Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Persistent Pandemics☆

Peter Z. Lin [b], Christopher M. Meissner [a,*]

[a] Department of Economics, University of California, Davis and National Bureau of Economic Research (NBER), United States
[b] California Center for Population Research, University of California, Los Angeles, United States

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ABSTRACT

We ask whether mortality from historical pandemics has any predictive content for mortality in the Covid-19 pandemic. We find strong persistence in public health performance. Places that performed worse in terms of mortality in the 1918 influenza pandemic also have higher Covid-19 mortality today. This is true across countries as well as across a sample of large US cities. Experience with SARS in 2003 is associated with slightly lower mortality today. We discuss some socio-political factors that may account for persistence including distrust of expert advice, lack of cooperation, over-confidence, and health care supply shortages. Multi-generational effects of past pandemics may also matter.

1. Introduction

The Covid-19 pandemic is one of the largest threats to global public health and the global economy since the influenza pandemic of 1918. Was the world better prepared for a highly contagious airborne pandemic in 2020 than it was in 1918? It might be expected that in the intervening 100 years societies would have made great progress in predicting, containing, mitigating, and managing pandemics (Morens and Fauci, 2007). Indeed, the global mortality rate from Covid-19 remains well below the influenza of 1918, despite having an estimated case fatality rate of half the magnitude of the 1918 influenza.[1]

Additionally, the most recent global public health scares such as SARS, MERS, Ebola, and H1N1 influenza were largely successfully contained without extraordinary levels of excess mortality at the global level. This track record suggests high preparedness and ability to manage pandemics.

On the other hand, Covid-19 has been a significant shock, and some places have been harder hit than others. What factors might explain this variation in public health outcomes? Our research focusses on the long-run correlates of population mortality rates from Covid-19. Specifically we relate mortality from Covid-19 to death rates in the 1918 influenza pandemic and other past pandemics. We perform this exercise in a broad sample of countries and for a sample of large US cities, and we control for a number of confounding factors.

We find strong evidence of long-run persistence in public health performance. In the first weeks and months of the Covid-19 pandemic, places that performed poorly in terms of mortality during the "Spanish flu" were more likely to have higher mortality. This is true across countries and across a sample of US cities. Our results are robust to inclusion of a range of fixed effects, control variables and corrections for spatial correlation. The results for the US city data are robust to endogeneity using an instrumental variables strategy.

We also find that there has been some recent success consistent with the possibility of learning (at the societal level) over time. Countries that were more strongly affected by SARS in 2002-03 are likely to have lower mortality rates from Covid-19 thus far in proportion to their experience with SARS. These places are mainly in East Asia where there is recent memory of a potentially highly lethal pandemic.

As we detail in the discussion, the mechanisms by which the past may affect us today can be numerous. Preparedness for disasters may

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* Corresponding author.
E-mail addresses: zxlin@ucla.edu (P.Z. Lin), cmm@ucdavis.edu (C.M. Meissner).

[1] According to Our World in Data as of April 6, 2021 2.87 million people have died from Covid-19 or about 0.035% of world population. The flu of 1957-58 and the flu of 1968-70 are estimated to have killed about 1 million people each for about 0.02% and 0.034% of world population (HYPERLINK \l "Ref12" \o "Centers for Disease Control and Prevention, 2007 Centers for Disease Control and Prevention 2007. Interim Pre-Pandemic Planning Guidance: Community Strategy for Pandemic Influenza Mitigation in the United States— Early, Targeted, Layered Use of Nonpharmaceutic" Centers for Disease Control and Prevention, 2007). The influenza of 1918 killed roughly 50 million people or about 2.6% of world population (Crosby, 1989).

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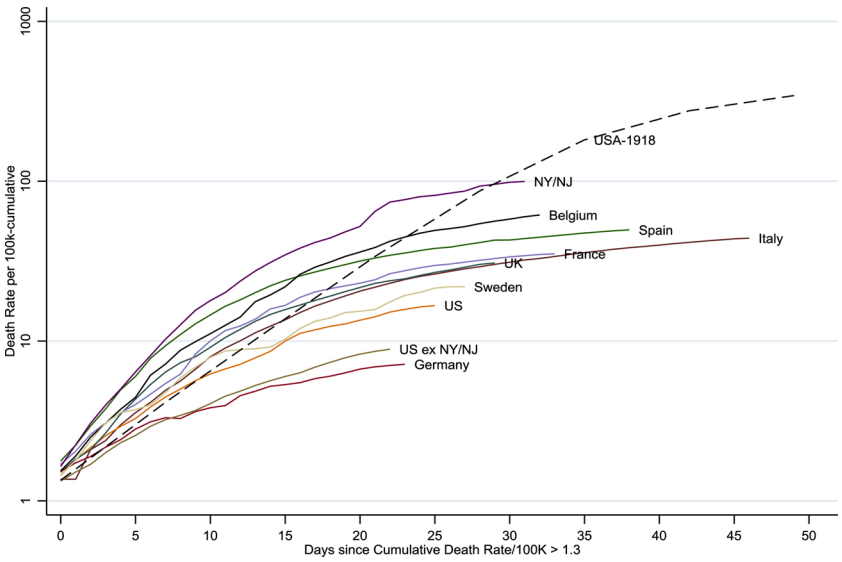

Fig. 1. Mortality Rate per 100,000 Covid-19 versus US Influenza Mortality in 1918: Cross-Country Evidence.
Notes: Figure shows the population mortality rates for Covid-19 based on data from CSSE (2020). We break the data for the US into three parts: mortality for the entire US, mortality rates for the states of New York and New Jersey, the hardest hit states to date, and for the US excluding these two states. Data for the influenza pandemic of 1918 are for total weekly deaths per 100k from influenza and pneumonia for data from 46 cities in the USA (Collins et al., 1930). Data are plotted for countries in 2020 that had reached a threshold of 1.34 deaths per 100,000. This is the first available level of the mortality rate in the 1918 for the national level data for the USA. Data from 2020 are as of 27 April, 2020.

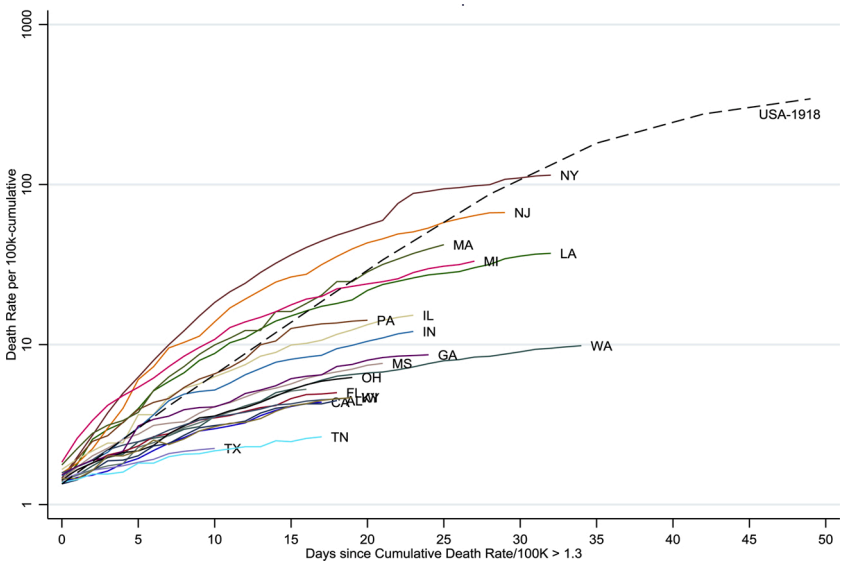

Fig. 2. Mortality Rate per 100,000 Covid-19 versus US Influenza Mortality in 1918: US States.
Notes: Figure shows the population mortality rates of Covid-19 based on data from CSSE (2020). Data for the Influenza pandemic of 1918 are as described in the notes to Fig. 1. Data for Covid-19 are as of 27 April, 2020.

increase when the event was recent, but this "collective" memory seems to fade over time (Fanta et al., 2019). Socio-political threats to preparedness such as "distrust, isolationism and hubris" are also a possibility (Parmet and Rothstein, 2018). Trust, a key factor in public health, evolves slowly and is driven by past pandemics (Aassve et al., 2020). Without doubt, society and politics have changed since the 2009 H1N1 pandemic and potentially in a direction that might work against public health efforts, particularly in the US.[2] Perhaps politics and social forces of 1918 and today have some similarities merely by coincidence. Finally, we also highlight the possibility of a multi-generational biological impact of the 1918 influenza.

2. Preliminary Discussion and Background

The public health response to the Covid-19 pandemic represents a significant test of preparedness and represents a new opportunity to study the key determinants of public health today. Could public health outcomes in 1918 and in the more recent past matter even today? Are the drivers of public health in the past related to those today?

At first glance, the past might seem irrelevant. Rapid geographic mobility due to air travel has increased dramatically in recent decades and significantly so with respect to the years 1918-1920. International connections and cooperation is more significant than in the past. The idea that international cooperation is waning is exemplified by the Trump administration's proposal to reduce funding and support for the WHO in 2020. Modern methods of communication and social media platforms complicate the search for accurate content and often create confusion.

Health infrastructure and accessibility is better than in the past in most countries. Still, experts predicted the possibility it would be

[2] See Iyengar et al. (2019) for evidence on the US and Boxell et al. (2020) for a recent comparative analysis.

