Pandemics and Cities: Evidence from the Black Death

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Abstract

The Black Death killed 40% of Europe's population between 1347 and 1352. Using a novel dataset on plague mortality at the city level, we study its effect on urban populations. We establish that plague mortality was unrelated to city-level characteristics and had a random component. Turning our attention to the recovery, we find that on average, cities recovered their pre-plague populations within two centuries. However, this masked considerable heterogeneity. Coastal and riverine cities grew fastest following the Black Death, as did cities that were part of trade networks such as the Hanseatic League.

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

Keywords: Pandemics; Black Death; Cities; Shocks; Path Dependence

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The Black Death was the largest demographic shock in modern European history, killing approximately 40% of its population between 1347-1352. Many cities were devastated, while others were hardly affected at all. On average, England, France, Italy and Spain lost 50-60% of their populations in just one or two years. While the Black Death has been extensively studied by historians and social scientists, little is know about its spatial effects, due to the lack of data on local mortality. We use city-level data on Black Death mortality to test whether cities that experienced high mortality shocks were permanently affected. And we identify the city-level characteristics associated with the speed of urban recovery.

We are not the first to study the Black Death. Important contributions to the historical literature include: Ziegler (1969); Gottfried (1983); Benedictow (2005); Clark (2016); Campbell (2016). Many scholars argue that Northwestern Europe rose to economic prominence due in part to the differential effects of the Black Death (North and Thomas, 1973; Brenner, 1976; Pamuk, 2007; van Zanden, 2009; Moor and Zanden, 2010; de Pleijt and van Zanden, 2013). Acemoglu and Robinson (2012) suggest that the Black Death was a critical juncture inaugurating an institutional divergence between western and eastern Europe.

The standard model of growth in the preindustrial world is Malthusian (Galor, 2005; Ashraf and Galor, 2011; Galor, 2011). In Malthusian theory, a demographic shock such as the Black Death should be associated with a rise in per capita income. Indeed, the Black Death is often viewed as a classic test in support of this theory (see Hatcher and Bailey, 2001; Clark, 2007; Jedwab et al., 2021). Voigtländer and Voth (2013b) adapt the Malthusian model to show how a large mortality shock can trigger a transition to a new steady state. When wages increase, non-homothetic preferences raise the demand for urban goods and spur urbanization. Since historically cities were unhealthy and because the plague returned frequently and conflict was endemic, mortality rates and incomes remained high, thus explaining the rise in European incomes prior to the Great Divergence.

We build on this body of research to test four important hypotheses using

novel city-level data on Black Death mortality.

H1: *What were the determinants of mortality from the Black Death and was the virulence of the Black Death as if random?* While previous literature has explored the macroeconomic effects of the Black Death, due to a lack of local data, it has been a challenge to establish the causal impact of the plague at the city and regional level. We investigate how mortality varied across cities and the extent to which these mortality rates had a plausibly random component. For example, we ask: were trade routes associated with higher mortality rates? We find mortality rates were uncorrelated with observables including access to trade routes. We also establish that higher mortality cities did not have different growth trajectories than low mortality cities before the Black Death. While this may be surprising given what we know about trade networks and the diffusion of pandemics, our results highlight the importance of differences in epidemiological and environmental characteristics for the diffusion of disease.

H2: *What was the causal impact of the Black Death on city populations in the short-run and in the long-run?* The literature on the Black Death at the country-level shows significant short-run impacts and aggregate recovery after a few centuries. However, at the country-level it is difficult to disentangle the effects of the plague from other changes occurring at the same time. Because we use local level data, we are able to exploit several causal techniques to corroborate these findings. The Black Death was not the only major shock in the fourteenth century. The Great Famine (1315-1317) caused a huge number of deaths and likely reduced the livestock population of Europe by 80%. It also coincided with the end of the Medieval Warm period and famines recurred across Europe for much of the remainder of the century (Campbell, 2016). The Hundred Years' War (1337-1453) also devastated many regions and disrupted trade patterns across Europe. Any one of these could have been correlated with plague mortality and city growth in both the short and long runs. As such, our causal investigation of city recovery is an important contribution to the existing literature.

