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Pandemics and cities: Evidence from the Black Death and the long-run[☆]

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ABSTRACT

The Black Death killed 40% of Europe's population between 1347 and 1352, making it one of the largest shocks in the history of mankind. Using a novel dataset that provides information on spatial variation in plague mortality at the city level, as well as various identification strategies, we explore the short-run *and* long-run impacts of Black Death mortality on city growth. On average, cities recovered their pre-plague populations within two centuries. However, aggregate convergence masked heterogeneity in urban recovery. Both of these facts are consistent with populations returning disproportionally to locations endowed with more rural and urban fixed factors of production. Land suitability and natural and historical trade networks played a vital role in recovery. Our study highlights the role played by the Black Death and physical and economic geography in determining the relative size of European cities.

We study the urban effects of the Black Death, the largest demographic shock in modern history. It killed around 40% of Europe's population between 1347–1352. Some regions and cities were spared, others were devastated—England, France, Italy and Spain lost 50%–60% of their populations. While the Black Death has been widely studied, little is known about its effects on city growth. Furthermore, it is important to study how population shocks affect cities as agglomeration effects play a crucial role in explaining the distribution of urban population and income.

The city-level effects of demographic shocks are theoretically ambiguous. (i) Local increasing returns could result in a large negative shock generating a negative feedback cycle in which hard hit cities continue to decline. Alternatively, (ii) If urban incomes rely on fixed factors of production such as land or natural resources, i.e. locational fundamentals, or if there are sunk investments in housing and infrastructure, net wages may rise due to labor scarcity, enabling population recovery through either migration or net fertility.

The implications of these models for long-run city growth differ: relative *decline* (i) or *recovery* (ii). The importance of increasing returns, locational fundamentals, sunk investments, and other factors may then vary across contexts. For example, locational fundamentals may play a disproportionately large role in less developed economies, thus increasing the likelihood of *recovery*.

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Using data for 165 cities, comprising 60% of the total urban population of Western Europe, we find that between 1300 and 1400 a 10 percentage point higher mortality rate was associated with an 8.7 percentage point fall in city population. After two centuries the impact of mortality was *zero*, in part due to migration from low-mortality areas. Examining the spill-over and general equilibrium effects of the Black Death, we find similarly negative effects in the short-run and nil effects in the long-run for both rural and urban areas.

While rural recovery was to be expected, given how important (fixed) land is for agriculture, large cities recovered as well, consistent with scenario (ii). Thus, in economies where locational fundamentals and sunk investments play an important role, mortality shocks may not strongly affect the relative distribution of city sizes. Consistent with this, we find that urban recovery is strongly explained by the interacted effects of mortality with city characteristics that proxy for *fixed factors of production*: rural fixed factors related to better land suitability, but also urban fixed factors related to natural advantages (e.g., coastal access and waterways) or historically sunk investments (e.g., roads and trade networks).

Aggregate recovery, however, hides permutations in the distribution of cities. Some cities collapsed after the shock whereas other cities gained in the long run. We provide evidence that these permutations were associated with the presence of fixed factors.[1] Also, many prominent present-day cities might have fallen into oblivion absent fixed factors. Lastly, since permutations favored cities with better land and trade potential, urban systems may have become more productive.

These results are plausibly causal. For example, they hold when implementing two instrumental variables strategies premised on the facts that: (A) the Black Death entered Europe through the Sicilian port of Messina (largely by chance) and was more virulent in its earlier stages (for pathogenic reasons). It was trade with Messina and not trade in general that mattered for plague virulence; and (B) the Black Death was more lethal in cities in which it reached its peak in the summer since the fleas that transmitted the disease were more active then.

We contribute to research on shocks and long-run persistence. Several studies have shown how path dependent spatial economic patterns are. Different causes have been advanced for this path dependence, including locational fundamentals (i.e. natural advantages), sunk investments (man-made advantages), agglomeration effects (the direct effect of scale), or local institutions (e.g., Bleakley and Lin, 2012; Combes, Duranton, Gobillon, Puga, and Roux, 2012; Hanlon, 2017; Henderson, Squires, Storeygard, and Weil, 2017; Cermeno and Enflo, 2019; Desmet, Greif, and Parente, 2020; Barsanetti, 2021).

Unlike other shocks considered in the literature, the Black Death was large and a comparatively "pure" population shock. Buildings and equipment were not destroyed, the event itself did not directly target a particular demographic group, and there was no government intervention. This makes our setting well suited to test for the path dependent effects of mortality shocks (see Bleakley and Lin, 2015; Hanlon and Heblich, 2022; Jedwab, Johnson, and Koyama, 2022a for recent surveys).[2]

We complement (Davis and Weinstein, 2002, 2008) in four ways. First, we study a larger shock. Mean plague mortality was 40% and all cities were impacted. In contrast, 20% and 8.5% of the populations of Hiroshima and Nagasaki were killed, respectively. Second, no buildings were destroyed and government assistance was non-existent in our context. Third, Davis and Weinstein explain that cities with strong defense capabilities, of historical significance, or with a specific topography, were spared by the bombings whereas plague virulence was apparently exogenous. Fourth, they do not interact the bombings with the characteristics of the cities to identify which locational fundamentals and sunk investments mattered for recovery.[3]

We also contribute to the literature on the economics of pandemics (see Baum-Snow, Glaeser, and Rosenthal, 2022 for a survey on COVID-19 and cities). Existing work on the Black Death focuses on its macro-level impacts (Voigtländer and Voth, 2013a,b) or on specific European regions (Bosshart and Dittmar, 2021). By contrast, our city-level data allow us to study local effects, spillovers, migration, and heterogeneous recovery. Finally, we provide suggestive evidence that the Black Death generated an urban reset by spurring population growth in areas better endowed with factors facilitating trade (as in Michaels and Rauch, 2018).

1. Data

Mortality. Data on cumulative Black Death mortality for the period 1347–1352 come from Christakos et al. (2005, 117–122). Christakos et al. (2005) compile mortality rates based on information from a wide array of historical sources including ecclesiastical and parish records, testaments, tax records, court rolls, chroniclers' reports, donations to the church, financial transactions, mortality of famous people, letters, edicts, guild records, hospital records, cemeteries and tombstones. Christakos et al. (2005) examine each data point and arbitrate between conflicting estimates based on the best available information. We have checked these data using other sources including Ziegler (1969), Russell (1972), Gottfried (1983), and Benedictow (2005) (see Web Appx. Section 1. for details). These data yield mortality estimates for 274 localities in 16 countries.

For 177 of these we have a percentage estimate. In other cases, the sources report more qualitative estimates: (i) For 49 cities, Christakos et al. (2005) provide a literary description of mortality. We rank these descriptions based on the implied magnitude of the shock and assign each one of them a numeric rate.[4] (ii) For 19 cities we know clergy mortality. Christakos et al. (2005) show that clergy mortality was 8% higher than general mortality, so we divide the clergy mortality rates by 1.08.[5] (iii) For 29 cities we know the desertion rate, which includes non-returnees. Following Christakos et al. (2005, 154–155), who show that desertion rates were 1.2 times higher than mortality rates, we divide desertion rates by 1.2. Lastly, for a significant number of cities (Christakos et al., 2005) report the years and the months the Black Death started and ended in each city.

Cities. Bairoch (1988) reports population estimates for 1726 cities between 800 and 1850. Observations are provided for every century up to 1700 and then for each fifty year interval. The criterion for inclusion is a city population greater than 1000 inhabitants. We update Bairoch

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[1] Henderson et al. (2017) shows how agriculture- and trade-related geographic variables explain the distribution of economic activity globally. Due to high transport costs, cities had to be closer to agriculturally suitable areas. Instead of studying how the influence of these factors has changed over time, we study their importance after a shock. Other studies of the role of geography include Bosker et al. (2013), Bosker and Buringh (2017) and Flückiger and Ludwig (2020).

[2] Wars, as studied by Davis and Weinstein (2002, 2008), Bosker et al. (2008), Glocker and Sturm (2014) and Redding and Sturm (2016) led to massive physical destruction and resulted in government reconstruction programs. Natural disasters as studied by Boustan et al. (2012), Boustan et al. (2020) and Hornbeck and Keniston (2017) kill few people and lead to physical destruction. Malaria, HIV, the 1918 influenza or COVID-19, as studied by Bleakley (2010), Young (2005), Beach et al. (2018) and Desmet and Wacziarg (2022) kill subgroups of the population.

[3] Our study is also related to studies of the effects of market access (e.g., Bosker et al., 2007; Ahlfeldt et al., 2015; Brülhart et al., 2020; Alix-Garcia and Sellars, 2020; Cuberes et al., 2021). We find that the mortality rate in a city affected not just that city but also the city's "neighbors".

[4] 5% for "spared"/"escaped", 10% for "partially spared"/"minimal", 20% for "low", 25% for "moderate", 50% for "high", 66% for "highly depopulated", and 80% for "decimated".

[5] Clergymen were the only exception to our statement that specific populations were not targeted. Clergymen, however, only comprised a few individuals so this should not matter overall.

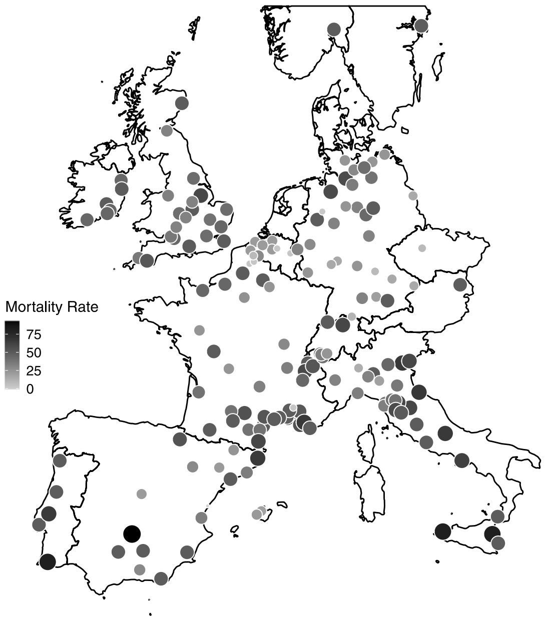

Fig. 1. Black Death mortality rates (%) in 1347–1352. *Notes*: This map plots the location of all 165 existing cities (i.e. loc. ≥ 1000 inh.) in 1300 for which we know their Black Death mortality rate (%) in 1347-52 and the modern boundaries of the 16 Western European countries of our analysis.

where scholars – Nicholas (1997), Campbell (2008), Bosker et al. (2013) and Voigtländer and Voth (2013b) – have revised population estimates. We add 76 cities mentioned in Christakos et al. (2005).[6] In the end, we obtain 1801 cities (see Web Appx. Section 2. for details).[7]

Sample. Our sample consists of 165 cities recorded in our dataset in 1300 and for which we know the plague mortality rate. They comprise 60% of the urban population of Western Europe in 1300. We map these along with their mortality rates in Fig. 1.

Controls for *locational fundamentals* include growing season temperature, elevation, soil suitability for cereal production, potato cultivation and pastoral farming, dummies for whether the city is within 10 km of a coast or river, and longitude and latitude. To proxy for *increasing returns*, we control for population and market access in 1300. We calculate market access for every city in our main sample to the cities of the full sample for which we have populations in 1300. Market access for town i is defined as $MA_i = \Sigma_j (L_j) \div (\tau_{ij}^{\sigma})$, with L_j being the population of town $j \neq i$, τ_{ij} the travel time between town i and town j, and $\sigma = 3.8$ (Donaldson, 2018). We compute the least cost travel paths via four transportation modes – sea, river, road and walking – using the plague diffusion data from Boerner and Severgnini (2014). To proxy for *sunk investments*, we control for the presence of major and minor Roman roads (and their intersections) (McCormick et al., 2013), medieval trade routes (and their intersections) (Shepherd, 1923), and dummies capturing the presence of market fairs, membership in the Hanseatic league (Dollinger, 1970), whether a city possessed a university (Bosker, Buringh, and van Zanden, 2013), and whether a city was within 10 km of an aqueduct (Talbert, 2000). To control for *institutions*, we distinguish between cities located in monarchies, self-governing cities, or whether the city was a state capital c. 1300 (Bosker et al., 2013; Stasavage, 2014). We also include measures of parliamentary activity during the 14th century (Zanden, Buringh, and Bosker, 2012) and control for whether a city was within 100 km of a battle between 1300-50. See Web Appx. Section 3. for details and Web Appx. Table A.1 for summary statistics.