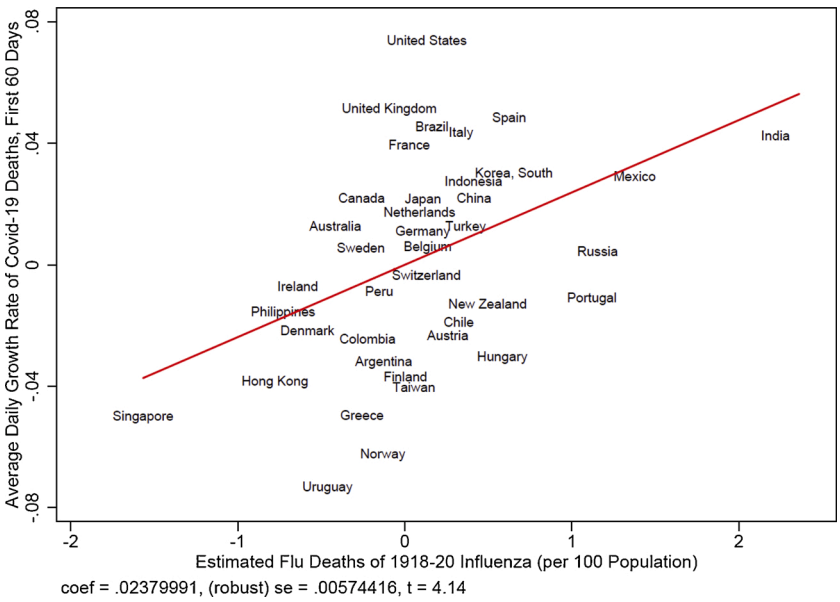

Fig. 3. Mortality of 1918-20 Influenza and Covid-19 Pandemics, 39 Countries.
Notes: This graph plots the average daily growth rate of total deaths from Covid-19 in the first 60 days since the first death in a sample of 39 countries against the excess mortality rate of the 1918 Influenza pandemic, both conditional on a set of regional fixed effects (we include fixed effects for the Americas, Europe, East Asia, South Asia, and Oceania). Data are described in the data appendix A. The average growth rate of total deaths for Covid-19 is calculated as $\sqrt[59]{cmdeath_{i60}/cmdeath_{i1}} - 1$. The robust t-statistic for the coefficient on deaths from influenza in 1918 is = 4.14, and the regression has an $R^2 = 0.5$.

incapable of meeting surging demands induced by a pandemic (Morens and Fauci, 2007). Such capacity bottlenecks can raise cumulative mortality even when modern health care provides a viable means of treatment for a disease.

Comparing outcomes today to the past is difficult. SARS-CoV-2 and the 1918 H1N1 influenza have different etiologies and epidemiological properties. Nevertheless, both pandemics seem to pose significant risks based on estimates of case fatality rates. An estimate of the case fatality rate (CFR) for Covid-19 is 1.38% while the CFR for the 1918-20 influenza has been estimated to be roughly 2.5% (Verity et al. 2020 and Short et al., 2018).[3]

Given the estimated case/infection fatality rates, most people would predict lower mortality in the early stages of the Covid-19 pandemic than in 1918-20. After all, humanity has a century of public health research and practice, along with experience gained from SARS, MERS and Ebola. Contingency plans have been formulated at the behest of the WHO and through national initiatives. Non-pharmaceutical interventions (e.g., social distancing) designed to lower peak mortality have been investigated and shown to be effective (Bootsma and Ferguson, 2007; Hatchett et al., 2007; Markel et al., 2007).

Recent data make us less sanguine. Fig. 1 illustrates that many countries, especially advanced western countries, had mortality rates above the frontier defined by US mortality rates from flu and pneumonia in 1918 at similar stages in the pandemic.[4] Similarly, Fig. 2 shows a number of US states also witnessed mortality rates per 100,000 population above those witnessed in 1918 at a similar stage. Clearly, public health knowledge and practices are not the only thing that matter for outcomes in a pandemic. Performance should have been better than in 1918 given the lower mortality rates

We emphasize that our goal in this paper is not to assess the level of mortality in one pandemic versus the other.[5] There are obvious problems comparing distinct diseases and many data measurement issues. Neither do we wish to argue that Covid-19 will be worse in terms of cumulative mortality than the 1918 pandemic. Instead, we compare relative outcomes, and we ask whether the mortality outcomes of historical pandemics have any predictive content for mortality in this contemporary pandemic. We find that historical experience helps predict recent experience even after controlling for a range of potentially confounding factors. This suggests, at the very least, that the "technology" of public health and preparedness are as of yet unable to overcome entirely the shadow of the past.

3. Data

3.1. Data Collection: Countries

We collect data on country–level population mortality from the influenza pandemic of 1918 and from Covid-19. Our baseline country sample covers 39 countries for up to 120 days since the 100thcase (corresponding roughly to February 2020 until mid-July 2020). The country sample is determined by availability of estimated mortality rates from the 1918 influenza pandemic, other control variables, and whether a country had recorded at least one death or confirmed case of Covid-19. Therefore, our sample for cross-country comparison covers those countries subjected to Covid-19 relatively early in the recent global pandemic.

Data on mortality from Covid-19 are expressed as the number of total deaths per 100 thousand population and are from CSSE Johns Hopkins University (2020). For Covid-19, excess mortality statistics are not readily available yet for our entire sample on a consistent basis. Under- or inconsistent reporting of death from Covid-19 might be a problem which deserves further scrutiny in later research. Nevertheless, these numbers are the official numbers, and those which have driven policy and response in the first year of the pandemic.

Data on mortality in the 1918 influenza pandemic are also expressed in deaths per 100,000 population and come from Barro et al. (2020).[6]

[3] Case fatality ratios for the 1957 and 1968 influenza pandemics were much lower at roughly 0.27 and 0.15 (Centers for Disease Control and Prevention, 2007).

[4] The mortality rates from flu and pneumonia in 1918 are based on a widely used data base of weekly death rates in a group of 35 cities (see Table 1, Collins et al., 1930).

[5] See the papers by Beach et al. (Forthcoming), He et al. (2020) and Petersen et al. (2020) for comparisons of pandemics over the last 100 years.

[6] Barro et al. (2020) develop an original database of excess mortality from influenza and pneumonia. When this is not available they rely on total excess mortality and report that "…comparisons of direct yearly estimates of death rates from influenza/pneumonia with all-cause excess mortality rates for countries with both types of data indicate a close correspondence for the two methods." The primary data sources they use include Johnson and Mueller (2002), Murray et al. (2006), Mitchell (2007), and the Human Mortality Database (www.mortality.org).

Table 1
Mortality Rates of Three Pandemics: 1918-20 Influenza, 2002-03 SARS, and Covid-19

Country	Mortality Rates of 1918-20 Influenza (per 100,000)	Mortality Rates of 2002-2003 SARS (per 100,000)	Mortality Rates of Covid-19 by Oct 1, 2020 (per 100,000)
Argentina	330	0	45.31
Australia	280	0	3.53
Austria	970	0	8.96
Belgium	830	0	86.86
Brazil	690	0	68.55
Canada	620	0.131	25.04
Chile	860	0	67.66
Colombia	460	0	52.04
Denmark	310	0	11.28
Finland	710	0	6.22
France	740	0.002	43.25
Germany	780	0	11.39
Greece	450	0	3.75
Hungary	1270	0	8.06
Ireland	430	0	36.99
Italy	1230	0	59.32
Mexico	2060	0	61.20
Netherlands	710	0	37.54
New Zealand	690	0	0.52
Norway	570	0	5.09
Peru	390	0	99.85
Portugal	1810	0	19.33
Russia	1870	0	14.26
Spain	1360	0	68.41
Sweden	630	0	58.72
Switzerland	760	0	24.14
Turkey	1080	0	9.90
United Kingdom	460	0	62.56
United States	650	0	63.15
Uruguay	220	0	1.39
Average	1128	0.0035	48.16
	Asian Countries		
China	1430	0.027	0.32
India	5220	0	7.30
Indonesia	3040	0	4.01
Japan	960	0	1.25
South Korea	1380	0	0.81
Philippines	1880	0.002	5.14
Singapore	1290	0.79	0.47
Taiwan	1070	0.799	0.03
Hong Kong	238	4.448	1.41
Average	2759	0.03	3.62
Asia Average (ex. China and Japan)	4637	0.04	6.37

Notes: Estimated mortality rates of 1918 Influenza come from the recalculation and compilation by Barro et al. (2020) except for Hong Kong, South Korea, and Singapore (see the data appendix A for additional sources of these three locations). Mortality rates for 2002-2003 SARS come from WHO and only include the SARS deaths between November 1, 2002 and July 31, 2003. Mortality rates for Covid-19 come from the CSSE (2020). Average mortality rates are weighted by country-specific population for certain regions.

These latter figures refer to estimates of excess mortality rates from influenza (or influenza and pneumonia as was common historically) between 1918 and 1920. Excess mortality is defined either relative to normal seasonal mortality from influenza or pneumonia or from all-causes. Barro et al. (2020) argue that these two types of measures "correspond closely". Given the speed with which the influenza pandemic overcame most populations in this period and historical mis-perceptions about the transmission of the influenza, excess mortality is a reasonable and readily available summary statistic for the severity of the 1918 influenza pandemic. Crosby (1989) notes that influenza was not a reportable cause of death in many cities of the US prior to mid-1918 and the same held true in many countries even throughout the pandemic.

