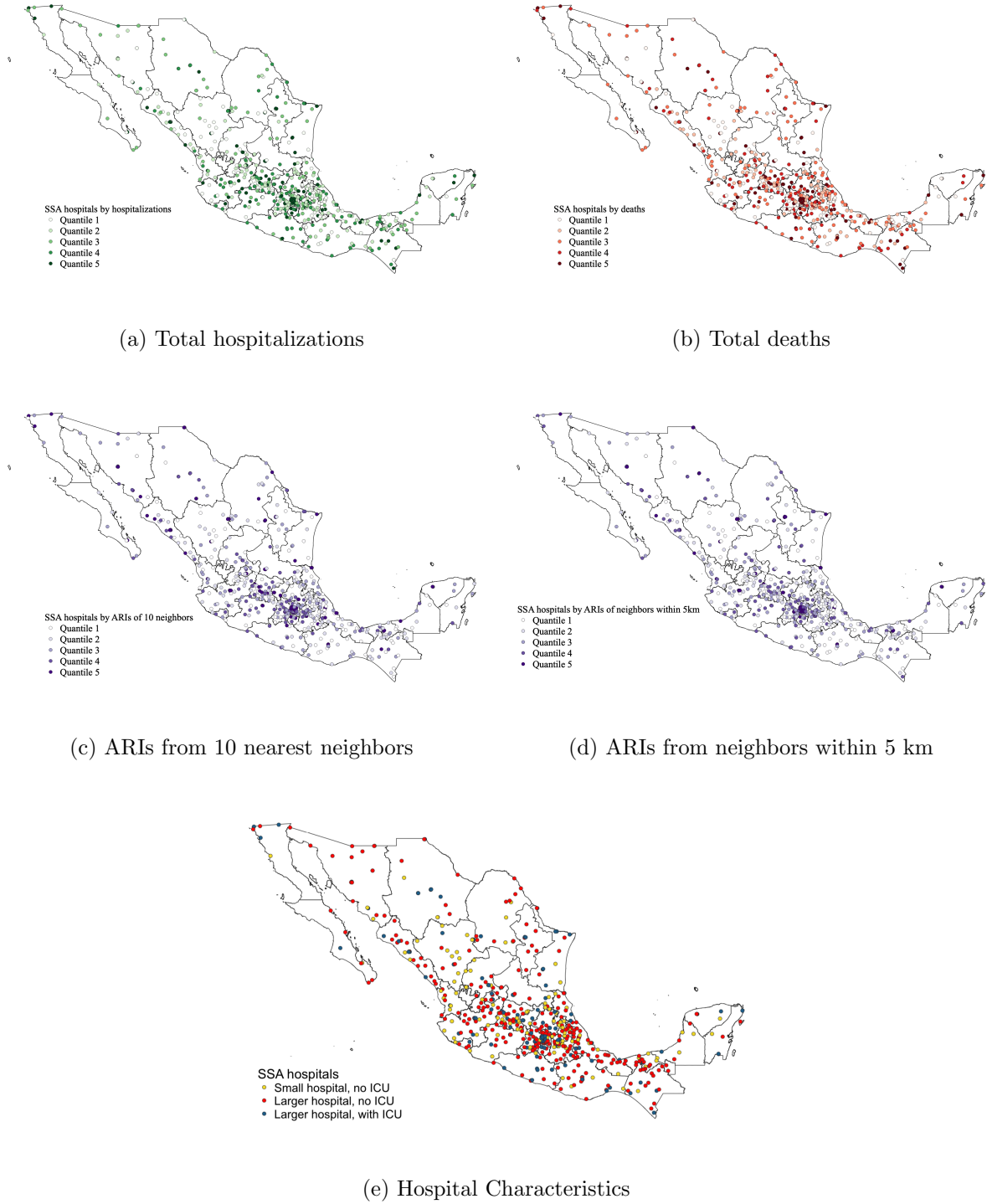
*** p<0.01, ** p<0.05, * p<0.1

Figure S1:
Hospital Beds and Medical Personnel per 1,000 in Different Regions of
the World



