Pandemics, Places, and Populations: Evidence from the Black Death

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Abstract

The Black Death killed 40% of Europe's population between 1347-1352, making it the largest demographic shock in modern history. 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 the Black Death on city growth. On average, cities recovered their pre-Plague populations within two centuries. In addition, aggregate convergence masked heterogeneity in urban recovery. We show that both of these facts are consistent with populations returning to high-mortality locations endowed with more rural and urban fixed factors of production. Land suitability and natural and historical trade networks played a vital role in urban recovery. Our study highlights the role played by pandemics and mortality shocks in determining both the sizes and placements of populations.

JEL: R11; R12; O11; O47; J11; N00; N13

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In a seminal study Davis and Weinstein (2002) use a "spatial catastrophe"—the bombing of Japanese cities during World War II—to investigate the relative importance of various theories of urban location. We build on their work by studying the effects of the Black Death which was the largest demographic shock in the history of mankind. It killed approximately 40% of Europe's population between 1347-1352. Some regions and cities were spared, others were devastated—England, France, Italy and Spain lost as much as 50-60% of their populations. While the Black Death has been extensively studied, little is know about its effects on city growth. More generally, little is known about the localized effects of continent-wide pandemics due to their rarity.

In this paper, we use city-level data on Black Death mortality to test whether cities that had relatively high mortality rates were permanently affected. A priori, the city-level effects of mortality are ambiguous: (i) If there are local increasing returns a large negative shock to a population could lead to a negative feedback cycle in which wages and population continue to decline; (ii) If production relies largely on spatially fixed factors of production such as land and other natural resources, i.e. locational fundamentals, wages go up due to labor scarcity, thus allowing population recovery by either causing migration from low to high-mortality areas or increasing fertility and decreasing mortality in the latter; alternatively (iii) If the population shock and the resulting higher wages permit investments in physical or human capital or leads to beneficial institutional changes, high-mortality areas may gain a long-run productivity advantage and keep growing. The implications of each of these models for long-run city growth are different: relative *decline* (i), *recovery* (ii) or *growth acceleration* (iii).

Our setting is particularly well suited to testing how localized shocks affect economic activity (see Bleakley and Lin (2015) for a survey of the literature on shocks and path dependence). The Black Death was a comparatively "pure" population shock. Unlike other shocks considered in the literature, buildings and physical capital were not destroyed and the event itself did not directly target a particular demographic group. Wars and bombings, as studied by Davis and Weinstein (2002, 2008) and Glocker and Sturm (2014) killed people but also led to massive physical destruction and resulted in government reconstruction programs. Disasters such as floods and fires, as studied by Boustan et al. (2017) and Hornbeck and Keniston (2017) kill far less people

but also lead to massive physical destruction.[1] Malaria, HIV, or the 1918 influenza pandemic, as studied by Bleakley (2010), Young (2005), Almond (2006), Donaldson and Keniston (2016) and Beach et al. (2018) disproportionately kill subgroups of the population.[2] Finally, some studies have examined the local effects of exogenous changes in technology, for example transportation technology (Bleakley and Lin, 2012).[3]

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 Black Death mortality rate was associated with a 8.7 percentage point fall in city population. After two centuries the impact of mortality was close to zero. When we examine the spill-over, general equilibrium, and rural effects of the Black Death, we find similarly negative effects in the short-run and mostly nil effects in the long-run. Next, we use data on deserted medieval villages in England to show that more settlements were abandoned in low, rather than high, mortality areas. Therefore, recovery in high-mortality areas must have been accelerated by migration from low-mortality areas.

We also show that urban recovery is almost entirely 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 and urban fixed factors related to natural advantages (e.g., coastal access) or sunk man-made advantages (e.g., roads) favoring trade. We show that aggregate urban recovery hides permutations in the distribution of cities. Some cities permanently collapsed after the Black Death whereas other cities gained in the long run. We provide evidence that these permutations were associated with the presence of fixed factors. Since permutations favored cities with better land and trade potential, urban systems may have become more productive.

We provide evidence that these results are causal. First, the virulence of the Plague was plausibly unrelated to factors related to future city growth. Second, the parallel trends assumption is verified. Third, results are robust to the inclusion of controls

[1] Genocides and expulsions, as studied by Acemoglu et al. (2011) and Chaney and Hornbeck (2015) target specific subpopulations and tend to result in physical destruction. Famines, as studied by Meng et al. (2015), kill large numbers of people but the poor die at higher rates and cognitive abilities are affected.

[2] Malaria tends to kill children whereas HIV kills working-age adults (Weil, 2010). The 1918 influenza disproportionately killed the "very young, those around age 30, and the elderly" (Almond, 2006).

[3] An overall mortality rate of 40% is also unprecedented. Other examples of shocks include the 1918 influenza pandemic (3-5% of the world's population), the Ukrainian Famine (10%), World War II (3.5% of the world's population), the Bengal Famine (6.6%), the Great Chinese Famine (2.5-6.8%), and the Rwandan Genocide (11%). The introduction of European diseases in the Americas after 1492 killed between 75% and 90% of the population, but this was over the course of a century (Koch et al., 2019).

for city characteristics, region fixed effects, and contemporaneous events. Fourth, our results hold when we implement instrumental variables strategies premised on the facts that: (i) the Black Death entered Europe through the Sicilian port of Messina and was more virulent in its earlier stages, for pathogenic reasons; (ii) 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; and (iii) it was connectedness to Messina and not connectedness to other important cities that mattered for plague virulence.

We complement the analysis of Davis and Weinstein (2002, 2008) in four ways. First, our shock is far larger than the shock they study. Mean Plague mortality was 40% and all cities were impacted, with mortality equal to 5% and 80% at the 1st and 99th percentiles. In contrast, 20% and 8.5% of the populations of Hiroshima and Nagasaki were killed during WWII, respectively, and 80% of Japan's cities were not targeted. Second, the Plague did not destroy buildings and government assistance was non-existent in our context. Thus, we isolate the effects of mortality. Third, Davis and Weinstein explain that cities with strong defense capabilities, of historical significance, or with a specific topography, were relatively spared by the bombings whereas Plague virulence was apparently exogenous. Fourth, their results imply that locational fundamentals explain urban recovery but they do not interact the bombings with the geographical characteristics of the cities to identify which locational fundamentals mattered for urban recovery and permutations. However, we do not have the richness of their population and industry data and in our context increasing returns must have been weaker than in 20th century Japan, making our results less relevant for richer countries.

We also add to the literature on the economics of pandemics. Most studies of their economic consequences use macroeconomic approaches (Young, 2005; Weil, 2010; Voigtländer and Voth, 2013b,a), notable exceptions being Almond (2006), Donaldson and Keniston (2016) and Beach et al. (2018) who study the 1918 influenza pandemic.[4] Likewise, there is a nascent literature on the effects of the West African Ebola epidemic (e.g., Bowles et al., 2016). However, this disease has killed only about 10,000 people. The currently spreading Coronavirus has killed only 2,000 people so far.[5] An uncontrolled

[4]There is also a recent literature on the effects of plague recurrences in Europe (Siuda and Sunde, 2017; Dittmar and Meisenzahl, 2019). However, these events were on average much less deadly than the Black Death and only affected a few cities at a time (Aberth, 2010, p.37).

[5]As of 2/17/2020. See https://www.worldometers.info/coronavirus/.

pandemic could, however, have dramatic localized economic effects (e.g. UNECA, 2015; Gates, 2018), but it is difficult to forecast which consequences without more evidence.[6]

Finally, in contrast to previous studies on the Black Death, we: (i) Provide econometric evidence that the Black Death had strong causal localized effects in the short-run, and, on average, no effects in the long-run. But there were stark permutations in the ranking of cities.[7] (ii) Our analysis uses city-level data on Black Death mortality and focuses on cities, which are not the focus of the macroeconomic and historical literatures (Galor and Weil, 2000; Galor, 2005; Clark, 2007; Ashraf and Galor, 2011; Galor, 2011; Voigtländer and Voth, 2013b,a).[8] In the standard Malthusian model cities proxy for productivity growth in the countryside.[9] However, cities were important centers of trade and production. In addition, while the macro-historical literature has highlighted the role of natural increase in aggregate population recovery, we document how migration disproportionately mattered for local urban recovery. Finally, understanding how the Black Death interacted with natural or sunk man-made advantages improves our understanding of how, in a Malthusian economy, shocks can lead to a potentially beneficial spatial reallocation of economic activity.

1. Data

This section presents our data (see Web Appendix Sections 1.-3. for more details and Web Appendix Table A.1 for summary statistics of the main variables used).

Black Death Mortality. Data on cumulative Black Death mortality for the period 1347-1352 come from Christakos et al. (2005, 117-122) who compile data on 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

[6]Understanding the effects of pandemics is important since their frequency and severity will increase with climate change (BBC, 2017). Bill Gates (2018) argues that "given the continual emergence of new pathogens, the increasing risk of a bioterror attack, and the ever-increasing connectedness of our world, there is a significant probability that a large and lethal modern-day pandemic will occur in our lifetime".

[7]In a related paper, Jebwab, Johnson and Koyama (2018) investigate the effects of the Black Death on the persecution of Jews. They show that higher-mortality cities persecuted Jewish communities less and that Jews were relatively skilled. Given this result, we will show that results hold when controlling for the occurrence of a persecution. The working paper of the current manuscript predates Jebwab et al. (2018).

[8]Contributions to the historical literature includes: Ziegler (1969); Gottfried (1983); Benedictow (2005); Borsch (2005); Pamuk (2007); Clark (2016); Campbell (2016); Alfani and Murphy (2017).

[9]However, Voigtländer and Voth (2013b) study the urban sector from a macroeconomic viewpoint. Boerner and Severgnini (2014) study the determinants of medieval trade using data on Plague diffusion.

records, hospital records, cemeteries and tombstones. Christakos et al. (2005) carefully 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). Details on data construction and which estimates were selected by Christakos et al. (2005) are provided in Web Appx. Section 1.. These data yield mortality estimates for 274 localities.

For 177 of these we have a percentage estimate of the mortality rate. For example, Venice had a mortality rate of 60%. 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 mortality rate.[10] (ii) For 19 cities we know the mortality rate of the clergy. Christakos et al. (2005) show that clergy mortality was 8% higher than general mortality, so we divide the clergy mortality rates by 1.08.[11] (iii) For 29 cities we know the desertion rate, which includes both people who died and people who never came back. 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.

Cities. Our main source of city population data is the Bairoch (1988) dataset. This source reports population estimates for 1,726 cities between 800 and 1850. Observations are provided for every century up to 1700 and then for each fifty year interval up to 1850. The criterion for inclusion in the Bairoch dataset is a city population greater than 1,000 inhabitants at any point between 800 and 1850. We update the Bairoch dataset where historians – Chandler (1974, 1987), Nicholas (1997), and Campbell (2008) – have revised estimates of the population of particular cities. We also add 76 cities that are mentioned as cities in Christakos et al. (2005) but not in Bairoch (1988). In the end, we obtain 1,801 cities and focus on the period 1100-1850.[12]

Sample. Our main sample consists of 165 cities existing in 1300 and for which we know the Black Death mortality rate. The cities comprise 60% of the total urban population of Western Europe in 1300. We map these along with their mortality rates in Figure 1.

[10] 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 "close to being depopulated"/"decimated".

[11] Clergymen were the only exception to our general statement that the Black Death did not target specific populations. Clergymen, however, only comprised a tiny share of the urban population (often just a few individuals) so this should not matter overall.

[12] Details on the choices we make are confined to Web Appendix Section 2..

Controls. 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 from a coast or river, and longitude and latitude. To proxy for *agglomeration effects*, 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 $MA_i = \Sigma_j \frac{L_j}{\tau_{ij}^{\sigma}}$, 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 data from Boerner and Severgnini (2014) who estimate the speed at which the Plague traveled via each mode of transportation. To proxy for *sunk investments*, we control for the presence of major and minor Roman roads (and their intersections) using the data from McCormick et al. (2013), medieval trade routes (and their intersections) after digitizing a map from Shepherd (1923), and dummies capturing the presence of medieval market fairs, membership in the Hanseatic league (Dollinger, 1970), whether a city possessed a university (Bosker et al., 2013), and whether a city was within 10 km of a Roman aqueduct (Talbert, ed, 2000). To control for *institutions*, we distinguish between cities that were located in monarchies, self-governing cities, or whether the city was a state capital around 1300 (Bosker et al., 2013; Stasavage, 2014). We measure parliamentary activity during the 14th century using data from van Zanden et al. (2012) and control for whether a city was within 100 km of a battle in 1300-1350.