2. The shock

The Black Death arrived in Europe in October 1347 after merchant ships carrying the plague from Kaffa in Crimea stopped in Messina in Sicily (Fig. 1). Over the next three years it spread across Europe killing 40% of the population (we obtain a mortality rate of 38.9% for the 274 localities in our sample). In this section we document that there was a plausible random component to mortality.

Epidemiology. Recent discoveries in plague pits have corroborated the hypothesis that the Black Death was Bubonic plague (Benedictow, 2005, 2010). The bacterium *Yersinia Pestis* was transmitted by the fleas of the black rat. Infected fleas suffer from a blocked esophagus. These "blocked" fleas are unable to state themselves and continue to bite rats or humans, regurgitating the bacterium into the bite wound. Within less than a week, the bacteria is transmitted from the bite to the lymph nodes causing them to become buboes. Once infected, death occurred within ten days with 70% probability.

Fleas cannot spread the disease far in the absence of hosts. A rat (or other small mammal) carrying infected fleas could board a ship or wagon and hide in the barrels, bags, or straw it transported. Likewise, the body or clothes of a person walking or on horseback could carry infected fleas. It is important to note that rats travel at low speeds and tend not to stray far from their home territories. Yet, dispersal occurs over long distances (10 km) if resources are scarce or for mate-searching (Byers et al., 2019). Thus, a rat may plausibly infect other rats 10 km away, and in turn that population cluster could infect other rats 10 km away, etc. Once a host carrying infected fleas arrives in an uninfected community, other potential hosts coming in close contact to the infected host (whether alive or dead) become infected as they themselves get bitten by infected fleas. The disease then spreads among the rat and human populations. As such, factors such as population density and trade may have been important determinants of the speed with which the disease spread, but not necessarily its mortality rate.

An important epidemiological fact about the plague that we exploit is that the virulence was far greater in cities affected *earlier* (Christakos et al., 2005, 212–213). Initially, epidemics spread exponentially. One possible explanation for this is that as more people have been infected and survive or die and the pool of susceptible hosts in the aggregate population decreases, the disease might mutate in favor of benign pathogens that facilitate transmission, but at the expense of mortality.[8] Pathogen mutation also increases individual immune responses due to "contacted individuals becoming infected only if they are exposed to strains that are significantly different from other strains in their memory repertoire" (Girvan et al., 2002). Pathogen mutation and natural immunization may eventually cause an epidemic to end.

Early exposure can explain the terrible mortality Sicilian cities experienced (two thirds on average). Other coastal cities such as Barcelona, Bristol, Edinburgh and Rostock experienced much lower

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[6] The 76 added cities are comparatively small, typically below a few thousand individuals. As such, it is likely that Bairoch (1988) captures almost all significant cities circa 1300.

[7] We supplement these historical data with present-day populations. Given that cities have grown dramatically since 1850, absorbing other settlements and becoming multi-city agglomerations, we read the webpage of each city in Wikipedia and selected the 2015 population of the city itself rather than the population of the agglomeration. We nonetheless verify that results hold if we use the agglomeration estimate or the mean of the two estimates (not shown).

[8] According to Berngruber et al. (2013): "[. . .] selection for pathogen virulence and horizontal transmission is highest at the onset [. . .] but decreases thereafter, as the epidemic depletes the pool of susceptible hosts [. . .] In the early stage of an epidemic susceptible hosts are abundant and virulent pathogens that invest more into horizontal transmission should win the competition. Later on, [a smaller pool of susceptible hosts favors] [. . .] benign pathogens [. . .]".

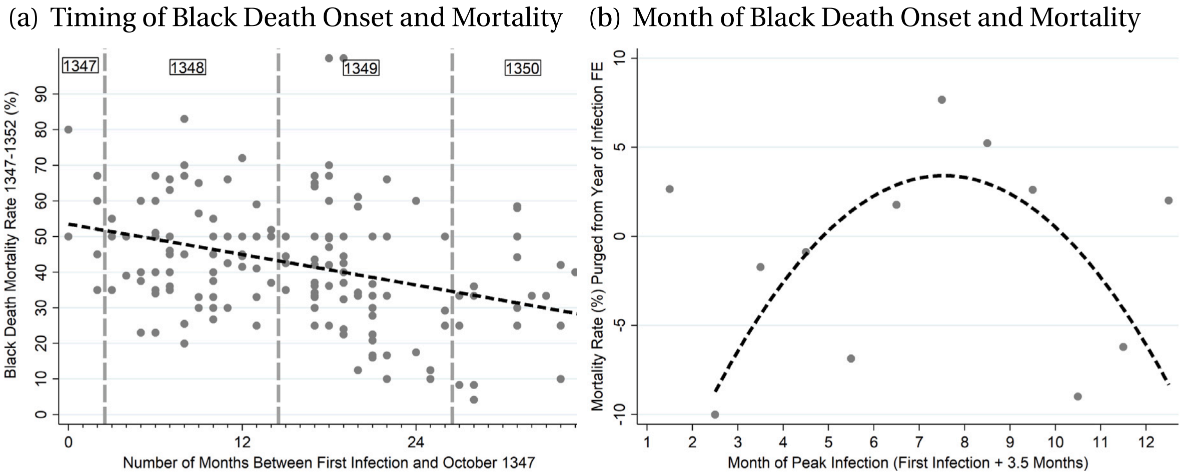
(a) Timing of Black Death Onset and Mortality
(b) Month of Black Death Onset and Mortality

Fig. 2. Timing of the onset of the Black Death and Black Death mortality. *Notes*: Fig. 2(a) shows for 124 cities the relationship between mortality and the timing of the onset of the Black Death in the city. *Number of months* is measured since Oct. 1347, the date Messina – the port of entry of the Black Death in Europe – was infected. Fig. 2(b) shows for 124 cities and for each month of peak infection (month of first infection +3.5) the average mortality rate purged of year of infection fixed effects. The quadratic fit shows that mortality was the highest when peak mortality was in the summer and the lowest in the winter. The quadratic fit omits January ("1"), which has high mortality rates due to pathogenic reasons and october being the month of onset of the Black Death in Europe.

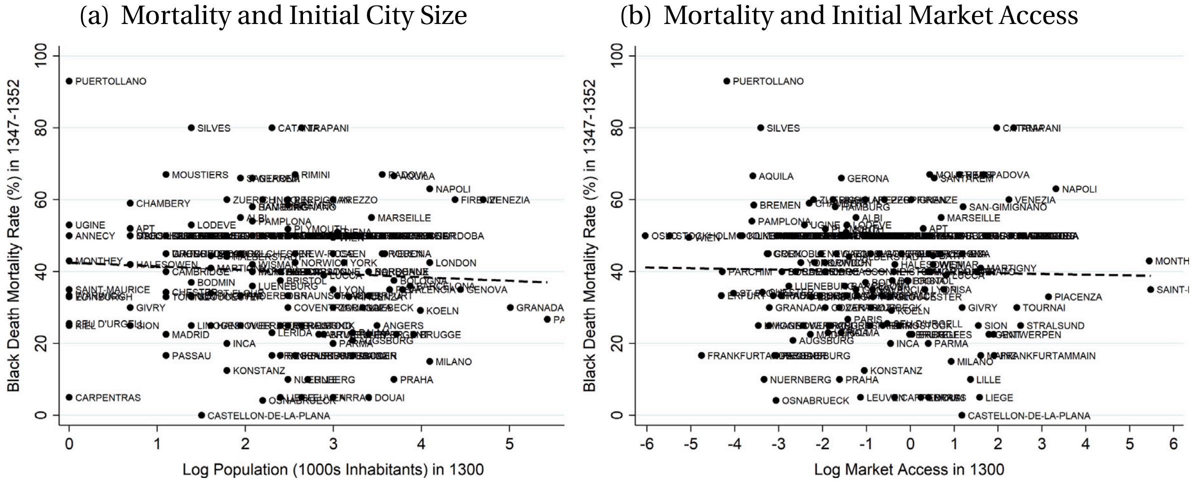
(a) Mortality and Initial City Size
(b) Mortality and Initial Market Access

Fig. 3. Mortality rates, city size and city market access in 1300. *Notes:* Fig. 3(a) shows the relationship between mortality (%, 1347–1352) and log city pop. in 1300 for our main sample of 165 cities (Y = 42.5*** −1.01 X; R^2 = 0.00). Fig. 3(b) shows for the same 165 cities the relationship between mortality (%, 1347–1352) and log market access to all 1801 cities in 1300 (Y = 40.0*** −0.20 X; R^2 = 0.00). Market access for city i is defined as $MA_i = \Sigma_j (P_j / D_{ij})^{\sigma}$, with P_j being the pop. of town $j \neq i$, D_{ij} the travel time between city i and city j, and $\sigma = 3.8$. To obtain the travel times, we compute the least cost travel paths via four transportation modes – by sea, by river, by road and by walk – with the transportation speeds from Boerner and Severgnini (2014).

mortality rates. Likewise, this also helps explain why average mortality decreased over time (see Fig. 2(a)) and why the disease eventually disappeared. If we compare the mortality rates of cities infected 1 month after the initial arrival of the plague in Messina to cities infected after 6, 12, 24 and 36 months, the average mortality difference is 9, 13, 22 and 39 percentage points. Thus, a difference of a few months in the arrival date of the plague in a city had dramatic effects on the city's cumulative mortality rate.

What determined why some cities were infected earlier than others? The literature on epidemic diseases has emphasized the importance of trade, both for the spread of plague (see Boerner and Severgnini, 2014) and other diseases (see Geloso et al., 2022). As we document below, what was relevant in the Black Death outbreak was less overall trade connectivity, but rather a city's trade connectivity to the city of Messina, the point of origin of the Black Death in Europe.

Why Messina? The disease first arrived in Messina in late 1347, which at the time was only the 45th largest city in Europe. While the exact origins of the Black Death are still the subject of scholarly debate (Green, 2020), we do know that Astrakhan, a trade center located on the Volga river near the Caspian Sea, was infected in 1345. Kaffa, a Genoese colony in Crimea, was then infected in 1346. It was from there that the Genoese galleys with infected rats and humans on their voyage home stopped in Messina in October 1347. Two months later, ships left for Genoa.

Messina did not have to be the point of entry for the plague. Genoa had other colonies in the Black Sea (Deletant, 1984) including Vinica along the banks of the Danube which led all the way to Vienna, a port of entry of plague recurrences in later centuries (Web Appx. Fig. A.1 maps the cities and routes mentioned in this paragraph). It also had colonies along the Dniester River, at the end of which was Halych, a town located on the East–West trade route that led to Leipzig via Prague. Thus, in 1346, the plague could have infected these other Genoese colonies and then traveled to Vienna or Leipzig. Moreover, Astrakhan was an important trading center connected via river to Moscow and Novgorod, which both had river access to the Gulf of Finland. Novgorod traded with Visby (Sweden), one of the centers of the Hanseatic League, a trade network between Northern European cities. Thus, Messina, Genoa, Vienna, Prague, Leipzig and Visby all could have been the port of entry for the plague and trade networks in Central or Northern Europe could have been infected before the Mediterranean basin. Indeed, when we compute the travel times between Astrakhan and each of the counterfactual ports of entry, we find that it would have taken 3 months for the disease to reach any of them *had* it spread in their direction resulting in one of these other cities being infected as early as 1346. Yet, it happened that the disease went a different direction towards Genoa, making a stop in Messina.

After Messina. When the disease arrived in Messina, it was extremely virulent and the cities closer to Messina that were infected first also had high mortality rates. Trade connectedness to Messina thus disproportionately determined the mortality rate of a city. Paris, London, Cologne and Lisboa were among the largest trading cities of Europe but were infected much later than smaller cities closer to Messina and, consequently, experienced relatively lower mortality. Even among cities directly connected to Messina, some were infected earlier than others due to chance. Within the Mediterranean basin, Barcelona, Naples, Rome, and Valencia were infected months after smaller cities such as Aix, Arles, Beziers and Tarragona. In the rest of Europe, smaller hinterland cities such as Grenoble, Lyon, Rouen, and Verona were infected before important coastal cities such as Bordeaux, Bruges, Plymouth, or Lübeck. Web Appx. Section 4. and Web Appx. Figure A.3 document visually how there was a plausibly random component to the spread of the Black Death in the first year of the pandemic.