H3: *What was the impact of a city's mortality rate from the Black Death on*

nearby cities? Our unique city-level data allow us to address this. We find that the mortality rate in a city affected not just that city but also the city's "neighbors". It has long been recognized that the Black Death led to massive market disintermediation in the short-run (Broadberry et al., 2015, Ch. X). However, there are no formal empirical tests of the existence and magnitude of this effect. We find a large negative, though imprecise, impact of neighboring cities mortality in the short-run and no long-run affect. When we examine mortality at the regional level we find that higher average regional mortality rates led to slower recovery than if a lone city were to experience that same rate. We discuss how this discrepancy may be explained by city-level spillovers.

H4: *What factors determined city recovery?* In a Malthusian economy, a pandemic might have no long-term spatial effects *on average*. That is, we would expect high-mortality cities to recover over time independent of the impact of the plague. Nonetheless, there could still be *permutations* between cities, as some large cities become relatively smaller, and small cities, relatively larger.

We corroborate that the European population recovered by 1600. However, we also show that there was a significant amount of heterogeneity in urban recovery. For a *given* mortality shock, some cities recovered faster than others. Our data allows us to do a quantitative analysis of the factors which may explain these differences. We find strong correlations between city recovery and access to markets and trade and local geography. Our analysis suggests that many prominent modern-day cities might have been marginal today absent these factors. Furthermore, we discuss why the Black Death might have led to people moving to better urban locations overall.

In addition to the macroeconomics and historical literatures on the economic effects of the Black Death, we contribute to a body of research on shocks and long-run urban persistence. Unlike other shocks considered in the literature, our shock was exceptionally large. The Black Death was also a comparatively "pure" population shock. More precisely, buildings and equipment were not destroyed and the event itself did not directly target a particular demographic group. This

makes our setting well suited to test for the path dependent effects of mortality shocks (see Bleakley and Lin (2015) and Hanlon and Heblich (2020) for surveys of the path dependence literature).[1] Different causes have been advanced for this path dependence, including locational fundamentals (i.e. natural advantages), sunk investments (i.e. man-made advantages), agglomeration effects (i.e. the direct effect of scale), or institutions (Henderson and Thisse, eds, 2004; Bleakley and Lin, 2012; Maloney and Caicedo, 2015; Hanlon, 2017; Dalgaard et al., 2018a).

Our findings on the importance of land suitability and natural and historical trade networks for recovery are related to Henderson et al. (2017b) who show how both agriculture- and trade-related geographic variables explain the long-run distribution of economic activity globally. Due to high transportation costs, cities had to be closer, or naturally connected, to agriculturally suitable areas. Instead of studying how the influence of these factors has changed over time, we study their importance after a massive population shock.[2]

Finally, most pre-COVID studies of their economic consequences employ macroeconomic approaches (Young, 2005; Weil, 2010; Voigtländer and Voth, 2013b,a), notable exceptions being Almond (2006) and Beach et al. (2018) who study the 1918 influenza pandemic. There is also research on subsequent outbreaks of the bubonic plague (Bosker et al., 2008; Wilde, 2017; Alfani and Murphy, 2017; Alfani and Bonetti, 2018; Alfani and Percoco, 2019; Dittmar and Meisenzahl, 2019; Siuda and Sunde, 2021). However, with the partial exception of the plague that hit Italy in the 17th century, these events were on average much less deadly than the Black Death and only affected a few areas at a time

[1] Wars and bombings, as studied by Davis and Weinstein (2002, 2008) and Caicedo and Riaño (2020) also led to massive physical destruction. 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 physical destruction. Climate change, as studied by Waldinger (2015) and Henderson et al. (2017a), kill people, but in this scenario physical geography is also, by construction, changing.

