Covid And The City: Intra-Hospital Transmission of Covid-19

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Abstract

Intra-hospital contagion is a potent and persistent channel of Covid-19 transmission, yet its role in the New York City outbreak has been relatively unexplored in the prevailing literature. Using event study and panel regression analyses, we investigate the effect of hospitalisation on the proportion of positive test results out of total tests conducted at the borough level in New York City. We find that the impact of hospital capacity on positive test rate is initially negative and becomes positive after roughly a month, reflecting the idea that greater capacity is initially a boon but ultimately a greater bane. Furthermore, we find a positive and statistically significant effect associated with hospitals in a borough on average being above median capacity (an above-median capacity 'shock'), after the first day of the shock; this suggests that on average, a surge in positive tests accompany hospitals in above-median capacity in a borough after the first day of experiencing the shock. Policy implications include the need to even out the burden of Covid cases across hospitals. We conclude with a discussion on limitations and the merits of obtaining better-quality data. Additional graphs that examine heterogeneity are included in the Appendix.

*This submission contains 5 pages of text, and 5 pages of Appendices, excluding references, cover page and table of contents.

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1 Introduction and Literature Review

Ironically, one of the most negative words in 2020 was 'positive'. A number of economic and behavioural mechanisms have contributed to the transmission of Covid-19, culminating in the high numbers of positive test results seen in the notorious New York City outbreak. One particular mechanism stands out as another sad irony - hospitals, meant to combat Covid, ended up being fertile grounds for its spread. The contribution of intra-hospital contagion to the spread of Covid in New York City is the main focus of our paper.

The bulk of Covid research has explored the channels of Covid transmission preceding the "stay at home" period (hereafter, lockdown). Among other factors, the contribution of public transport, partisan differences, and occupational characteristics have been assessed (Harris, 2020; Allcott et al., 2020; Almagro and Orane-Hutchinson, 2020). However, how Covid continues to spread during lockdown deserves closer attention. Investigation of key drivers of Covid transmission *during lockdown* can provide policymakers with crucial insights on the best ways to tackle the channels of spread that persist even when harsh restrictions are already in place.

Lockdown has proven effective in closing off some of the most prominent channels of Covid transmission in many countries (Lau et al., 2020; Alfano and Ercolano, 2020). Yet, some channels persist after its implementation, taking centre stage. It is no surprise that intra-hospital transmission is one such channel, with hospitals worldwide generally ill-prepared to meet the influx of cases. Mixing of Covid-infected patients with uninfected patients, lapses in protection in interactions of hospital staff with Covid patients, and their subsequent contact with colleagues are some media-documented ways that hospitals contributed to the spread of Covid in New York City (Ramachandran et al., 2020; Evans, 2020). What is surprising is the dearth of scholarly attention paid to intra-hospital transmission in New York City, especially in comparison to its coverage in the media. The devastating role played by intra-hospital contagion in exacerbating outbreaks in other places, such as Brisbane and Wuhan (see Robertson, 2021; Wang et al., 2020), prompts us to explore its contribution to the outbreak in New York City.

We investigate the role of intra-hospital contagion in the New York City outbreak by testing two hypotheses: (1) whether hospitals with greater capacity contribute to higher infection rates; (2) whether overcapacity of hospitals leads to higher infection rates. The first hypothesis stems from the idea that hospitals can be breeding grounds for the spread of Covid. If true, it would follow that hospitals with larger capacity would, by virtue of housing more Covid patients, be more susceptible to in-house spread of Covid within these hospitals. The second hypothesis stems from the idea that hospitals which are more overburdened are less able to coherently execute measures designed to prevent in-house contagion of Covid. For instance, Personal Protective Equipment (PPE) shortages may be likelier or more dire in overburdened hospitals. Hospital staff, under high pressure from an influx of patients, may also be less attentive in keeping to protocol for preventing in-house contagion. All these increase the chances of in-house contagion, leading to higher infection rates on average.

2 Data and Empirical Methods

2.1 Data

We choose to supplement the *metricsgame2.dta* dataset with data from (1) the New York State Department of Health, (2) the Department of Health and Mental Hygiene, and (3) the Department of Health & Human Services. From (1), we identify hospitals in NYC by zipcode and their associated (pre-pandemic) bed count; from (2), we take the number of positive tests at the borough level; from (3), we use the number of admissions associated with Covid at the hospital level.