What mattered was which city, due to chance, received an infected host early. Infected rats and fleas were not choosing merchant ships or wagons depending on the economic importance of their final destination. Likewise, among human travelers, some going to smaller cities were already infected and some going to larger cities were not. Plague diffusion also depended on the local populations of black rats. Since they are territorial, i.e. a territory is chosen because enough rats have randomly made similar locational decisions, their numbers were not correlated with population density (Benedictow, 2005). For example, similar death rates are recorded in urban and in rural areas (Herlihy, 1965).[9]

Bubonic plague was most virulent during the summer (Benedictow, 2005, 233–235). Fleas become most active when it is warm and humid (Gottfried, 1983, 9). Christakos et al. (2005, 230) notes that mortality displayed seasonal patterns with deaths diminishing with colder weather "without the epidemic coming to a complete halt". Using available data on the year and month of first and last infection for 61 cities, the average duration of the Black Death was 7 months (see Web Appx. Fig. A.2). According to Christakos et al. (2005, 212–213), mortality on average peaked 3.5 months after the first infection. Therefore, one source of random variation in plague virulence depends on the fact that cities infected in late fall escaped relatively unscathed compared to cities infected in spring.

Plague virulence had a significant random component which depended on a city's proximity to Messina, whether infected humans and rats visited the city early by mischance, the size of its rat population, and whether the disease arrived in spring (see Web Appx. Section 5. for qualitative evidence). When studying variation in mortality rates across space, historical accounts have been unable to rationalize the patterns in the data (Ziegler, 1969; Gottfried, 1983; Theilmann and Cate, 2007; Cohn and Alfani, 2007). Venice had high mortality (60%) while Milan escaped comparatively unscathed (c. 15%). Paris' mortality rate was 20 points lower than London's. Highly urbanized Sicily suffered heavily. Equally urbanized Flanders had low death rates. Southern Europe and the Mediterranean were hit hard, but so were the British Isles and Scandinavia. Christakos et al. (2005, 150) explain that some scholars have "argued that Black Death hit harder the ports and large cities along trade routes" but that "the generalization is logically valid at a regional level at best" and that "examples and counterexamples abound".

Fig. 3(a) illustrates the lack of a relationship between mortality (1347-52) and city population in 1300 (Y = 42.5*** −1.01 X; Obs. = 165; $R^2 = 0.00$) in our sample of 165 cities. For the 88 cities with data on walled area we also find no relationship with population density (Web Appx. Fig. A.4). Likewise, Fig. 3(b) indicates no relationship between mortality and log market access in 1300 (Y = 40.0*** −0.20 X; Obs. = 124; $R^2 = 0.00$). Note that random measurement error in dependent variables (mortality) does not lead to bias. However, random measurement error in market access produces a downward bias, and non-classical measurement error is possible with historical data. Web Appx. Table A.3 finds no correlation between mortality and market access when: (i) using lower trade elasticities in the market access calculation (2 or 1, instead of 3.8);[10] (ii) using alternative measures of transport costs or Euclidean distance, which has the advantage of not having to rely on travel speeds related to plague diffusion itself[11]; (iii) including each city in its own calculation of its market access;[12] (iv) including other (mostly Eastern) European cities not in our main sample of 16 countries; and (v) including cities of the Middle East and North Africa.[13]

Subsequent plague outbreaks took place for two and a half centuries following the Black Death (Alfani and Murphy, 2017). Epidemiologists and historians have long noted the virulence, spread, and associated mortality of the Black Death differed from the pattern associated with later outbreaks of bubonic plague (Web Appendix Section 5.). These plague recurrences were caused either by local plague reservoirs or the reintroduction of the bacteria from Asia (Schmid et al., 2015). Though on occasion later outbreaks could devastate a city, in general mortality was significantly lower than in the initial pandemic (Aberth, 2010).[14]

3. The Black Death shock and city recovery

3.1. Baseline results

To estimate the short- and long-run effects of Black Death mortality on city growth we estimate a series of city-level regressions based on:

$$\%\Delta \mathrm{Pop}_{i,t} = \alpha + \beta_t \mathrm{Mort}_{i,1347-52} + \epsilon_{i,t} \tag{1}$$

where $\%\Delta \mathrm{Pop}_{i,t}$ is the percentage population growth (%) in city i over period t-1 to t, and $\mathrm{Mort}_{i,1347-52}$ is the city-level cumulative mortality rate (%; 1347-52). We weight observations by their population size in year $t-1$ to minimize issues arising from smaller cities mechanically experiencing larger percentage changes.[15]

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[9] Unlike today's brown rats that prefer to live in urban areas, black rats were as likely to be found in rural areas as in urban areas.

[10] We use a high sigma because trade costs were high and trade was limited then (relative to today), much like in 19th century India for which sigma = 3.8 was estimated (Donaldson, 2018).

[11] Boerner and Severgnini (2014) find that traveling by sea was 1.4 and 2.9 times faster than traveling by river or road. We then assume that walking on a path was twice slower than traveling by road (and thus 5.7 slower than traveling by sea). They also cite other estimates from Pryor (1992) and McCormick (2001) that lead to a different combination: (3.8; 3.8; 7.7).

[12] To avoid a zero trade cost, we use the travel cost between Paris and Saint-Denis, two localities 7 km away from each other (Saint-Denis is now part of Paris). Paris' radius was smaller then. However, to account for likely intracity congestion, we do not adjust down the travel cost.

[13] We use data from Bosker et al. (2013) which includes cities with more than 10,000 people.

[14] Only the plague of 1629-30 in Italy came close to the Black Death's virulence (Alfani, 2020).

[15] Growth for a city of 1000 in $t-1$ and 5000 in t is 400%. Large cities rarely experience such growth rates. While this is a standard issue when using percentage growth outcomes, we choose this as our main specification because the interpretation of the coefficient is straightforward.

Table 1
Black Death mortality rates and city growth, 1100–1750.

Dep. Var.	*Percentage change in city population (%) in Period t*							
t:	1300–1400 (1)	1300–1500 (2)	1300–1600 (3)	1300–1700 (4)	1300–1750 (5)	1300–1850 (6)	1300–2015 (7)	1200–1300 (8)
β	−0.87***	−0.28	0.36	0.47	0.85	0.99	−0.53	0.16
	[0.28]	[0.38]	[0.80]	[1.00]	[1.17]	[2.24]	[14.26]	[0.59]
CI	[−1.4, −0.3]	[−1.0, 0.5]	[−1.2 , 1.9]	[−1.5, 2.4]	[−1.5, 3.2]	[−3.4, 5.4]	[−28.7, 27.6]	[−1.0, 1.3]
Beta Coef.	−0.35	−0.07	0.05	0.03	0.04	0.02	0.00	0.04
Mean	−9.4	−2.8	29.7	69.9	95.7	275.6	1791.2	44.3
Z-Score	−0.019***	−0.002	0.002	0.001	0.001	0.001	−0.000	0.002
	[0.006]	[0.002]	[0.004]	[0.002]	[0.002]	[0.001]	[0.001]	[0.006]
CI	[−0.03,−0.01]	[−0.01,0.00]	[−0.01,0.01]	[−0.00,0.00]	[−0.00,0.00]	[−0.00,0.00]	[−0.00,0.00]	[−0.01,0.01]
Obs	165	164	164	164	164	165	165	93
R^2	0.12	0.01	0.00	0.00	0.00	0.00	0.00	0.00

Notes: The main sample consists of 165 cities (i.e. loc. ≥ 1000 inh.) in the Bairoch dataset for 1300 and for which mortality is available. We use city pop. in the initial year of period *t* as regression weights. See Web Appendix for data sources. Robust SE's.

Table 2
City characteristics and Black Death mortality rates.

Dependent variable:	Black Death mortality rate (%, 1347–1352)							
	(1)		(2)		(3)		(4)	
Average Temperature 1500–1600 (d)	0.16	[0.66]					0.55	[0.95]
Elevation (m)	0.00	[0.01]					0.00	[0.01]
Cereal Suitability Index	1.08	[1.60]					2.11	[1.77]
Potato Suitability Index	0.16	[1.90]					−1.05	[2.03]
Pastoral Suitability Index	0.58	[4.25]					1.30	[4.55]
Coast 10 Km Dummy	4.64	[3.19]					4.08	[3.86]
Rivers 10 Km Dummy	−5.29**	[2.63]					−4.81	[3.25]
Longitude (d)	−0.12	[0.21]					0.09	[0.32]
Latitude (d)	−0.88**	[0.42]					−0.61	[0.55]
Log City Population in 1300			−0.56	[1.34]			−2.02	[1.90]
Log Market Access in 1300			−0.49	[0.71]			−0.34	[0.82]
Maj.Roman Rd (MRR) 10 Km Dummy			−3.35	[7.57]			−1.99	[6.04]
MRR Intersection 10 Km Dummy			3.86	[4.15]			5.56	[4.09]
Any Roman Rd (ARR) 10 Km Dummy			7.55	[8.08]			4.73	[6.65]
ARR Intersection 10 Km Dummy			−1.99	[4.59]			−1.44	[4.50]
Medieval Route (MR) 10 Km Dummy			0.80	[3.12]			2.40	[3.07]
MR Intersection 10 Km Dummy			−5.52	[4.82]			−6.25	[4.99]
Market and Fair Dummy			−5.10	[3.55]			−2.89	[4.06]
Hanseatic League Dummy			0.46	[4.77]			4.44	[5.97]
Aqueduct 10 Km Dummy			2.72	[3.77]			−0.10	[3.86]
University Dummy			6.56	[4.26]			5.82	[4.52]
Monarchy in 1300 Dummy					4.02	[4.43]	2.60	[4.58]
State Capital in 1300 Dummy					3.73	[4.40]	1.49	[4.78]
Representative Body in 1300 Dummy					−4.08	[3.50]	0.34	[3.88]
Parliamentary Activity in 1300–1400					0.50	[3.99]	−0.11	[4.13]
Log Distance to Parliament in 1300					0.59	[0.48]	0.06	[0.45]
Battle w/i 100 Km in 1300–1350 Dummy					−3.80	[2.80]	−2.49	[2.95]
Obs.; R^2	165; 0.16		165; 0.08		165; 0.07		165; 0.23	

Notes: This table shows the effects of city characteristics on mortality (%, 1347–52). Robust SE's: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Col. (1) of Table 1 measures the short-run impact in 1300–1400. The coefficient, −0.87***, should be interpreted relative to the immediate effect in 1347-52 (−1.00 by construction). The fact that the coefficient is not significantly different from −1.00 suggests little recovery in the decades directly following the onset of the Black Death. The effect is large as shown by its beta coefficient: a one standard deviation increase in mortality is associated with a 0.35 standard deviation decrease in population growth. The effect in 1300–1500 is still negative (−0.28, col. (2)) but smaller in size (beta coefficient of −0.07).

Col. (3)–(7) show the cumulative effect up to 2015. The coefficient increases over time, but this is simply a reflection of the fact that the dependent variable grows over time whereas the variable of interest – the mortality rate – always remains the same. When we standardize the coefficients using beta coefficients or z-scores – both reported in the table –, they are small in the long run.[16]

3.2. Identification

Parallel Trends. Col. (8) shows that prior to 1300, there is no difference in growth between cities most affected and those comparatively unaffected by the plague.

Correlates of Mortality. Table 2 shows that mortality rates were uncorrelated with various city characteristics capturing physical geography (1), access to markets and trade (2) or institutions (3). The

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[16] We use 1600, 1700, 1750, 1800 and 1850 because these are the years available in the Bairoch dataset. We also test if any traces of the impact of Black Death lasted to the present day (2015).

only variables that have explanatory power are proximity to rivers and latitude. However, the sign on proximity to rivers is negative, not positive. Other measures of transportation and trade networks do not predict mortality. The coefficient on latitude reflects the fact that the Black Death hit southern Europe first and was more virulent in the early years of the epidemic. Finally, no effect is significant once all controls are included.[17]

Row 2 of Table 5 shows the baseline results hold when we include all the controls of Table 2 simultaneously. The effect in 1300–1400 is now less negative. Indeed, we will show in Section 6 that city characteristics affected the recovery of higher-mortality cities in 1353–1400 and beyond. Over-controlling then leads us to under-estimate the negative short-run effects. Lastly, note that the 1300–1600 effect is small once standardized (Z-score of −0.002 vs. -0.013*** for 1300–1400).