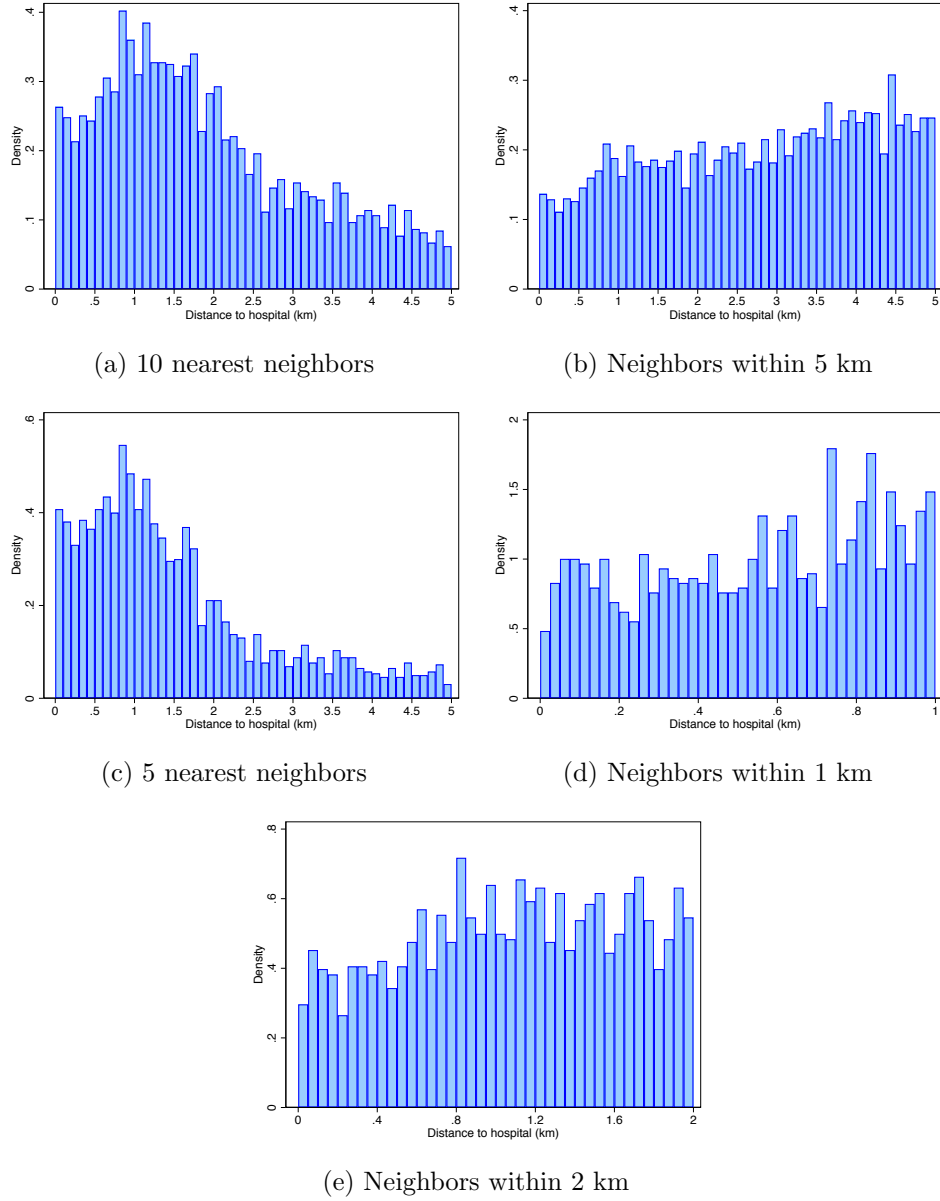
Notes: The graph on the left shows hospital beds per 1,000 people and the one on the right shows medical staff (doctors, nurses and midwives) for different regions and countries in the world. Data correspond to the available measures closest to 2009, considering 2007 to 2010. Data available at <https://ourworldindata.org/grapher/hospital-beds-per-1000-people> and <https://data.worldbank.org>.

Figure S2:
Spatial Distribution of Hospitalizations, Deaths, and Neighboring ARIs



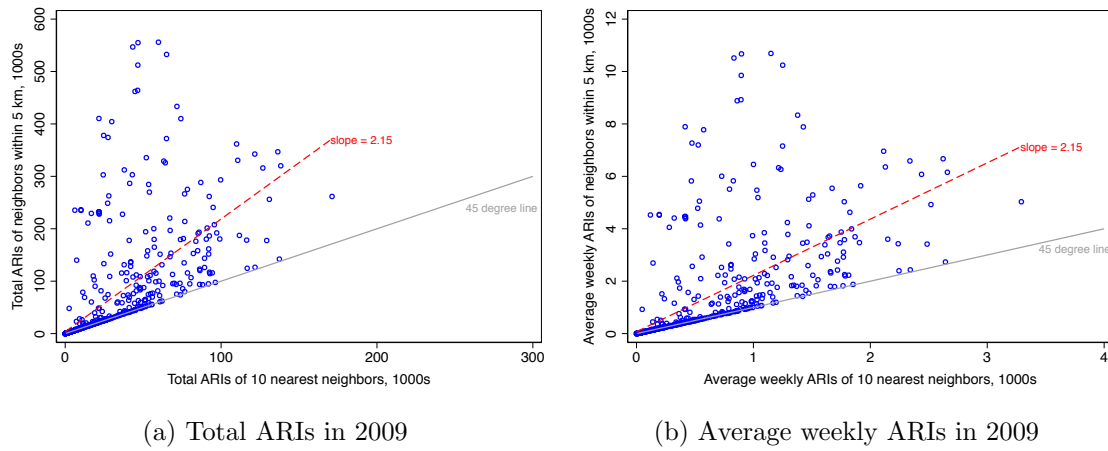
Notes: These maps show the spatial distribution of SSA hospitals used in the estimating sample. The top left map classifies hospitals by quintiles of the total number of hospitalizations in 2009, the top right map by the total number of deaths, the middle left map by ARIs from the 10 nearest neighboring healthcare facilities, the middle right map from all neighbors within 5 km, and the bottom map shows hospitals by size and whether they have an ICU. Small hospitals are in the bottom quintile of number of beds.