[2] Other studies finding a strong impact of geography on spatial development include Bosker, Buringh and van Zanden (2013); Maloney and Caicedo (2015); Andersen, Dalgaard and Selaya (2016); Bosker and Buringh (2017); Dalgaard, Knudsen and Selaya (2020). Several papers also shed light on aspects of medieval cities, for example de la Croix, Doepke and Mokyr (2018) on guilds, Croix et al. (2019); de la Croix and Morault (2020) on universities, and Becker et al. (2020) on fiscal institutions. See Jedwab et al. (2020) for a survey of the literature on medieval cities.

(Aberth, 2010, p.37). Likewise, there is a nascent literature on the effects of the West African Ebola epidemic (2013–2016) (e.g., Bowles et al., 2016). However, this disease has killed only about 10,000 people which is 0.003% of West Africa's population. Pandemics like the Black Death differ from epidemics in that they affect a very large number of areas and people, so their effects are likely to differ.

1. Data

Mortality. Data on cumulative Black Death mortality for the period 1347-1352 come from Christakos et al. (2005, 117-122) who compile mortality rates based on information from a wide array of historical sources including ecclesiastical and parish records, testaments, tax records, court rolls, chroniclers' reports, donations to the church, financial transactions, mortality of famous people, letters, edicts, guild records, hospital records, cemeteries and tombstones. Christakos et al. (2005) 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) (see Web Appx. Section 1. for details). These data yield mortality estimates for 274 localities in 16 countries.

For 177 of these we have a percentage estimate. In other cases the sources report more qualitative estimates: (i) For 49 cities Christakos et al. (2005) provide a literary description of mortality. We rank these descriptions based on the implied magnitude of the shock and assign each one of them a numeric rate.[3] (ii) For 19 cities we know clergy mortality. Christakos et al. (2005) show that clergy mortality was 8% higher than general mortality, so we divide the clergy mortality rates by 1.08.[4] (iii) For 29 cities we know the desertion rate, which includes non-returnees. Following Christakos et al. (2005, 154-155), who show that desertion rates were 1.2 times higher than mortality rates, we divide desertion rates by 1.2.

Cities. Our main source is the Bairoch (1988) dataset, which reports population

[3] 5% for "spared"/"escaped", 10% for "partially spared"/"minimal", 20% for "low", 25% for "moderate", 50% for "high", 66% for "highly depopulated", and 80% for "decimated".

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

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. The criterion for inclusion in the dataset is a city population greater than 1,000 inhabitants. We update Bairoch where scholars—Nicholas (1997), Campbell (2008), Bosker et al. (2013) and Voigtländer and Voth (2013b)—have revised population estimates. We also add 76 cities mentioned in Christakos et al. (2005). In the end, we obtain 1,801 cities and focus on 1100-1850 (see Web Appx. Section 2. for details).[5]

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

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

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

measures of parliamentary activity during the 14th century (Zanden et al., 2012) and control for whether a city was within 100 km of a battle between 1300-50. See Web Appx. Section 3. for details and Table A.1 for summary statistics.

2. The Shock

The Black Death arrived in Europe in October 1347 after ships carrying the plague from Kaffa in Crimea stopped in Messina in Sicily (Figure 1). Over the next three years it spread across Europe killing 40% of the population (we obtain a mortality rate of 38.9% for the 274 localities in our sample). In this section we document that there was a plausible random component to mortality (Hypothesis H1).

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

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

the speed with which the disease spread, but not necessarily its mortality rate.

An important epidemiological fact about the plague that we exploit is that the virulence was far greater in cities affected *earlier* (Christakos et al., 2005, 212-213). Initially, epidemics spread exponentially. One possible explanation for this is that as more people have been infected and survive or die and the pool of susceptible hosts in the aggregate population decreases, the disease might mutate in favor of benign pathogens that facilitate transmission, but at the expense of mortality.[6] Pathogen mutation also increases individual immune responses due to "contacted individuals becoming infected only if they are exposed to strains that are significantly different from other strains in their memory repertoire" (Girvan et al., 2002). Pathogen mutation and natural immunization may eventually cause an epidemic to end.