2. Historical Setting

2.1. The Epidemiological Shock

The Black Death arrived in Europe in October 1347. Over the next few years it spread across the continent killing between 30% and 50% of the population.[13] Recent discoveries in plague pits have corroborated the hypothesis that the Black Death was Bubonic plague. 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 sate themselves and continue to bite rats or humans, regurgitating the bacterium into

[13]Conventionally, the death rate was estimated at 1/3. Recent studies suggest higher rates (see Benedictow, 2005, 2010; Aberth, 2010). For the 165 cities, the population-weighted average is 38.8%.

the bite wound thereby infecting rats or humans. Within less than a week, the bacteria is transmitted from the bite to the lymph nodes causing them to swell painfully as they become buboes. Once infected, death occurred within ten days with 70% probability.[14]

While the vector for bubonic plague is infected fleas, they cannot spread the disease far in the absence of hosts such as rats. The spread of the Plague was rapid and its trajectory was largely determined by chance. For example, one important means of transmission depended on which ships became inhabited with infected fleas. It was largely coincidence that the Plague spread first from Kaffa in the Black Sea to Messina in Sicily in October 1347 as the ships carrying the Plague were originally bound to Genoa (but the ships could have been bound to other ports). Figure 1 shows the locations of Messina and Kaffa. Similarly, it was coincidental that the Plague spread from Messina to Marseille rather than to, say, Barcelona, Lisbon, or Antwerp. From the various coasts where infected ships docked the Plague then spread inland along rivers and roads.[15]

Virulence was greater in cities affected earlier (Christakos et al., 2005, 212-213). Initially, epidemics spread exponentially and then, as they run out of victims, the disease mutates in favor of benign pathogens.[16] People also develop immunities and pathogen mutation 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). Thus, pathogen mutation and natural immunization eventually cause an epidemic to end.

The Black Death was at its most virulent during the summer months (Benedictow, 2005, 233-235). Fleas become most active when it is warm and humid (Gottfried, 1983, 9). Christakos et al. (2005, 230) notes that Black Death 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

[14]See Benedictow (2005, 2010). The importance of blocked fleas as the main vector of transmission is under debate. Other vectors (e.g., lice) may also have been at work. The literature agrees, however, that person-to-person transmission was rare and cannot account for Plague diffusion (Campbell, 2016, 235).

[15]The local spread of the Plague thus also depended on the local populations of black rats. Since black rats are territorial, 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).

[16]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, the spread of the infection reduces the pool of susceptible hosts [...] This may favor benign pathogens [...]."

for 61 out of the 165 towns, the average duration of the Black Death was 7 months (see Web Appx. Fig. A.1). According to Christakos et al. (2005, 212-213), mortality on average peaked 3.5 months after the first infection. Cities infected in late fall thus escaped relatively unscathed compared to cities infected in spring.

Therefore, the local virulence, of the Plague had a significant random component (see Web Appx. Section 4. for more 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). To illustrate, Venice had high mortality (60%) while Milan escaped comparatively unscathed (15%). Highly urbanized Sicily suffered heavily from the Plague. Equally urbanized Flanders, however, had low death rates. Southern Europe and the Mediterranean were hit especially hard, but so were the British Isles and Scandinavia.[17] Likewise, 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, making it impossible to reach any definite conclusion." Consistent with this, Figure 2(a) illustrates the lack of a relationship between mortality rates (1347-52) and city population in 1300 (Y = 42.5*** - 1.01 X; Obs. = 165;$R^2 = 0.00$). Likewise, Figure 2(b) shows that there is no relationship between mortality rates and city market access in 1300 (Y = 40.0*** -0.20 X; Obs. = 124; $R^2 = 0.00$).[18]

Subsequent outbreaks of bubonic plague reoccurred in Europe for two and a half centuries following the Black Death. 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 (see discussion in Web Appendix

[17]Variation in sanitation does not explain this pattern. Gottfried (1983, 69) notes that this explanation does not hold given the "failure of Venice's excellent sanitation to stem the deadly effect of the plague".

[18]Random measurement error in dependent variables, here mortality, does not lead to bias, only to higher standard error. Random measurement error in market access then produces a downward bias. However, we use an elasticity that is now standard (e.g., Donaldson, 2018). In addition, Web Appx. Table A.3 shows no correlation when market access is computed using a lower elasticity – 2 or 1 – or when relying on Euclidean distance in case the speed parameters are misestimated Lastly, we will show that there is no positive significant effect of other measures of transportation and trade networks. Similarly, some scholars have argued that death rates were lower in mountainous regions, but mortality rates in mountainous Savoy were high whereas "despite Switzerland having the most rugged terrain in Europe, the Black Death reached almost every inhabited region of the country" (Christakos et al., 2005, 150). We also find no relationship with the density of population within walled cities (see Web Appx. Figure A.4).

Section 4.). These plague recurrences were caused either by local plague reservoirs or the repeated 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, 37). Europe-wide studies of later outbreaks focus on the extensive margin of the plague and not on the intensive margin.

Finally, the Black Death affected all segments of the population, rulers and commoners, rich and poor, adults and children, men and women. Neither the medical profession nor authorities were able to respond effectively. Medical knowledge was rudimentary: Boccaccio (2005, 1371) wrote that "all the advice of physicians and all the power of medicine were profitless and unavailing". Individuals, regardless of wealth, were unable to protect themselves. Institutional measures of prevention were nonexistent: the practice of quarantine was not employed until 20 years later.[19]

The Black Death represents as close to a *pure* population shock as we are aware of in the literature. Death rates were similar across social, age, gender, ethnic or skill groups (see Web Appendix Section 5. for qualitative evidence). The available evidence suggests that the Black Death was "massive and indiscriminate, making no exception to factors such as personal hygiene, health, age, sex, or social class" (Christakos et al., 2005, 150). This is supported by recent studies of plague outbreaks (Alfani and Bonetti, 2018). Christakos et al. (2005, 150) note that the claim that the poor died more than the rich is a plausible sounding statement "rather than an independent scientific conclusion derived from the analysis of real Black Death data". For contrary evidence, see DeWitte and Wood (2008). However, they study data for one cemetery only. Thus, even if the Black Death had *indirect* longer run effects on human capital, possibly due to the economy changing in its aftermath (see Donaldson and Keniston (2016) for an example of how fertility, health and education responded in the aftermath of the 1918 Influenza in India), the shock we study did not *directly* impact human capital.

2.2. Economic Consequences of the Black Death

The Black Death caused immediate economic damage. In rural areas, harvests went uncollected. In cities, trade was disrupted. As local economies collapsed, there were food shortages and inflation. Campbell (2016, 355) notes that "the simultaneous shock that plague then inflicted upon the supply of labour and the demand for goods and

[19]The term quarantine was first used in Ragusa in Sicily in 1377 (Gensini et al., 2004).

services set in train an immediate and enduring contraction in economic activity". Nominal wages increased immediately after the shock. Real wages, however, rose only gradually due to inflation (Munro, 2003). In England, for example, it was not until the 1380s that real wages reached their pre-Plague levels.[20] In the long run wages did increase but this rise was gradual—peaking in many parts of Europe in the mid-15th century before declining as a result of population growth after 1500 (see Web Appendix Section 6. for more qualitative evidence on wage patterns after the Black Death).

The demographic recovery was slow. Figure 3(a) presents estimates of total population (source: McEvedy and Jones (1978)) and urbanization for the 16 countries which contain our cities. Europe regained its pre-Plague population by 1600. Urbanization, here defined as the population share of localities above 1,000 inhabitants, rose in the aftermath of the Black Death from 8% to 11% by 1400, and 13% in 1600. This was due to urban areas recovering faster than rural areas (see Figure 3(b)).

3. Main Results: Black Death Shock and City Recovery

We estimate a series of city-level regressions based on:

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

where $\%\Delta \text{Pop}_{i,t}$ is the percentage population growth (%) in city i over period t-1 to t, and $\text{Mort}_{i,1347-52}$ is the city-level cumulative mortality rate (%) between 1347 and 1352. We weight observations by their initial population size in year t-1 to minimize issues arising from smaller cities mechanically experiencing larger percentage changes.[21]

3.1. Short-Run and Long-Run Effects

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 1347-1352 which is -1.00 by construction. The fact that the coefficient is not significantly different from -1.00 suggests little recovery in population in the decades directly following the onset of the Plague. In terms of magnitude, the effect is large: a one standard deviation increase

[20] Landlords complained about scarcity of labor and workers demanding higher wages. This lag is partly due to legislation after the Black Death to restrict wage increases (Cohn, 2007, 481).

[21] For example, the growth rate of a city of 1,000 in t-1 and 5,000 in t is 400%. Larger cities rarely experience growth rates as high. While this is a standard issue when using percentage growth-based outcomes, we choose this as our main specification because the interpretation of the coefficient is straightforward. We will show later that results hold with alternative specifications.

in mortality is associated with a 0.31 standard deviation decrease in population growth.

The effect in 1300-1500 is negative (-0.28, col. (2)) but smaller in size compared to the effect in 1300-1400 and significantly different from -1. Columns (3)-(5) examine the cumulative effect up to 1750. The coefficient increases to 0.36, 0.47 and 0.85 by 1600, 1700 and 1750 respectively. However, the magnitudes are small: A one standard deviation increase in mortality is associated with a 0.02-0.03 standard deviation increase in population growth, implying total recovery. The effects are also not significant.

3.2. Investigating Causality

Our discussion in Section 2.1. suggests that the intensity of the Plague was not well explained by characteristics of the cities affected. We now provide further evidence that the impact of the Plague was plausibly exogenous to other factors affecting city size.

Biases that Matter. For the short-run a downward bias is more problematic than an upward bias as we then overestimate the effect of the Plague (the true effect in 1300-1400 must be higher than -0.87). The short-run effect could be downward biased if cities that were inherently growing slower (faster) were also affected by higher (lower) mortality rates. For the long-run, an upward bias is more problematic as we then overestimate how fast cities recover (the true effect in 1300-1600 is lower than 0.36).

Correlates of Mortality. In Table 2 we show that mortality rates were uncorrelated with city characteristics that could also have caused future city growth. We define city level characteristics according to whether they proxy for locational fundamentals (1), increasing returns (2) (i.e. agglomeration effects or sunk investments) or institutions (3). The only variables that have explanatory power are proximity to rivers and latitude (column (1)). Proximity to rivers is negatively correlated with mortality, which is inconsistent with the claim that trade routes were correlated with Plague virulence. Other measures of transportation and trade networks do not have economically or statistically significant effects. 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.[22]

Controls. If larger cities had higher mortality rates due to higher densities propagating

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

the disease, and if larger cities were larger due to the presence of sectors driving future growth, this would be a source of upward bias. Likewise, if being on a trade route was positively correlated with both mortality and a city's growth potential, this would lead to an upward bias. If anything, an upward bias makes us under-estimate the short-term negative effect of the plague, which is inconsequential for our analysis. In row 2 of Table 3, we show 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 4.4. 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.

Spatial Fixed Effects. Results hold if we employ 13 modern country fixed effects (row 3), to control for regionally correlated unobservables. Modern country borders differ from the political units of the fourteenth century so in row 4 we assign a separate dummy variable to each of the independent polities with at least 5 cities in our dataset.[23]

Outliers. In row 3, we drop towns with the 5% highest and the 5% lowest mortality rates to ensure that our results are not driven by outliers. In general, no community was prepared to deal with the Black Death. It was attributed to the "vengeance of God" or the "'conjunction of certain stars and planets" (Horrox, ed, 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 (row 4).

Parallel Trends Columns (6)-(7) of Table 1 show that prior to 1300, there is no difference in growth between cities most affected and those comparatively unaffected by the Plague. As can be seen in the lower sample sizes, many cities did not exist (i.e. had populations below 1,000) in 1100 and 1200. Since columns (6)-(7) examine the intensive margin of city growth, we show in columns (8)-(9) that the likelihood of being above 1,000 by 1200 or 1300 is also not correlated with mortality.[24]

Panel. We restrict the sample to the 165 cities, focusing on the years 1100, 1200, 1300, 1400, 1500, 1600, 1700 and 1750. We run a panel regression where the dependent variable is the percentage change in population between t-1 and t (1100 is dropped),

[23] We use the information on borders from Nussli (2011). This gives us 44 independent states. The sheer number of states raises a potential problem as many were small principalities with only a single major city. Hence we use fixed effects for 7 larger states (i.e. with at least 5 cities).

[24] Likewise, city population in 1200 and 1300 is not correlated with mortality among cities that passed the 1,000 threshold in 1100-1200 and 1200-1300, respectively (not shown, but available upon request).

where city fixed effects and year fixed effects are included, and where the variables of interest are Black Death mortality in 1347-1352 interacted with the year effects (1750 is the omitted year). We use as weights city population in t-1 and cluster standard errors at the city level. Figure 4 shows: (i) The interacted effects ("Panel"); (ii) The corresponding effects when running the cross-sectional regression for each year one by one ("Non-Panel"). For the period 1400-1750, these differ from col. (2)-(5) in Table 1 which showed long-difference effects with 1300 as the start year; and (iii) The panel effects when the dependent variable is the change in log population size between t-1 and t ("Panel w/ Log"). The negative effects in 1300-1400 ("1400"; -0.87***, -0.91*** and -0.94***, respectively) are offset by positive effects in 1400-1500 ("1500") and 1500-1600 ("1600"). Results are very similar across the three specifications (coefficients shown in Web Appx. Table A.5), implying city effects do not matter.

Note that these results hold if we control for log population size in t-1 (not shown, but available upon request). This is unsurprising as mortality was uncorrelated with initial population size (Figure 2(a)). As initial population is a "bad" control in a panel regression due to dynamic panel bias (Nickell, 1981), we ignore these results.