We added several data points for the 1918 flu pandemic from secondary sources including Singapore, Hong Kong, and Korea. These are the official deaths from influenza and pneumonia without reference to a baseline level of mortality. Deaths and confirmed cases of Covid-19 were last updated on October 1, 2020. Our data begin as early as January 21, 2020. For the 39 sample countries, the inter-quartile range of mortality in the 1918 pandemic is 620-1360 deaths per 100,000 population with a median of 780 and a mean value of 1,120. This compares to the inter-quartile range across countries (as of May 21, 2020) for reported Covid-19 deaths of 0.52 to 16.75 per 100,000 and a median of 4.88.

We supplement the country mortality data with population mortality rates from SARS in 2002-03, GDP per capita in 2018, population density in 2019, share of the population over 70 years old, some measures of cultural differences such as an index of individualism in a country, and a dummy variable for a tradition of Confucianism. Places coded as Confucianist include mainland China, Taiwan, Hong Kong, Singapore, Japan, and South Korea. We include regional fixed effects for five regions: East Asia, South Asia, Europe, the Americas, and Oceania.

3.2. *Data Collection: US Cities*

We also explore a historical data base of 46 US cities. Data are from Collins et al. (1930). The total population in these cities totaled 20.4 million or about 18% of the 1918 US population. Data cover most of the largest cities in the US. We include deaths attributed to influenza and pneumonia, as the rest of the literature has done in the face of historical problems in coding precise causes of death. In these years, the two were often confounded, although they were strongly related.

For the 1918-1920 influenza pandemic in these US cities, Collins et al. (1930) report only monthly or weekly excess mortality per 100,000 population of 1920. We use weekly data for the period 10 September 1918 to 13 November 1918, covering the first six weeks of the 1918-20 pandemic for US cities. The excess mortality rate is calculated as the difference between the actual mortality rate and the (within city) median monthly mortality rate from influenza and pneumonia in previous non-epidemic years in those cities. Given the speed with which the influenza pandemic overcame most populations in this period and the lack of understanding of the transmission, excess mortality is a reasonable and readily available summary statistic for the severity of the influenza pandemic. To make data even more comparable to our Covid-19 data, we convert the weekly excess deaths to daily observations by linear interpolation within the week. This yields daily cumulative excess influenza and pneumonia deaths by city from the first week of September 1918.

We match US cities with continuous historical data to modern city or county-level data. One issue associated with the long-run city-level comparison is that Covid-19 data are separately reported only for some cities in our sample (New York City, St. Louis, Richmond, etc.) while most data from 2020 is reported at the county level. For cities in the historical sample without separately reported Covid-19 data at the city-level, we use data from the counties where the cities are located. For example, we pair the 1918 data for Detroit with Wayne County today. Our Covid-19 mortality data run through November 16, 2020 for our sample of U.S. cities.

To calculate event time, we set a threshold level of mortality at the city level of 0.5 per 100,000 for each pandemic. Event time and observations begin as per this threshold mortality rate. This threshold was chosen since this is the lowest recorded threshold for excess deaths from influenza and pneumonia we have available in the historical city-level data in 1918-20.

4. Analysis

4.1. *Determinants of Country Level Mortality*

Our first test finds significant persistence of mortality outcomes between the 1918 influenza pandemic and the current Covid-19 pandemic across countries. In Fig. 3, we plot the average daily growth rate of total

Table 2
Covid-19 Pandemics and Mortality from 1918 Influenza and SARS, Country-Level Evidence

	(1) Average Daily Growth Rate of Total Deaths of Covid-19 60 Days since First Death	(2) Average Daily Growth Rate of Total Deaths of Covid-19 90 Days since First Death	(3) Average Daily Growth Rate of Total Deaths of Covid-19 120 Days since First Death	(4) Average Daily Growth Rate of Total Cases of Covid-19 60 Days since 100^{th} Case	(5) Average Daily Growth Rate of Total Cases of Covid-19 90 Days since 100^{th} Case	(6) Average Daily Growth Rate of Total Cases of Covid-19 120 Days since 100^{th} Case
Total Mortality Rate of 1918 Influenza	0.022**	0.014**	0.011**	0.018**	0.013**	0.010**
	(0.009)	(0.006)	(0.004)	(0.009)	(0.006)	(0.004)
Total Mortality Rate of SARS	−0.077***	−0.057***	−0.045***	−0.071**	−0.050**	−0.039**
	(0.023)	(0.018)	(0.014)	(0.029)	(0.020)	(0.015)
Population Density in 2019 (100k per km^2)	0.004***	0.003***	0.003***	0.004**	0.003**	0.002**
	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)
Log (GDP per capita in 2019)	0.004	0.0001	−0.001	0.005	0.002	0.0002
	(0.018)	(0.011)	(0.008)	(0.015)	(0.010)	(0.008)
Confucianism Tradition (0/1)	−0.310**	−0.232**	−0.186***	−0.255*	−0.181*	−0.146*
	(0.114)	(0.085)	(0.065)	(0.147)	(0.100)	(0.075)
Individualism Index (0/100)	0.001*	0.0004	0.0003	0.0005	0.0002	0.0002
	(0.0004)	(0.0003)	(0.0002)	(0.0004)	(0.0003)	(0.0002)
Percentage of Population, Age >= 70	−0.002	−0.001	−0.001	−0.002	−0.001	−0.001
	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)
Observations	39	39	39	39	39	39
R^2	0.664	0.660	0.683	0.473	0.531	0.575

Notes: Dependent variable in columns (1)-(3) is the average daily growth rate of total deaths of Covid-19 in the first 60, 90, and 120 days since first death case, respectively. Dependent variable in columns (4)-(6) is the average daily growth rate of total confirmed cases of Covid-19 in the first 60, 90, and 120 days since the 100^{th} confirmed cases, respectively. Estimation is obtained by OLS. The total mortality rate of 1918 influenza is measured as total deaths per 100 population, and the total mortality rate of SARS is measured as total deaths per 100 thousand population. All columns control for region fixed effects (the sample countries are in 5 regions: East Asia, South Asia, Europe, Americas, and Oceania). Robust standard errors are reported in parentheses.

* p < 0.1.
** p < 0.05.
*** p < 0.01.

deaths from Covid-19 in the first sixty days since each country reported their first death case against the excess mortality rates from the 1918 influenza pandemic. The scatter plot is conditional on a set of fixed effects for geographic regions. The conditional scatter plot suggests a positive and statistically significant correlation between the two pandemics (robust t-statistic = 4.14, adjusted $R^2 = 0.5$)

The positive correlation reveals that some countries performing poorly in terms of mortality in the 1918 pandemic, such as Spain and Italy, also experienced fast mortality growth in the recent Covid-19 pandemic. However, the persistence between 1918 influenza and the current Covid-19 pandemic might not be a universal phenomenon. We note that some places such as Singapore, Hong Kong, and Taiwan, fall below the regression line, suggesting these countries are performing much better than what their 1918 performance would predict.

Results from formal regression analysis are reported in Table 2. Besides country-level mortality in the 1918 pandemic, we also include these countries' mortality rates from the SARS pandemic in 2002-03 as well as other control variables. We run cross sectional regressions of the following form

$$M_i^{Covid} = \beta_1 M_i^{1918} + \beta_2 M_i^{SARS} + X_i'\gamma + \rho_r + \varepsilon_i$$

where i indexes countries, M_i^{Covid} is the average daily growth rate of deaths from Covid-19 in the first 60, 90, or 120 days since the first reported death, M_i^{1918} is the total excess mortality rate from the 1918 influenza pandemic, M_i^{SARS} is the total mortality from SARS in 2002-03, X_i includes a set of control variables, ρ_r is a set of regional fixed effects, and ε_i is an error term. We use heteroscedasticity robust standard errors in all specifications.

Our baseline results are reported in columns (1)-(3) of Table 2. We also explore the dependent variable of the average daily growth rate of confirmed cases of Covid-19 in the first 60, 90, 120 days after the 100^{th} confirmed case. These results are reported in column (4)-(6).

All of our findings suggest that, even conditional on a set of observable characteristics, including population density, 2018 GDP per capita, and cultural controls, countries performing poorly in the 1918 pandemic tended to fail to control mortality growth of Covid-19 in the first several months of the outbreak.[7] The point estimate of the coefficient on 1918 mortality for mortality growth of Covid-19 in the first 60 days is 0.022 (p-value: 0.017, 95% C.I.: 0.004 to 0.04)[8] . This implies that a one standard deviation rise in 1918 mortality is associated with a 0.52 standard deviation rise in the growth rate of deaths from Covid-19.

On the other hand, there is some evidence of learning. The negative correlation between mortality from SARS and Covid-19 mortality reveals that the countries hit harder by the more recent epidemic have been more successful in slowing down the development of Covid-19 in the first several weeks and months. The point estimate of the coefficient on 2002-03 SARS mortality is -0.077 (p-value: 0.003, 95% C.I.: -0.125 to -0.029). This implies a one standard deviation rise in 2002-03 SARS mortality is associated with a fall in the growth rate of deaths from Covid-19 of 1.63 standard deviations. This is suggestive evidence that some countries strongly learned from their more recent experience.