Early exposure can explain the terrible mortality Sicilian cities experienced (two thirds on average). Other coastal cities such as Barcelona, Bristol, Edinburgh and Rostock experienced much lower mortality rates. Likewise, this also helps explain why average mortality decreased over time (see Figure 2(a)) and why the disease eventually disappeared. If we compare the mortality rates of cities infected 1 month after the initial arrival of the plague in Messina to cities infected after 6, 12, 24 and 36 months, the average mortality difference is 9, 13, 22 and 39 percentage points. Thus, a difference of a few months in the arrival date of the plague in a city had dramatic effects on the city's cumulative mortality rate.

What determined why some cities were infected earlier than others? While density and trade could have mattered in theory, since the largest and most connected cities may have received infected people/cargoes before other cities, as we explain below, it wasn't trade potential *in general* that mattered, but rather how connected a city was to the origin point of the disease in Europe—Messina.

Why Messina? The disease first arrived in Messina in late 1347, which at the time

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

was only the 55th largest city in Europe. While the exact origins of the Black Death are unknown, we do know that Astrakhan, a trade centre located on the Volga river near the Caspian Sea, was infected in 1345. Kaffa, a Genoese colony in Crimea, was then infected in 1346. It was from there that the Genoese galleys with infected rats and humans on their voyage home stopped in Messina in October 1347. Two months later ships left for Genoa. Other infected ships probably also traveled from Messina to other Mediterranean cities around the same time.

Messina did not have to be the point of entry for the Plague. Genoa had other colonies in the Black Sea (Deletant, 1984) including Vinica along the banks of the Danube which led all the way to Vienna, a port of entry of plague recurrences in later centuries (Web Appx. Fig. A.1 maps the cities and routes mentioned in this paragraph). It also had colonies along the Dniester River, at the end of which was Halych, a town located on the East-West trade route that led to Leipzig via Prague. Thus, in 1346, the plague could have infected these other Genoese colonies and then traveled to Vienna or Leipzig. Moreover, Astrakhan was an important trading centre connected via river to Moscow and Novgorod, which both had river access to the Gulf of Finland. Novgorod traded with Visby (Sweden), one of the centers of the Hanseatic League, a trade network between Northern European cities. Thus, Messina, Genoa, Vienna, Prague, Leipzig and Visby all could have been the port of entry for the plague and trade networks in Central or Northern Europe could have been infected before the Mediterranean basin. Indeed, when we compute the travel times between Astrakhan and each of the counterfactual ports of entry, we find that it would have taken 3 months for the disease to reach any of them *had* it spread in their direction resulting in one of these other cities being infected as early as 1346. Yet, it happened that the disease went a different direction towards Genoa, making a stop in Messina. For our main sample of 165 cities, if we sequentially regress their mortality rates on their Euclidean distances to Messina and each of these alternative ports of entry, we indeed only find a significant negative effect for Messina (Web Appx. Table A.2).

After Messina. When the disease arrived in Messina, it was extremely virulent

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

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

Bubonic plague was most virulent during the summer (Benedictow, 2005, 233-235). Fleas become most active when it is warm and humid (Gottfried, 1983, 9). Christakos et al. (2005, 230) notes that mortality displayed seasonal patterns with deaths diminishing with colder weather "without the epidemic coming to a complete halt". Using available data on the year and month of first and last infection for 61 cities, the average duration of the Black Death was 7 months (see

Web Appx. Fig. A.2). According to Christakos et al. (2005, 212-213), mortality on average peaked 3.5 months after the first infection. Therefore, cities infected in late fall escaped relatively unscathed compared to cities infected in spring.