Given how insensitive our results are to the specification used and the inclusion of city effects, we use long-difference regressions for the rest of the analysis. Furthermore, the IV strategies developed below do not suit a panel framework. Indeed, the instruments would need to be interacted with the year fixed effects, thus creating multiple first-stage regressions and generating multiple weak instruments.[25]

Correlated Shocks. The plague reoccurred following the Black Death. This could be a potential source of bias if subsequent plague outbreaks were correlated with the initial pandemic. In row 7 we use data from Biraben (1975) and show results hold if we include a dummy for plague recurrence and the number of recurrences within 50 km of the city in the period of interest 1353-1400 or 1353-1600, respectively.[26]

The immediate impact of the Black Death reduced the intensity and scale of conflict (in the Hundred Years War, for example, as documented by Sumpton (1999)). However, as Voigtländer and Voth (2013a) and Dincecco and Onorato (2017) argue, warfare

[25]For example, when the endogenous variable is mortality*1400, only instrument*1400 should be relevant and instrument*1200, instrument*1300, etc. should be mechanically less relevant.

[26]Subsequent plagues were not correlated with mortality (see Web Appendix Table A.4). These results on plague recurrences are also robust to using other distance thresholds (not shown). Later recurrences had a different epidemiology to the initial outbreak (see Web Appendix Section 4.).

ultimately intensified and contributed to increases in urbanization in late medieval and early modern Europe. Row 8 shows that results hold if we include a dummy for battle occurrence and the number of battles in the period of interest 1353-1400 or 1353-1600.[27] Results also hold if we control for the number of famines experienced by the region or the country of the city in 1300-1400 or 1300-1600 (row 9).

Jebwab et al. (2018) show that lower-mortality cities were more likely to persecute their Jewish community, which then impacted their growth. Our results hold if we add three dummies for whether the city had a Jewish community, a persecution took place, and the persecution took the form of a pogrom during the Black Death (row 11). In row 12, we show results hold if we drop any city with a persecution. In addition, Jewish populations were very small relative to the non-Jewish city populations. As such, these pogroms were unlikely to affect human capital in the short to medium terms.

In Table 4, we implement three IV strategies: IV1, IV2 and IV3. The first two strategies rely on the date of first infection in the city, which is only available for 124 cities.[28] Also, since the three IV strategies rely on the spatial diffusion of the Plague, we cluster standard errors at the state (1300) level (N = 64) for these analyses.

IV1. Our first IV exploits the fact that there was a lot of randomness in the local patterns of the Plague depending on where infected rats and fleas went. We exploit this exogenous variation in mortality by creating a variable for date of first infection for a city. Figure 5(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. Using the number of months since October 1347 as an IV, and adding the controls of Table 2 (incl. longitude and latitude) and the squares and cubes of longitude and latitude to exploit the more random component of the spread of the Plague, we find coefficients similar to our OLS estimates (row 2; IV-F stat = 11.8).

IV2. Our second IV uses the variation in mortality generated by differences in the *month* of first infection *within* a single year. Figure 5(b) suggests that this IV has explanatory power. For 124 cities of the main sample for which we have data on the onset of the Black Death, it shows the relationship between mortality rates and the month of peak infection in the city (= month of onset + 3.5 months). The Black Death was more

[27] The respective number of soldiers involved, and whether the cities were burned or sacked, may be a better proxy for conflict intensity. Our results hold if we use these controls (not shown).

[28] See Web Appx. Table A.6 for the full first-stage regressions for IV1, IV2 and IV3.

virulent when peak mortality in the city occurred during summer (6-8) (the quadratic fit omits January (1), which has abnormally high mortality rates due to October being the month of onset of the Black Death in Europe). We report results using our second IV, eleven dummies for the month of peak infection (since we exclude cities first infected in October), while simultaneously adding the controls used for IV1 and dummies for the year of first infection to control for the fact that cities infected in earlier years had higher mortality. We obtain similar results to the OLS results (row 3; IV-F stat = 7.1).

IV3. Proximity to Messina should predict Plague mortality, since the Plague was more virulent initially. We use as an IV the log Euclidean distance to Messina, conditional on average log Euclidean distance to all cities. Controlling for average distance to all cities captures the fact that some cities were better connected overall. Hence, we exploit the fact that it was the specific connectedness to Messina, and not connectedness overall, that mattered for mortality.[29] We report estimates using this instrument in row 4 (we add the same controls as for IV1 and Messina is dropped from the regressions; IV F-stat = 23.0). The short-run coefficients are similar to our OLS estimates. However, the long-run effect becomes large and negative, but not significant. Given that the IV is identifying a LATE, one possibility is that it is being driven by the economic collapse of Sicily in the early modern period. Indeed, the IVs give more weight to the LATE compliers, here the cities close to Messina. Row 5 shows the 1300-1600 effect becomes half as negative when Sicily is dropped. Results also hold when using IV1 and IV3 simultaneously (row 6). We do not use IV2 because of the lower IV F-stat.[30]

According to the latest scientific research the Black Death was transmitted by several vectors. The most important of these were the fleas of the black rat.

Summary. The identification strategies return effects that are not significantly different from our baseline OLS effects, suggesting that the impact of the Black Death was exogenous. In the rest of the analysis we will employ OLS estimates as our baseline.

3.3. Robustness Checks

We now show that our results are robust to potential concerns about specification, data measurement, and sampling issues. Row 1 of Table 5 reports our baseline estimates (for

[29]The correlation between the two distance measures is indeed much lower than 1, at 0.09.

[30]We also do not exploit the distribution of rats. The population of black rats declined after the introduction of the brown rat in the 18th century (Christakos et al., 2005), which coincided with the end of plague reoccurrences. Little is known about the historical geographical distribution of black rats.

both the short-run effect in column (1) and the long-run effect in column (2)).

Specification and Standard Errors. Results are unchanged when: (i) we control for past population trends, i.e. city population growth in 1200-1300 (row 2); (ii) use as the dependent variable the absolute change in population and as the variable of interest the number of deaths (row 3); and (iii) cluster standard errors at the state (1300) level (row 4) or employ Conley (2008) standard errors with a radius of 500 km (row 5).

Measurement Concerns. Classical measurement error in mortality should bias the short-run estimates towards zero. In that case, our effect is less negative than the true effect, which is less of an issue than a downward bias. Additionally, if measurement error is classical, the long-term effect should be as biased as the short-term effect, which would not change our results. However, measurement error could be non-classical, hence the need to examine how the effects vary depending on the sources of mortality data. Our estimates do not systematically differ when: we (i) include dummies for different sources of mortality data (row 6);[31] (ii) drop estimates based on literary descriptions (row 7); (iii) drop estimates based on desertion rates (row 8); (iv) drop estimates based on clergy mortality (row 9); and (v) only use the raw numerical mortality estimates directly provided by Christakos et al. (2005) (row 10), thus omitting the 25 description-based mortality estimates. Among these, "high" (assigned 50%) appears 15 times, "spared" (5%) 3 times, and the other seven descriptions only once. We verify that results hold if we use a different rate for "high" (e.g., 60 or 40%) and spared (e.g., 10 or 0%), or drop each description one by one (not shown, but available upon request). Finally, in row 11 we 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 relatively similar estimated rates.

Classical measurement error in the dependent variable should increase standard errors. However, our results remain precise. We can also employ alternative population estimates. Row 9 reports estimates using the uncorrected Bairoch data. Row 10 reports results using only observations from the Chandler (1974, 1987) dataset. Results hold.

External Validity. There could be concerns about sample size. We employ as regression weights populations in 1300 ensuring less weight is placed on small towns. Given that our 165 cities capture 60% of the total population of the 466 existing cities in 1300 (see

[31] These are: population records, literary descriptions, desertion rates, and clergy morality.

Web Appx. Fig. A.5 for their respective locations), having more cities should not change the results. We also use several methods to obtain mortality estimates for more cities. Results hold if we use: (i) the mortality of the closest city with data if this city is within 50 km (row 11, N = 290–286); (ii) the average mortality of the cities in the same state (row 12, 380–274, SEs clustered at the state level); and (iii) estimates of mortality based on spatial extrapolation (row 13, 464–457).[32] Lastly, we study for all 466 cities in 1300 the reduced-form effect of the inverse of log distance to Messina, conditional on average log distance to all cities and the controls used for IV3. The short-term effect remains negative and significant (not shown, but available upon request).

Our sample is biased towards large cities. Kernel distributions of 1300 populations and Kolmogorov-Smirnov tests confirm that. In row 14 we reweight observations to match the distribution of city populations in 1300.[33] Initial population size is not the only characteristic in which our sample is selected. For the 466 cities, we regress a dummy equal to one if mortality data is available on the controls of Table 2. We find significant differences for temperature, monarchy, capital cities, and representative cities. If we reweight observations to match the distribution of each characteristic one by one, results are unchanged (not shown, but available upon request).

Finally, we drop cities located within France, Germany, Italy, the United Kingdom or Spain (Web Appx. Table A.7). Other countries contribute few cities.

4. Mechanisms and Heterogenous Recovery

We now show that: (a) urban systems and rural areas close to cities relatively recovered to their pre-Plague population levels; (b) migration drove local recovery; (c) urban recovery is explained by the interacted effects of mortality with city characteristics that proxy for fixed factors of production; and (d) aggregate urban recovery hides permutations in the distribution of cities which are explained by fixed factors.

[32] We create a two-dimensional surface of predicted plague mortality using an inverse distance weighted function of known mortality rates for the full sample of 274 cities with mortality data. For every point on the surface a predicted mortality rate is then generated using the closest 15 cities within an approximately 1,000 km radius circle around the point. Details can be found in Web Appendix Section 1..

[33] We divide the cities into bins of 1,000 inhabitants and estimate the share of each bin in our sample and in the full sample. We then correct our weights so that our distribution does not significantly differ from the full distribution, which we then confirm using Kolmogorov-Smirnov tests.

4.1. Effects on Urban Systems

Spillovers. Cities could be affected by mortality in nearby towns. Web Appx. Table A.8 examines the effects of a city's own mortality rate and the population-weighted average mortality of neighboring towns, defined in four ways:[34] (i) of the same state (1300); (ii) of the same country (2019); (iii) within the bottom 10% of distance to the city; and (iv) or using all 1,801 cities but relying on the total change in market access between 1300 and 1353.[35] Cities that experienced a large direct shock did not always experience a large indirect shock: The correlation between city mortality and indirect mortality is about 0.43-0.51. The indirect effect is always negative, but not significant. The combined direct and indirect effects are about -1.00 to -1.25, and significant. By contrast, the long-run direct and spillover effects are not significantly different from 0 (while some coefficients appear large, the beta coefficients are actually very small).

Aggregate Effects. There may also have been effects at the state level. In columns (1)-(5) of Table 6, we estimate the effects of population-weighted average mortality at the state/country level on the percentage change in urban population at the state/country level.[36] Column (1) of Panel A replicates our baseline short-run effect (1300-1400) at the city level (-0.87***). Columns (2) and (4) show the effects at the state and country levels, for cities that existed in 1300. The effect is now more negative and close to -1.15.

In columns (3) and (5), we examine the state- and country-level 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, at -1.27 to -1.47 implying that in high-mortality areas, fewer new cities emerged. We verify this in column (6)-(7) using 1,335 cities in the Bairoch data set that did not exist in 1300. These cities can be thought of as the universe of potential city locations. We test whether their emergence in 1400 — via a simple dummy — is related to the Black Death. Cities were less likely to emerge when their extrapolated mortality rate was high (col. 6). Likewise, we regress the log population of these 1,335 cities (using

[34]We include all 1,801 towns. We give 500 inhabitants to the towns with less than 1,000 in 1300.

[35]To construct market access in 1353, we use the predicted population of the other towns in 1353 (= pop. in 1300 x (100-mortality)/100). Since mortality is only available for 274 cities, we use spatially extrapolated mortality rates for 1,527 cities. For each of the 165 city-observations, the extrapolated mortality rates of the other towns are constructed excluding the mortality rate of the observation itself.

[36]As before, we include all 1,801 towns, and use spatially extrapolated mortality rates for towns without mortality data and population = 500 inhabitants for towns with population below 1,000. Note that when running regressions we lose 20 states and 1 country (Luxembourg) without any urban population in 1300.

500 for cities below 1,000) on mortality and find that fewer locations became urbanized in high-mortality areas (col. 7). Consistent with previous results, we find however that these negative effects of the Black Death disappeared by 1600 (Panel B).[37]

4.2. Effects on the Countryside

In the aftermath of the Black Death, contemporaries noted the depopulation of rural areas. Gottfried (1983, 135) observes that "However much plague depleted urban populations, there were always country folk ready to replace them. This migration to towns combined with the effects of disease in rural areas caused a pronounced shortage of agricultural workers". The Black Death thus led to massive reforestation as the need for land and wood dramatically declined and marginal soils were abandoned (Campbell, 2016, 363) (see Web Appx. Sections 9.-10. for qualitative evidence). While urban populations recovered by the 16th century, rural population recovered in the aggregate only by 1600 (see Figure 3(b)). Unfortunately, we do not have localized data on rural populations. Land use data, however, provides a proxy for rural population.

Land Use. Kaplan et al. (2009) recreates localized data on land use from 1100 to 1850 at the 5 by 5 minute (10 x 10 km) grid-cell level by combining information on country population, historical forest cover maps, and soil suitability.[38] Using these data, we obtain the mean land use share (%) of the 16 countries in our sample. The share reached two thirds by 1300 and decreased by 15 percentage points by 1400. Land use then did not fully recover until 1800. The delayed recovery of land use may reflect the employment of more capital per worker and the switch to pastoral farming. We obtain the mean land share within a 10 km radius of each of the 165 cities and examine how land use varied.[39] Web Appx. Table A.9 shows that mortality led to reforestation in 1300-1400, which remained significant until 1500. No effect is found after 1600.[40]

[37] Some coefficients are still negative, in particular for the analysis at the state/country level (cols. (2)-(5)). However, these effects are relatively small once standardized, with beta coefficients equal to -0.05/-0.11 for states (vs. -0.35/-0.40 in 1300-1400) and -0.05/-0.06 for countries (vs. -0.23/-0.26 in 1300-1400).