4.2. US Cities: Panel Data

Next, we examine the persistence of mortality outcomes in a group of large U.S. cities. We compare the early trajectories of population mortality rates in the 1918 influenza and the contemporary Covid-19 pandemic. Data are for 46 cities for which high frequency data in

[7] We also included the number of years of educational attainment for the population aged 15-64 in 2010. The coefficient on this variable is insignificant in all specifications with no changes in the reported coefficients.

[8] The coefficients and 95% confidence intervals are multiplied by 1000 for better presentation.

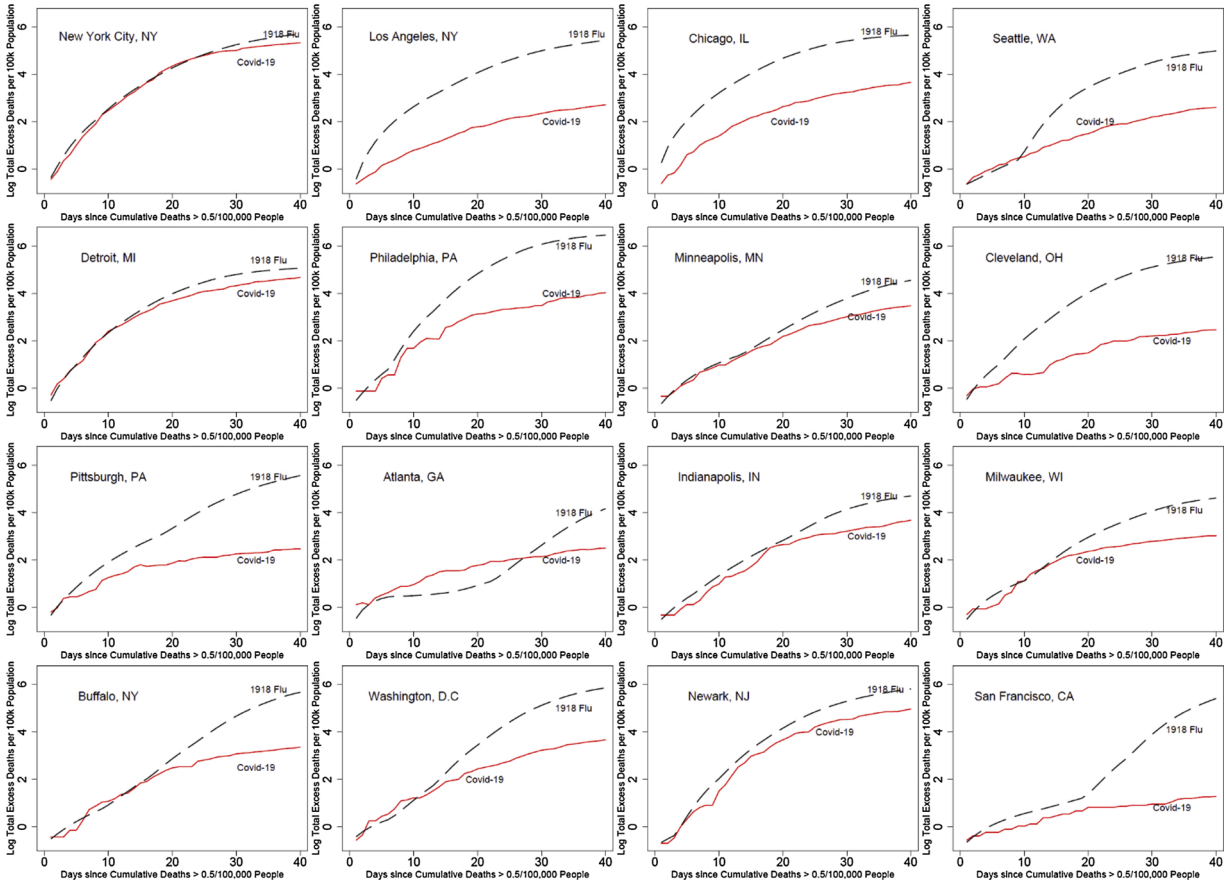

Fig. 4. Mortality Curves of Covid-19 and 1918 Influenza in Selected U.S. Cities.
Notes: These graphs plot the mortality curve of Covid-19 and the 1918 Influenza pandemic. The y-axis for each city is the log of total deaths per 100k population (for Covid-19) or total excess deaths per 100k population (for 1918 influenza); and the x-axis is the days since mortality rates reached 0.5/100,000 population. The cities shown here are the 16 cities with the largest population in 2019.

1918 are available.

In Fig. 4, we plot the trajectory of the mortality rate (excess deaths per 100,000 population) from influenza and pneumonia and Covid-19 in the days after total deaths crossed the 0.5 per 100,000 people in those cities.[9] The city-by-city comparison of historical and contemporary mortality trajectories reveals high similarity of the two epidemics in most cities.

We next conduct regression analysis with daily observations for the 46 large cities. We start with regressions of the following form

$$\ln\left(D_{c\tau}^{Covid}\right) = \pi_1 \ln\left(D_{c\tau}^{1918}\right) + \tau + \tau^2 + \kappa_c + \varepsilon_{c\tau}$$

where c indexes cities, $D_{c\tau}^{Covid}$ is the number of cumulative deaths from Covid-19, D_{ct}^{1918} is the number of cumulative deaths from influenza and pneumonia in 1918 in event time, τ indexes event time, κ_c is a set of city fixed effects and ε is an error term. To compare the mortality from Covid-19 and 1918 influenza at a similar stage, we line up the event time for both epidemics by the number of days since the death rate reached the threshold of 0.5 per 100,000 population.

For robustness we try specifications with state fixed effects to control for spatial correlation, and also including calendar day fixed effects instead of event time trends for more flexibility in the trend. As for spatial correlation, correcting the standard errors with arbitrary spatial weights makes little qualitative difference to the results. Calendar time fixed effects are highly correlated with event time which makes it hard to control for both at the same time.

Regression results are reported in Table 3 and indicate that Covid-19 deaths are positively correlated with total excess deaths in the 1918 influenza pandemic conditional on being at a similar stage in the epidemic (point estimate: 0.38, p-value: 0.000, 95% C.I.: 0.29-0.47). The results are robust to the alternative specifications and are not driven by the city of New York (Column 4 omits New York).

In addition Table A6 and A7 present two other approaches. Table A6 changes the dependent variable to the log change in mortality andincludes the lagged mortality level from Covid-19 and city fixed effects. Results are similar to our baseline models. Table A7 converts the modern data from daily to weekly data since the historical data are originally at the weekly level. We find our results are also highly qualitatively similar to those in the baseline.

We also compare the growth rate of total deaths from the 1918 flu to the growth rate of deaths from Covid-19 over the first 3, 4, or 5 weeks after total mortality reached 0.5 per 100,000 population in each pandemic. In Fig. 5, we plot the average daily growth rate of total deaths during the two pandemics in the first 4 weeks after mortality reached 0.5 per 100,000 population. The positive correlation suggests that the cities experiencing faster mortality growth in 1918 tend to experience fast growth in the initial phase of Covid-19.

4.3. U.S. Cities: Cross Section & Instrumental Variable Approach

In this section we aim to alleviate concerns about endogeneity by

[9] The threshold of 0.5/100,000 is chosen to attain comparable starting mortality rates for the two epidemics across cities. Most cities in our sample reached this threshold early in both epidemics. Our results are robust to other alternative thresholds such as 1/100,000.

Table 3
Mortality of Covid-19 and 1918 Influenza in 46 U.S. Cities, Daily Data

	(1) Log Total Deaths per 100,000, Covid-19, All Cities	(2) Log Total Deaths per 100,000, Covid-19, All Cities	(3) Log Total Deaths per 100,000, Covid-19, All Cities, Weighted	(4) Log Total Deaths per 100,000, Covid-19, Exc. NYC	(5) Log Total Deaths per 100,000, Covid-19, All Cities
ln (Total Excess Deaths per 100,000, 1918 Flu)	0.378***	0.381***	0.473***	0.371***	0.223***
	(0.066)	(0.046)	(0.075)	(0.045)	(0.066)
Event Days	0.015***	0.015***	0.014***	0.016***	
	(0.003)	(0.002)	(0.003)	(0.002)	
(Event Days)^2	−0.042***	−0.042***	−0.038***	−0.043***	
	(0.001)	(0.005)	(0.008)	(0.005)	
Observations	10212	10212	10212	9990	10202
R^2	0.796	0.924	0.939	0.923	0.942
Number of Cities	46	46	46	45	46
Calendar Date F.E.	No	No	No	No	Yes
State F.E.	Yes	No	No	No	No
City F.E.	No	Yes	Yes	Yes	Yes

Notes: Dependent variables are the logarithm of total deaths of Covid-19 per 100,000 population. Regressions are conducted with data at the daily level. Event days are the number of days since total deaths (for Covid-19) or total excess deaths (for 1918 Influenza) reached 0.5/100,000 population. To keep a balanced panel data, the event days have a maximum of 222 days. The data were last updated on Nov.16, 2020. The full list of cities can be found in data appendix A and are the same as those from Collins et al. (1930). All regressions are unweighted, except that in column (3), where the regression is weighed by 2019 population as a robustness check. Coefficients and standard errors for square of event days are multiplied by 1,000 for better display. Standard errors are clustered at the city level (clustered at the state level for first column) and reported in the parentheses. * $p < 0.1$, ** $p < 0.05$.
*** $p < 0.01$.

using an instrumental variable approach. Our excluded instrument, explained below, is not time varying, so we employ a cross-sectional model which collapses the panel data from the previous section.