2.2 Event Study

We estimate the effect of borough-level hospital capacity on the rate of positive test results - i.e. the ratio of positive tests to total tests. We choose to weight the positive tests this way to account for heterogeneous populations by borough, as boroughs with greater populations have a higher number of tests. We verify this by calculating the correlation between these two variables, and find a strong positive correlation of 0.9126. The rationale for this weighting is that the number of positive tests is affected by the total number of tests carried out, and so it makes sense to weight this by testing numbers rather than population.

$$y_{it} = \alpha_i + \rho_t + \beta \sum_{i=beginning, i \neq n}^{end} X_i \times Event_t + \epsilon_{it} \tag{1}$$

Where y_{it} is the log ratio of positive tests to total tests at the zipcode level, α_i are zipcode-level fixed effects, ρ_t are day-of-the-week fixed effects, X_i is our measure of hospital capacity defined as either the number of hospitals in a borough or the number of beds in all hospitals in a borough, and $Event_t$ is the full set of event-time indicators.

β is our parameter of interest which reflects the effect of how the impact of hospital capacity on ratio of positive tests to total tests evolves over time.

The subscript i denotes zipcode and t denotes time (in days). Day-of-the-week fixed effects non-parametrically accounts for specific trends in positive ratios due to different days of the week, such as different secular trends in testing rates due to work schedules. Zipcode fixed effects accounts for time-invariant observable and unobservable confounders that may affect the intensity of hospitalisation, such as the race composition of the borough or attitude to risk. Arguably, attitudes to risk may evolve over time as the pandemic develops and hence motivates the need for time fixed effects.

In the *metricsgame2.dta* dataset, observable characteristics such as local income levels, ethnicity, and occupation are all time-invariant due to them being collected from the cross-section American Community Survey in 2018. With these sets of fixed effects, we identify our effect of interest by comparing borough areas with higher hospital intensity with those with

lower hospital intensity. Our event-study approach allows us to examine trends in positive rates over time and flexibly accounts for time-invariant and time-variant at the borough level.

2.3 Difference-In-Differences Analysis

Having collected data on hospitalisation rates over time from the Department of Health & Human Services, we intend to exploit time variation in when hospitals on average in a borough reach critical capacity, defined as a shock to their hospitalisation capacity.

y_{it} is the total number of cases in borough i and day t, $Shock_{it}$ is a dummy variable that equals 1 if the borough was observed on or after the day its hospitalisation capacity - defined as total increase in hospitalisation over existing beds had exceeded its overall median level. We interpret $Shock_{it}$ as a time-variant, borough-specific shock. The coefficient of interest is β, which measures the average effect of shock to hospitalisation capacity on

We control for borough fixed effects and day fixed effects to account for time-invariant, borough-specific heterogeneity (income levels, infrastructure levels, existing level differences in hospital qualities), as well as time-variant shocks (changes in commuting costs, weather shocks).

$$y_{it} = \alpha_i + \rho_t + \beta Shock_i \times Post_t + \epsilon_{it} \tag{2}$$

Given the small number of clusters in this case (we only have 5 boroughs), we reason that clustering the standard errors at one-way still results in poor effective coverage rates of confidence intervals and over-rejection of the null hypothesis. To partially mitigate this, we two-away cluster the standard errors at the borough-day level, while applying cluster-robust standard errors corrections developed by Cameron and Miller (2015).

3 Discussion of Results

3.1 Event Study Results

For both measures of the explanatory variable (number of hospitals and bed-to-hospital ratio) in Figure 1 and **??**, we observe that there is a slight initial decrease in the point estimate impact on the positivity rate, but this coefficient soon becomes positive. The initial decrease is a sensible result if we consider greater hospital capacity to be initially an advantage for boroughs in identifying, treating and containing Covid cases. The increase in the coefficient has remained stable after a month, and is potentially a source of concern, as this indicates that higher hospital capacity is associated with a higher rate of positive tests. This is consistent with our first hypothesis that hospitals are a breeding ground for Covid cases.

We also explore heterogeneity of the effects depending on the existing occupational share

at zip-code level working in health services (as shown by Figure 3), as a potential mechanism of higher hospital intensity resulting in higher infection spreads. This was motivated by the hypothesis that zip-code areas with higher hospital intensity led to higher infection spreads due to more high risk healthcare workers residing in the area. Our results provide little evidence in support of this hypothesis.