[38] Other sources have used data on tree rings or fossil pollen for plants used by humans, but only for selected regions (e.g. van Hoof et al., 2006). As such, we cannot use this data for our analysis.

[39] Since country populations are one input used in the creation of these data, we verify that land use changes are not mechanically correlated with population changes. The correlation between the percentage change in land use within 10 km of a city and the percentage change in the population of that city is 0.14 in 1300-1400 and -0.03 in 1300-1600. The correlations with the percentage change of that city's country population are higher, but still low, at -0.30 and 0.44 respectively. Additionally, we control for the contemporaneous changes in both city population and country population in all regressions.

[40] We test for parallel trends and results hold if we cluster observations at the country level (not shown).

Timing. Overall, cities recovered their populations by the 16th century and their rural areas recovered theirs by 1600. Obviously, we expect cities and their rural areas to recover simultaneously: (i) As land is a fixed factor, rural wages increase when rural population declines, which, combined with non-homothetic preferences, increases the demand for urban goods, thus attracting people to cities; and (ii) Fixed factors in cities means that urban wages increase when urban population declines, thus raising the demand for food and attracting people to villages. However, cities recovered slightly before land use, suggesting that either peasants were initially replaced by technology or livestock or that urban fixed factors played an important role in urban recovery.

If both cities and their immediate hinterlands recovered by 1500-1600, population 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.[41] Thus, marginal rural areas suffered relatively greater population losses following the Black Death. Indeed, while land use recovered locally by 1600, it did not recover in the aggregate before the 18th century. Accordingly, using grid cell-level data on cereal suitability, cereal suitability decreases with distance to existing cities in 1300 (not shown).[42]

Now, was the relative decline of these marginal rural areas correlated with their own mortality rate? If fixed factors increase demand for labor in high-mortality areas, it could well be that marginal areas are more depopulated in low-mortality, not high-mortality, areas. We address this using data on the abandonment of settlements in England.

Deserted Medieval Villages (DMVs). Historians document how the Black Death led to the desertion of villages in England (Beresford, 1954), France, and Germany (Braudel, ed, 1965; Pounds, 1974).[43] In the longer run, since labor was in short supply and peasants demanded better pay, many landowners switched to sheep rearing, which required much less labor than arable farming. This gave rise to Thomas Moore's

[41] Of course, it could have been that people left areas close to low-mortality cities for areas close to high-mortality cities to be in turn replaced by migrants from rural areas farther away from cities.

[42] Pastoral suitability also decreases with distance to cities in 1300. Potato suitability does increase with distance from cities in 1300, which is not surprising since it did not matter before the introduction of the potato in the 16th century (results not shown, but available upon request).

[43] The Plague directly erased rural communities. Others declined gradually: "... a steady hemorrhage of labor, whether through migration or early deaths, caused rural settlement to fall back. Some of the smaller villages were lost in these circumstances, many more shrank dramatically..." (Platt, 1996, 16).

observation in the 16th century that sheep "devour men themselves". Peasants left their villages to seek newly available economic opportunities in high-mortality areas. This immigration "topped up otherwise diminishing urban communities" (Platt, 1996, 20). Immigrants had to come from somewhere and it was often the countryside that lost population (see Web Appx. Section 11. for qualitative evidence).

Data on the location of DMVs exist for all 41 English counties during the medieval period (Fenwick and Turner, 2015). For 28 of these counties, we know from Shrewsbury (1970) and Scott and Duncan (2001) the mortality rate of the clergy, which we use as a proxy for the overall mortality rate. This allows us to study how the number of deserted villages varied with Black Death mortality, depending on their proximity to cities. Since this sample is different from the main sample of 165 cities, we verify in columns (1)-(3) of Table 7 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 coefficient of -0.10 vs. -0.35 in 1290-1377). When using 1801 (England's first census), the standardized effect is small (-0.08, not shown). We then check parallel trends and find no effect in 1086-1290 (0.05, beta coeff. of 0.00, see col. (3)).[44]

In columns (4) - (6) we use the log number of DMVs per 1000 sq. km as the dependent variable. We control for the county's log area and log population in 1290 since the density of DMVs depends on pre-Plague human density.[45] We find a negative effect of mortality, at -0.46*** (col. (4), beta coefficient of -0.51).[46] Low-mortality areas had more DMVs than high-mortality areas. Therefore, people disproportionately left the relatively Plague-free rural areas. Web Appx. Table A.9 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-

[44]We weight observations by their population in 1290 and exclude Cornwall whose population in 1290 is severely underestimated due to the lack of reliable data on their large mining population (see Broadberry et al. (2010, , 14)). Middlesex is not included in the analysis, due to the lack of data on its mortality. If we impute overall mortality from other sources, results still hold (not shown but available upon request).

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

[46]We include Cornwall for this regression, 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).

mortality areas *beyond* 10 km from cities. For 39 counties, we obtain from Fenwick and Turner (2015) the precise location of each DMV in England and compute the minimal distance to an existing city in 1300. 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 then verify in columns (5)-(6) that the loss of villages is driven by areas farther away from cities.[47]

In column (7), we regress the absolute change in the urban share (%) on mortality (using our city-level data to estimate each county's urban population) and find a small and insignificant negative effect.[48] DMVs were small. Therefore, 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 allowed by migration from both urban and rural areas in low-mortality areas.

4.3. Wages and Natural Increase vs. Migration

Wages. As explained in Section 2.2., real wages eventually increased in high-mortality areas. Unfortunately, data on nominal wages and prices does not exist for enough cities during our period of study. For example, welfare ratios as collected by economic historians are only available for a very few cities, preventing us from conducting econometric analyses.[49] Instead, Web Appx. Section 6. provides qualitative evidence on wage patterns after the Black Death. Overall, evidence is too sparse to be able to explicitly compare selected high- and low-mortality cities over time. Yet, the historical literature has consistently found that in Western European localities where mortality was high living standards on average improved for both skilled and unskilled workers.

Natural Increase. The relative recovery of high-mortality areas could have been due to higher real wages there 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 may have reduced fertility (Hajnal, 1965; Voigtländer and Voth, 2013a). The EMP was

[47] Our analysis focuses on 28 counties because mortality is only available for 28 counties. We verify for the 41 counties that the number of DMVs per 1000 sq km is not correlated with a dummy for whether mortality is available. Likewise, results hold if we impute mortality from other sources (not shown).

[48] We exclude Cornwall from these regressions for reasons discussed above.

[49] See, for example, data sets inspired by the work of Robert Allen: http://www.iisg.nl/hpw/data.php.

stronger in Northern than in Southern Europe (Moor and Zanden, 2010; Dennison and Ogilvie, 2014). The EMP also functioned at the macro level, affecting social norms about the appropriate age of first marriage that were common across entire regions, rather than varying at the local level. Thus, natural increase likely only played a minor role in *local* recovery. Migration must have been the source, especially for cities (Section 2.2.).[50]

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 1,000) 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). 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 wage earners 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". Cities attracted migrants from the nearby countryside. The number of freeman admitted into York increased by 365% in the year of the Plague (Dobson, 1973, 17). However, as the countryside was often as hard hit as the cities by the Plague, population sometimes took decades to recover. London, for example, saw a "great concourse of aliens and denizens to the city and suburbs, now that the pestilence is stayed" but the city remained depopulated for years (Sloane, 2011).[51]

EMP. We test if the speed at which high-mortality cities relatively recover depends on whether the cities belong to a Northern Europe region or a region characterized by the EMP. We classify our cities into North/Strong EMP vs. South/Weak EMP, based on data from Dennison and Ogilvie (2014) on the average age at first marriage and the female celibacy rate (%) at the country or regional level (e.g., Southern France). For the 165 cities, we use the same specification as before but interact mortality with a North/Strong EMP dummy and test if the interacted is negative and significant. Indeed,

[50] See Web Appendix Section 7. for more qualitative evidence.

[51] Migration may have been a consequence of both an improved bargaining position of peasants *and* an increase in labor coercion in some areas forcing peasants to escape these. We discuss serfdom and how its decline was related to the Black Death in Web Appendix Section 8.. As discussed by Wolitzky and Acemoglu (2011) the effects of greater labor scarcity on the use of labor coercion are ambiguous.

if natural increase was important for local recovery, we should expect high-mortality cities in North/Strong EMP regions to recover relatively 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. A lack of any significant difference between North/Strong EMP and South/Weak EMP cities implies that natural increase was not significant in either Southern cities or cities where the EMP was weaker.

Web Appx. Table A.10 shows the effects of mortality, the North/Strong EMP dummy and their interaction for the long-run period 1300-1600. The North dummy is based on 9 Northern European countries or the same 9 countries except France. The Strong EMP dummy is equal to one for cities in countries or 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 North/Strong EMP cities did not recover relatively slower, since the coefficients are not negative and significant, thus suggesting that migration, and not natural increase, was indeed the main driver behind local urban recovery.[52]

4.4. Heterogeneity in Population Recovery

Permutations. Historical evidence suggests there was heterogeneity across cities in the response to the Black Death. Campbell (2016, 365) notes that "as demand subsided and markets shrank, towns competed with each other in an urban survival of the fittest. Those able to secure a new commercial niche — Milan, Antwerp, Geneva, Nuremberg and Lisbon — fared well and grew, but 'success' for most meant the avoidance of decline. London out-performed most other leading English towns and just about held its own, but only did so by claiming a greater share of the nation's trade and commerce, to the disadvantage of once prosperous regional capitals such as York".

Relatedly, for our sample of 165 cities, we regress the rank of each city in 1600 on its rank in 1300 and find a slope of 0.86***. Therefore, large cities tended to remain large cities and small cities tended to remain small cities after the Black Death. However, aggregate urban recovery hides *permutations* in that the R2 of the regression is 0.56. Figure 6 illustrates this, with many cities far from the regression line, having permanently declined or grown relative to other cities. After World War II, Hiroshima,

[52]The dummies are not correlated so they capture different aspects of the EMP. Results hold if we classify Austria and Switzerland as Southern or only consider French cities above Paris as Northern.

Nagasaki and other Japanese cities regained almost their exact pre-war rank. This would imply a slope coefficient close to 1.00 but a R2 also closer to 1.00.[53]

We now test whether these permutations were associated with the presence or absence of fixed factors. Indeed, when mortality is high and labor becomes scarce, if there are fixed factors complementary to labor, wages should increase, attracting people. We modify Equation 1 by interacting mortality ($\text{Mort}_{i,1347-52}$) with selected fixed factors (FixFact_i) while controlling for the fixed factors themselves and mortality:

$$\%\Delta\text{Pop}_{i,t} = \alpha + \beta_t \text{Mort}_{i,1347-52} + \text{Mort}_{i,1347-52} * \text{FixFact}_i \boldsymbol{\theta} + \text{FixFact}_i \boldsymbol{\xi} + \epsilon_{i,t} \quad (2)$$

For cities experiencing the *same* mortality shock, the vector $\boldsymbol{\theta}$ captures the differential recovery effects of each factor. Throughout, we focus on our main sample of 165 cities, for the period 1300-2015. From the variables in Table 2, we select those that proxy for: (i) *rural fixed factors*: the three agricultural suitability measures (cereal, potato, pastoral); and (ii) *urban fixed factors*: coastal and river dummies, Roman road or medieval land route intersections, and the Hanseatic League dummy.[54] While the coast and rivers are by construction "fixed", they lowered transportation costs. Hence, coastal and riverine cities were more likely to develop a trading sector. Roads and the Hanseatic league can be treated as fixed factors, since they represent past sunk investments in transportation and trade networks. Roman roads remained the basis of the road network in the medieval era (Dalgaard et al., 2018). Medieval trade routes reflected long-established trading linkages. The origins of the Hanseatic league go back to its establishment in Lübeck in 1159 and it rose to prominence in the century before the Black Death (Dollinger, 1970, xviii). Lastly, we include factors proxying for agglomeration effects and institutions. These variable factors include the log of the estimated population of the city in 1353 (= pop. in 1300 x (100-mortality)/100), since larger cities in the aftermath of the Black Death may have recovered faster due to agglomeration effects. We also include three dummies for whether the city was part of a monarchy, was a state capital,

[53] Examples of cities that permanently declined after the Black Death included L'Aquila, Almeria, Cordoba, Montpellier, York and Winchester. These were very important cities at the European level or in their respective country before the Black Death. They are not thought of "large" cities today. Conversely, some cities became important after the Black Death, such as Hamburg, Munich, Stockholm and Turin (see Web Appendix Section 12. for a lengthier discussion of some of these permutations).

[54] We classify fixed factors as "rural" or "urban" depending on their relative importance in the production function of the rural areas surrounding cities or of the city itself. Also, since we have only 165 cities, we do not add all 27 variables of Table 2 and their interactions with mortality.

and whether it had a representative body (ca. 1300).