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

Figure 3(a) illustrates the lack of a relationship between mortality (1347-52) and city population in 1300 (Y = 42.5*** - 1.01 X; Obs. = 165; $R^2 = 0.00$) in our sample of 165 cities. For the 88 cities with data on walled area we also find no relationship with population density (Web Appx. Fig. A.4). Likewise, Fig. 3(b) indicates no relationship between mortality and log market access in 1300 (Y = 40.0*** -0.20 X; Obs. = 124; $R^2 = 0.00$). Note that random measurement error in dependent variables (mortality) does not lead to bias. However, random measurement error in market access produces a downward bias, and non-classical measurement error is possible. Reassuringly, Web Appx. Table A.3 shows no correlation when: (i) using a lower trade elasticities in the market access calculation (2 or 1, instead of 3.8);[7] (ii) using alternative measures of transport costs or Euclidean distance, which has the advantage of not having to rely

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

on speeds related to plague diffusion itself;[8] (iii) including the city itself;[9] (iv) including other (mostly Eastern) European cities not in our main sample of 16 countries; and (v) including cities of the Middle East and North Africa.[10]

Subsequent outbreaks of bubonic plague took place 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 Web Appendix Section 5.). These plague recurrences were caused either by local plague reservoirs or the reintroduction of the bacteria from Asia (Schmid et al., 2015). Though on occasion later outbreaks could devastate a city, in general mortality was significantly lower than in the initial pandemic (Aberth, 2010, 37).[11]

To summarize, on average, random factors must have compensated for non-random factors, making Black Death mortality apparently locally exogenous.

3. The Black Death Shock and City Recovery

To estimate the short- and long-run effects of Black Death mortality on city growth we estimate a series of city-level regressions based on (Hypothesis H2):

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

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 (%; 1347-52). We weight observations by their population size in year t-1 to minimize issues arising from smaller cities mechanically experiencing larger percentage changes.[12]

Col. (1) of Table 1 measures the short-run impact in 1300-1400. The

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

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

[10] We use the data of Bosker et al. (2013). Consequently, only 10,000+ cities are included.

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

[12] Growth for a city of 1,000 in t-1 and 5,000 in t is 400%. Large cities rarely experience such growth rates. While this is a standard issue when using percentage growth outcomes, we choose this as our main specification because the interpretation of the coefficient is straightforward.

coefficient, -0.87***, should be interpreted relative to the immediate effect in 1347-52, which is -1.00 by construction. The fact that the coefficient is not significantly different from -1.00 suggests little recovery in the decades directly following the onset of the Black Death. The effect is large: a one standard deviation increase 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. Col. (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 between a 0.03 and 0.05 standard deviation increase in population growth in columns (3)-(5) implying total recovery.

Parallel Trends. Col. (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. However, standard errors are not nil, so the pre-Black Death effects are imprecisely estimated. The sample sizes show that many cities also did not exist (i.e. were below 1,000 population) before 1300. Since col. (6)-(7) examine the intensive margin of city growth, we show in col. (8)-(9) that the likelihood of being above 1,000 by 1200 or 1300 is not correlated with mortality.

Correlates of Mortality. Table 2 shows that mortality rates were uncorrelated with various city characteristics capturing physical geography (1), access to markets and trade (2) or institutions (3). The only variables that have explanatory power are proximity to rivers and latitude. However, the sign on proximity to rivers is negative which is inconsistent with the claim that trade routes were correlated with plague virulence. Other measures of transportation and trade networks do not predict mortality. The coefficient on latitude reflects the fact that the Black Death hit southern Europe first and was more virulent in the early years of the epidemic. Finally, no effect is significant once all controls are included.[13]

[13]The R2 in Col. (1) falls to 0.08 when we exclude latitude and temperature (correlation with latitude of 0.77). If we re-run the specification in Col. (4) while dropping latitude and temperature, the coefficients of the other controls remain insignificant and the R2 decreases to 0.18. It does not

In row 2 of Table 3, we show the baseline results hold when we include all the controls of Table 2 simultaneously. The effect in 1300-1400 is now less negative. Indeed, we will show in Section 5. that city characteristics affected the recovery of higher-mortality cities in 1353-1400 and beyond. Over-controlling might then lead us to under-estimate the negative short-run effects.