Figure S3:
Distribution of Distance from Hospital to Assigned Neighbors



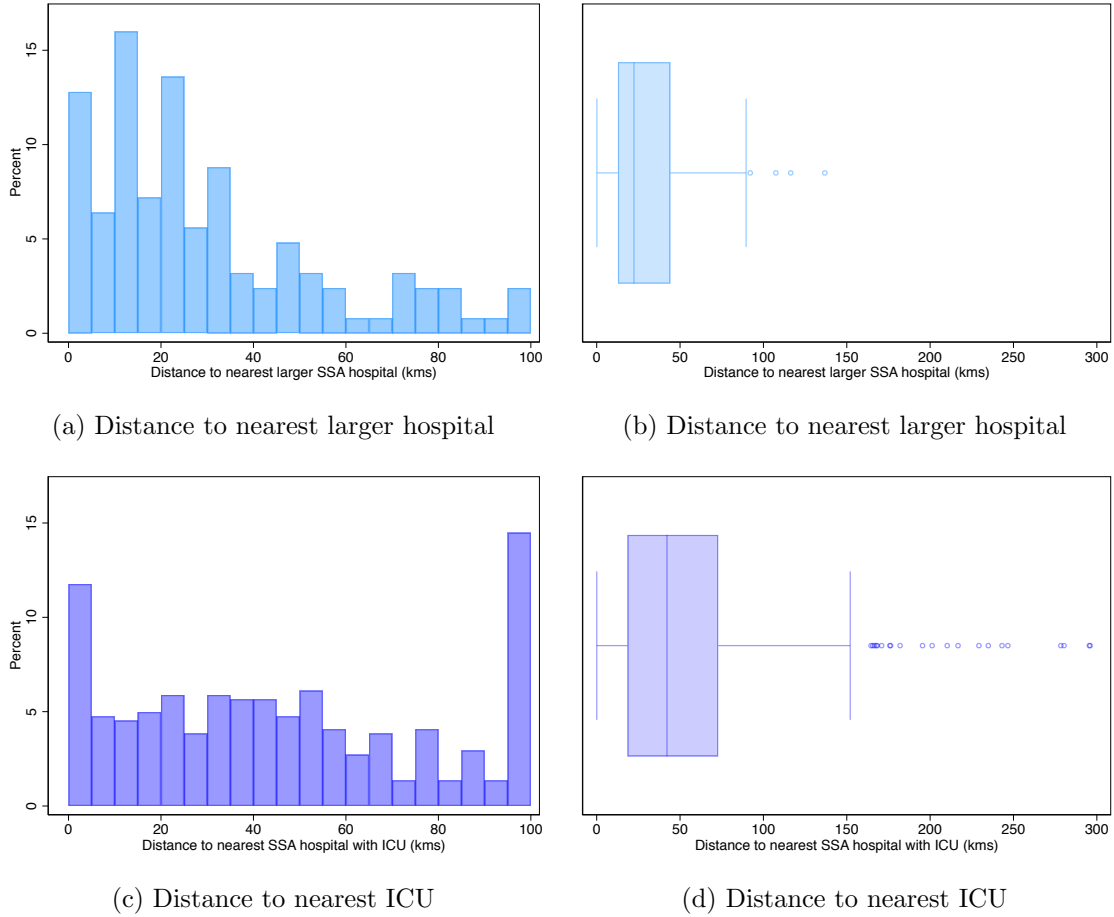
Notes: These graphs show histograms for the distribution of distance from the SSA hospitals to their assigned neighbors, under each of the five definitions. The first two definitions are the main ones used in the text, while the other three are used in the robustness checks.

Figure S4:
Correlation between ARIs from 10 Nearest Neighbors and All
Neighbors Within 5 km



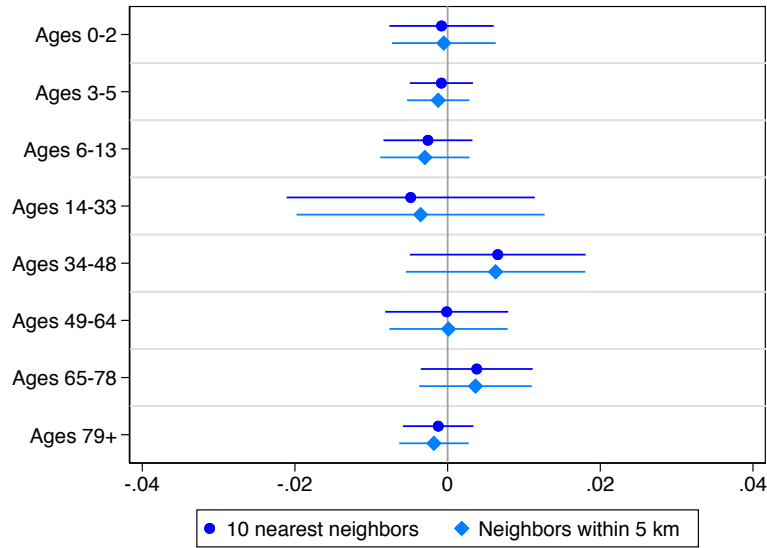
Notes: These plots correlate the ARIs assigned from the 10 nearest neighbors to the hospital with those from all neighboring healthcare facilities within 5 km. The plot on the right uses the total ARIs reported in 2009, while the plot on the left calculates the average ARIs per week. The 45 degree line is shown, as well as the line of best fit from a simple OLS regression.