Table 8 shows the 11 interacted effects, for 1300-1750 (col. (1)-(5)) and 1300-2015 (col. (6)). The 11 interacted effects being *simultaneously* included, they show the recovery effect associated with each factor *conditional* on the recovery effect associated with each other factor. With 165 observations and 23 variables, this makes our test particularly stringent. Note that we show the interacted effects for the period 1300-1400 because cities started recovering in 1353-1400. We then use 1300 as the start year instead of 1353 because we do not know the true population of each city in 1353. Finally, we predict that the recovery effects of the fixed factors increase with their economic value, which may exogenously change over time, for example with new technologies. As such, since the factors we consider are likely to affect nominal wages much more than housing and non-housing prices and amenities, especially as pre-modern construction technologies were not particularly advanced or distinct across European cities, we interpret these recovery effects as reflecting nominal wage changes. [55]

Rural Fixed Factors. The coefficients of cereal suitability becomes positive (but are not significant) after 1400 (col. (2)). However, the effect is meaningful since the beta coefficient (henceforth, "beta") reached 0.47 by 1600 and remained high after (0.17 in 2015). Potato suitability also helped cities recover from the 17th century onwards (col. (4)). Nunn and Qian (2011) indeed show that countries that were relatively more suitable for potato cultivation urbanized faster after potato cultivation diffused in Europe (the non-effects in col. (1)-(3) are reassuring). Note that their country-level effects appear in 1750, whereas our interacted effects with mortality appear in 1700 because we focus on the local level. Indeed, the local cultivation of the potato started in the late 16th century and became widespread in the late 17th century (Nunn and Qian, 2011, p.601-603). Our effect is still large, and significant, in 2015 (beta = 1.06).

Next, in high-mortality areas that were 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). We believe this is caused by higher wages due to labor shortages that created incentives

[55] One issue with 2015 is that the Bairoch data set stops in 1850. Cities have also grown dramatically since 1850, becoming multi-city agglomerations. We read the webpage of each city in Wikipedia and selected the population of the city itself rather than the population of the agglomeration. Results, however, hold if we use the agglomeration estimate or the mean of the two estimates (not shown).

for landlords to specialize in pastoral agriculture, thus further reducing the need for labor (Voigtländer and Voth, 2013a, p. 2255). This effect then diminishes after 1750. This may reflect the rise of proto-industry in rural areas, in particular textile production, which was associated with more rapid population growth (Mendels, 1972; Pfister, 1989). Indeed, wool was then the most common textile used in making clothing.

Urban Fixed Factors. The interacted effect for coastal proximity is one of the only two significant coefficients in 1300-1400 (col. (1) of Table 8), along with the interacted effect for the Hanseatic league, suggesting that these factors can help account for the rapid relative recovery of some cities in 1353-1400. 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. The coastal effect is strong throughout the period (beta = 0.99 in 1500, slowly decreasing to 0.33 in 2015). The Hanseatic league effect is strong (beta = 0.72) in 1300-1400 and significant until 1700 (col. (4)), by which time the Hanseatic league was in decline (Dollinger, 1970).

Rivers have positive and significant effects from the 17th century onwards (col. (4)). River transportation was important throughout the medieval period (Masschaele, 1993). But we find the interacted effects of mortality and rivers on city population to be much stronger after 1600. The effect in 1700 is fairly strong (beta = 0.40) and remained so up to the present day. This may reflect greater investment in riverine technologies and canals as documented for England by Bogart (2011). Similar improvements in riverine transport also occurred in 17th-18th century France and elsewhere in Europe (albeit on a small scale). In France, Colbert passed laws ensuring that all rivers had to be traversable by private towpath companies. Note that investment in canals often raised the value of being on a river as canals were often dug to connect two previously separate riverine systems (see Geiger (1994) for a broader discussion).

Next, we find that being at the intersection of two or more roads/trade routes has a positive, significant, and economically large effect in 1500 (col. (2), beta = 0.51). The effect weakens in later years, as alternative rail and road networks expanded.[56]

In rows 1-3 of Table 9, we examine whether specific coastal and riverine cities recovered faster. In our main analysis we consider cities within 10 km of the coast.

[56]The effects of road intersections are stronger for Roman roads than for Medieval roads (not shown). Indeed, Roman roads were often stone-paved, and thus durable. We then find insignificant negative or nil interacted effects for elevation and ruggedness (not shown, but available upon request).

However, cities located *directly* on the coast may have recovered faster than those several kilometers inland. A 10 km band also does not include some estuarine cities with a free connection to the open sea (e.g., Bordeaux). We replace the coast dummy and its interaction with mortality by three dummies for being located directly on the coast, for being within 50 km from the coast and its estuaries, and for being within 50 km of the coast and on a river, and their respective interactions with mortality (the river dummy is adjusted accordingly to exclude such cities). As seen in row 1, the effects are stronger for truly coastal or estuarine locations, which we call the "best" coastal locations.

Row 2 shows the results when we instead replace the coast dummy and its interaction with mortality with two dummies for being one of the best Mediterranean coastal locations and for being one of the best Atlantic locations.[57] We find strong recovery effects for the Mediterranean. Indeed, Mediterranean trade was particularly important around the time of the Black Death. We also observe significant recovery effects for Atlantic cities starting in the 17th century. This is consistent with Acemoglu et al. (2005) who show that the Atlantic trade led to Atlantic ports with good institutions over-taking Mediterranean ports starting around 1600. However, in their analysis, identification comes from comparing the growth of Atlantic and non-Atlantic cities over time, whereas in our case it comes from the triple interaction between mortality, the coastal locations considered optimal, *and* the timing of the boom in Atlantic trade. As such, it is reassuring that we find no recovery effect in the earlier centuries.

Lastly, in row 3, we interact the best coastal location dummy with dummies for whether the city belongs to a monarchy or not and add the interactions with mortality. The river dummy now includes only riverine cities that are not on the best coastal locations. We also create interactions with mortality and the monarchy dummy. We find that the best coastal cities and riverine cities recovered faster when they were part of a more unified state, possibly because they served a larger market since within-state tariffs tended to be lower than between-state tariffs.

Agglomeration Economies. The urban literature suggests that a larger population should increase productivity and wages, which may cause in-migration. Traditionally, the literature (e.g., Duranton and Puga, 2004) distinguishes economies of scale – in

[57] There are 15 and 19 such cities, respectively. Atlantic ports include, for example, Antwerp, Bordeaux, Hamburg, Lisbon and Plymouth. As such, it does not include Atlantic ports that were either too small in 1300 (e.g., Cherbourg and Liverpool) or for which mortality is unavailable (e.g., Bristol and La Rochelle).

production, market places, and consumption – and agglomeration economies strictly defined, which include: (i) *sharing* the gains from access to varied inputs, a deepening of the division of labor, and risk pooling across sectors; (ii) *matching*, for example between firms and suppliers and between employers and employees; and (iii) *learning*, i.e. the generation, accumulation or diffusion of knowledge. Table 8 shows no significant recovery effect for cities with a larger residual population in 1353.

In the right panel of Table 9, we further examine the role of agglomeration economies in urban recovery following the Black Death. Using equation 2 but adding log population in 1300 and its interaction with mortality, we find no effect (row 6). It is possible that cities that were larger before the Black Death had more durable structures and infrastructure by 1353 and thus recovered faster. The city's historical "reputation" may also have mattered. But we find no evidence for either of these effects. We also find no positive, significant recovery effect of estimated market access (row 7) or estimated state population size (row 8) in 1353. Taken together these results suggest that agglomeration economies were not a significant factor in the post-Black Death recovery.

Turning to the economies of scale mechanisms, we do not find positive significant effects when adding the following variables and their interaction with mortality: a dummy for guild presence before the Black Death (row 9; source: Ogilvie (2019)), a dummy for the presence of a market fair circa 1300 (row 10), and log walled area circa 1300 (controlling for log city population size circa 1300) (row 11). Guilds reflect whether there was an industry large enough to organize and hence can proxy for economies of scale in production. However, guilds also reflected the political power of craftsmen and artisans (Ogilvie, 2019). The presence of a market fair can proxy for economies of scale in market places. The extent of a city's walls may proxy for economies of scale in production, market places, and consumption. Due to warfare between European states, walled cities offered protections to craftsmen, merchants, and more broadly residents, which Dincecco and Onorato (2017) and others have labeled the "safe harbor" effect. As Weber (1958, p.77) put it, the Medieval city was a "fusion of fortress and market."

We also include a dummy if there was any bishopric or archbishopric circa 1300 (row 12) and if there was a university circa 1300 (row 13). Both of these measures can be thought of as potentially capturing economies of scale in human capital, and thus production, and institutional capacity. We only find a positive significant effect

for the presence of a (circa 1300) university in 1600. This is perhaps unsurprising as universities were largely training grounds for the church and the law and did not provide their graduates with commercial or engineering skills (see Miethke et al., eds, 2000).

Labor Mobility. Serfdom and other forms of labor coercion restricted the ability of peasants to migrate to cities. While serfdom disappeared in Western Europe following the Black Death, it remained in place in Central and Eastern Europe for several centuries. We use information on where serfdom persisted through to the end of the 18th century in order to classify cities as either within or outside the zone of labor coercion (Source: Web Appx. Section 8.). As can be seen in row 14, we find for the 16th and 17th centuries negative recovery effects in areas affected by serfdom.

Causality. The question is to what extent the interacted effects are causal. In all regressions presented here, we *simultaneously* control for mortality, the individual effects of the 11 city characteristics used for the interactions, and the 11 interactions. Reassuringly, the effects only turn significant when expected. In addition, most important effects of Table 8 remain strong and significant when including 13 modern country fixed effects (Web Appx. Table A.11). Identification then comes from comparing over time cities experiencing the same plausibly exogenous initial shock, having the same 11 characteristics, and belonging to the same political entity. With 165 observations, 23 variables and 13 fixed effects, this makes our test even more stringent.

Interpretation. In summary, we find few significant effects of population or institutions. Adding the absolute values of the beta coefficients for the different types of factors shown in Table 8, we find that fixed factors are particularly important in the first centuries after the Black Death and that their importance decreases over time (see Figure 7(a)). Adding the beta coefficients also helps taking into account substitution effects possibly due to the interacted effects capturing each other's effect. Other factors such as population and institutions are less important. However, one caveat is that local institutions likely changed as a result of the Black Death (Brenner, 1976; Acemoglu and Robinson, 2012). This is not the focus of this paper, however, and our test imposes that we study the effects of pre-, not post-, Plague institutions.[58]

Table 1 showed no long-term effects of the Black Death at the city level. In Table

[58]Note that we find similar patterns if we compare the average beta coefficients across the three sets of variables or also add the beta coefficients of the variables in the right panel of Table 9.

8, the significant negative effects of mortality once we control for the fixed factors and their interactions with mortality implies that any city without fixed factors would have possibly remained small (the point estimate becomes more negative but the beta coefficient remains similar across years, at -1.5 in 1300-1400 and -1.1 in 1300-2015). In other words, for cities without access to river networks or the coast or other fixed factors, a large Black Death shock would have had permanent, negative local effects.

Next, for our sample of 165 cities existing in 1300, we can predict which high-mortality cities would have relatively lost out by 1750 or 2015 had they not had fixed factors. First off, to use our model of Table 8 to predict the counterfactual population level of each city in 1750 or 2015 absent the recovery effects related to fixed factors, one must account for the fact that predicted percentage population growth in 1300-1750 or 1300-2015 must be left-censored at -100. We thus re-run the regression but with a Tobit model. The effects on the latent variable are almost the same as with the OLS model (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 percentage change in 1300-1750 – is strongly correlated with the actual 1750 rank of each city among the 165 cities (correlation of 0.79). For 2015, the correlation is weaker – at 0.61 – due to factors that appeared in the Industrial Era. We then replicate the same analysis to predict the 1750 or 2015 rank of each city had the interacted effects of mortality and the fixed factors set to 0. Comparing the predicted ranks of each city excluding the recovery effects of fixed factors with their predicted ranks when the same recovery effects are included, one can identify cities that would have lost a lot otherwise.

As seen in Figure 8, examples of such cities in 2015 include some of the largest cities in 1300: Venice (3rd largest, mortality of about 60%), Florence (5th, 60%), Cordoba (8th, 50%), Naples (9th, 65%), Cologne (10th, 30%), Cordoba (8th, 50%), Pisa (19th, 35%), Toulouse (23st, 50%), Rouen (24th, 45%), and Marseille (28th, 55%).[59] These cities have in common to have been hit hard by the Plague and either coastal, riverine, on a road intersection and/or part of the Hansa league. Some of these cities have become relatively smaller over time, due to other factors. But in the absence of important fixed factors aiding their recovery, they could have been at the bottom of the list of the 165 cities in 1750 and/or 2015. Thus, our analysis suggests that, absent the presence of

[59] Somewhat similar results are observed for 1750 (Web Appx. Fig. A.6).

important fixed factors, these cities would not have recovered, and subsequently grown, following the Black Death, for example during the Renaissance.

Implications. Our results suggest that people disproportionately left (and/or did not move to) marginal rural areas as well as cities with "worse" rural and urban fixed factors. Thus, the relative population decline of "worse" areas suggests that the growth potential of Europe's distribution of population might have improved in the aggregate. Indeed, if, among the 165 cities existing in 1300, cities with better land or trade potential recovered faster after the Black Death, we should observe over time higher shares of the total population of these 165 cities living in locations with such advantages.