Spatial Fixed Effects. In row 3 we include fixed effects corresponding to modern country borders. As modern country borders differ from the political units of the fourteenth century, in row 4 we assign a separate dummy variable to each of the independent polities with at least 5 cities in our dataset (Web Appx. Fig. A.5 shows state boundaries).[18] Alternatively, we use fixed effects corresponding to seven 5 × 5 degree cells (row 5). The results that we obtain are qualitatively similar.

Next, we employ two instrumental variable strategies: IV-A and IV-B.[19] Since the IV strategies rely on the spatial diffusion of the plague, we cluster standard errors at the state (1300) level (N = 64) for these analyses.

IV-A: Proximity to Messina We begin by creating a variable for date of first infection for each city in our dataset (available for 124 cities). Fig. 2(a) plots mortality rates against the *date* that the city was first infected (number of months since October 1347). Cities infected later, indeed, had lower mortality. As such, we could try to use the number of months since October 1347 *conditional* on year of first infection fixed effects, as an IV. This would exploit *within-year* variation in the timing of the plague. However, there are concerns that even looking at month of arrival within a given year, the timing of the plague could be correlated with city-specific characteristics. IV-A—therefore builds on the intuition above by exploiting the idea that as the virus was more virulent initially, locations that were connected to, and thus more likely to trade more with, Messina were more likely to be infected earlier and hence more likely to suffer high rates of mortality.

We use as an IV the Euclidean distance to Messina, *conditional* on average Euclidean distance to *all* cities in Western and Eastern Europe and the Middle East and North Africa (using their 1300 population as weights). Controlling for average distance to all cities captures the fact that some cities were better connected and trading more overall. Hence, we exploit the fact that it was the specific *trade connectedness to Messina*, and not trade connectedness overall, that mattered for mortality. In addition, since we use Euclidean distances, our IV is not built using the (possibly endogenous) speeds of plague transmission. We add the same controls as above, including the transportation controls. The logic here is similar to Borusyak and Hull (2020) who discuss the problems posed by non-random exposure to exogenous shocks in similar settings. By controlling for network centrality, our aim is to exploit a specific shock to network links.

Controlling for longitude and latitude and allowing them to have non-linear effects is important because it captures any South vs. North and East vs. West effects. Row 7 shows the estimates. The short-run coefficient (−1.17**) is similar to our OLS estimate (IV F-stat = 22.3). The long-run effect is negative, but one-ninth the size of the short-run effect (Z-score = −0.003 vs. −0.026** in 1300–1400).[20]

Borusyak and Hull (2020) show how shock counterfactuals "that were as likely to have occurred" can be used for inference. We argued in Section 2 that Genoa, Vienna, Prague, Leipzig and Visby all could have been the port of entry for the plague. We exploit this fact to investigate the validity of IV-A. For our sample of 165 cities, if we sequentially regress their mortality rates on their Euclidean distances to Messina and each of these alternative ports of entry, we only find a significant negative effect for Messina (Web Appx. Table A.2).[21] Web Appx Fig. A.6 generalizes this by showing the distribution of the coefficient for the top 50 cities before the Black Death (1300). Messina's estimate (−0.06) is particularly skewed to the left whereas the mean coefficient is 0.02, suggesting slightly *lower*, not higher, mortality close to top cities. Larger, more connected cities were not associated with higher mortality rates. These results confirm that there was something specific about Messina, consistent with the historical literature.

IV-B: Month of First Infection IV-B uses the variation in mortality generated by differences in the *month* of first infection *within* a single year. For 124 cities for which we have data on the onset of the plague, Fig. 2(b) shows the relationship between mortality and the *month of peak infection* in the city (= *month of onset* + 3.5 months). The plague was less virulent when peak mortality occurred during the winter and more virulent when peak mortality occurred during summer (the quadratic fit omits January, which has abnormally high mortality due to October being the month of onset of the plague in Europe). We report results using IV-B, dummies for the month of peak infection, while adding the controls used above (including the year of first infection fixed effects). We obtain similar results (row 8; −0.93*** and −0.23). The IV F-statistic is smaller (6.0), possibly due to the fact that we use 11 instruments (month dummies).

If we focus on the relationship between mortality and the month of peak infection in a city for warmer regions vs. colder regions (based on mean temperature in the sample), we find even lower mortality rates in the winter in cities located in colder regions (Web Appx. Fig. A.7). Using as an IV, dummies for the month of peak infection interacted with the log of a city location's average temperature, the IV F-statistic increases to 6.6. The coefficients remain comparable to the coefficients for IV-B (rows 14–15 of Web Appx. Table A.6).

Panel Analysis. We restrict the sample to the same 165 cities as before and focus our analysis on the years 1200, 1300, 1400, 1500, 1600, 1700 and 1750. We then estimate the following panel regression equation:

$$\%\Delta \text{Pop}_{i,t-1\rightarrow t} = \alpha + \beta_t \text{Mort}_{i,1347-52} + \kappa_i + \theta_t + \epsilon_{i,t} \tag{2}$$

where the dependent variable is the percentage change in population between t-1 and t (1100 is dropped), where city (κ_i) and year (θ_t) fixed effects are included, and where the variables of interest are mortality in 1347-52 interacted with the year dummies (β_t shows the effect in each

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[17] The R^2 in Col. (1) falls to 0.08 when we exclude latitude and temperature (correlation with latitude of 0.77). If we re-run the specification in Col. (4) while dropping latitude and temperature, the coefficients of the other controls remain insignificant and the R^2 decreases to 0.18. It does not decrease to 0 because some of the remaining variables are still correlated with latitude.

[18] The sheer number of states (44; source: Nussli, 2011) raises a potential problem as many had only a single major city. Hence we use fixed effects for 7 larger states with at least 5 cities.

[19] See Web Appx. Table A.4 for the full first-stage regressions for IV-A and IV-B.

[20] For this IV, we use cities above 1000 in Europe and cities above 10,000 in the Middle East and North Africa (estimates not available below). What could matter for trade to influence mortality could be proximity to large cities only, or proximity to many cities. We take an intermediary approach and use as weights log population in 1300, thus giving less weight to the largest cities. Results hold if we: (i) control for average distance to all cities above 10,000 only; and (ii) use as weights unlogged population – giving more weight to large cities – or no weights – making a high spatial density of cities important – when computing the distance to all cities (not shown).

[21] Note that we also include the same controls as for the first-stage regression of IV-A.

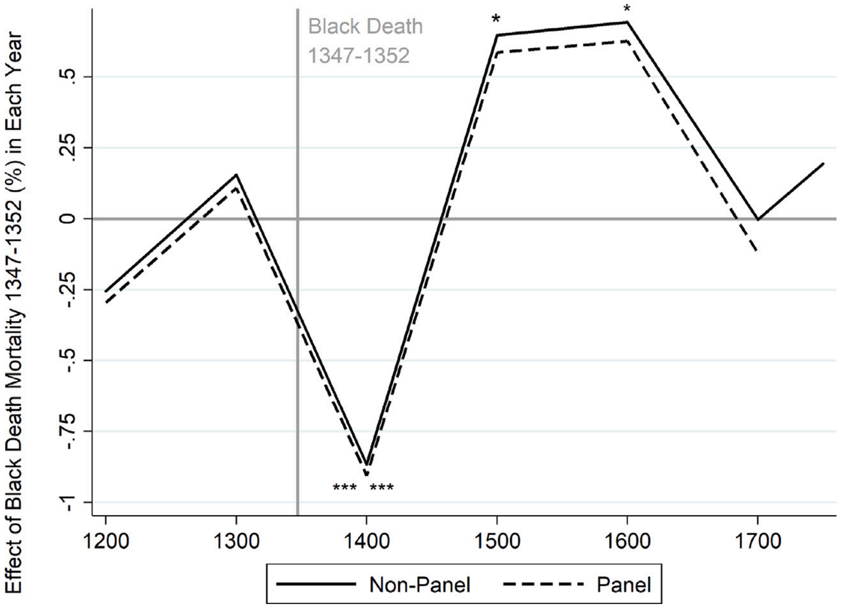

Fig. 4. Yearly effect of Black Death mortality 1347-52 (%), panel regressions. *Notes:* The figure shows the year-specific effects of Black Death mortality (%) in 1347–1352. The omitted year for the panel regressions is 1750. Non-panel regressions consist of repeated cross-sectional regressions for each century. See text for details. Robust SE's (clustered at the city level for the panel regressions): * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

year relative to the omitted year 1750). We use as weights population in t-1 and cluster standard errors at the city level.

Fig. 4 shows the interacted effects (“Panel”) and the corresponding effects when running the cross-sectional regression for each year one by one (“Non-Panel”). The negative effects in 1300–1400 (“1400”; ≈ -0.9***) are offset by positive effects in 1400–1500 (“1500”) and 1500–1600 (“1600”) (coefficients shown in Web Appx. Table A.5). No pre-Black Death effect is observed. We also find similar patterns if the dependent variable is the log of city population in t, then capturing cumulative effects rather than period-specific ones (Web Appx Fig. A.8).

Robustness. These results are robust to additional concerns about causality, specification, data measurement, sample size, and sampling.

The Black Death was attributed to the “vengeance of God” or the “‘conjunction of certain stars and planets” (Horrox, 1994, 48-49). Thus, there was little variation in a city's ability to deal with it. Historians report that some cities had either natural baths (Bath, Nuremberg) or tried to take action in response to the plague (Milan, Venice). Results hold when we drop these (Web Appx. Table A.6). Neither the medical profession nor authorities could respond to the initial shock (Aberth, 2021). Medical knowledge was rudimentary: Boccaccio (2005) wrote that “all the advice of physicians and all the power of medicine were profitless and unavailing”. Individuals, regardless of wealth, could not protect themselves. Outside of the cities mentioned above, preventative measures were almost nonexistent: the practice of quarantine was not employed until 1377.[22]

Bubonic plague reoccurred following the Black Death. This could be a source of bias if subsequent outbreaks were correlated with the initial pandemic. We use data from Biraben (1975) and show results hold if we control for plague recurrences (see Web Appx. Table A.6 for details).[23] The Black Death initially reduced the intensity of conflict (see Sumpton, 1999). However, warfare ultimately intensified and, according to some accounts, led to urbanization (Voigtländer and Voth, 2013a). We show results hold if we control for contemporaneous or past battles (Web Appx. Table A.6), or control for proximity to a battle of the Hundred Years' War prior to the onset of the Black Death. Results also hold if we control for the number of famines experienced by the city's region or country, or the possible magnitude of the Great Famine of 1315–1317 (same table). Finally, Jedwab, Johnson, and Koyama (2019) show that higher-mortality cities persecuted Jewish communities less. Results nonetheless hold if we control for persecutions or drop any city with a persecution.

Similarly, our findings are robust when we: (i) consider other specifications, control for past population trends or study absolute changes in population; (ii) cluster standard errors differently; (iii) take into account measurement error arising from the coding of mortality rates; (iv) focus on cities that are either in the bottom 10% of least affected cities or in the top 10% of most affected cities, since measurement errors in mortality rates are more likely when comparing cities with similar rates; (v) crudely categorize the shock in order to minimize the influence of measurement error[24]; (vi) use alternative population estimates; and (vii) address concerns regarding the external validity of our results, for example increase sample size by using imputed mortality rates for cities outside our main sample (Web Appx. Table A.8). Lastly, we drop cities located within France, Germany, Italy, the UK or Spain (Web Appx. Table A.9).

The various regressions return similar estimates to those obtained using the baseline OLS cross-sectional regressions. This reassures us that mortality had a strong locally exogenous component. In the rest of the analysis, and for the sake of simplicity, we employ the baseline OLS specification.

Overall, we find strong negative effects in the short-run and no effects on average after two centuries. That so many cities that were hit by such a massive population shock recovered may be surprising. However, we are considering economies in which forces of persistence,

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[22] The term quarantine was first used in Ragusa in Croatia in 1377 (Gensini et al., 2004). Other practices such as separating the sick and burning the homes of the infected were also introduced after the Black Death period (1347–1352).