Regressions relating the average growth rate of deaths in Covid-19 to the average growth rate of deaths from the 1918 pandemic are reported in Table 4. Regressions take the form

$$\widehat{D}_c^{Covid} = \theta \widehat{D}_c^{1918} + X_c^{'}\phi + \nu_c$$

where $\widehat{D}_c^{Covid}$ denotes the average daily growth rate of deaths from Covid-19 for city c, $\widehat{D}_c^{1918}$ is the average growth rate of deaths from influenza and pneumonia in 1918, X is a set of control variables, and ν is an error term. With X we condition on historical and contemporary population density and age distribution.

A positive and statistically significant correlation between the growth rates in the first 3, 4, and 5 weeks after mortality rates reached the given threshold is evident. For the first 4 weeks, the estimated coefficient of mortality growth in the 1918 pandemic is 0.30 (p-value: 0.00, 95% C.I.: 0.08 to 0.52). This implies a one standard deviation rise in mortality growth of the 1918 influenza is associated with a 0.33 standard deviation rise in the mortality growth from Covid-19 in the first 4 weeks.

The strong persistence of mortality outcomes between the 1918 pandemic and Covid-19 in U.S. major cities could be explained by some long-run persistent, but unobservable characteristics of the cities driving growth rates of mortality in both pandemics. To understand whether the identified persistence might be driven by such factors or whether historical mortality plays a more direct role in influencing health outcomes today, we employ an instrumental variable strategy.

Our excluded instrument for mortality in the 1918 flu during the early phase leverages information on the distance between cities and 39 historical U.S. Army training camps. Historical evidence suggests that military personnel were among those who were affected early and contributed significantly to disease spread in nearby cities (Crosby, 1989; Barry, 2004; Byerly, 2010). Crosby (1989) argues strongly that the

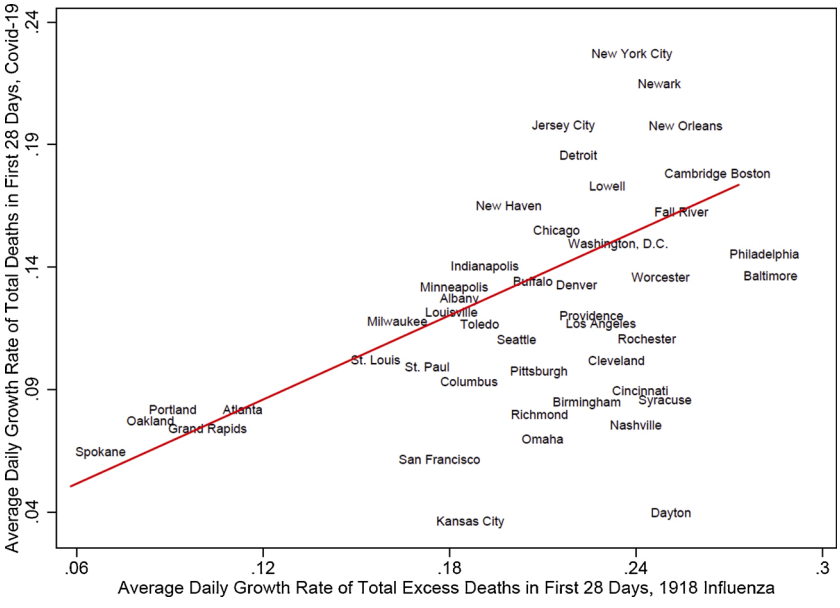

Fig. 5. Average Daily Growth of Total Deaths from Covid-19 and 1918 Influenza in U.S. Cities: First 28 Days.
Notes: This figure shows the unconditional correlation between the average daily growth rate of total deaths of Covid-19 and the average growth rate of total excess deaths of the 1918-20 Influenza pandemic in the first 28 days since the total deaths (for Covid-19) or total excess deaths (for 1918 Influenza) reached 0.5 for every 100,000 population. The coefficient of the regression (which includes a constant) is 0.355 with a robust t-statistic of 4.09 and a 95% C.I. of 0.179 to 0.531. The average daily growth rates of total deaths (or total excess deaths for 1918 influenza) in the first 28 days are calculated by $\sqrt[27]{cmdeath_{it}/cmdeath_{it-27}} - 1$.

Table 4
Growth of Total Deaths of Covid-19 and 1918 Influenza in U.S. Cities: OLS Estimates

	(1) Average Daily Growth Rate of Total Deaths, Covid-19 First 28 Days	(2) Average Daily Growth Rate of Total Deaths, Covid-19 First 28 Days	(3) Average Daily Growth Rate of Total Deaths, Covid-19 First 28 Days	(4) Average Daily Growth Rate of Total Deaths, Covid-19 First 21 Days	(5) Average Daily Growth Rate of Total Deaths, Covid-19 First 35 Days
Average Daily Growth Rate of Total Excess Deaths, 1918 Influenza, First *N* Days	0.573***	0.281***	0.301***	0.246***	0.366**
	(0.121)	(0.0992)	(0.105)	(0.0798)	(0.150)
Population Density, 2019		0.096	0.056	0.119	0.020
		(0.077)	(0.075)	(0.119)	(0.0509)
Population Density, 1920		0.410***	0.434***	0.476**	0.379***
		(0.0893)	(0.132)	(0.200)	(0.0995)
Percentage of Population Age 65 and Over, 2019			−0.004	−0.004	−0.004
			(0.003)	(0.004)	(0.003)
Percentage of Population Age 65 and Over, 1920			−0.003	−0.006	−0.002
			(0.005)	(0.006)	(0.004)
# Cities	46	46	46	46	46
R^2	0.247	0.681	0.698	0.693	0.671

Notes: The average daily growth rates of total deaths for first *n* days are calculated by $\sqrt[n-1]{cmdeath_{it}/cmdeath_{in-1}} - 1$. The first *n* days refers to the days since total deaths (for Covid-19) and total excess deaths (for 1918 Influenza) reached 0.5 for every 100,000 people. All regressions are weighted by population in 2019. Standard errors are clustered at the state level and reported in the parentheses. * $p < 0.1$.
** $p < 0.05$.
*** $p < 0.01$.

most deadly wave began on the east coast amongst military personnel in August and September 1918. He then makes the case that movement of infected personnel, which occurred for strategic military reasons, between east coast Naval ports and military bases to other camps around the nation helps explain the timing of accelerations of civilian influenza. Cities closer to military camps would have been at higher risk due to the higher possibility of interacting with infected military personnel.

The military cantonments we use in our data are located across the US and were for training recruits for World War I in the early years of World War I. Their precise location was likely determined by strategic concerns rather than being associated with the drivers of public health in a pandemic. It is unlikely that the camp locations exercise much influence on the spread of Covid-19 since only 8 of the 39 camps are in operation currently.

Clay et al. (2018) uses a measure of proximity to the nearest military training camps as a determinant of mortality from in the 1918 pandemic. Hilt and Rahn. (2020) use average distance to military camps as an excluded instrumental variable to predict excess mortality in October 1918 while Correia, Luck, and Verner (2020) use distance weighted by the number of personnel at the bases as an excluded instrument to predict mortality from influenza in late 1918.

We construct our own index of exposure to influenza in military camps. We leverage not only distance to the camps but also the intensity of infection in the camps in August and September – largely before the onset in the general population which occurred from mid-September onwards.

For each city, exposure is defined as $\sum_j \frac{\ln(admission_j)}{\ln(distance_{cj})}$, where $admission_j$ is the number of (camp) hospital admissions due to influenza and pneumonia in military camp *j* in the two months of August and September 1918. The variable $distance_{cj}$ is the geodesic distance between city *c* and camp. For each city, we include the five closest camps in this calculation. To control for potential direct effects of proximity to military camps, we also include the log of distance between each city and the nearest camp in all 2SLS regressions. The data on location and flu-related admissions for the major army camps come from the 1919 *Annual Report of the War Department*.

Fig. 6 shows a simple correlation between the growth rate of mortality from influenza in 1918 and exposure to influenza in nearby military camps. The positive correlation is consistent with our hypothesis that cities more exposed to the influenza outbreaks in nearby camps had faster mortality growth in the early phase of the 1918 pandemic. This could be explained by the fact that the intensity tended to be higher in the South and East where the pandemic started and where local public health systems and populations were caught off-guard. We next re-estimate the relationship between mortality growth of 1918 influenza and contemporary Covid-19, with two-stage least squares (2SLS).

Two stage least squares estimates are reported in Table 5. The first stage results show large F-statistics. A weak instrumental variable is not a concern. The coefficients on mortality growth in the 1918 flu remain statistically significant and positive. These findings suggest that the identified positive correlation between the early performance in these two pandemics is not solely driven by potential unobservable pre-existing city characteristics. These results suggest that there is a plausible link between contemporary and historical drivers of mortality in pandemics. We discuss some potential explanations for this result below.

We also explored the impact of mortality in 1918 and another influenza pandemic (that of 1968) in Table A4 and A5. While daily mortality in 1918 seems to be related to daily mortality in 1968 in US cities (Table A4), this result is not robust to the instrumental variables specification in Table A5. One explanation for the lack of robust correlation may be the significantly smaller scale of the 1968 influenza pandemic. We could not readily locate city-level data for 1957-58, but this pandemic was also not comparable to Covid-19 nor the Influenza of 1918 (Doshi, 2008).