This is what we find. For example, high-mortality cities along a river started growing significantly faster than other cities from the 17th century. As a result, an increasing share of the total urban population of Europe must have resided along a river over time. Figure 7(b) shows that the unconditional population share of the 165 cities located along a river did increase from 1400-2015. Figure 7(b) 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 population growth (i.e. the interacted effect of mortality and the river dummy times mortality times the river dummy). As can be seen, the conditional population share along rivers has increased over time, close to the unconditional share. We find similar patterns when we perform the same analysis for the other fixed factors (not shown, but available upon request).

We do not pursue this analysis further as it would require a causal analysis of the effects of these factors on long-run economic development, and a theory-grounded analysis of what a dynamically optimal distribution of population involves. But we note that 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 opened up between Europe and the rest of the world after 1700 and the Little Divergence that took place within Europe itself (Pamuk, 2007; Voigtländer and Voth, 2013b).

5. Conclusion

The Black Death killed 40% of Europe's population between 1347-1352. Using a novel dataset that provides information on spatial variation in Plague mortality at the city level, as well as various identification strategies, we explored the short-run and long-

run impacts of the Black Death on city growth. On average, cities recovered their pre-Plague populations within two centuries. In addition, aggregate convergence masked heterogeneity in urban recovery. We showed that both of these facts are consistent with populations returning to high-mortality locations endowed with more fixed factors of production. Land suitability and natural and historical trade networks played a vital role in urban recovery. Our study highlights the role played by pandemics and mortality shocks in determining both the size and location of urban populations.

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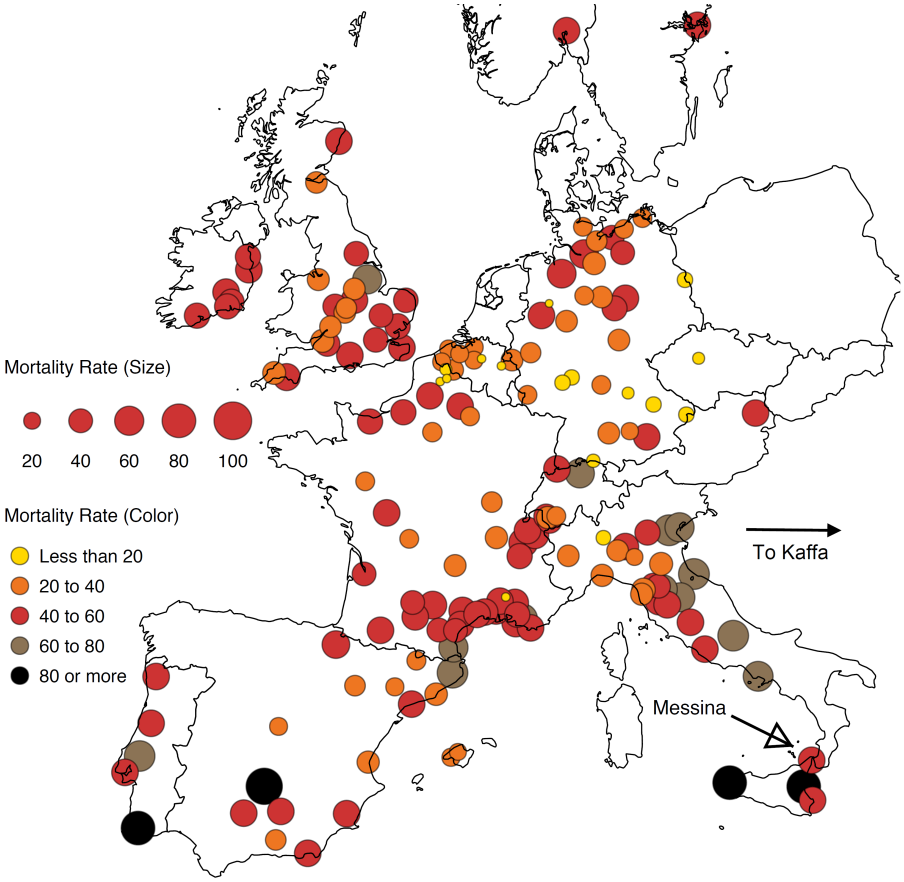
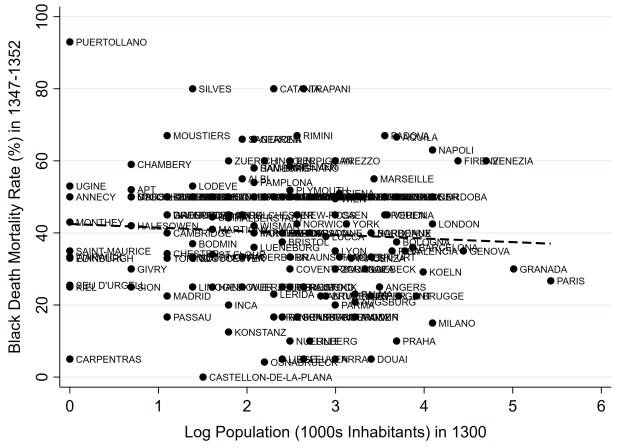
Figure 1: Black Death Mortality Rates (%) in 1347-1352



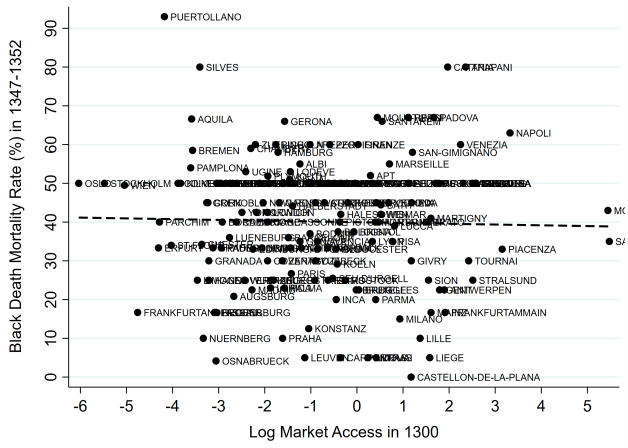
Notes: This map plots the location of all 165 existing cities (i.e. localities $\geq$ 1,000 inhabitants) in 1300 for which we know their Black Death mortality rate (%) in 1347-1352 as well as the modern boundaries of the 16 Western European countries of our main analysis (Austria, Belgium, Czech Republic, Denmark, France, Germany, Italy, Luxembourg, Norway, Poland, Portugal, Spain, Sweden, Switzerland, the Netherlands, and the United Kingdom). See Web Data Appendix for data sources.

Figure 2: Mortality Rates, City Size and City Market Access in 1300

(a) Mortality and Initial City Size

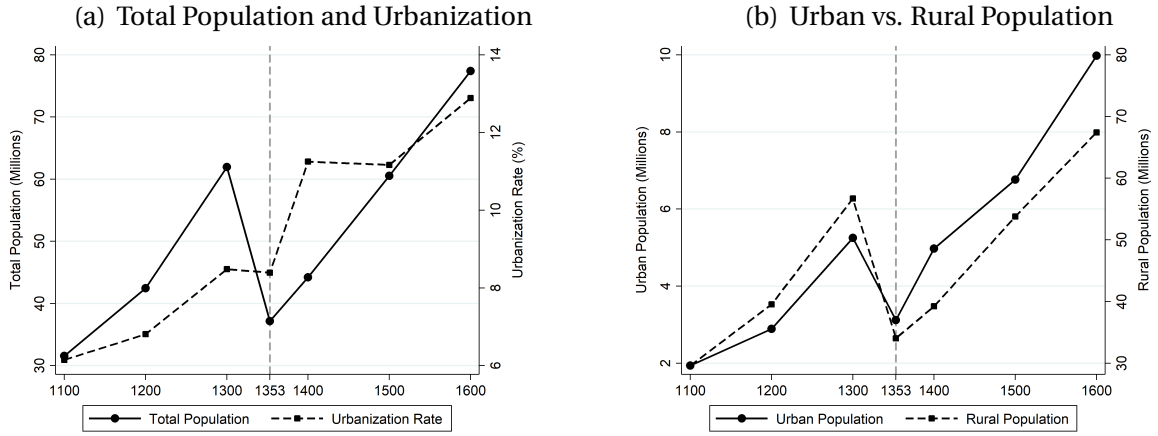


(b) Mortality and Initial Market Access



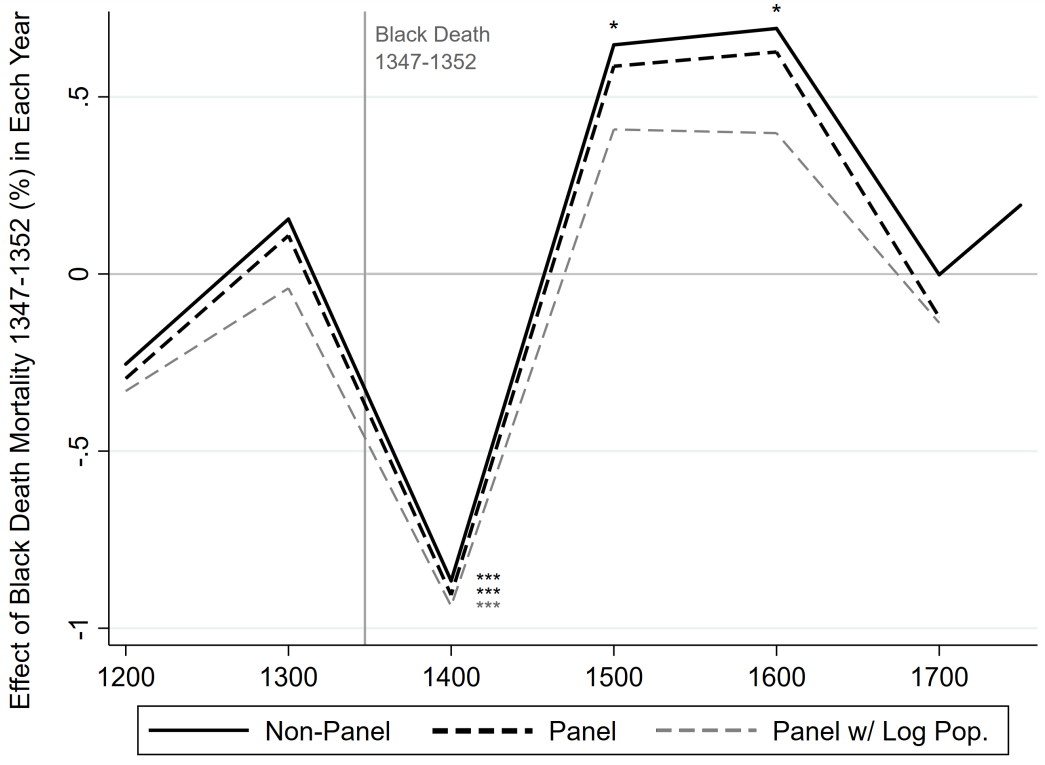
Notes: Subfigure 2(a) shows the relationship between mortality rates (%, 1347-1352) and log city population in 1300 for our main sample of 165 cities (Y = 42.5*** -1.01 X; R2 = 0.00). Subfigure 2(b) shows for the same 165 cities the relationship between mortality rates (%, 1347-1352) and log market access to all 1,801 cities in 1300 (Y = 40.0*** -0.20 X; R2 = 0.00). Market access for city i is defined as $MA_i = \Sigma_j (P_j / D_{ij})^{\sigma}$, with P_j being the population 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). See Web Appendix for data sources.

Figure 3: Evolution of Europe's Total, Urban and Rural Populations, 1100-1600



Notes: The subfigures show the respective evolutions of the total population (millions) and urbanization rate (%) (Subfigure 3(a)) and the total urban and rural populations (Subfigure 3(b)) of the 16 European countries in 1100-1600. Total (urban) population in 1353 is proxied by the total (urban) population in 1300 times the population-weighted average total (urban) mortality rate in 1347-1352 (40% and 38.8%). Rural populations are estimated residually. See Web Appendix for data sources.

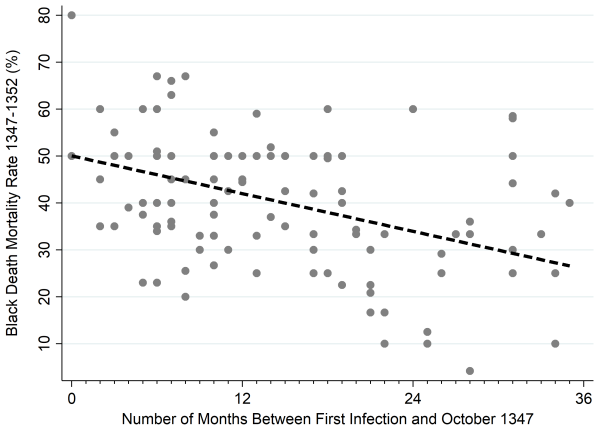
Figure 4: Effect of Black Death Mortality 1347-1352 (%) in Each Year, Panel Regressions



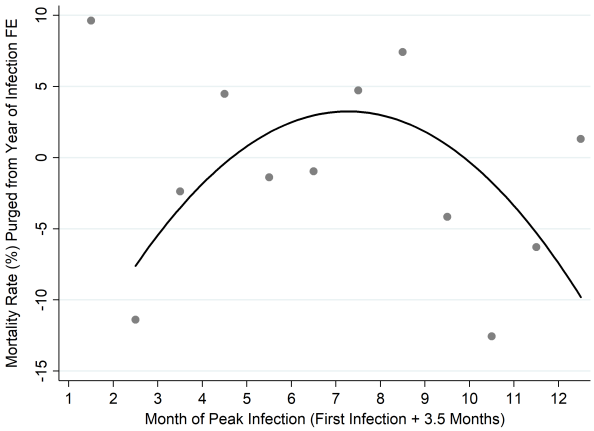
Notes: The figure shows the year-specific effects of the Black Death Mortality rate (%) in 1347-1352. The omitted year for the panel regressions is 1750. Non-panel regressions consist of repeated cross-sectional regressions for each century. See text for details and Web Appendix for data sources. Robust SE's (clustered at the city level for the panel regressions): * p<0.10, ** p<0.05, *** p<0.01.