[23] Subsequent plagues were not correlated with mortality (Web Appx. Table A.7). Later occurrences also had a different epidemiology to the initial outbreak (Web Appx. Section 5.).

[24] The 1300–1400 effect of mortality is decreasing convex, which suggests that each additional point of mortality was “recovered” proportionally faster in 1353–1400. Given durable housing, it could be that housing prices decreased dramatically in highly impacted cities, which attracted individuals with low levels of human capital (Glaeser and Gyourko, 2005) and more children.

such as locational fundamentals and sunk investments, were comparatively important. The fact that it took two centuries for cities to recover on average may raise questions about the respective roles of migration and natural increase. We will investigate both of these in sections 5 and 6. In the next section, we exploit the Black Death as a market access shock in order to study its effects on urban systems.

4. The Black Death as a regional shock

So far, we have only discussed the effects of a city's own mortality rate on that city's subsequent growth. However, we need to quantify spillover and general equilibrium effects in order to test the recovery hypothesis for urban systems, not just individual cities.

These effects are important because the population declines associated with the plague reduced market potential. In this sense, it was a massive shock to trade, one which we can exploit to study the effect of a market access shock on city growth (albeit with the caveat that we lack trade data).

Table 6 studies the effects of a city's own mortality and the spillover effects from mortality in other cities. Col. (1) reproduces the city-level results. Col. (2)–(3) show the effects of population-weighted average mortality at the state level on the percentage change in urban population at the state level.[25] Col. (2) shows the effects for cities that existed in 1300. Our baseline estimates suggest that a city with a 10% mortality rate was only 8.7% smaller by 1400. In contrast, col. (2) suggests that if an entire region experienced average mortality of 10%, then a city with a 10% mortality rate would have shrunk by 11.5% by 1400. Col. (3) examines the effects on all cities that are in the dataset in 1400 (including cities not in the dataset in 1300). The effects are larger than before (−1.47**) implying that in high-mortality areas, fewer new cities emerged, a result we explore in more detail below. This is consistent with the Black Death shock having initially strong disintermediation effects (Broadberry et al., 2015, Ch. X).

When we look at the long-run effects in Panel B, we find that unlike the impact of a city's own mortality rate, the effect of aggregate mortality is negative. Yet, the standardized effects – i.e., the Z-scores – are 4–7 times smaller than the short-run effects. Most urban systems were resilient in the long run.

In columns (4) and (5), we define "indirect mortality" as the average mortality rate of the cities of the same state and of the closest 10% of cities, respectively.[26] This is similar to a market potential shock where market potential is often estimated as the inverse distance-weighted sum of population in other locations in the spirit of Harris (1954) and Hanson (2005). In this case, we give the same weight to all cities of the same state or all cities within the bottom 10% of Euclidean distance to the city (332 km).[27] Market potential can then change due to changes in transportation technology (Donaldson and Hornbeck, 2016), or as it is the case in our study changes in population. Finally, market potential could have positive effects if it increases the numbers of consumers and suppliers for the city's producers. Alternatively, it could have negative effects if the city's producers cannot sustain other cities' firms competition (Duranton, 2016).

Note that cities that experienced high mortality did not always experience high indirect mortality (the correlation between the two measures is less than 0.5 in both cases). We again find suggestive evidence that indirect mortality had a negative impact on population between 1300–1400. The coefficient is large and negative (as shown by the Z-scores), but imprecisely estimated. In the longer run (1300–1600), the standardized effects become positive but remain small, suggesting that disintermediation effects disappeared after two centuries.[28]

Since mortality rates may be correlated spatially, Web Appx. Table A.10 shows comparable results when we exclude from the calculations cities within 25 km or 50 km from the city, much like Donaldson and Hornbeck (2016) who exclude from their market access measure (endogenous) *local* railroad changes.[29]

Next, to explore how cities responded when nearby cities were impacted, we examine the interacted effect of a city's own mortality and nearby cities' mortality in Table 3. The interacted effect (Own*Other) is positive and significant in the short run (1300–1400), and zero in the long run (1300–1600). Magnitude-wise, if one considers two cities A and B facing a 20% mortality rate but city A is surrounded by cities with 50% mortality and city B is surrounded by cities with 75% mortality, the growth impact is $(75–50) * 0.06 = 1.5$ (if considering cities in the same state) or $(75–50) * 0.04 = 1.0$ (cities within the bottom 10% of distance). This is a large effect compared to a mean growth rate of −9.4% in 1300–1400.

The negative direct effect of nearby cities' mortality suggests disintermediation effects. In contrast, the positive interacted effect suggests a "survival of the fittest" effect whereby impacted cities did not lose as much when other nearby cities were impacted. Possibly, with a limited pool of migrants in the plague's aftermath, losing fewer people compared to neighboring cities might have helped a city remain attractive regionally, thus aiding its recovery. However, in the long run, once there was less competition for potential migrants, the interacted effect became small as indicated by the Z-scores for 1300–1600.

Finally, we explore how the Black Death shaped the emergence of new cities and the transition of smaller settlements into cities. Recall that our dataset contains 1801 cities but that 1335 of these were not present in the year 1300. These cities can be thought of as the universe of potential city locations. In column (6) of Table 6, we look at the effect of Black Death mortality rate on whether a city enters ours dataset in 1400. To do this we use our extrapolated mortality rate estimates. We find that cities were less likely to emerge where extrapolated mortality rate was high. Likewise, regressing the log population of these 1335 cities (using 500 for cities below 1000) on mortality, we find that fewer locations became urbanized in high-mortality areas (column (7)). These negative effects had disappeared by 1600 (Panel B). This suggests that in the long-run, the Black Death did not delay the transition of villages into cities.

Overall, we provide evidence that short-run disintermediation effects and competition effects played out in some regions. Yet, the fact that urban systems were resilient overall after the shock is consistent with the city level evidence. In the next section, we use both qualitative and econometric evidence on natural increase and migration to shed light on the potential mechanisms by which cities and urban regions recovered.

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[25] For this analysis, we include all 1801 towns, and use spatially extrapolated mortality rates for towns without mortality data and population = 500 inhabitants for towns with population below 1000. We lose 20 states and 1 country (Luxembourg) without any urban population in 1300.

[26] Since mortality is only available for 274 out of the 1801 European cities, we use spatially extrapolated mortality rates for 1527 cities. For each of the 165 observations, the mortality rates of the other towns are constructed excluding the mortality rate of the observation itself.

[27] Results nonetheless hold if we additionally use Euclidean distance to compute the weights or rely on network-based distance using data on transportation costs at the time (not shown).

[28] Results hold if indirect mortality is constructed using cities of the same modern country or relying on the change in market potential 1300–1353 (not shown). To construct market potential in 1353, we use the predicted population of the other towns in 1353 (= pop_{1300}*(100-mort.)).

[29] Results hold if we consider cities within the bottom 5% (219 km) or 1% (86 km) of Euclidean distance to the city, whether considering exclusion zones of 0, 25 or 50 km (not shown).

Table 3
Effects of own mortality × regional mortality.

Dep. Var.:	Change city Pop. 1300–1400				Change city Pop. 1300–1600			
	Pct change (%)		Z-Score of it		Pct change (%)		Z-Score of it	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Own Mort. (%)	−3.19***	−2.38**	−0.069***	−0.051**	−4.47	−3.64	−0.021	−0.017
	[1.08]	[1.08]	[0.023]	[0.023]	[3.28]	[4.00]	[0.015]	[0.018]
Mort. Other (%)	−2.89***	−2.23**	−0.062***	−0.048**	−2.96	−3.09	−0.014	−0.014
	[0.99]	[1.01]	[0.021]	[0.022]	[3.85]	[3.81]	[0.018]	[0.017]
Own × Other	0.06***	0.04*	0.001***	0.001*	0.11	0.09	0.000	0.000
	[0.02]	[0.02]	[0.000]	[0.000]	[0.09]	[0.10]	[0.000]	[0.000]
Definition "Other"	State	Dist. 10%	State	Dist. 10%	State	Dist. 10%	State	Dist. 10%
Observations	160	165	160	165	159	164	159	164

Notes: Own Mort. (%): Mortality rate of the city. *Mort. Other (%): State:* Avg. mortality rate of other cities in the same state (1300). *Mort. Other (%): Dist. 10%:* Avg. mortality rate of other cities within the bottom 10% of Euclidean distance to the city. Robust SE's.

Table 4
Black Death mortality and deserted villages, England.

Dep. Var.:	Percentage Change in Population (%) in Period t			Number of DMVs per 1000 Sq Km			Abs. Change Urban Share
	1290–1377 (1)	1290–1756 (2)	1086–1290 (3)	All (4)	≤10 Km (5)	>10 Km (6)	1290–1756 (7)
β_t	−0.64**	−0.96	0.05	−0.46***	−0.04	−0.41***	−0.16
	[0.31]	[2.06]	[2.77]	[0.33]	[0.10]	[0.30]	[0.21]
Beta Coef.	−0.32	−0.06	0.00	−0.51	−0.25	−0.50	−0.13
Mean	−46.3	22.5	158.9	8.39	0.53	7.44	8.46
Z-Score	−0.05**	−0.01	0.00	−0.07***	−0.03	−0.04**	−0.02
	[0.02]	[0.03]	[0.02]	[0.02]	[0.03]	[0.02]	[0.02]
Obs.; R^2	27; 0.13	27; 0.01	24; 0.00	28; 0.31	28; 0.06	28; 0.35	27; 0.02

Notes: We show for 27–28 counties the effect β_t of mortality (%) on: (1)–(3) the percentage change in total population (%) in different periods; (4)–(6) the number of deserted medieval villages (DMVs) per 1000 sq km (col. (5): Within 10 km from an existing city in 1300; col. (6): Beyond 10 km). We use county pop. in the initial years of the period as weights. Col. (1)–(3) and (7): We exclude Cornwall whose pop. in 1290 is not known. Col. (4)–(6): We control for log pop. in 1290 and log area. Robust SE's.

Table 5
Mortality and city growth, investigation of causality.

Dependent Variable: Percentage change in city population (%) in Period t

Regression:	(1) t = 1300–1400			(2) t = 1300–1600			Obs.
	Coef.	SE	Z-Score	Coef.	SE	Z-Score	
1. Baseline (Columns (1) and (3) of Table 1)	−0.87***	[0.28]	−0.019***	0.36	[0.80]	0.002	165
2. Controls: All	−0.59***	[0.21]	−0.013***	−0.37	[0.70]	−0.002	165
3. 13 Country (2018) FE	−0.62**	[0.26]	−0.013**	0.03	[0.76]	0.000	165
4. 7 States (1300) FE (for States ≥ 5 Cities)	−0.82**	[0.35]	−0.018**	−0.29	[0.68]	−0.001	105
5. 7 5 × 5° Cell FE	−1.12***	[0.29]	−0.025***	−0.45	[0.53]	−0.002	140
6. IVA: Messina w/ Ctrls (IV F = 22.3)	−1.17**	[0.56]	−0.026**	−0.68	[1.69]	−0.003	163
7. IVB: Month w/ Ctrls, Year FE (IV F = 6.0)	−0.93***	[0.33]	−0.021***	−0.23	[0.58]	−0.002	124

Notes: Row 2: Adding the controls of Table 2. Row 3: Adding 13 country FE. Rows 4–5: Adding 44 state FE or 7 cell FE but excluding states with less than 5 cities. Row 6: IV = number of months between the city-specific date of first infection and October 1347. Row 6: IV = Euclidean distance to Messina, controlling for (population-weighted) average Euclidean distance to European and MENA cities in 1300 (Messina is dropped). Row 7: IV = 11 dummies for the month of peak infection (= month of onset (October is omitted) + 3.5). Rows 6–7: Adding the controls of Table 2 as well as the squares and cubes of longitude and latitude. Robust SE's (clustered at the state (1300) level in rows 6–7): * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

5. Natural increase and rural-to-urban migration

The Countryside. Urban areas are less reliant than rural areas on fixed geographical factors, in particular land. In the Malthusian model, a decline in labor-to-land ratios temporarily increases agricultural incomes, until population increases due to higher net fertility (Galor, 2005, 2011). As the demand for urban products increases, the urban sector expands through rural-to-urban migration. More generally, in the macro-historical literature, the urban sector recovers because rural areas recover. However, there is, to our knowledge, little econometric evidence on the latter, which is what we focus on now.