Our results paint an overall picture of greater hospital capacity being a mild advantage for combating Covid at the start, but later backfiring due to their severity as breeding grounds for Covid. However, we caution that our results are generally statistically insignificant, and the informativeness of the point estimates are convoluted by the large standard errors.

3.2 Panel Regression Results

As seen in Table 1, we find a statistically significant and positive effect of the coefficient of interest. This indicates that on average in a borough, hospitals that are experiencing above-median capacity are associated with a greater number of positive cases in the period after the first day of experiencing the shock.

3.3 Policy Implications

The hospital capacity results point to the importance of quality of measures aimed at prevention of in-house contagion. With more robustly designed preventative measures, hospitals may avoid having greater capacity being more of a bane than a boon. This is in line with observations that intra-hospital contagion became less of an issue over spring, as hospitals became more experienced in dealing with Covid patient influxes (Almagro and Orane-Hutchinson (2020)).

At the borough-level, panel data regressions gives suggestive evidence that shocks to hospital capacity, potential causing hospital exceeding their attainable capacity of caring patients, was associated with spikes in new cases. While we caution that the result should be taken with caution, this highlights potential mechanism of overloaded hospitals raising spread of infections, such as due to high patient intensity and community infections.

Our results from the event studies also suggest that it would be beneficial to maintain an even spread of patients across hospitals in New York City. This would prevent any particular hospital becoming (relatively) overburdened and potentially limit the effect of rising caseloads at each hospital on further transmission. A clear example of this is the 'Public-Private Hospital Plan' which could be further expanded to fully integrate treatment and PPE stocks as well as sharing case loads.

One reason cases may be rising over time is that PPE may be becoming exhausted as overburdened hospitals struggle to cope with demand. This would explain how such hospitals may be seeing a positive effect on cases as time progresses, as a falling stock of PPE reduces the ability of staff to protect themselves and hence other patients from Covid. It would therefore be important to ensure that PPE stocks are diverted from relatively underwhelmed hospitals to those which are experiencing a higher case load.

4 Conclusion

We make a sincere attempt to address a gap in the existing literature, quantitatively investigating the contribution of intra-hospital contagion *during* lockdown.

From our event study, our findings suggest that density of hospitals in an area may be positively associated with Covid cases especially in the medium-to-long run. This clearly has policy implications for a city undergoing lockdown: overcapacity in a hospital can easily spur in-house contagion, so efforts to even out the burden across hospitals are especially important.

Changes in policy necessitate revisions to our research and may provide additional sources of variation between and within geographical units, which in turn allow the use of other empirical methodologies.

A potential extension to our paper would be to account for hospitals close to the border of each borough. Currently, we do not account for the fact that hospitals close to a borough border will, under the assumptions of our model, impact the positive test rate of inhabitants in zipcodes just beyond the border. As such, the Stable Unit Treatment Value Assumption (SUTVA), that our unit of analysis does not affect the treatment status or potential outcomes of other units, may not hold.

Another extension we could employ would be to account for behavioural factors in our research, such as behavioural fatigue due to the ongoing nature of the pandemic. Such factors may be difficult to quantify, however.

Finally, better data can help us build a more robust empirical strategy. With Covid data on pre-lockdown time periods, we would be able to verify the non-existence of pre-trends that would support the validity of our event study. Additionally, more data at the zipcode level would provide an escape from the limitation imposed by the number of boroughs we have in our data for our difference-in-differences strategy, which prevents any meaningful clustering of standard errors using Arellano's White standard errors at the borough level. We may wish to cluster our standard errors Arellano (1987) at the zipcode level as we are concerned about serial correlation in the ratio of positive to total tests within zipcode-level regions. Failure to cluster will mean that we assume the i.i.d. of our observations and hence that each additional time-period observation of a unit gives us more information than we actually have, and this fails to reflect the true uncertainty in our sample. It is also more likely that zipcode characteristics would allow us to control for greater heterogeneity than is currently feasible at the borough level.