Figure 5: Timing of the Onset of the Black Death and Black Death Mortality

(a) Timing of Black Death Onset and Mortality

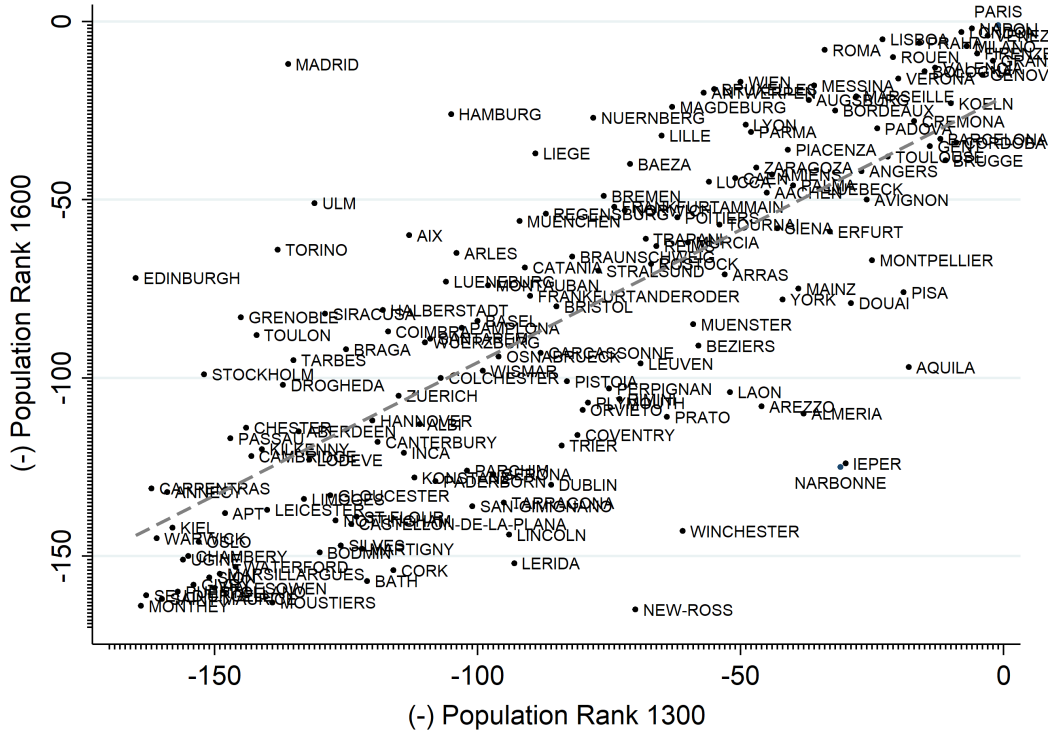


(b) Month of Black Death Onset and Mortality



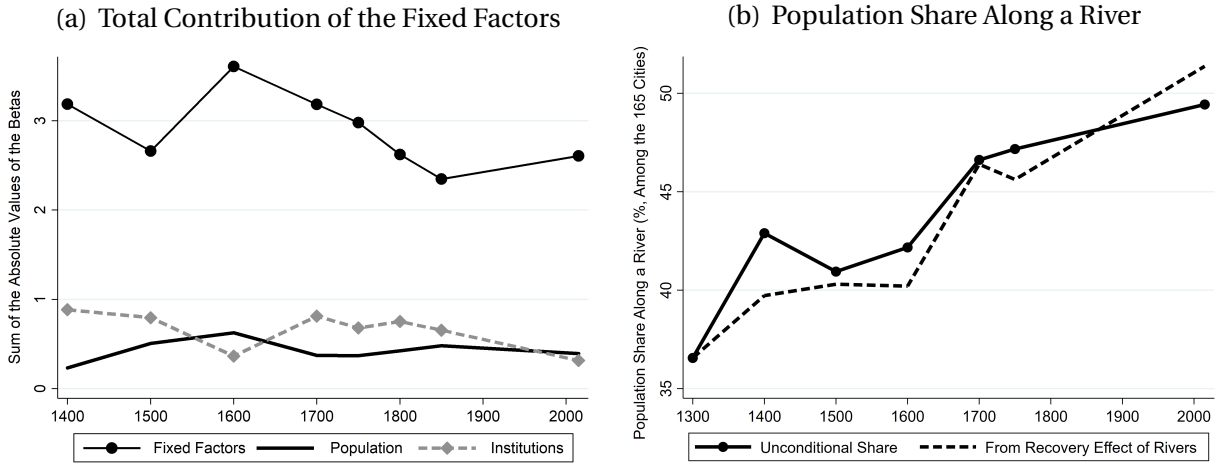
Notes: Subfigure 5(a) shows for 165 cities the relationship between mortality (%) and the timing of the onset of the Black Death in the city (Y = 50.0*** - 0.67***; R2 = 0.19). Number of months is measured since October 1347, the date Messina – the port of entry of the Black Death in Europe – was infected. Subfigure 5(b) shows for the same 165 cities and for each month of peak infection (month of first infection + 3.5 months) 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 October, which has high mortality rates due to being the month of onset of the Black Death in Europe. See Web Appendix for data sources.

Figure 6: Changes in Population Ranks Among the 165 Cities 1300-1600



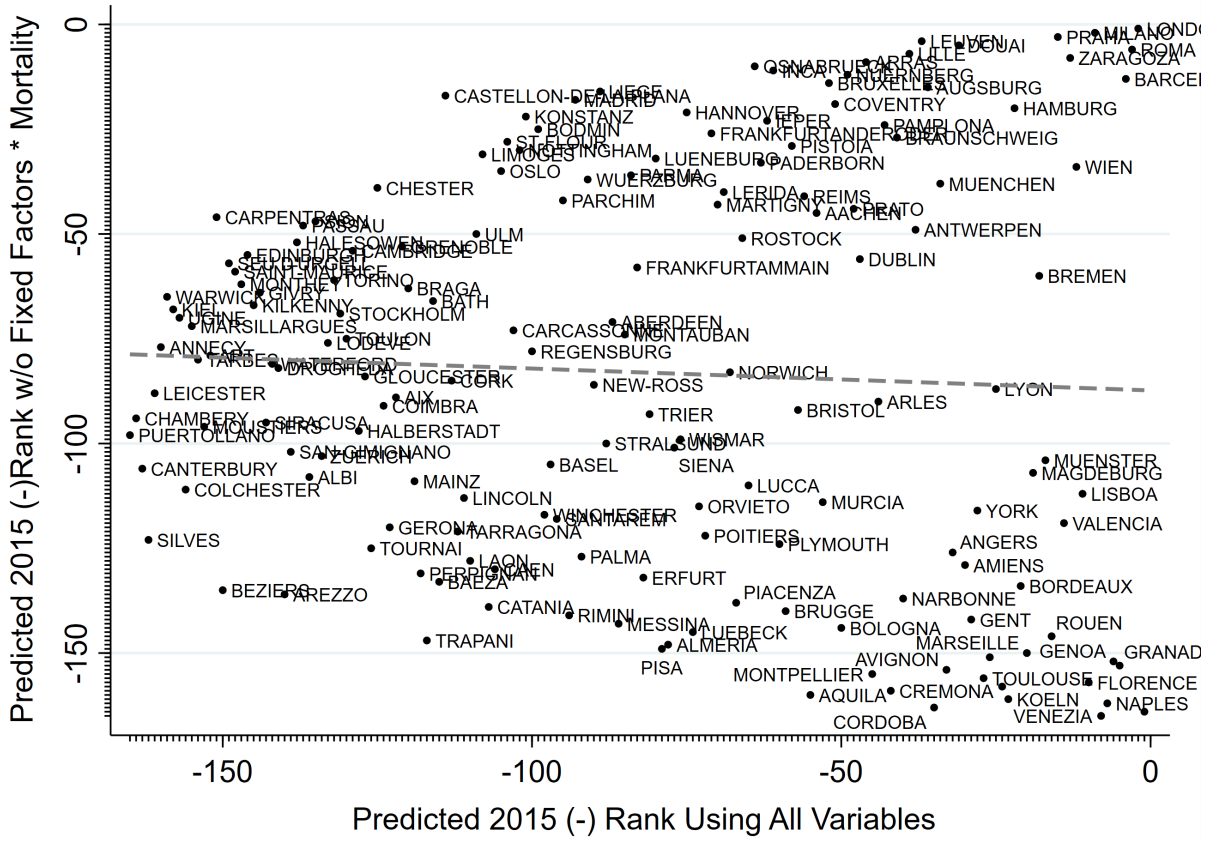
Notes: The figure shows for the 165 cities of the main sample the relationship between their inverted population rank in 1300 (among the 165 cities; 0 = largest city) and their inverted population rank in 1600 (among the 165 cities; 0 = largest city).

Figure 7: Total Contribution of the Fixed Factors to Aggregate Patterns



Notes: Subfigure 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). Subfigure 7(b) shows the percentage share of the total population of the 165 main cities that is located along a river in the raw population data ("unconditional") and based on 1300 population $\times$ the recovery effect of rivers to predicted city population growth ("from recovery effect of rivers"). See Web Appendix for details on data sources.

Figure 8: Fixed Factors and Counterfactual Population Ranks, 2015



Notes: This figure shows the relationship between the predicted 2015 inverted population rank (among the 165 main cities; 0 = largest city) when using their respective populations in 1300 and the Tobit-estimated regression results of Table 8 (including the independent effects of the variables shown at left) and the predicted 2015 inverted population rank (0 = largest city) when ignoring the interacted effects of mortality with the fixed factors (cereal, potato, pastoral, coast, rivers, road, Hanseatic).

Table 1: BLACK DEATH MORTALITY RATES AND CITY GROWTH, 1100-1750

Dep. Var.	*Col. (1)-(7): Percentage Change in City Population (%) in Period t*							*City Pop. $\geq$ 1,000 in t*	
t:	1300-1400 (1)	1300-1500 (2)	1300-1600 (3)	1300-1700 (4)	1300-1750 (5)	1100-1200 (6)	1200-1300 (7)	1100-1200 (8)	1200-1300 (9)
β	-0.87***	-0.28	0.36	0.47	0.85	-0.25	0.16	0.00	0.00
	[0.28]	[0.38]	[0.80]	[1.00]	[1.17]	[0.34]	[0.59]	[0.00]	[0.00]
Obs.	165	164	164	164	164	62	93	165	165
R^2	0.12	0.01	0.00	0.00	0.00	0.01	0.00	0.00	0.01

Notes: The main sample consists of 165 cities (i.e. loc. $\geq$ 1,000 inh.) that existed in 1300 and for which mortality is available. Col. (1)-(7) show the effect β_t of the mortality rate (%) in 1347-1352 on the percentage change in city pop. (%) for each period t. We use city pop. in the initial year of period t as regression weights. Col. (8)-(9) show the effect of the mortality rate on the likelihood of being above 1,000. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.

Table 2: CITY CHARACTERISTICS AND BLACK DEATH MORTALITY RATES

Dependent Variable:	Black Death Mortality Rate (%, 1347-1352)							
	(1)		(2)		(3)		(4)	
Locational Fundamentals:								
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]
Increasing Returns:								
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]
Institutions:								
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 (%, 1347-1352). See the text for a description of the variables. We use the main sample of 165 cities. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.

Table 3: 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		
1. Baseline (See Columns (1) and (3) of Table 1)	-0.87***	[0.28]	165	0.36	[0.80]	164
2. Controls: All	-0.59***	[0.21]	165	-0.37	[0.70]	164
3. 13 Country (2018) FE	-0.62**	[0.26]	165	0.03	[0.76]	164
4. 7 States (1300) FE (for States >= 5 Cities)	-0.82**	[0.35]	105	-0.29	[0.68]	104
5. Dropping Top and Bottom 5% in Mortality	-0.67**	[0.29]	145	1.36	[1.12]	144
6. Dropping More Hygienic Cities	-0.89***	[0.30]	161	0.59	[0.90]	160
7. Ctrls for Dummy & Num. Plague Recur. 50km	-0.87***	[0.27]	165	0.41	[0.81]	164
8. Ctrls for Dummy & Num. Battles 50km	-0.84***	[0.28]	165	0.77	[0.78]	164
9. Ctrl for Num. of Famines in Region/Country	-0.80***	[0.29]	165	0.45	[0.77]	164
10. Ctrls for Jewish Pres., Pers., Pogr. 1347-1352	-0.83***	[0.30]	165	0.28	[0.81]	164
11. Drop if Jewish Persecution 1347-1352	-0.71***	[0.30]	115	0.67	[0.96]	114

Notes: This table shows the effect β_t of the mortality rate (%) on the percentage change in city population (%) for period t. Row 2: Adding the controls of Table 2. Row 3: Adding 13 country FE. Rows 4: Adding 44 state FE but excl. states with less than 5 cities in our sample. Row 5: Dropping the top and bottom 5% mortality rates. Row 6: Dropping cities with a better hygiene system. Rows 7-8: Adding a dummy if there was a plague recurrence/battle and the number of recurrences/battles in 1353-1400 or 1353-1600. Row 9: Adding the number of famines experienced by the region or country of the city in 1300-1400 or 1300-1600. Row 10: Adding dummies if Jews were present, and if a persecution, and a pogrom in particular, took place in 1347-1352. Row 11: Dropping cities with Jewish persecutions in 1347-1352. Robust SE's: † p=0.17, * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.