Spatial Fixed Effects. In row 3 we include fixed effects corresponding to modern country borders. As modern country borders differ from the political units of the fourteenth century, in row 4 we assign a separate dummy variable to each of the independent polities with at least 5 cities in our data set (Web Appx. Fig. A.5 shows state boundaries).[14] Alternatively, we use fixed effects corresponding to twelve 5x5 degree cells (row 5). The results that we obtain are qualitatively similar.

We then employ three instrumental variable strategies: IV1, IV2 and IV3. IV1 and IV3 rely on the date of first infection in the city, which is available for 124 cities.[15] Also, since the IV strategies rely on the spatial diffusion of the Plague, we cluster standard errors at the state (1300) level (N = 64) for these analyses.

IV1: Timing of Infection IV1 exploits the randomness in plague intensity generated by the travel path of the disease. As discussed above, the Black Death was most virulent initially, and over time virulence declined. We create a variable for date of first infection for each city in our dataset. Fig. 2(a) plots mortality rates against the *date* that the city was first infected (number of months since October 1347). Cities infected later, indeed, had lower mortality. We therefore use the number of months since October 1347 as an instrumental variable. We add the controls of Table 2 (incl. longitude and latitude) and include the squares and cubes of longitude and latitude. The estimates we obtain are similar to our OLS estimates (row 6 of Table 3; -1.07** and 0.05; IV-F stat = 11.8).

IV2: Proximity to Messina IV2 is based on distance to the point of first infection, Messina. The logic behind IV2 is similar to IV1: as the virus was more virulent

decrease to 0 because some of the remaining variables are still correlated with latitude.

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

[15] See Web Appx. Table A.4 for the full first-stage regressions for IV1, IV2 and IV3.

initially, locations that were connected to Messina were more likely to be infected earlier and hence more likely to suffer high rates of mortality.

We use as an IV the Euclidean distance to Messina, conditional on average Euclidean distance to *all* cities in Western and Eastern Europe and the Middle East and North Africa (using their 1300 population as weights). Controlling for average distance to all cities captures the fact that some cities were better connected overall. Hence, we exploit the fact that it was the specific *connectedness to Messina*, and not connectedness overall, that mattered for mortality. In addition, since we use Euclidean distances, our IV is not built using the (possibly endogenous) speeds of plague transmission. We add the same controls as for IV1, including the controls for the various means of transportation. Controlling for longitude and latitude (and allowing them to have non-linear effects) is important because it captures any South vs. North and East vs. West effects. We report the IV estimates in row 7. The short-run coefficient (-1.20**) is similar to our OLS estimate (IV F-stat = 22.6). The long-run effect is negative (-0.68), but half the size of the short-run effect and not significant.[16]

IV3: Month of First Infection IV3 uses the variation in mortality generated by differences in the *month* of first infection *within* a single year. For 124 cities for which we have data on the onset of the plague, Fig. 2(b) shows the relationship between mortality and the month of peak infection in the city (= month of onset + 3.5 months). The plague was more virulent when peak mortality occurred during summer (6-8) (the quadratic fit omits January, which has abnormally high mortality due to October being the month of onset of the plague in Europe). We report results using IV3, dummies for the month of peak infection, while 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

[16]For this IV, we use cities above 1,000 in Europe and cities above 10,000 in the Middle East and North Africa (estimates not available below). What could matter for trade to influence mortality could be proximity to large cities only, or proximity to many cities. We take an intermediary approach and use as weights log population in 1300, thus giving less weight to the largest cities. Results hold if we: (i) control for average distance to all cities above 10,000 only; and (ii) use as weights unlogged population – giving more weight to large cities – or no weights – making a high spatial density of cities important – when computing the distance to all cities (not shown).

mortality. We obtain similar results (row 8; -0.93*** and -0.23; IV-F stat = 6.0).[17] Lastly, results hold when using the three IVs simultaneously (row 9).