5. Discussion

What mechanisms link the past to today in this context? We are left with several possibilities all of which deserve more research. First, localities and countries may have bad luck. It is possible that public health inaction in the past and today was due to a lack of leadership or was hamstrung by politics. This seems unlikely because multiple cities and countries would have to be systematically unlucky in multiple periods.[10]

Second, there is the possibility the influenza of 1918 affected some unobservable variable which is now affecting mortality from Covid-19. Such a variable could be overall trust of others in society and/or trust in the government. Aassve et al. (2020) argue greater mortality in the 1918-20 pandemic generated lower trust of others in the long run.[11] This

[10] Abad and Maurer (2020) show that the 1918 pandemic does not seem to have had a major impact on the national elections of 1918.

[11] The measure of trust is based on the General Social Survey question: "Generally speaking, would you say that most people can be trusted or that you can't be too careful in dealing with people?"

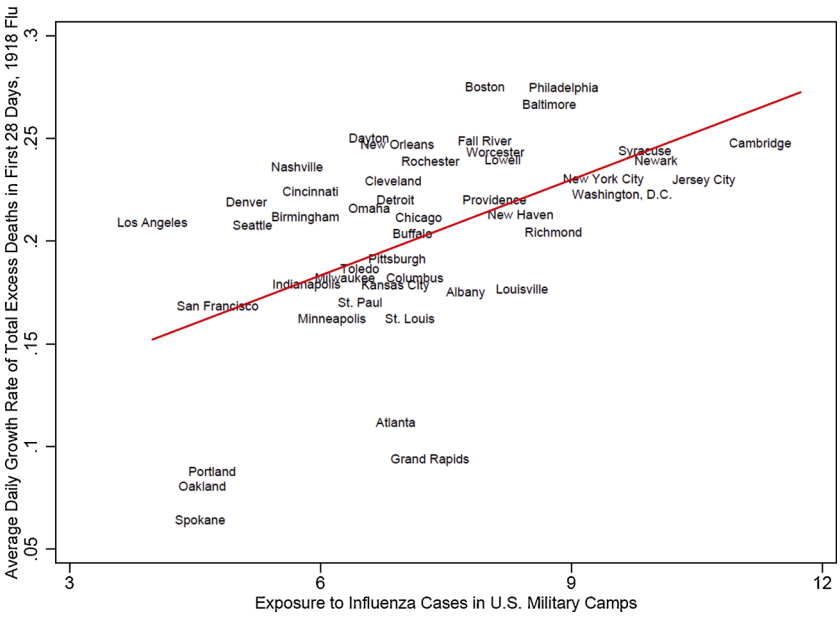

Fig. 6. Mortality Growth of 1918 Influenza and Exposure to U. S. Army Camps.
Note: The data for influenza and pneumonia admissions by major military camps are from the Report of the Surgeon General, which is Volume 1 of the War Department Annual Report of 1919. We have 39 major army camps and the distances between cities and camps are determined by the distance between cities and the counties in which these camps are located. The definition of the exposure to influenza cases in U. S. Military camps is given in the text.

Table 5
Growth of Total Deaths of Covid-19 and 1918 Influenza in U.S. Cities: 2SLS Estimates

	(1) Average Daily Growth Rate of Total Deaths, Covid-19 First 28 Days	(2) Average Daily Growth Rate of Total Deaths, Covid-19 First 28 Days	(3) Average Daily Growth Rate of Total Deaths, Covid-19 First 28 Days	(4) Average Daily Growth Rate of Total Deaths, Covid-19 First 21 Days	(5) Average Daily Growth Rate of Total Deaths, Covid-19 First 35 Days
Average Daily Growth Rate of Total Excess Deaths, 1918 Flu, First *N* Days	0.909***	0.621***	0.630***	0.531***	1.032***
	(0.199)	(0.191)	(0.177)	(0.151)	(0.349)
Population Density, 2019		0.050	0.013	0.066	−0.009
		(0.041)	(0.042)	(0.083)	(0.035)
Population Density, 1920		0.226*	0.262*	0.196	0.204
		(0.137)	(0.137)	(0.213)	(0.125)
Percentage of Population Age 65 and Over, 2019			−0.004	−0.00263	−0.004
			(0.004)	(0.00452)	(0.003)
Percentage of Population Age 65 and Over, 1920			−0.004	−0.003	−0.004
			(0.004)	(0.008)	(0.003)
	First Stage: Dependent Var is Average Daily Growth Rate of Total Excess Deaths, 1918 Flu				
Exposure to Influenza in Military Camps	0.020***	0.017***	0.017***	0.025***	0.009***
	(0.004)	(0.003)	(0.003)	(0.007)	(0.002)
K-P F-Statistics for IV	29.082	27.220	25.370	12.119	22.269
# Cities	46	46	46	46	46

Notes: The instrumental variable for the daily growth rates of 1918 influenza is an estimated exposure to flu-related admissions in military camps in August and September in 1918. The average daily growth rates of total deaths for first n days are calculated by $\sqrt[n-1]{cmdeath_{it}/cmdeath_{in-1}} - 1$. The first n days refers to the days since total deaths (for Covid-19) and total excess deaths (for 1918 Influenza) reached 0.5 for every 100,000 people. All regressions also control for the log of distance between each city to the nearest military camp. Standard errors are clustered at the state level and reported in the parentheses. ** $p < 0.05$.
* $p < 0.1$.
*** $p < 0.01$.

lack of trust could be vertically transmitted to the next generation as well. This could help explain some of the persistence in mortality we see in the data both across countries and within the US. Parmet and Rothstein (2018) highlight three factors that determine public health responses to a pandemic: "distrust, isolationism and hubris". The lack of trust engendered by the level of exposure to the influenza pandemic of 1918 could explain why our instrumental variables results show that mortality in 1918 matters today. More detailed data by city of residence and trust attitudes would be helpful in this regard, but high quality, representative data like these are not readily available to the best of our knowledge.

A third possibility relates to the fetal origins hypothesis. The fetal origins hypothesis argues that in utero exposure an infectious disease like influenza is associated with worse SES outcomes later in life. Almond (2006) finds evidence that exposure to influenza in 1918 in utero was associated with worse SES later in life. Taking the next step, a growing literature emphasizes the multi-generation effects of health shocks. If morbidity of offspring is higher due to health shocks, then it is likely that the second generation could also suffer from the shocks from the previous generation. Richter and Robling (2013) discuss the possibility of a direct biological effect on the germ cells of the exposed fetus as well as "indirect" effects to the children of those exposed in utero via their mothers who are more likely to have lower SES outcomes. These multi-generational effects have been explored in a nascent literature.[12]

A sufficient driver of a multi-generational effect would be significant spatial persistence in population over multiple generations in the

[12] For a recent review see East and Page (2019). For evidence on multigenerational effects of famines and health shocks see Painter et al. (2008), Kaati et al. (2007), Roseboom et al. (2011), East et al. (2019) and Richter and Robling (2013).

context of population level findings. An individual level assessment of this hypothesis deserves further scrutiny. In the end, multiple channels are likely to be driving the effect found here. The persistence of pandemics and health shocks highlights the urgency and importance of public health and other efforts to prepare for the next pandemic and to allow better mechanisms to cope with health shocks.

Finally, experience with SARS is associated with lower mortality today. Meanwhile, where mortality of the 1918 influenza was high, mortality is likely to be higher today. Why? A potential explanation lies in evidence from the literature on natural disasters. Fanta et al. (2019) provide evidence that in the aftermath of major floods societies tend to build new settlements away from the danger zone "for a period of one generation". However, from the second generation onwards, settlements tend to be built closer to the flood zone. It is quite plausible that outcomes were better where SARS was more prevalent for similar reasons.

In conclusion, it is unlikely that one and only one mechanism explains the bottom line result that past experience with pandemics matters for contemporary pandemics. Two things are worth emphasizing at this point. First, our results are robust to a number of alternative specifications, control variables, and a plausible instrumental variables model. Second, more research into the exact mechanisms with the assistance of new data at a greater level of detail is of importance in order to understand the historical drivers of modern pandemics.

Declaration of Competing Interest

The authors of "Persistent Pandemics", Christopher M. Meissner and Peter Z. Lin have no competing interests to declare.

Appendix A

Tables A1–A3

Table A1
Covid-19 Pandemics and Mortality from 1918 Influenza and SARS, Country-Level Evidence with Spatial HAC Standard Errors

	(1) Average Daily Growth Rate of Total Deaths of Covid-19	(2) Average Daily Growth Rate of Total Deaths of Covid-19	(3) Average Daily Growth Rate of Total Deaths of Covid-19	(4) Average Daily Growth Rate of Total Cases of Covid-19	(5) Average Daily Growth Rate of Total Cases of Covid-19	(6) Average Daily Growth Rate of Total Cases of Covid-19
	60 Days since First Death	90 Days since First Death	120 Days since First Death	60 Days since 100th Case	90 Days since 100th Case	120 Days since 100th Case
Total Mortality Rate of 1918 Influenza	0.0219***	0.0144***	0.0113***	0.0184**	0.0133***	0.00999***
	(0.007)	(0.005)	(0.004)	(0.007)	(0.005)	(0.003)
Total Mortality Rate of SARS	−0.0768***	−0.0568***	−0.0448***	−0.0708**	−0.0504***	−0.0393***
	(0.018)	(0.014)	(0.011)	(0.023)	(0.017)	(0.013)
Observations	39	39	39	39	39	39
R^2	0.664	0.660	0.683	0.473	0.531	0.575

Notes: This table presents the same estimation as Table 2, however with HAC (heteroskedasticity and autocorrelation consistent) standard errors allowing spatial correlation of error terms. We adopt the approach introduced by Kelly (2020), which relies on the Matern function (Gneiting and Gutthorp, 2010) as the kernel function characterizing spatial correlation. The estimated coefficients remained unchanged from Table 2, while most standard errors changed. Allowing error terms to be spatially correlated does not alter our results significantly. All regressions also include the control variables reported in Table 2 as well as the region fixed effects. Spatial HAC standard errors are reported in parentheses. * $p < 0.1$.
** $p < 0.05$.
*** $p < 0.01$.