Table 4: MORTALITY AND CITY GROWTH, IV REGRESSIONS

Dependent Variable: Percentage Change in City Population (%) in Period t

Regression:	(1) t = 1300-1400			(2) t = 1300-1600		
1. Baseline (See Columns (1) and (3) of Table 1)	-0.87***	[0.28]	165	0.36	[0.80]	164
2. IV1: Timing w/ Controls (IV F-stat = 11.8)	-1.07**	[0.50]	124	0.05	[1.32]	124
3. IV2: Month w/ Controls (IV F-stat = 7.1)	-0.92***	[0.35]	114	-0.17	[0.68]	114
4. IV3: Messina w/ Controls (IV F-stat = 23.0)	-0.93**	[0.46]	164	-1.67	[1.72]	163
5. Row 4, Excl. Sicily (IV F-stat = 14.3)	-1.12*	[0.63]	161	-0.72	[2.14]	160
6. IV1 + IV3 w/ Controls (IV F-stat = 14.6)	-1.16***	[0.45]	123	-0.26	[1.21]	123

Notes: Row 2: Instrumenting by the number of months between the city-specific date of first infection and Oct. 1347. Row 3: Instrumenting by eleven dummies for the month of peak infection (= month of onset (Oct. is omitted) + 3.5 months) while adding dummies for the year of infection. Row 4: Instrumenting by log Euclidean dist. to Messina while controlling for average log Euclidean dist. to all 1,801 towns in 1300 (Messina is dropped from the regression). Rows 2-6: We add the controls of Table 2 as well as latitude and longitude and their squares and cubes to flexibly control for Plague diffusion coming from the South and from the East. Robust SE's (clustered at the state (1300) level in rows 2-5): * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.

Table 5: MORTALITY AND CITY GROWTH, ROBUSTNESS CHECKS

Dependent Variable: Percentage Change in City Population (%) in Period t						
Regression:	(1) t = 1300-1400			(2) t = 1300-1600		
1. Baseline (See Columns (1) and (3) of Table 1)	-0.87***	[0.28]	165	0.36	[0.80]	164
2. Add Pct. Change in Pop. 1200-1300 as Control	-1.07***	[0.36]	93	0.83	[1.04]	93
3. Absolute △Pop. 1300-1400 & Mortality 1347-52	-0.86**	[0.42]	162	0.83	[0.99]	161
4. SEs Clustered at State ca 1300 Level (N = 64)	-0.87***	[0.27]	165	0.36	[0.68]	164
5. Conley Standard Errors (500 km)	-0.87***	[0.17]	165	0.36	[0.58]	164
2. Dummies for Type of Mortality Data	-0.94***	[0.30]	165	0.27	[0.84]	164
3. Excl. Description-Based Mortality Data (N = 25)	-0.83***	[0.32]	140	0.37	[0.97]	139
4. Excl. Desertion-Based Mortality Data (N = 21)	-0.98***	[0.31]	144	0.31	[0.86]	143
5. Excl. Clergy-Based Mortality Data (N = 5)	-0.86***	[0.28]	160	0.38	[0.80]	158
6. Use Only Number-Based Mortality Data	-0.96***	[0.36]	114	0.35	[1.06]	113
7. Keeping Top and Bottom 10% in Mortality	-0.99**	[0.39]	34	-0.21	[1.01]	34
8. City Population Data: Bairoch Only	-0.78*	[0.41]	151	0.47	[0.80]	150
9. City Population Data: Chandler Only	-0.85**	[0.36]	59	1.37	[1.10]	60
10. Use Mortality of Nearest Avail. City w/i 50 km	-0.60***	[0.22]	290	0.41	[0.59]	286
11. Use Mortality of Other Cities in Same State	-0.70***	[0.17]	380	0.33	[0.63]	374
12. Extrapolated Rates Based on 274 Cities in 1300	-0.68***	[0.21]	464	0.43	[0.54]	457
13. Reweighting to Match Full City Size Dist.	-0.74***	[0.24]	165	-0.06	[0.65]	164

Notes: This table shows the effect β_t of the mortality rate (%) on the percentage change in city population (%) for period t. See text for details on each robustness check. Robust SE's: † p=0.17, * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.

Table 6: MORTALITY AND CITY GROWTH, AGGREGATE EFFECTS, 1300-1600

Panel A: Dep. Var.:	Percentage Change in Total City Population (%) in Period 1300-1400					Dummy if Exists 1400	Log Pop. 1400
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mortality Rate (%)	-0.87***	-1.15***	-1.47**	-1.13*	-1.27**	-0.002***	-0.004***
	[0.28]	[0.40]	[0.57]	[0.62]	[0.58]	[0.001]	[0.001]
Unit	City	State	State	Country	Country	City	City
Population	Intensive	Intensive	Total	Intensive	Total	Extensive	Extensive
Observations	165	68	68	15	15	1,335	1,335
R^2	0.12	0.16	0.12	0.05	0.07	0.01	0.01
Panel B: Dep. Var.:	Percentage Change in Total City Population (%) in Period 1300-1600					Dummy if Exists 1600	Log Pop. 1600
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mortality Rate (%)	0.36	-1.49	-1.34	-1.64	-2.49	-0.001	0.002
	[0.80]	[1.32]	[3.17]	[2.71]	[5.35]	[0.001]	[0.002]
Unit	City	State	State	Country	Country	City	City
Population	Intensive	Intensive	Total	Intensive	Total	Extensive	Extensive
Observations	164	68	68	15	15	1,335	1,335
R^2	0.00	0.01	0.00	0.00	0.00	0.00	0.00

Notes: Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. State/Country: We run the main regression at the state (1300)/country (2018) level. Intensive/Total: The cities considered to construct total city population are the cities that already existed in 1300 / all cities. Extensive: We consider cities that did not already exist in 1300 but existed at one point in the Bairoch (1988) data set (800-1850). The 1,335 cities can thus be thought of the universe of potential new city locations in 1600. See Web Appendix for data sources.

Table 7: 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	1290-1756	1086-1290	All	$\leq$10Km	>10Km	1290-1756
	(1)	(2)	(3)	(4)	(5)	(6)	(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]
Obs.	27	27	27	28	28	28	27
R2	0.13	0.01	0.00	0.31	0.06	0.35	0.02

Notes: This table shows for 27-28 English 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 per 1000 sq km (col. (5): Within 10 km from an existing city in 1300; col. (6): Beyond 10 km from an existing city in 1300); and (7) the absolute change in the urban share (%) in 1290-1756. In all regressions, we use county populations in the initial years of the period as weights. Col. (1)-(3) and (7): We exclude Cornwall whose population in 1290 is severely underestimated. Columns (4)-(6): We control for log population in 1290 and log area. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.

Table 8: BLACK DEATH MORTALITY RATES AND POPULATION RECOVERY, 1300-2015

Dependent Variable: Percentage Change in City Population (%) in Period 1300-t

Period 1300-t:	1400 (1)	1500 (2)	1600 (3)	1700 (4)	1750 (5)	2015 (6)
Mort.*Cereal Suitability Index	-0.1	0.2	0.6	0.9	1.0	8.8
	[0.3]	[0.3]	[0.7]	[1.5]	[1.7]	[33.0]
Mort.*Potato Suitability Index	0.3	-0.2	0.9	2.6**	3.0**	51.6**
	[0.3]	[0.4]	[0.6]	[1.3]	[1.5]	[23.9]
Mort.*Pastoral Suitability Index	0.6	-0.3	-4.1*	-3.2	-5.7*	-63.7
	[0.7]	[1.1]	[2.1]	[2.8]	[3.2]	[61.4]
Mort.*Coast 10 Km Dummy	1.2**	2.9***	4.8***	7.3*	7.6*	72.6
	[0.5]	[0.7]	[1.8]	[3.7]	[4.3]	[79.3]
Mort.*Rivers 10 Km Dummy	-0.5	0.3	1.7	5.0**	6.0**	112.4***
	[0.5]	[0.7]	[1.1]	[2.1]	[2.4]	[39.7]
Mort.*Road Intersection 10 Km Dummy	0.6	1.5*	1.3	2.2	2.7	31.3
	[0.6]	[0.8]	[1.5]	[2.7]	[3.1]	[57.7]
Mort.*Hanseatic League Dummy	2.9***	2.3*	4.1*	7.3*	8.5	92.2
	[0.9]	[1.2]	[2.4]	[4.4]	[5.8]	[104.2]
Mort.*Log Est .City Population 1353	-0.2	0.6	1.4	1.7	2.2	35.7
	[0.2]	[0.4]	[1.0]	[2.0]	[2.3]	[42.0]
Mort.*Monarchy 1300 Dummy	-0.2	0.7	1.2	2.3	1.9	-26.4
	[0.5]	[0.6]	[1.2]	[2.1]	[2.4]	[43.0]
Mort.*State Capital 1300 Dummy	-0.6	-1.5	-0.3	4.7	4.7	-19.1
	[0.8]	[1.3]	[2.5]	[4.3]	[5.3]	[87.1]
Mort.*Representative Body 1300 Dummy	0.8	-0.2	-0.5	-2.1	-3.1	-22.7
	[0.6]	[0.7]	[1.1]	[2.0]	[2.4]	[39.5]
Mortality	-3.9***	-1.8	-6.1*	-19.6**	-20.7**	-373.8**
	[1.4]	[1.9]	[3.5]	[9.2]	[10.2]	[156.9]
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 165 cities the effects of 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 factors are used as controls. We use as weights city pop. in 1300. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. See Web Appx. for data sources.

Table 9: BLACK DEATH MORTALITY AND POPULATION RECOVERY, OTHER RESULTS

Dependent Variable: Percentage Change in City Population (%) in Period 1300-t

Period 1300-t:	1500 (1)	1600 (2)	1700 (3)	1750 (4)		1500 (5)	1600 (6)	1700 (7)	1750 (8)
Each row is for a separate regression and shows the effect of mortality * the variable shown at left									
1. Coastal Coast	2.8***	4.4**	6.7**	7.2*	6. Log Pop 1300	1.5	3.4	-11.0	-17.2
	[0.7]	[1.7]	[3.3]	[3.7]		[7.0]	[12.0]	[23.2]	[27.7]
50KmCoast Estuary	2.7*	5.2	18.3**	26.2**	7 Market Access	0.2	0.3	-0.6	-0.9*
	[1.6]	[3.6]	[7.7]	[10.1]		[0.2]	[0.3]	[0.4]	[0.5]
50KmCoast Ot.River	1.9*	3.8*	4.9	3.3	8 State Pop. Size	0.0	0.0	-0.7	-1.0
	[1.0]	[2.0]	[4.1]	[4.8]		[0.3]	[0.4]	[0.8]	[1.1]
2. BestCoast Medi.	3.3***	5.0***	7.8**	8.4**	9. Guild	0.3	-0.1	-1.8	-3.0
	[0.8]	[1.8]	[3.5]	[4.1]		[0.8]	[1.1]	[1.9]	[2.3]
BestCoast Atlantic	1.9	3.2	10.0*	16.2*	10. Market Fair	-1.4	-2.1*	-2.4	-1.4
	[1.7]	[3.1]	[5.9]	[8.3]		[0.9]	[1.2]	[1.8]	[2.1]
3. BestCoast Mon	3.8***	5.7***	8.6**	8.8**	11. Log Wall Area	-1.1	-2.0	-6.7*	-6.9
	[0.9]	[1.8]	[3.7]	[4.5]		[1.4]	[1.5]	[3.7]	[4.5]
BestCoast No-Mon	1.5	1.7	1.4	2.4	12. Bishopric	0.0	-0.7	-1.9	-3.8*
	[1.0]	[1.8]	[3.0]	[3.6]		[0.6]	[0.9]	[1.6]	[2.0]
Ot.River Mon	0.6	1.9	4.8*	5.1*	13. University	1.1	3.5**	3.0	1.7
	[0.9]	[1.9]	[2.5]	[2.9]		[1.0]	[1.6]	[2.6]	[3.1]
Ot.River No-Mon	0.9	2.1	3.5	3.8	14. Serfdom	-2.5	-5.7**	-9.8*	-10.9
	[0.9]	[1.3]	[2.6]	[3.5]		[1.8]	[2.6]	[5.6]	[9.3]

Notes: This table shows for the 165 cities of the main sample the effect of mortality (%) interacted with selected city characteristics when using the same regressions as in Table 8 but adding the selected city characteristic and its interaction with mortality. We use as weights city populations in 1300. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.