Panel Analysis. To do this we restrict the sample to the 165 cities that are in our dataset in 1300 and for which we know Black Death mortality rates. We focus our analysis on the years 1100, 1200, 1300, 1400, 1500, 1600, 1700 and 1750. We then estimate the following regression equation:

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

where the dependent variable is the percentage change in population between t-1 and t (1100 is dropped), where city (κ_i) and year (θ_t) fixed effects are included, and where the variables of interest are mortality in 1347-52 interacted with the year dummies (β_t shows the effect in each year relative to the omitted year 1750). We use as weights population in t-1 and cluster standard errors at the city level.

Figure 4 shows the interacted effects ("Panel") and the corresponding effects when running the cross-sectional regression for each year one by one ("Non-Panel"). The negative effects in 1300-1400 ("1400"; about -0.9***) are offset by positive effects in 1400-1500 ("1500") and 1500-1600 ("1600") (coefficients shown in Web Appx. Table A.5), implying city effects do not have important effects.

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

The Black Death was attributed to the "vengeance of God" or the "'conjunction of certain stars and planets" (Horrox, 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 (Web Appx. Table A.6). Neither the medical profession nor authorities could respond. Medical knowledge was rudimentary: Boccaccio (2005, 1371) wrote that "all the advice of physi-

[17] If we study the relationship between mortality and the month of peak infection in the city for warmer regions vs. colder regions, we find even lower mortality rates in the winter in cities located in colder regions (Web Appx. Fig. A.6). Using as an IV dummies for the month of peak infection interacted with the log of a city location's average temperature, the IV-F stat increases to 7.2. The coefficients remain very similar to the coefficients for IV3 (row 15 of Web Appx. Table A.6).

cians and all the power of medicine were profitless and unavailing". Individuals, regardless of wealth, could not protect themselves. Prevention measures were nonexistent: the practice of quarantine was not employed until 1377.[18] Other practices such as separating the sick and burning the homes of the infected were also introduced after the Black Death period (1347-1352).

Bubonic plague reoccurred following the Black Death. This could be a source of bias if subsequent outbreaks were correlated with the initial pandemic. We use data from Biraben (1975) and show results hold if we control for plague recurrences (see Web Appx. Table A.6 for details).[19] The Black Death initially reduced the intensity of conflict (see Sumpton (1999)). However, warfare ultimately intensified and, according to some accounts, led to urbanization (Voigtländer and Voth, 2013a). We show results hold if we control for contemporaneous or past battles (Web Appx. Table A.6), or control for proximity to a battle of the Hundred Years' War prior to the onset of the Black Death. Results also hold if we control for the number of famines experienced by the city's region or country, or the possible magnitude of the Great Famine of 1315-1317 (same table). Finally, Jedwab et al. (2019) show that higher-mortality cities persecuted Jewish communities less. Results nonetheless hold if we control for persecutions or drop any city with a persecution (Ibid.).

Similarly, our findings are robust when we: (i) consider other specifications, for example control for past population trends or study absolute changes in population; (ii) cluster standard errors differently; (iii) take into account measurement error arising from the coding of mortality rates; (iv) focus on cities that are either in the bottom 10% of least affected cities or in the top 10% of most affected cities, since measurement errors in mortality rates are more likely when comparing cities with similar rates; (v) use alternative population estimates; and (vi) address concerns regarding the external validity of our results, for example increase sample size by using imputed mortality rates for cities outside our main

[18] The term quarantine was indeed first used in Ragusa in Croatia in 1377 (Gensini et al., 2004).

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

sample (Web Appx. Table A.8). Lastly, we drop cities located within France, Germany, Italy, the United Kingdom or Spain (Web Appx. Table A.9).

Overall, the various regressions return similar estimates to those obtained using the baseline OLS cross-sectional regressions. This reassures us that plague mortality had a strong locally exogenous component (H1). In the rest of the analysis, and for the sake of simplicity, we employ the baseline OLS specification.