Figure S5:
Distribution of Distance from Small Hospitals to Nearest Larger
Hospital and from Hospitals without ICU to Nearest ICU



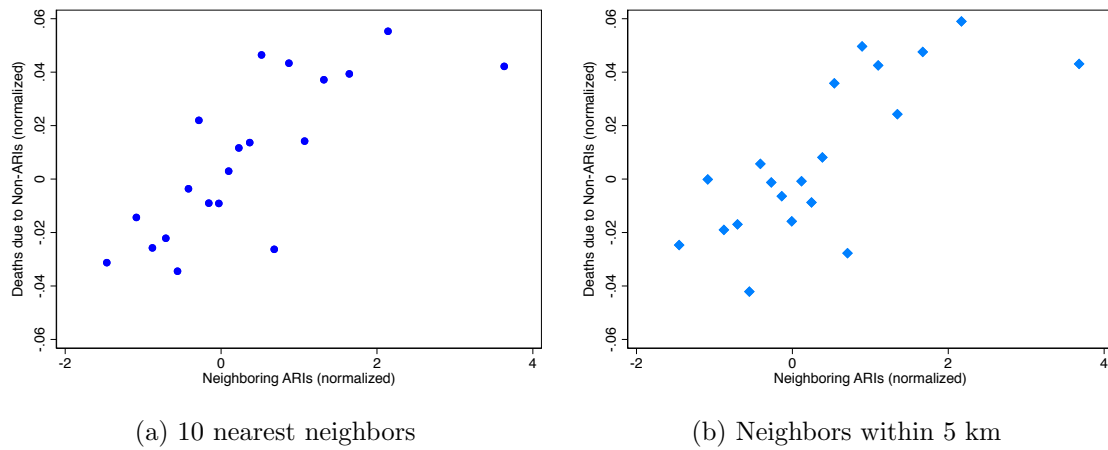
Notes: The plot on the top left shows a histogram for small hospitals (those in the bottom quintile of hospital beds) of the distance to the nearest larger hospital (any hospital not in the bottom quintile). The graph on the top right shows the corresponding box plot. The plot on the bottom left shows a histogram for hospitals without an ICU for the distance to the nearest ICU. The graph on the bottom right shows the corresponding box plot. Any distance above 100 km is capped at 100 for the histograms.

Figure S6:
Effect of ARI Outbreaks on Age Distribution of Non-ARI Hospitalizations



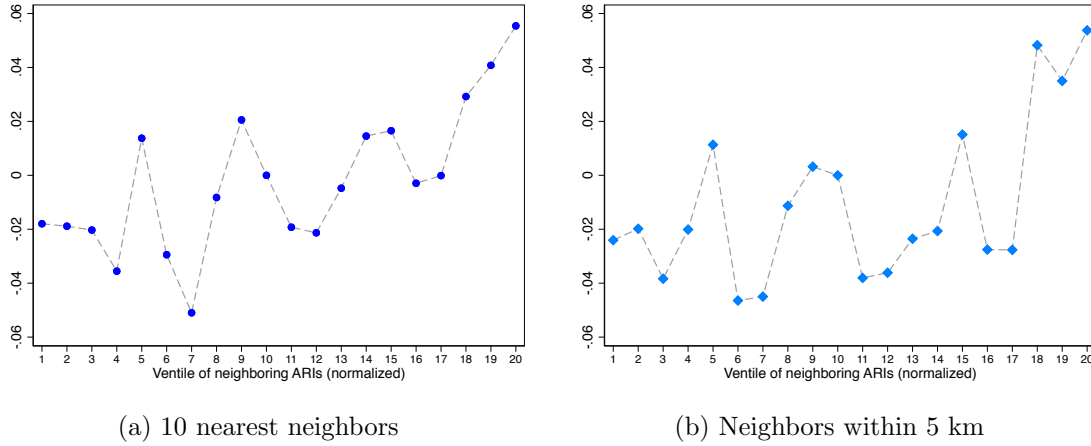
Notes: This plot shows IV estimates of shares of hospitalizations due to non-ARIs for different age groups, instrumenting (normalized) hospitalizations due to ARIs with the (normalized) measure of neighboring ARIs. Each coefficient corresponds to a separate regression. Each series corresponds to a different definition of neighboring healthcare facilities. Regressions include hospital and week FE. Bars correspond to 95% confidence intervals, from standard errors clustered at the hospital level.

Figure S7:
Relationship Between Non-ARI Mortality and ARI Outbreaks



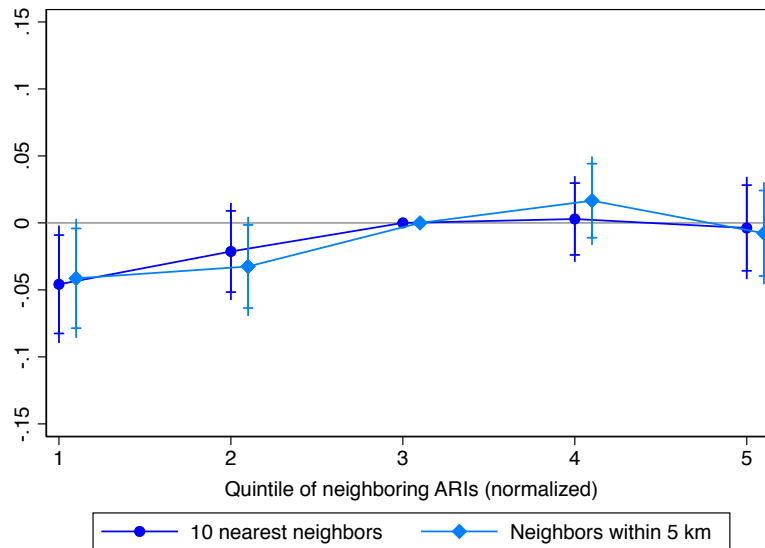
Notes: These graphs show a scatterplot of non-ARI deaths and ARI outbreaks. The plot on the left considers the 10 nearest neighbors, the one on the right all neighbors within 5 km. We partition the (normalized) neighboring ARIs into 20 bins and calculate the mean (normalized) deaths due to non-ARIs.