5 Appendix

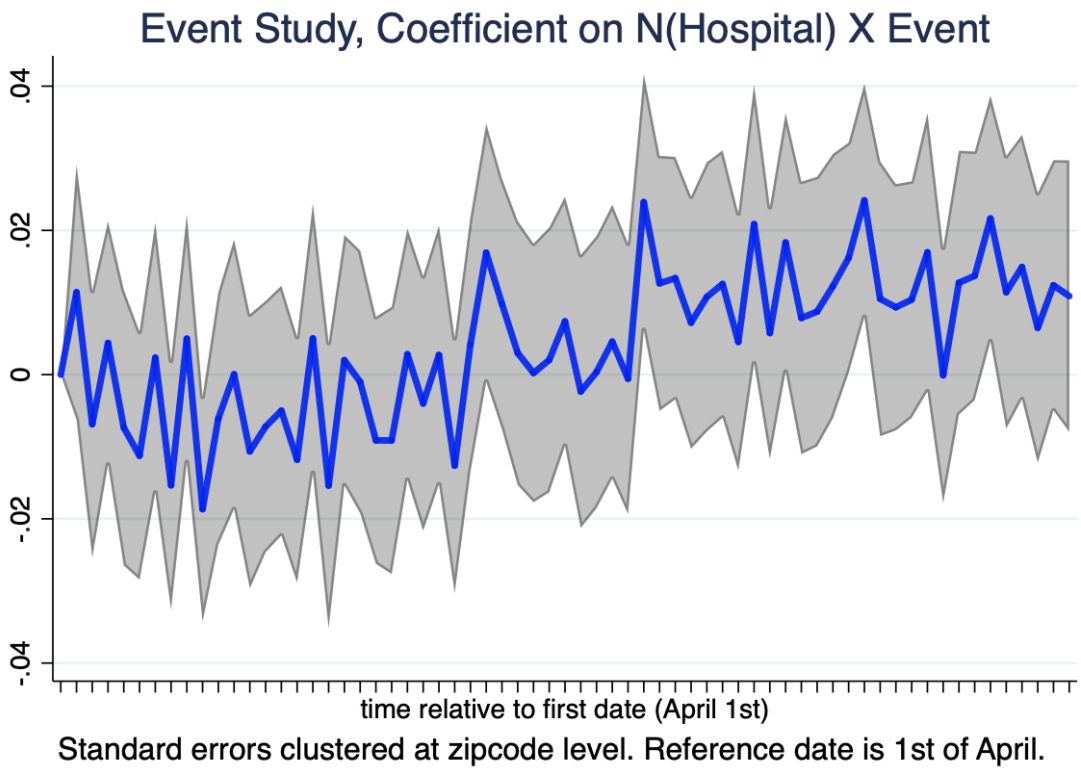

Figure 1: Event study plot of coefficient using number of hospitals as explanatory variable.