Land use data provides a proxy for rural population. Indeed, the plague led to reforestation as the need for land and wood declined and marginal soils were abandoned (Campbell, 2016, 363) (Web Appx. Section 6.). Kaplan et al. (2009) recreate data on land use from 1200 to 1850 at the 5 by 5 min (10 × 10 km) cell level by combining information on country population, historical forest cover maps, and soil suitability. Using these data, we obtain the land use share of the in-sample countries, i.e. the share of the land used for crops vs. naturally forested (forests were not managed). The share was two thirds in 1300 and decreased by 15 percentage points by 1400. Land use recovered by 1800.

We obtain the mean land share within a 10 km radius of each of the 165 cities and examine how land use varied. Table 7 shows that mortality led to reforestation in 1300–1400, which remained significant until 1500.[30] No effect is found after 1600.[31] Similar patterns are

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[30] Since country populations are one input used in the creation of these data, we verify land use changes are not mechanically correlated with population

Table 6
Mortality and city growth, aggregate effects, 1300–1600.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel A: Dep. Var.:	% Change in total city Population, 1300–1400			% Change city Pop., 1300–1400		Dummy if Exists 1400	Log City Pop. 1400
Own Mortality (%)	−0.87*** [0.28]	−1.15*** [0.40]	−1.47** [0.57]	−0.72** [0.31]	−0.68** [0.33]	−0.002*** [0.001]	−0.004*** [0.001]
Mort. Other Cities (%)				−0.39 [0.43]	−0.57 [0.47]		
Z-Score Mortality	−0.019***	−0.033***	−0.029**	−0.015**	−0.015**	−0.007***	−0.008***
Z Mort. Other Cities				−0.008	−0.012		
Panel B: Dep. Var.:	% Change in Total City Population, 1300–1600			% Change City Pop., 1300–1600		Dummy if Exists 1600	Log City Pop. 1600
Own Mortality (%)	0.36 [0.80]	−1.49 [1.32]	−1.34 [3.17]	−0.08 [0.82]	0.16 [0.84]	−0.001 [0.001]	0.002 [0.002]
Mort. Other Cities (%)				1.49 [1.50]	0.60 [1.78]		
Z-Score Mortality	0.002	−0.009	−0.004	−0.000	0.001	−0.001	0.002
Z Mort. Other Cities				0.007	0.003		
Sample	City	State	State	State	Dist. 10%	City	City
Population	Intensive	Intensive	Total	Other	Other	Extensive	Intensive
Observations	165	68	68	165	165	1335	1335

Notes: (1) Baseline city-level regressions. (2)–(3) *State:* We run the main regressions at the state (1300) level. *Intensive/Total:* The cities considered to construct total city population are the cities that already existed in 1300/all cities. (4)–(5) *Mort. Other Cities: State:* Average mortality rate of other cities in the same state (1300). *Mort. Other Cities: Dist. 10%:* Average mortality rate of other cities within the bottom 10% of Euclidean distance to the city. (6) *Extensive:* We consider 1335 cities that did not already exist in 1300 but existed at one point in Bairoch (1988) (800–1850). Robust SE's: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 7
Black Death mortality rates and land use share, 1200–1750.

Dependent Variable: Percentage Change in Land Use Share (%) in Period *t*
(Land Use Share = Share of the Land Used for Crops Instead of Being Naturally Forested)

t:	1300–1400 (1)	1300–1500 (2)	1300–1600 (3)	1300–1700 (4)	1300–1750 (5)	1200–1300 (6)
Panel A:	Controlling for the contemporaneous changes in both city pop. and country pop.					
β_t	−0.28** [0.12]	−0.21** [0.08]	−0.01 [0.05]	−0.01 [0.07]	0.02 [0.09]	0.04 [0.05]
Beta Coef.	−0.20	−0.18	−0.02	−0.01	0.02	0.11
Z-Score	−0.013**	−0.013**	−0.002	−0.001	0.001	0.007
Panel B:	Not controlling for the contemporaneous changes in both city pop. and country pop.					
β_t	−0.37*** [0.13]	−0.09 [0.11]	−0.01 [0.06]	−0.00 [0.10]	0.00 [0.10]	0.02 [0.03]
Beta Coef.	−0.26	−0.08	−0.01	0.00	0.00	0.06
Z-Score	−0.018***	−0.006	−0.001	−0.000	−0.000	0.011
Mean	−26.0	−15.3	−6.6	−9.2	−9.2	5.7
Obs.	160	160	160	160	160	89

Notes: This table shows for the 165 cities the effect β_t of mortality (%) on the percentage change in the mean land use share (%) within 10 km for each period *t*. The land use share is the share of the land that is used for crops instead of being naturally forested. Panel A: The percentage changes in city population (%) and country population (%) in *t* are added as controls (we lose 5 cities due to missing country population data). Panel B: We do not include the total and city population growth controls. In both panels, we use as weights populations of the cities in the initial year of the period. Robust SE's.

observed if we consider a panel regression with city and year fixed effects instead (Web Appx. Figure A.9). Nor are pre-trends present in either the cross-section or the panel. Finally, in the aggregate, urban areas recovered their total population by 1500 and their rural areas recovered theirs one century later (Web Appx. Fig. 11(b)).

This is consistent with the mechanism emphasized by Voigtländer and Voth (2013b). In their model, a large mortality shock triggers a transition to a new steady state. Given non-homothetic preferences, as wages increase demand for urban goods spurs urbanization. Since pre-industrial cities were unhealthy and because the plague returned frequently and conflict was endemic, death rates and incomes remained high. One can also envision different mechanisms: trade-related fixed factors in cities – e.g., a good location on the coast – means that urban wages increase when urban population declined, thus raising the demand for food and driving rural recovery *locally*.

Recovery in high-mortality areas must have been driven by either differentially increasing fertility and decreasing mortality in high-mortality areas or migration from low- to high-mortality areas. Since rural areas close to cities also recovered, migrants must have come *on net* from rural areas farther away. Thus, marginal rural areas suffered relatively greater population losses following the Black Death. Was the relative decline of these marginal rural areas correlated with their mortality rate? If fixed factors increase demand for labor in high-mortality

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changes. The correlation between the change in land use within 10 km of a city and the change in the population of that city is 0.14 in 1300–1400 and −0.03 in 1300–1600. The correlations with the change of that city's country population are higher, but still low, at −0.30 and 0.44 respectively. Indeed, changes in the forest maps also cause local land use changes. Lastly, results hold whether we control (Panel A) or not (Panel B) for the contemporaneous changes in both city population and country population.

[31] We test for parallel trends and results hold if we cluster standard errors at the country level.

areas, it could well be that marginal areas are more depopulated in *low-mortality*, not *high-mortality*, areas, which we test now.

Deserted Medieval Villages (DMVs). Historians document that villages were slowly deserted in the years following the Black Death (Beresford, 1954; Braudel, 1965). Peasants left their villages to seek newly available economic opportunities in high-mortality cities. This immigration "topped up otherwise diminishing urban communities" (Platt, 1996, 20) (see Web Appx. Section 7.). Since labor was in short supply and peasants demanded better pay, many landowners switched to sheep rearing, which required less labor than arable farming (Voigtländer and Voth, 2013a).

Data on the location of DMVs exist for all 41 English counties during the medieval era (Fenwick and Turner, 2015). For 28 of these, we know from Shrewsbury (1970) and Scott and Duncan (2001) clergy mortality, which we use as a proxy for overall mortality. This allows us to study how the number of DMVs varied with mortality, *depending* on their proximity to cities.

Since this sample differs from the sample of 165 cities, we verify in col. (1)–(3) of Table 4 that mortality had a negative effect in the short run and no effect in the long run. For the same 28 counties, we obtained population in 1086, 1290, 1377, 1756 and 1801 (data unavailable ca. 1600). For the period 1290–1377, we find a negative effect, at −0.64** (col. (1)). For the period 1290–1756, we find an insignificant effect, at −0.96 (col. (2)), but the effect is smaller than the short-run effect once standardized (beta coef. of −0.10 vs. −0.35 in 1290–1377). When using 1801 (first census), the standardized effect is small (−0.08, not shown). There are no pre-trends for 1086–1290 (0.05, beta coef. of 0.00, col. (3)).[32]

In col. (4)–(6) the dependent variable is the log number of DMVs per 1000 sq. km. We control for the county's log area and log population in 1290 since the density of DMVs depends on pre-plague human density.[33] We find a negative effect of mortality, at −0.46*** (col. (4), beta coef. of −0.51).[34] Low-mortality areas had *more* DMVs than high-mortality areas. Therefore, people disproportionately left the relatively plague-free rural areas. Table 7 showed that rural areas in the vicinity of cities – within 10 km – were not affected by the Black Death in the long-run. By 1600 they were completely repopulated. We should thus expect relatively more DMVs in low-mortality areas *beyond* 10 km from cities.

For 39 counties, we obtain from Fenwick and Turner (2015) the location of each DMV in England and compute the minimal distance to a 1300 city. For each of the 28 counties, we construct the number of DMVs (per 1000 sq km) both within and beyond 10 km from a city. We verify in col. (5)–(6) that the loss of villages is driven by areas farther away from cities.[35] In col. (7), we regress the absolute change in the urban share (%) on mortality and find a small and insignificant negative effect. DMVs were small. Thus, the loss of villages in low-mortality areas may have not been large enough to affect urbanization patterns across counties. This also suggests that the repopulation of high-mortality areas was driven by migration from both urban and rural areas in low-mortality areas.

Net Wages. Data on wages and prices does not exist for enough cities during our period of study for a formal analysis. Instead, Web Appx. Section 8. provides qualitative evidence on wage patterns after the Black Death. Overall, the historical literature has consistently found that in cities where mortality was high living standards significantly improved for all workers.

Natural Increase. The relative recovery of high-mortality areas could have been due to higher real wages raising fertility and lowering mortality relative to low-mortality areas. While the population recovery of Europe's total population by 1600 was only possible due to natural increase, it is less clear whether natural increase was responsible for local recovery. The literature on the European Marriage Pattern (EMP) – a higher age of first marriage and high rates of female celibacy – shows how the Black Death reduced fertility (Voigtländer and Voth, 2013a). Thus, natural increase likely played a minor role in *local* recovery.[36]

Migration. The rate of urban recovery we observe can only have occurred via migration. First, various cities had already recovered before 1400. Barcelona (mortality of 36%), Florence (60%), Lübeck (30%) and Venice (60%) recovered their pre-plague population levels in just 5, 30, 10 and 25 years respectively. Their rate of natural increase would have needed to be above 30 (per 1000) for natural increase to explain recovery. These rates were unheard of until the 20th century, particularly in preindustrial cities where such rates were typically negative (Woods, 2003; Jedwab and Vollrath, 2019; Gindelsky and Jedwab, 2022).

Second, historians speculate that "the first few years after the epidemic witnessed especially high migration rates" (Poos, 1991, 108). Penn and Dyer (1990, 363) note that late medieval workers had a great "capacity for geographical mobility" evident "from the indirect testimony of locative surnames which reflect migration into towns, and the patterns of immigration and emigration". Likewise, the number of freemen admitted into York increased by 365% in the year of the plague (Dobson, 1973, 17). London saw a "great concourse of aliens and denizens to the city and suburbs, now that the pestilence is stayed" (Sloane, 2011).[37]

Fertility. For a same mortality shock, we should expect fertility to be lower in a Northern European region or a region characterized by the EMP (Dennison and Ogilvie, 2014; Voigtländer and Voth, 2013a). We thus test if the speed at which high-mortality cities relatively recover depends on where the cities were located. We classify our cities into North vs. South or Strong EMP vs. Weak EMP (based on historical data from Dennison and Ogilvie (2014) on the average age at first marriage and the female celibacy rate (%) at the country or regional level).[38]

Using the same specification as before, we interact mortality with a North dummy or a Strong EMP dummy and test if the interacted effect is negative. If natural increase was important for local recovery, we should expect high-mortality cities in North/Strong EMP regions to recover slower than high-mortality cities in South/Weak EMP regions, because North/Strong EMP cities were more likely to recover solely through migration whereas South/Weak-EMP cities were more likely to experience both migration *and* natural increase.