Figure S8:
Effect of ARI Outbreaks on Non-ARI Mortality by Ventiles of the
Outbreak



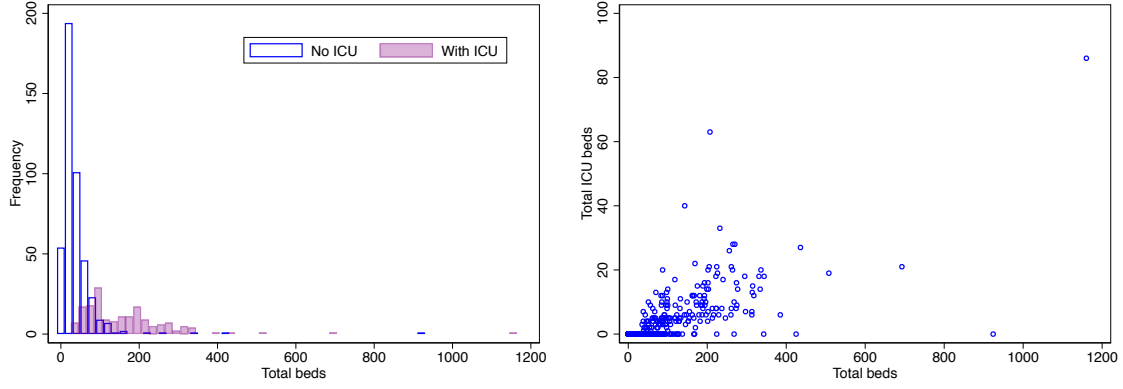
Notes: These graphs show OLS estimates of (normalized) deaths due to non-ARIs on the (normalized) measure of neighboring ARIs by ventiles. Each plot corresponds to a different definition of neighboring healthcare facilities. Regressions include hospital and week FE. For clarity, we plot the point estimates only.

Figure S9:
Effect of ARI Outbreaks on Non-ARI Hospitalizations by Size of the
Outbreak



Notes: This plot shows OLS estimates of hospitalizations due to non-ARIs on the (normalized) measure of neighboring ARIs by quintiles. Each coefficient series corresponds to a different definition of neighboring healthcare facilities. Regressions include hospital and week FE. Bars correspond to 90 and 95% confidence intervals, from standard errors clustered at the hospital level.

Figure S10:
Total Hospital Beds and ICU Beds

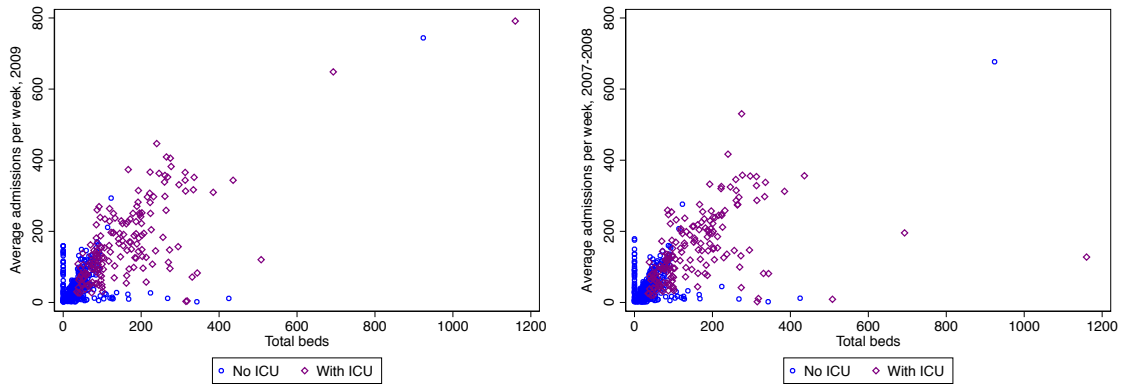


(a) Total hospital beds

(b) Total beds and total ICU beds

Notes: These plots describe hospital capacity as measured in 2013. The plot on the left shows histograms of total hospital beds, stratifying the sample between hospitals with and without an ICU. The plot on the right shows the correlation between total beds and total ICU beds for all hospitals.