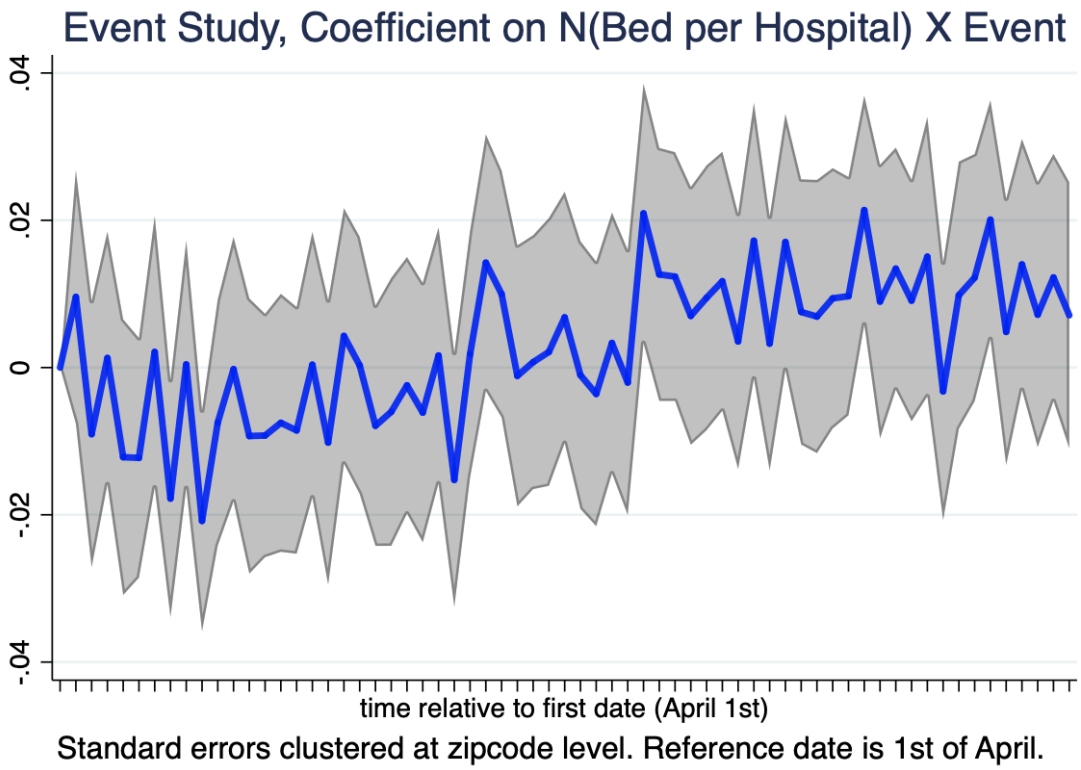

Figure 2: Caption

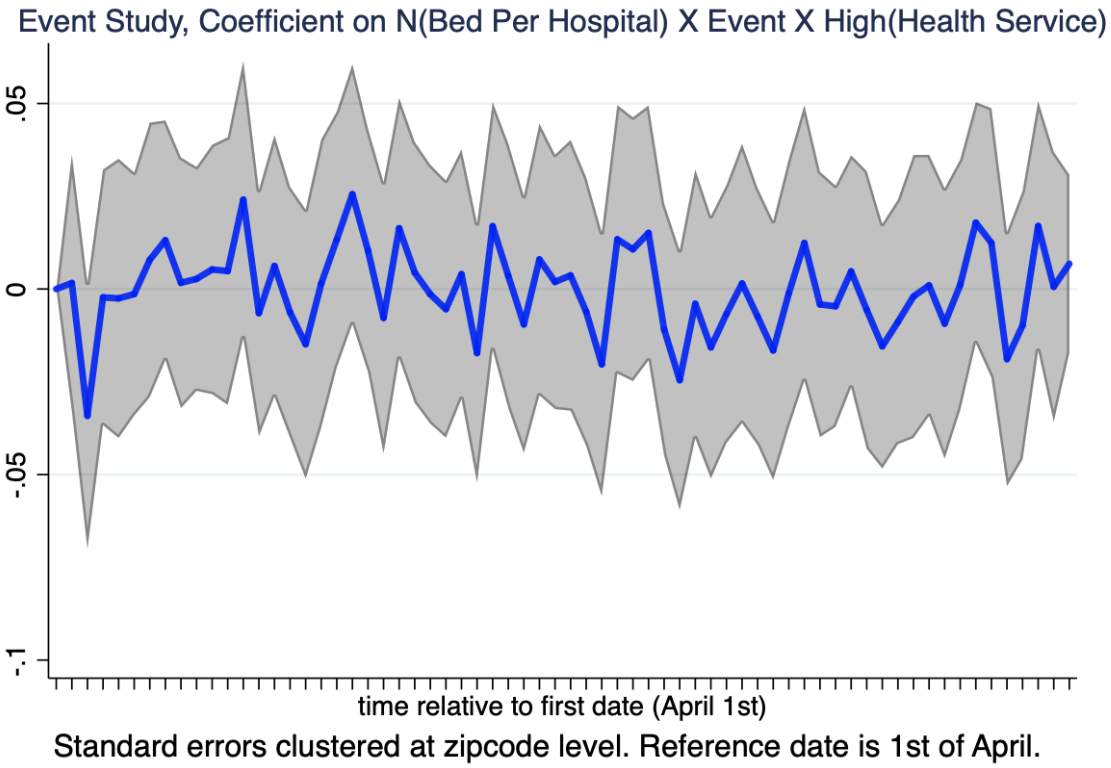

Figure 3: Heterogeneity, HealthService

VARIABLES	(1) Positive Case Count
$Shock_i \times Post_t$	1,130*** (84.23)
Observations	1,116
R-squared	0.026
Borough FE	Yes
Day FE	Yes
Two-way Clustering	Yes

Table 1: Difference-in-difference results.

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