Table A2
Mortality of Covid-19 and 1918 Influenza in 46 U.S. Cities, Daily Data with Spatial HAC Standard Errors

	(1) Log Total Deaths per 100,000, Covid-19, All Cities	(2) Log Total Deaths per 100,000, Covid-19, All Cities	(3) Log Total Deaths per 100,000, Covid-19, All Cities, Weighted	(4) Log Total Deaths per 100,000, Covid-19, Exc. NYC	(5) Log Total Deaths per 100,000, Covid-19, All Cities
ln (Total Excess Deaths per 100,000, 1918 Flu)	0.378***	0.381***	0.473***	0.371***	0.223***
	(0.050)	(0.053)	(0.068)	(0.051)	(0.071)
Event Days	0.015***	0.015***	0.014***	0.016***	
	(0.003)	(0.002)	(0.003)	(0.002)	
(Event Days)^2	−0.042***	−0.042***	−0.038***	−0.043***	
	(0.009)	(0.007)	(0.008)	(0.007)	
Observations	10212	10212	10212	9990	10202
R^2	0.796	0.924	0.939	0.923	0.942
Number of Cities	46	46	46	45	46
Calendar Date F.E.	No	No	No	No	Yes
State F.E.	Yes	No	No	No	No
City F.E.	No	Yes	Yes	Yes	Yes

Notes: This table presents the same estimation as Table 3, but with HAC (heteroskedasticity and autocorrelation consistent) standard errors allowing spatial correlation of error terms. We adopt the approach introduced by Kelly (2020), which relies on the Matern function (Gneiting and Gutthorp, 2010) as the kernel function characterizing spatial correlation. The estimated coefficients remained unchanged from Table 3, while standard errors changed. Allowing error terms to be spatially correlated does not alter our results significantly. Spatial HAC standard errors are reported in parentheses. * $p < 0.1$,** $p < 0.05$.
*** $p < 0.01$.

Table A3
Growth of Total Deaths of Covid-19 and 1918 Influenza in U.S. Cities: Spatial HAC Standard Errors

	(1) Average Daily Growth Rate of Total Deaths, Covid-19 First 28 Days	(2) Average Daily Growth Rate of Total Deaths, Covid-19 First 28 Days	(3) Average Daily Growth Rate of Total Deaths, Covid-19 First 28 Days	(4) Average Daily Growth Rate of Total Deaths, Covid-19 First 21 Days	(5) Average Daily Growth Rate of Total Deaths, Covid-19 First 35 Days
	Ordinary Least Square Estimates				
Average Daily Growth Rate of Total Excess Deaths, 1918 Influenza, First *N* Days	0.573***	0.281***	0.301***	0.246***	0.366**
	(0.149)	(0.074)	(0.083)	(0.066)	(0.118)
	Two-Stage Least Square Estimates				
Average Daily Growth Rate of Total Excess Deaths, 1918 Influenza, First *N* Days	0.909***	0.621*	0.630*	0.531**	1.032
	(0.318)	(0.353)	(0.341)	(0.252)	(0.684)
# Cities	46	46	46	46	46

Notes: This table presents the estimation of the specifications of Table 4 (for OLS estimates) and Table 5 (for 2SLS estimates), but with HAC (heteroskedasticity and autocorrelation consistent) standard errors allowing spatial correlation of error terms. We adopt the approach introduced by Kelly (2020), which relies on the Matern function (Gneiting and Gutthorp, 2010) as the kernel function characterizing spatial correlation. The estimated coefficients remained unchanged, while most standard errors changed. Allowing error terms to be spatially correlated does not alter our results significantly. All regressions also include control variables reported in Table 4 and 5. Spatial HAC standard errors are reported in parentheses.
* $p < 0.1$.
** $p < 0.05$.
*** $p < 0.01$.

Table A4
Mortality of 1968 H3N2 Influenza and 1918 Influenza in 46 U.S. Cities, Daily Data

	(1) Log Total Excess Deaths per 100,000, 1968 Flu, All Cities	(2) Log Total Excess Deaths per 100,000, 1968 Flu, All Cities	(3) Log Total Excess Deaths per 100,000, 1968 Flu, All Cities, Weighted	(4) Log Total Excess Deaths per 100,000, 1968 Flu, Exc. NYC	(5) Log Total Excess Deaths per 100,000, 1968 Flu, All Cities
ln (Total Excess Deaths per 100,000, 1918 Flu)	0.230***	0.271***	0.296***	0.268***	0.0648
	(0.051)	(0.031)	(0.055)	(0.031)	(0.062)
Event Days	0.012***	0.008***	0.006**	0.008***	
	(0.004)	(0.002)	(0.003)	(0.002)	
$(\text{Event Days})^2$	−0.0000414***	−0.0000302***	−0.0000216**	−0.0000311***	
	(0.0000118)	(0.00000801)	(0.00000912)	(0.00000822)	
Observations	9366	9366	9366	9144	9366
R^2	0.517	0.790	0.806	0.790	0.850
Number of Cities	44	44	44	43	44
Calendar Date F.E.	No	No	No	No	Yes
State F.E.	Yes	No	No	No	No
City F.E.	No	Yes	Yes	Yes	Yes

Notes: Dependent variables are the logarithm of total excess deaths of 1968 H3N2 Influenza per 100,000 population. Data come from the 122 Cities Mortality Reporting System by CDC, and the data report weekly deaths caused by pneumonia or influenza in U.S. cities from 1962 to 2016. The calculation of total excess death rates is detailed in data appendix A. Regressions are conducted at the daily level. Event days are the number of days since total excess deaths (for either 1968 or 1918 Influenza) reached 0.5/100,000 population. The mortality rate of 1968 influenza is not available for Oakland (CA) and Louisville (KY); leaving 44 cities in the baseline regressions. All regressions are unweighted, except that in column (3), the regression is weighed by 2019 population for robustness check. Coefficients and standard errors for the square of event days are multiplied by 1,000. Standard errors are clustered at the city level (clustered at the state level for first column) and reported in the parentheses. * $p < 0.1$.
** $p < 0.05$.
*** $p < 0.01$.

Data Appendix

Cross-Country Data, 1918

Mortality rates: Barro et al. (2020). This data set is uses excess mortality from influenza and pneumonia and total excess mortality depending on data availability and country. The primary data sources they rely on include Johnson and Mueller (2002), Murray et al. (2006), and the Human Mortality Database (www.mortality.org).

For countries not in Barro et al. (2020): Singapore from Lee et al. (2007); Korea from Hong and Yun (2017); Hong Kong from Cheng and Leung (2007). Hong Kong population in 1919 calculated from Swee--Hock and Chiu wing (1975); Singapore, population Dodge (1980)

Covid-19 Data, 2020

Data for cases and deaths by country and US cities for Covid-19 come from Covid-19 Data Repository by the Center for Systems Science and Engineering (CSSE) at Johns Hopkins University.

https://github.com/CSSEGISandData/COVID-19, downloaded on October 4, 2020.

SARS Mortality, 2002-2003

World Health Organization: https://www.who.int/csr/sars/country/2003_07_04/en/

Accessed on October 4, 2020.

Regional Fixed Effects

The 39 countries in our cross-country sample are categorized into 5 major regions:

East Asia: Mainland China, Hong Kong, Taiwan, Japan, and South Korea.

South Asia: India, Indonesia, Philippines, and Singapore.

Europe: Austria, Belgium, Denmark, Finland, France, Germany,

Table A5
Growth of Total Deaths of 1968 H3N2 Influenza and 1918 Influenza in U.S. Cities, Two Stage Least Squares.