Figure S11:
Correlation between Total Hospital Beds and Hospitalizations

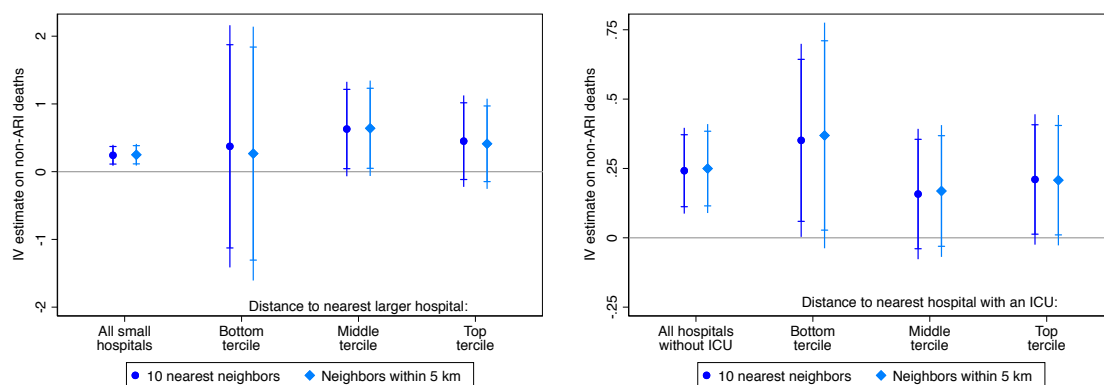


(a) Data for 2009

(b) Data for 2007 and 2008

Notes: These plots correlate the total number of hospital beds with the average weekly hospital admissions, distinguishing between hospital with and without an ICU. The plot on the left uses data from 2009, while the plot on the right uses pre-pandemic data.

Figure S12:
IV Estimates by Distance to Larger Hospitals and Hospitals with an ICU