Web Appx. Table A.11 shows the interacted effects in 1300–1600. The North dummy is based on 9 Northern European countries. The Strong EMP dummy is equal to one for cities in countries/regions with an age at first marriage or a female celibacy rate above the mean or median in the sample. The interacted effects show that Northern cities did not recover relatively slower, since the coefficients are positive,

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[32] Observations are weighted by their population in 1290. We exclude Cornwall whose population in 1290 is underestimated due to the lack of data on their large mining population (see Broadberry et al., 2010, 14). Middlesex is omitted as it lacks mortality data.

[33] We also weight observations by their populations in 1290.

[34] We include Cornwall, since we only use populations as weights and as controls. Removing Cornwall or adding London or extra counties does not affect the results (not shown).

[35] Our analysis focuses on 28 counties because mortality is only available for 28 counties. We verify for the 41 counties that the density of DMVs is uncorrelated with a dummy for whether mortality is available. Results hold if we impute mortality from other sources (not shown).

[36] See Web Appendix Section 9. for more qualitative evidence.

[37] See Web Appx. Section 10. for qualitative evidence. Migration was caused by both an improved bargaining position of peasants *and* an increase in labor coercion in some areas forcing peasants to escape these (see Web Appx. Section 11. for more qualitative evidence).

[38] See Web Appendix Section 9. for details on the data.

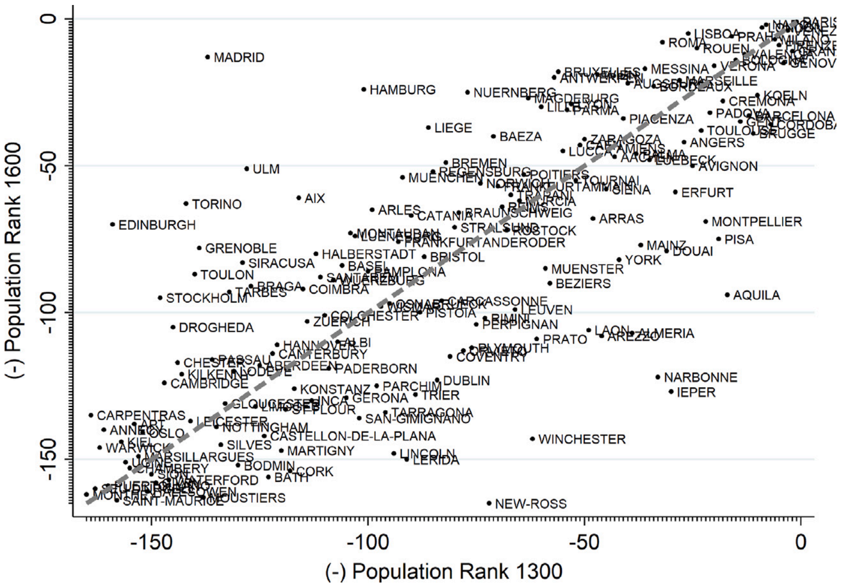

Fig. 5. Permutations in the distribution of cities. *Notes:* The figure shows for the 165 cities of the main sample the relationship between their inverted pop. rank in 1300 (among the 165 cities; 0 = largest city) and their inverted pop. rank in 1600 (among the 165 cities; 0 = largest city).

Fig. 6. Evidence of post-plague reset closer to trade-related factors. *Notes:* The figure shows for the 1801 cities of the full sample (16 Western European countries) in 1100–1750 the share of their total population that resides within 10 km from the coast, a river, or a (Roman and/or Medieval) road intersection, or that resides within a city belonging to the Hanseatic league (circa 1300). The dashed line shows the counterfactual share in 1300–1750 had the share evolved as it did between 1200 and 1300, before the Black Death (1347–1352) occurred.

thus suggesting that migration was the main driver behind recovery. We find overall similar results for the EMP dummy.

The evidence suggests that migration from low to high-mortality locations, driven by net wage differentials, was the main driver of urban recovery.

6. Heterogeneous recovery: Land quality and trade

A pandemic might have no long-term spatial effects *on average*, but there could still be *permutations*. For example, as some large cities become relatively smaller and small cities relatively larger. These permutations may in turn be affected by how mortality and the pre-pandemic characteristics of these cities interact.

Regressing the rank of each city in 1600 on its rank in 1300, we find a slope of 0.86. Hence, large cities tended to remain large and small cities tended to remain small. However, the R^2 is 0.56. Aggregate recovery thus hides *permutations*. Fig. 5 illustrates this, with many cities far from the forty-five degree line.[39]

were these permutations in recovery associated with an urban reset? That is, were cities that recovered more fully from the Black Death better located? We explore this by examining if the total population of the 1801 cities of our full European sample "moved" closer to areas with better trade potential.

We first consider four trade factors: proxmity to the coast (10 km dummy), rivers (10 km dummy), roads (Roman and/or medieval) and

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[39] Campbell (2016, 365) notes that "towns competed with each other in an urban survival of the fittest". See Web Appx. Section 12. for a lengthier discussion of the permutations in the data.

road intersection (10 km dummy), and whether a city belonged to the Hanseatic League (circa 1300) dummy. The solid line in Fig. 6 shows that 90% of the population lived close to a trade factor in 1100 and that this share declined to 83% in 1300. Had this trend continued, the extrapolated dashed line suggests the share of the population living in locations well-endowed with trade factors would have eventually reached 60% in 1700. However, in actuality, there was a break in this trend immediately after the Black Death. The actual share of the population in well-endowed locations is 77% in 1700. This suggests that the Black Death shock may have contributed to 17% of the population of Europe living in economically more advantageous regions.[40]

While this exercise is suggestive, in what follows we are interested in quantifying whether places endowed with certain types of factors did, indeed, recover more fully than those not endowed for a given mortality shock. We are also interested in when these factors mattered most. We thus modify Eq. (1) by interacting mortality ($\text{Mort}_{i,1347-52}$) with selected city characteristics (Char_i) while controlling for the characteristics themselves and mortality:

$$\%\Delta\text{Pop}_{i,t} = \alpha + \beta_t\text{Mort}_{i,1347-52} + \text{Mort}_{i,1347-52} * \text{Char}_i\theta + \text{Char}_i\xi + \epsilon_{i,t} \quad (3)$$

Throughout, we focus on our main sample of 165 cities, for the period 1300–2015. For two cities experiencing the *same* mortality shock (e.g., 50%), the vector $\boldsymbol{\theta}$ captures the differential recovery effects of each characteristic.[41]

Factor Selection. With 165 cities, we cannot add all 27 variables of Table 2 and their interactions with mortality. Instead, we select those that proxy for: (i) *land quality*: the three agricultural suitability measures (cereal, potato, pastoral); and (ii) *access to markets and trade*: coastal and river dummies, Roman road or medieval land route intersections, and the Hanseatic League dummy. We include factors proxying for (iii) *agglomeration effects* as the log of the estimated population of the city in 1353 (= pop._{1300} x (100-mort.)/100). We proxy for (iv) *institutions* using three dummies for whether the city was part of a monarchy, was a state capital, and whether it had a representative body (c. 1300).

Identification. Table 8 shows the 11 interacted effects (using Z-scores), for 1300–1750 (col. (1)–(5)) and 1300–2015 (col. (6)). The 11 interacted effects are *simultaneously* included in the model and show the recovery effect associated with each factor *conditional* on the recovery effect associated with each other factor. Note that we show the interacted effects for the period 1300–1400 because cities started recovering in 1353–1400. We thus use 1300 as the start year instead of 1353 because we do not know the true population of each city in 1353.

We do not use panel or IV regressions for this analysis as these methods return results that are similar to OLS. However, since we cannot be sure that mortality was indeed exogenous, the results in this section should be taken with caution.[42]

Land Quality. The coefficient on mortality*cereal suitability is positive but not significant after 1400 (col. (2)). However, the impact is meaningful since the corresponding estimated beta coefficient (henceforth, "beta") reaches 0.47 by 1600 and remains high thereafter (0.17 in 2015).[43] Potato suitability may have also helped highly impacted cities escape their post-plague low-population equilibrium from the 17th century onwards (col. (4)).[44]

In high-mortality areas suitable for pastoral farming we find a negative effect in 1500–1600 (col. (3)) and no effects before (col. (1)–(2)). The effect in 1500–1600 is strong (beta = -0.64) and becomes weaker over time (beta = -0.25 in 2015). Indeed, higher wages due to labor shortages created incentives for landlords to specialize in pastoral agriculture, thus reducing the need for labor (Voigtländer and Voth, 2013a, p. 2255).[45] As seen, the effect only became significant in the 16th century. Indeed, pastoral farming as a solution to labor scarcity and rising wages did not arise immediately across Europe (Jedwab et al., 2022b).

Agglomeration Economies. The literature (e.g., Duranton and Puga, 2004) distinguishes economies of scale – in production, market places, and consumption – and agglomeration economies strictly defined, according to which a larger population increases productivity and wages, which should cause in-migration. We thus interact estimated city population in the immediate aftermath of the Black Death (1353) with mortality. However, we find no effects, suggesting that agglomeration economies did not play a major role in city growth at this time.[46]

Access to Markets and Trade. The interacted effect for coastal proximity is one of the only two significant coefficients in 1300–1400 (col. (1)) along with the interacted effect for the Hanseatic league. While the standardized coefficient of mortality is −0.083, the coefficient of mortality*coastal proximity is 0.026. Thus, relative to non-coastal cities, coastal cities recovered almost 33% faster by 1400. In 1500, the interacted effect was strong enough relative to the direct mortality effect that coastal high-mortality cities had on average fully recovered relative to low-mortality cities.[47] The coastal effect remained strong for most of the pre-industrial era (beta = 0.99 in 1500, slowly decreasing to 0.33 in 2015).[48][49]

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[40] Of course, this assumes that the trend observed in 1200–1300 would have continued. We hypothesize that this downward trend was the result of the population expansion that took place in Europe from 1100–1300 (from 50–60 million to 75–80 million). This increase in population likely showed up in the formation of new towns, many of which had less favorable trade geography.

[41] If labor becomes scarce in both cities but one city has local factors complementary to labor, its net wages increase, attracting people. The city's population then recovers relative to lower-mortality cities. With high migration costs (incl. information costs), recovery may be slow. Alternatively, one city might have a local factor that is not valuable immediately after the shock (e.g., given the state of technology). Initially, the city will not recover faster than another equally impacted city. However, once the factor becomes valuable (e.g., a few centuries later), it could help the city escape the low-population post-plague equilibrium.

[42] We have 23 variables and 165 cities. Adding interactions of each factor with instruments would leave us with no variation, and creates multiple weak instruments. Since what matters are the interactions with mortality, we also do not report the independent effect of each factor.

[43] Calculations suggest that places with 1 SD higher cereal suitability recovered twice as fast as cities with poor cereal suitability experiencing the same mortality rate.

[44] The country-level effects of Nunn and Qian (2011) appear in 1750, whereas our interacted effects appear in 1700 because the local cultivation of the potato started in the late 16th century.

[45] Thomas Moore wrote in *Utopia* (1516): "Your sheep. . . that commonly are so meek and so little, now, as I hear, they have become so greedy and fierce that they devour men themselves".

[46] Web Appx. Table A.14 also shows limited recovery effects for: (i) larger cities, market access, or state population size in 1300, further confirming a limited role for agglomeration economies; (ii) craft guilds before the Black Death (source: Ogilvie, 2019), market fairs c. 1300, and log walled area c. 1300 (controlling for city population size c. 1300), which suggests limited recovery from economies in scale in production, market places, and consumption; and (iii) the seats of a bishopric/archbishopric or university towns c. 1300, which indicate limited recovery from economies of scale in human capital and institutional capacity.

[47] Stark examples include Barcelona (mortality of 36%; full recovery by 1355) and Venice (60%; 1375) for coastal cities and Lübeck (30%; 1360) as an example of a major Hansa town.

[48] By 2015, it is not significant, consistent with coastal proximity having become less of an advantage given advances in transportation technologies (e.g., railroads and roads) (Henderson, Squires, Storeygard, and Weil,

Table 8
Black Death mortality and city population recovery (Z-scores): geography, agglomeration effects, and institutions.