	(1) Average Daily Growth Rate of Total Excess Deaths, 1968 Flu First 28 Days	(2) Average Daily Growth Rate of Total Excess Deaths, 1968 Flu First 28 Days	(3) Average Daily Growth Rate of Total Excess Deaths, 1968 Flu First 28 Days	(4) Average Daily Growth Rate of Total Excess Deaths, 1968 Flu First 21 Days	(5) Average Daily Growth Rate of Total Excess Deaths, 1968 Flu First 35 Days
	Ordinary Least Square Estimates				
Average Daily Growth Rate of Total Excess Deaths, 1918 Influenza, First *N* Days	−0.0962	−0.181	−0.212	−0.101	−0.392
	(0.152)	(0.167)	(0.167)	(0.0955)	(0.301)
	Two-Stage Least Square Estimates				
Average Daily Growth Rate of Total Excess Deaths, 1918 Influenza, First *N* Days	0.370	0.319	0.277	0.296	0.460
	(0.235)	(0.208)	(0.262)	(0.302)	(0.429)
# Cities	43	43	43	44	43

Notes: This table presents the same specifications of Table 4 (for OLS estimates) and Table 5 (for 2SLS estimates), however with the average daily growth rate of total excess deaths of 1968 H3N2 Influenza as a dependent variable. All regressions control for population density in 1920 and 1968. We also control for the percentage of population aged 65 and over in 1920 and 1968 in all specifications. The 2SLS estimates are subject to potential weak instrument issue as the first stage Kleibergen-Paap F-statistics were all below 7. We do not include the city of Buffalo in the regressions with growth rate of excess deaths in first 28 and 35 days because the excess deaths fell below the median death rates before the pandemic reached 28 days. We also do not include the city of Oakland (CA) and Louisville (KY) because mortality data for the 1968 influenza is unavailable. The standard errors are clustered at state level and reported in parentheses. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A6
Mortality of Covid-19 and 1918 Influenza in 46 U.S. Cities, Daily Changes

	(1) Change of Log Total Deaths per 100,000, Covid-19, All Cities	(2) Change of Log Total Deaths per 100,000, Covid-19, All Cities	(3) Change of Log Total Deaths per 100,000, Covid-19, All Cities, Weighted	(4) Change of Log Total Deaths per 100,000, Covid-19, Exc. NYC	(5) Change of Log Total Deaths per 100,000, Covid-19, All Cities
Change of Log Total Excess Deaths per 100,000, 1918 Flu	0.217***	0.088**	0.129***	0.084**	0.065**
	(0.041)	(0.035)	(0.035)	(0.035)	(0.032)
Lag of Log Total Excess Deaths per 100,000, 1918 Flu	−0.014***	−0.039***	−0.043***	−0.039***	−0.022***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)
Event Days	−0.0006***	0.0001	0.0001***	0.00005	
	(0.0001)	(0.0001)	(0.00004)	(0.00006)	
$(\text{Event Days})^2$	0.002***	0.0003	−0.00002	0.0003	
	(0.0004)	(0.0002)	(0.0001)	(0.0002)	
Observations	10166	10166	10166	9945	10156
R^2	0.367	0.410	0.576	0.397	0.474
Number of Cities	46	46	46	45	46
Calendar Date F.E.	No	No	No	No	Yes
State F.E.	Yes	No	No	No	No
City F.E.	No	Yes	Yes	Yes	Yes

Notes: Dependent variables are the daily changes of the logarithm of total deaths of Covid-19 per 100,000 population. Regressions are conducted with data at the weekly level. Event days are the number of days since total deaths (for Covid-19) or total excess deaths (for 1918 Influenza) reached 0.5/100,000 population. The data is lastly updated on Nov.16, 2020. The full list of cities can be found in data appendix A and are the same as those from Collins et al. (1930). All regressions are unweighted, except that in column (3), where the regression is weighed by 2019 population. Coefficients and standard errors for the square of event days are multiplied by 1,000. Standard errors are clustered at the city level (clustered at the state level for first column) and reported in the parentheses. * $p < 0.1$.
** $p < 0.05$.
*** $p < 0.0$.

Greece, Hungary, Italy, Ireland, Netherlands, Norway, Portugal, Russia, Spain, Sweden, Switzerland, Turkey, and United Kingdom.

The Americas: Argentina, Brazil, Canada, Colombia, Chile, Mexico, Peru, United States, and Uruguay.

Oceania: Australia and New Zealand.

Density, GDP per capita, and other Control Variables

The population density and age distribution of sample countries come from the World Population Prospects 2019, by the United Nations. Details can be found: https://population.un.org/wpp/Download/Standard/CSV/

GDP per capita in 2018 comes from the World Bank and is in constant 2010 U.S. dollars. Details can be found:
https://data.worldbank.org/indicator/NY.GDP.PCAP.KD

Countries coded to have a Confucian tradition include Mainland China, Hong Kong, Taiwan, Japan, South Korea, and Singapore.

The individualism index comes from the 6-dimensional model of national culture by Geert Hofstede. The index is based on the Values Survey in 2013 (Geert Hofstede and Michael Minkov, 2013). Details of the 6-dimensional model can be found at
https://geerthofstede.com/culture-geert-hofstede-gert-jan-hofstede/6d-model-of-national-culture/

Cities in the US Sample

The 46 cities and states in parentheses in our analysis include: Albany (NY), Atlanta (GA), Baltimore (MD), Birmingham (AL), Boston (MA), Buffalo (NY), Cambridge (MA), Chicago (IL), Cincinnati (OH), Cleveland (OH), Columbus (OH), Dayton (OH), Denver (CO), Detroit

Table A7
Mortality of Covid-19 and 1918 Influenza in 46 U.S. Cities, Weekly Data

	(1) Log Total Deaths per 100,000, Covid-19, All Cities	(2) Log Total Deaths per 100,000, Covid-19, All Cities	(3) Log Total Deaths per 100,000, Covid-19, All Cities, Weighted	(4) Log Total Deaths per 100,000, Covid-19, Exc. NYC	(5) Log Total Deaths per 100,000, Covid-19, All Cities
ln (Total Excess Deaths per 100,000, 1918 Flu)	0.318***	0.318***	0.408***	0.309***	0.187***
	(0.070)	(0.048)	(0.078)	(0.048)	(0.063)
Event Weeks	0.120***	0.120***	0.109***	0.123***	
	(0.023)	(0.012)	(0.020)	(0.012)	
(Event Weeks)^2	−0.002***	−0.002***	−0.002***	−0.002***	
	(0.001)	(0.0002)	(0.0004)	(0.0002)	
Observations	1426	1426	1426	1395	1424
R^2	0.778	0.927	0.941	0.925	0.940
Number of Cities	46	46	46	45	46
Calendar Date F.E.	No	No	No	No	Yes
State F.E.	Yes	No	No	No	No
City F.E.	No	Yes	Yes	Yes	Yes

Notes: Dependent variable is the logarithm of total deaths of Covid-19 per 100,000 population. Regressions are conducted with data at the weekly level. Event days are the number of days since total deaths (for Covid-19) or total excess deaths (for 1918 Influenza) reached 0.5/100,000 population. To keep a balanced panel dataset, the event weeks are at the maximum of 31 weeks. The data is last updated on Nov.16, 2020. The full list of cities can be found in data appendix A and are the same as those from Collins et al. (1930). All regressions are unweighted, except that in column (3), where the regression is weighed by 2019 population. Coefficients and standard errors for the square of event days are multiplied by 1,000. Standard errors are clustered at the city level (clustered at the state level for first column) and reported in the parentheses. * $p < 0.1$, ** $p < 0.05$.
*** $p < 0.0$.

(MI), Fall River (MA), Grand Rapids (MI), Indianapolis (IN), Jersey City (NJ), Kansas City (MO), Los Angeles (CA), Louisville (KY), Lowell (MA), Milwaukee (WI), Minneapolis (MN), Nashville (TN), New Haven (CT), New Orleans (LA), New York City (NY), Newark (NJ), Oakland (CA), Omaha (NE), Philadelphia (PA), Pittsburgh (PA), Portland (OR), Providence (RI), Richmond (VA), Rochester (NY), Saint Louis (MO), Saint Paul (MN), San Francisco (CA), Seattle (WA), Spokane (WA), Syracuse (NY), Toledo (OH), Washington D.C., Worcester (MA).

We use data from 46 cities although according to Collins et al. (1930), there are 50 cities with documented excess monthly death rates from influenza and pneumonia between 1910 and 1929. However, only 47 of the 50 cities have available data of excess weekly death rates, which are the primary sources we use to calculate the city-level epidemic curves. The three cities with missing weekly data are Bridgeport (Conn.), Paterson (NJ), and Scranton (PA). In addition, we drop Memphis (Tenn.) from our sample because its weekly data are not available until the week of Oct. 12, 1918, when it had already seen a significant surge in excess deaths, and we are not able to characterize the early phase of the epidemic.

13. 1918-1919 Influenza Pandemic in U.S. Cities

The mortality data in 46 major U.S. cities come from the public health reports (Collins et al., 1930).[13] We interpolate the weekly excess deaths to daily basis by linear interpolation. Then, we calculate the cumulative excess deaths from the week of September 14, 1918, when continuous weekly deaths became available in Collins et al. (1930). For cities with negative excess deaths in the mid-September, we start counting total deaths when excess deaths became continuously positive.

14. 1968-1969 H3N2 Influenza Pandemic in U.S. Cities

The mortality data of 1968-1969 H3N2 Influenza in U.S. cities come from the 122 Cities Mortality Reporting System, which is provided by CDC and downloaded from HeathData.gov. (https://healthdata.gov/dataset/Deaths-in-122-U-S-cities-1962-2016-122-Cities-Mort/m36n-nf4p)

The reporting system reports pneumonia or influenza related deaths each week from 1962 to 2016. We have 44 cities (out of the 46 cities with available 1918 Influenza mortality data) in this reporting system.[14] To calculate the excess mortality rate, we firstly calculate median mortality rate for a specific week based on the weekly deaths between 1962 and 1980. Second, we calculate the excess mortality rate by subtracting the median mortality rate from actual mortality rate. Then, we calculate the cumulative excess deaths from the first week of December, 1968 when the Influenza pandemic start wide spreading in the United States.

[13] The original reports by Collins et al. (1930) includes 47 cities. We exclude the city of Memphis from our analysis as its weekly mortality data are not available until the week of October 12, 1918.

[14] The city of Oakland (CA) and Louisville (KY) are not included in this reporting system.

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