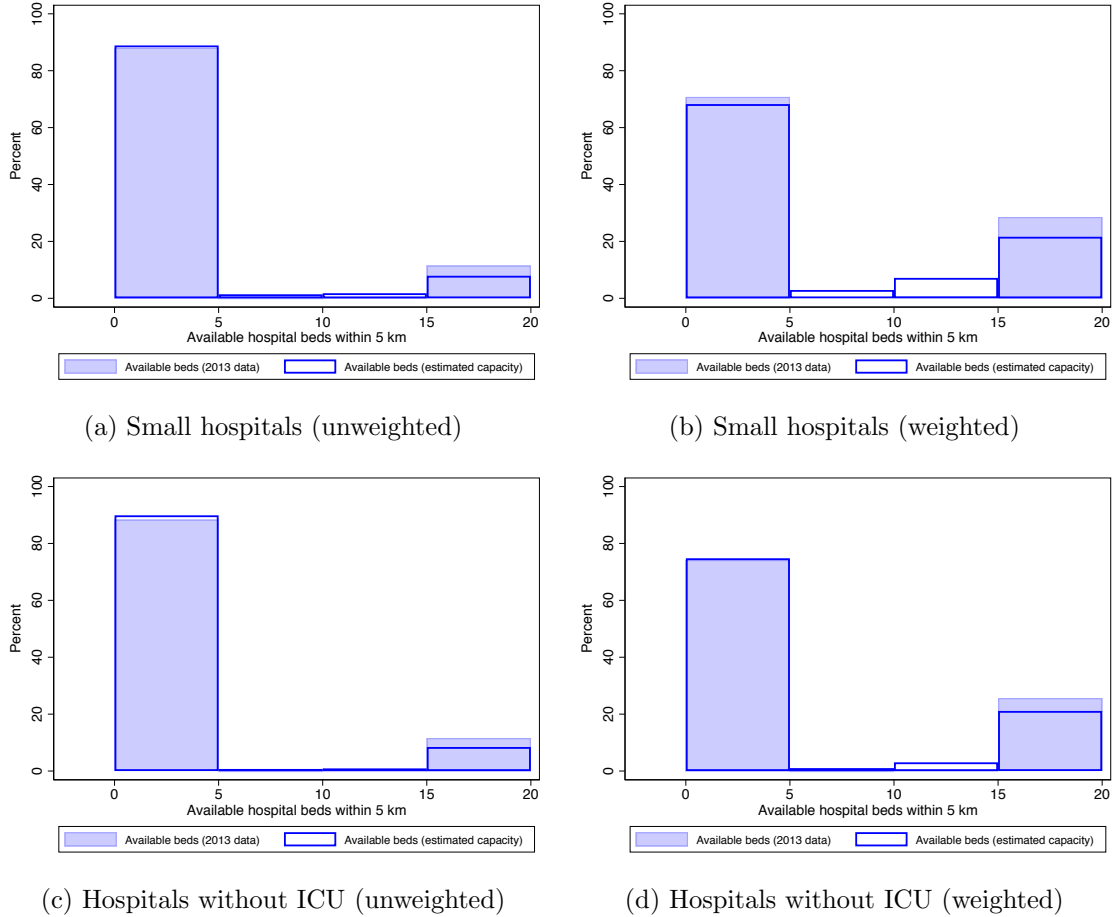


(a) Distance to nearest larger hospital

(b) Distance to nearest ICU

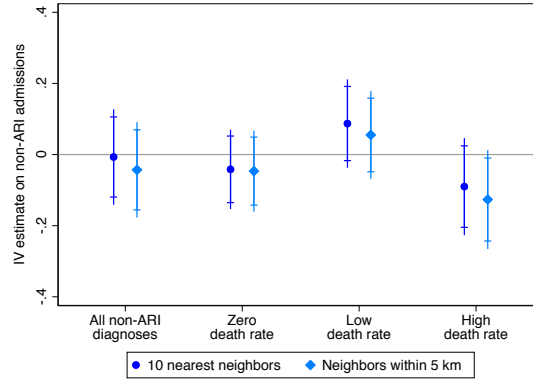
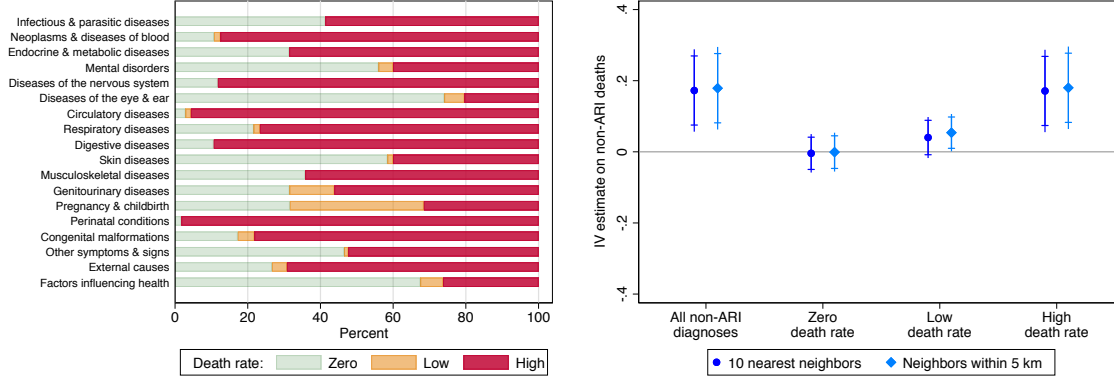
Notes: The plot on the left shows IV estimates on non-ARI deaths for small hospitals (bottom quintile of hospital beds) by terciles of distance to the nearest larger hospital (any hospital not in the bottom quintile), instrumenting (normalized) hospitalizations due to ARIs with the (normalized) measure of neighboring ARIs. The plot on the right repeats the exercise for hospitals without an ICU, stratifying by distance to the nearest ICU. Each coefficient corresponds to a separate regression. Each series corresponds to a different definition of neighboring healthcare facilities. Regressions include hospital and week FE. Bars correspond to 90 and 95% confidence intervals, from standard errors clustered at the hospital level.

Figure S13:
Available Beds within 5 km by Hospital Type



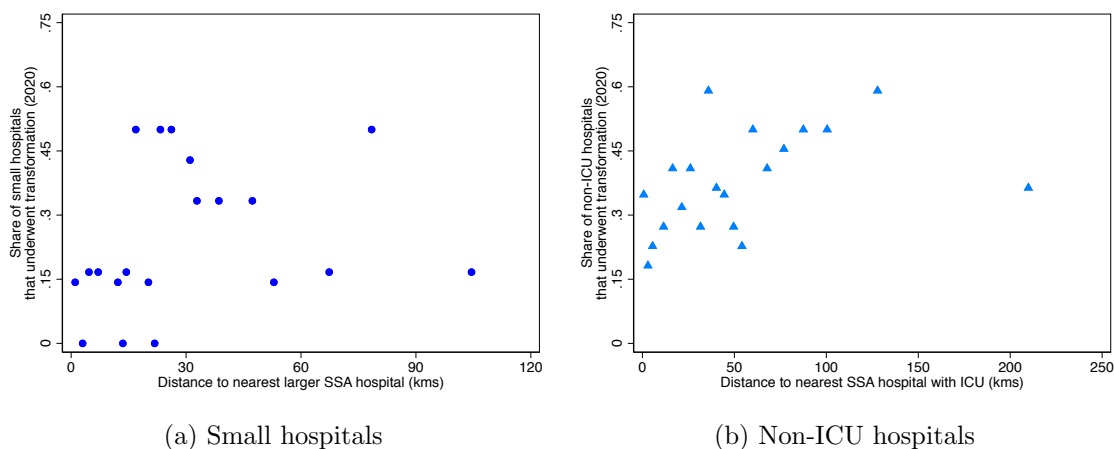
Notes: These plots show the average number of available beds within a 5 km radius for each hospital-week. The top panel considers available beds at large hospitals located within 5 km of each small hospital (bottom quintile). The bottom panel shows available beds at ICU hospitals within 5 km of each non-ICU hospital. Plots on the left show unweighted averages, and plots on the right weight each hospital-week by the number of non-ARI deaths. Any bed availability above 20 is capped at 20. We measure capacity of beds by taking either the 2013 infrastructure data on capacity or using hospitalizations data for 2007 and 2008 to construct an estimated capacity. Available beds are just the difference between capacity and observed hospitalizations.