Dependent Variable: Z-Score of the Percentage Change in City Population (%) in Period 1300-*t*						
Period 1300-*t*: *All Vars Simultaneously Included:*	1400 (1)	1500 (2)	1600 (3)	1700 (4)	1750 (5)	2015 (6)
Land Quality:						
Mort.*Cereal Suitability Index	−0.002	0.002	0.003	0.002	0.002	0.001
	[0.006]	[0.002]	[0.003]	[0.003]	[0.003]	[0.003]
Mort.*Potato Suitability Index	0.006	−0.001	0.004	0.005**	0.004**	0.005**
	[0.007]	[0.003]	[0.003]	[0.003]	[0.002]	[0.002]
Mort.*Pastoral Suitability Index	0.013	−0.002	−0.019*	−0.007	−0.008*	−0.006
	[0.014]	[0.007]	[0.010]	[0.006]	[0.005]	[0.006]
Access to Markets & Trade:						
Mort.*Coast 10 Km Dummy	0.026**	0.019***	0.022***	0.015*	0.011*	0.007
	[0.010]	[0.005]	[0.008]	[0.007]	[0.006]	[0.008]
Mort.*Rivers 10 Km Dummy	−0.011	0.002	0.008	0.010**	0.009**	0.011***
	[0.012]	[0.004]	[0.005]	[0.004]	[0.004]	[0.004]
Mort.*Road Intersect. 10 Km Dummy	0.013	0.010*	0.006	0.004	0.004	0.003
	[0.013]	[0.005]	[0.007]	[0.006]	[0.005]	[0.006]
Mort.*Hanseatic League Dummy	0.062***	0.015*	0.019*	0.015*	0.012	0.009
	[0.020]	[0.008]	[0.011]	[0.009]	[0.008]	[0.010]
Mort.*Log Est. City Population 1353	−0.004	0.004	0.007	0.003	0.003	0.003
	[0.005]	[0.003]	[0.004]	[0.004]	[0.003]	[0.004]
Institutions:						
Mort.*Monarchy 1300 Dummy	−0.004	0.004	0.005	0.005	0.003	−0.003
	[0.011]	[0.004]	[0.005]	[0.004]	[0.004]	[0.004]
Mort.*State Capital 1300 Dummy	−0.014	−0.010	−0.001	0.009	0.007	−0.002
	[0.018]	[0.009]	[0.011]	[0.009]	[0.008]	[0.008]
Mort.*Representative 1300 Dummy	0.018	−0.001	−0.002	−0.004	−0.004	−0.002
	[0.012]	[0.005]	[0.005]	[0.004]	[0.004]	[0.004]
Mortality	−0.083***	−0.012	−0.028*	−0.039**	−0.030**	−0.036**
	[0.029]	[0.012]	[0.016]	[0.018]	[0.015]	[0.015]
Observations	165	164	164	164	164	165
R-squared	0.45	0.29	0.39	0.35	0.35	0.25

Notes: This table shows for the 165 cities the effects of Black Death mortality (%) interacted with 11 selected factors (the 11 interacted effects are simultaneously included). We only show the interacted effects and the effect of mortality but the 11 factors are also used as controls. We use as weights city population in 1300. Robust SE's: * p < 0.10, ** p < 0.05, *** p < 0.01.

According to Dollinger (1970, viii), by the mid-13th century, Hansa merchants had a near monopoly of trade between Germany and England. By 1300 it comprised a network of cities across the North Sea, capable of boycotting trade with states that violated its rights. As the network developed, cities were able to invest in infrastructure (e.g. warehouses) and armed guards for their merchants.

The interacted Hansa effect is strong (beta = 0.72) in 1300–1400. A standardized coefficient of −0.062 compared to −0.083 for mortality implies that a Hansa town had already relatively recovered three fourths (−0.062/−0.083 = 0.74) of their population by 1400. The interacted Hansa effect then remained significant until 1700, by which time the league was in decline (Dollinger, 1970).

Rivers exhibited positive and significant effects from the 17th century onwards (col. (4)), indicating that river access might have helped some highly impacted cities escape their post-plague low-population equilibrium. River transportation was important throughout the medieval period (Masschaele, 1993). But the interacted effects of mortality and rivers are much stronger after 1600. This might reflect greater investment in riverine technologies and canals as documented for England by Bogart (2011). Similar improvements in riverine transport also occurred elsewhere in Europe.[50]

Being at the intersection of roads/trade routes has a positive, significant, and economically large effect in 1500 (col. (2), beta = 0.51). The effect weakens subsequently, as alternative transport networks expanded.

To summarize, cities with better market access and geographic endowments recovered faster. Cities lacking these were more likely to stagnate after the Black Death—sometimes taking centuries to regain their former populations.

Most of the important effects we discuss in Table 8 remain strong and significant when including 13 modern country fixed effects (Web Appx. Table A.13). Identification comes from comparing cities experiencing the same initial shock, having the same 11 characteristics, and belonging to the same entity.

Lastly, adding the absolute values of the beta coefficients for the different types of factors from Table 8, we find that land quality and trade potential were particularly important in the first centuries after the Black Death and their importance decreased over time (see Fig. 7(a)).

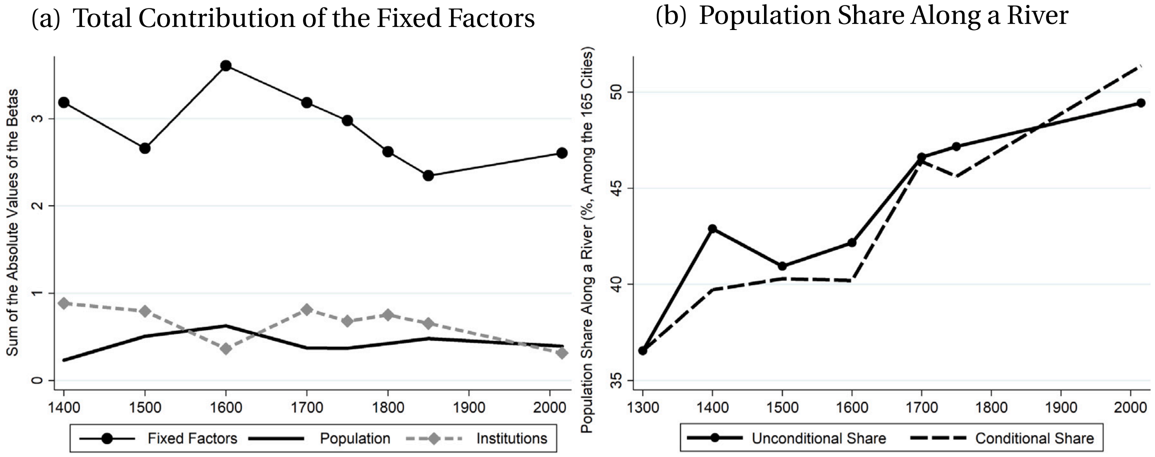
7. Concluding discussion: Counter-factual analyses

In this final section, we discuss two important implications of our results for the evolution of Europe's urban system after the Black Death.

2017) and port concentration, for example due to containerization (Brooks, Gendron-Carrier, and Rua, 2018).

[49] Web Appx. Table A.14 shows stronger effects for: (i) cities located *directly* on the coast as well as estuarine locations, especially when they were part of an unified state (as proxied by monarchy), likely due to market size effects; and (ii) coastal Mediterranean cities, as Mediterranean trade was particularly important in the medieval era. We then observe significant effects for Atlantic cities starting in the 17th century, consistent with Acemoglu, Johnson, and Robinson (2005).

[50] In France, in the seventeenth century Colbert passed laws ensuring that all rivers had to be traversable by private towpath companies. Canal investments often raised the value of being on a river as canals were often dug to connect two previously separate riverine systems (Geiger, 1994).


(a) Total Contribution of the Fixed Factors

(b) Population Share Along a River

Fig. 7. Total contribution of the fixed factors to aggregate patterns. *Notes:* Fig. 7(a) shows for each period from 1300–1400 ("1400") to 1300–2015 ("2015") the sum of the absolute values of the beta coefficients for the interacted effects of mortality with: (i) the fixed factors (cereal, potato, pastoral, coast, rivers, road intersection, Hanseatic League); (ii) the population in the aftermath of the Black Death (estimated for the year 1353); and (iii) institutions (monarchy, state capital, representative body). Fig. 7(b) shows the percentage share of the total pop. of the 165 main cities that is located along a river in the raw population data ("unconditional") and based on 1300 population × the recovery effect of rivers to predicted city population growth ("from recovery effect of rivers").

First, we ask: *Could some cities have permanently declined after the Black Death absent good land or trade factors?* Table 1 showed no long-term effects of the plague at the city level. Table 8, however, suggests that cities with favorable fixed factors of production (e.g., proximity to the coast or a river) recovered faster. For example, for mortality (last row), the standardized coefficient of −0.036 in 1300–2015 is more than two fifths of the 1300–1400 coefficient, −0.083. This implies that for cities without important fixed factors, a large Black Death shock would have permanently reduced their size relative to other cities.

Our estimates can now be used to predict which cities might have failed to recover had they not been endowed with favorable factors. We rely on the estimates in Table 8 to predict the counterfactual population level and rank of each city in 1750 or 2015 *absent* good endowments.[51] Comparing the predicted ranks of each city excluding the recovery effects of the factors with their predicted ranks when the same effects are included, we then identify cities that would have counterfactually lost of a lot of population had such factors been irrelevant.

Examples of cities that would be much smaller today absent these favorable recovery factors include major cities in 1300, for example: Venice (3rd largest, mortality ≈ 60%), Florence (5th, 60%), Cordoba (8th, 50%), Naples (9th, 65%), Cologne (10th, 30%), Pisa (19th, 35%), Toulouse (23rd, 50%), Rouen (24th, 45%), and Marseille (28th, 55%) (Web Appx. Fig. A.12; Web Appx. Fig. A.13 for 1750). What these cities all have in common is that they were hit hard by the plague and were either coastal, riverine, located on a road intersection, or part of the Hanseatic league.

Second, we ask: *Could the Black Death have led to a beneficial urban reset?* Section 6 suggested that people moved to better endowed cities. The aggregate growth potential of the post-Black Death urban network may therefore have increased. Fig. 6, for example, suggests that cities with better trade potential recovered more fully. Consequentially, the share of the European population living in areas with favorable factors for trade increased.

We next examine if cities along a river recovered faster. We find an increasing share of the urban population resided along a river over time. Fig. 7(b) shows that the unconditional population share of the 165 cities located along a river grew from 1400–2015. It also reports the predicted increase in the population share of riverine cities based on their population in 1300 and the estimated contribution of the recovery effect of rivers to predicted city growth. The conditional population share along rivers has increased over time and explains almost all of the unconditional share.[52]

Pursuing the possibility that the Black Death led to a beneficial "urban reset" further would require a theory of what a dynamically optimal distribution of population involves. Nonetheless, the impact of the Black Death on Europe's spatial distribution of population might have been one factor contributing to both the Great Divergence that emerged between Europe and the rest of the world and the Little Divergence that took place within Europe.

CRediT authorship contribution statement

Remi Jedwab: Formal analysis, Investigation, Writing – original draft, Writing – review & editing. **Noel D. Johnson:** Formal analysis, Investigation, Visualization, Writing – original draft, Writing – review & editing. **Mark Koyama:** Investigation, Writing – original draft, Writing – review & editing.

Appendix A. Supplementary data

Supplementary material related to this article can be found online at https://doi.org/10.1016/j.jue.2023.103628.

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[51] We account for the fact that predicted percentage growth in 1300–1750 or 1300–2015 must be left-censored at −100 by estimating Tobit models. The effects on the latent variable are almost the same as with OLS (not shown). We also verify that the predicted 1750 rank of each city among the 165 cities – based on predicted 1750 population levels calculated using 1300 populations and the predicted change in 1300–1750 – strongly correlates (0.79) with the actual 1750 rank of each city among the 165 cities. For 2015, the correlation is weaker (0.61) due to Industrial Era factors.

[52] We cannot, of course, rule out the possibility that other shocks could have produced similar patterns. We are, therefore, cautious in attributing this urban reset to the Black Death alone. In this sense, the 1300–1400 vs. 1200–1300 comparison is cleaner, as it reduces the possible influence from other events.

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