Figure S14:
IV Estimates by Diagnosis-Level Mortality Rates



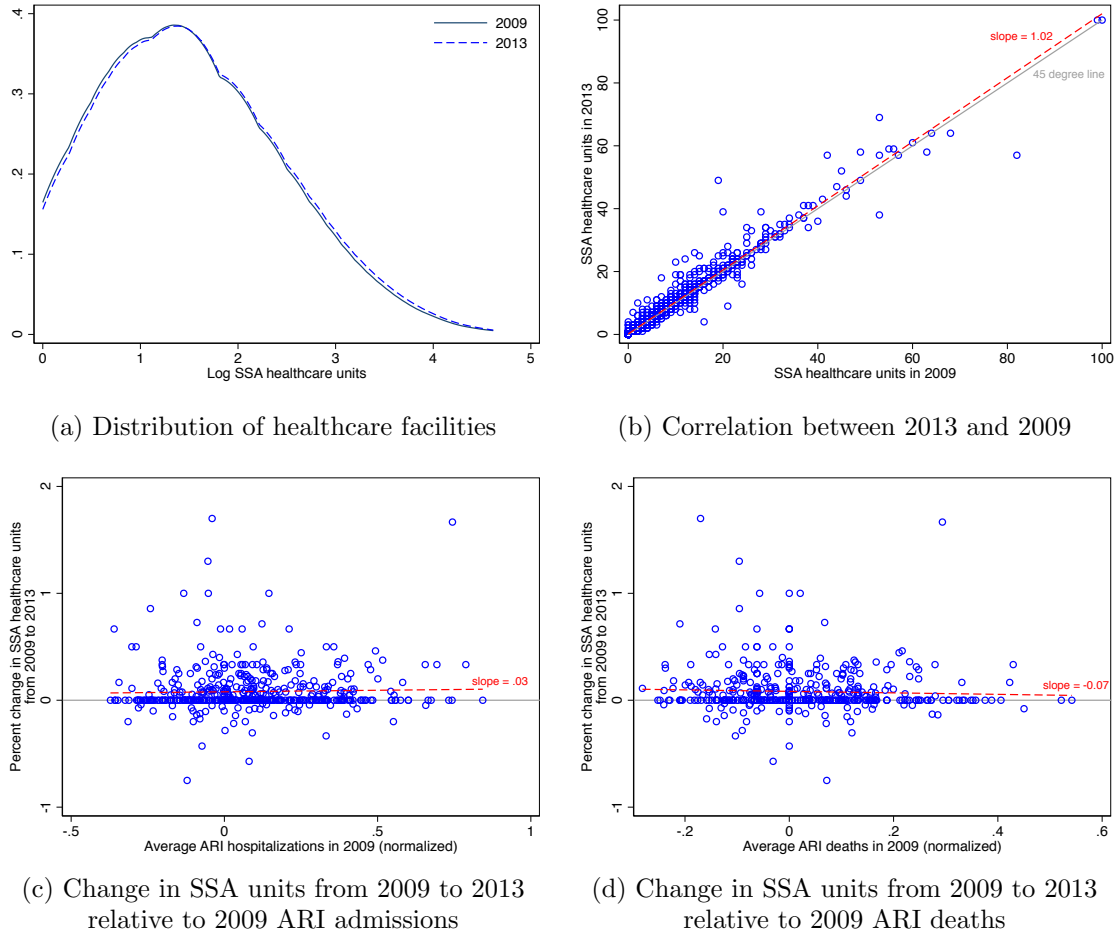
Notes: The plot on the top left shows the distribution of observed mortality rates during 2007 and 2008 by major diagnosis group. We stratify positive rates into low and high based on the admissions-weighted median. The plot on the top right shows IV estimates on non-ARI deaths by pre-2009 diagnosis-specific mortality rates, instrumenting (normalized) hospitalizations due to ARIs with the (normalized) measure of neighboring ARIs. The plot on the bottom shows IV estimates on non-ARI hospitalizations by pre-2009 diagnosis-specific mortality rates, instrumenting (normalized) hospitalizations due to ARIs with the (normalized) measure of neighboring ARIs. Each coefficient corresponds to a separate regression. Each series corresponds to a different definition of neighboring healthcare facilities. Regressions include hospital and week FE. Bars correspond to 90 and 95% confidence intervals, from standard errors clustered at the hospital level.

Figure S15:
Hospital Transformation of Small and Non-ICU Hospitals during the
Covid-19 Outbreak by Distance



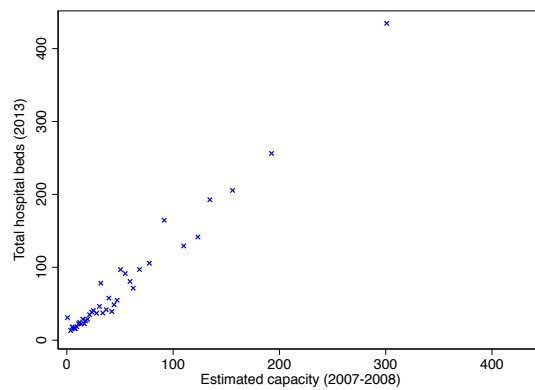
Notes: The graph on the left shows a binned scatterplot for small hospitals of the relationship between distance to the nearest larger hospital and the share of those small hospitals that underwent a transformation during the Covid-19 outbreak. The graph on the right shows the same for non-ICU hospitals by distance to the nearest ICU. Each graph considers 20 bins of distance, with each marker representing the within-bin mean. Hospital transformation refers to increases in capacity as measured by hospital beds, training of medical personnel to deal with increased demand and larger shares of severely-ill patients, and setting up a triage system. Data on hospital transformation were obtained from the government via a freedom of information request (October 2020).

Figure S16:
Municipality-Level Healthcare Facilities in 2013 and 2009



Notes: These plots show municipality-level infrastructure data for the SSA system for 2013 and 2009. Healthcare units include both hospitals and clinics. The plot on the top left shows the distribution of number of facilities in a municipality for each year. The plot on the top right shows the correlation between the number of units each year. The plots on the bottom correlate the municipality-level averages of the (normalized) 2009 ARI hospitalizations and ARI deaths with the percentage change in the number of SSA units from 2009 to 2013.

Figure S17:
Hospital Beds in Measured 2013 and Estimated with 2007-2008 Data



Notes: This plot shows the correlation between hospital beds as measured in 2013 and an estimate of beds from the 2007-2008 hospitalizations data. We use the maximum stock of patients in a given hospital during 2007 and 2008 as a proxy for the total bed capacity of the hospital.