4. The Black Death as a Regional Shock

So far, we have only discussed the effects of own city mortality on city growth. However, we need to quantify spillover and general equilibrium effects in order to test the recovery hypothesis for urban systems, not just individual cities. These effects are interesting in their own right because the population declines associated with the Black Death reduced market potential across Europe. In this sense, it was a massive trade shock that allows us to investigate Hypothesis H3.

Table 4 studies the effects of a city's own mortality and the spillover effects from mortality in other cities. Col. (1) reproduces the city-level results. In col. (2)-(3), we estimate the effects of population-weighted average mortality at the state level on the percentage change in urban population at the state level.[20] Col. (2) shows the effects for cities that existed in 1300. Our baseline estimate of the short-run impact of the Plague suggests that a city with a 10% mortality rate was only 8.7% smaller fifty years later. In contrast, col. (2) suggests that if an entire region experienced average mortality of 10%, then a city with a 10% mortality rate would have shrunk by 11.5% by 1400. Col. (3) examines the effects on all cities that are in the dataset in 1400 (including cities not in the dataset in 1300). The effects are larger than before (-1.47**) implying that in high-mortality areas, fewer new cities emerged, a result we explore in more detail below.

Similarly, when we look at the long-run effects in Panel B, we find that unlike the impact of a city's own mortality rate, the effect of aggregate mortality is negative (though the standard errors are very large, implying heterogeneity).

[20] For this analysis, 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. We lose 20 states and 1 country (Luxembourg) without any urban population in 1300.

This is consistent with the Black Death shock having broader, negative, disintermediation effects on local economies (Broadberry et al., 2015, Ch. X).

In columns (4) and (5), we define "indirect mortality" as the average mortality rate of the cities of the same state and of the closest 10% of cities, respectively. Note that cities that experienced high mortality did not always experience high indirect mortality (the correlation between the two measures is less than 0.5 in both cases). We again find suggestive evidence that indirect mortality had a negative impact on population between 1300-1400 (the coefficient is large and negative but imprecisely estimated). The combined effects of mortality and indirect mortality are about -1.00, and significant (not shown).[21]

We also explore how the Black Death shaped the emergence of new cities and the transition of smaller urban settlements into cities. Recall that our dataset contains 1,801 cities but that 1,335 of these cities are not present in the year 1300. These cities can be thought of as the universe of potential city locations. In column (6), we look at the effect of Black Death mortality rate on whether a city enters ours dataset in 1400. To do this we use our extrapolated mortality rate estimates. We find that cities were less likely to emerge when their extrapolated mortality rate was high. Likewise, we regress the log population of these 1,335 cities (using 500 for cities below 1,000) on mortality and find that fewer locations became urbanized in high-mortality areas (column (7)). Consistent with previous results, we find that these negative effects of the Black Death disappeared by 1600 (Panel B). This suggests that in the long-run, the Black Death did not delay the transition of villages into cities.

Finally, we investigate how the Black Death might have impacted rural areas. In Web Appx. Section 6., we use historical deforestation data to show how, in high-mortality regions, rural areas recovered slower than urban areas. Since

[21] Results hold if indirect mortality is constructed using cities of the same modern country or using all 1,801 cities but relying on the change in market potential 1300-1353 (not shown). To construct market potential in 1353, we use the predicted population of the other towns in 1353 (= pop_{1300}*(100-mort.)). Since mortality is only available for 274 cities, we use spatially extrapolated mortality rates for 1,527 cities. For each of the 165 observations, the mortality rates of the other towns are constructed excluding the mortality rate of the observation itself.

land use recovered slowly in the aggregate, it must be that marginal rural areas suffered relatively greater population losses following the Black Death. Data on the desertion of villages in England confirm this result, suggesting that many of the individuals who came to repopulate high-mortality cities and regions came from more rural areas in lower-mortality regions. In the Web Appendix we explain that this was probably driven by wages rising relatively more in high-mortality urban areas in the aftermath of the shock. Lastly, we further support this explanation using localized data on fertility regimes, confirming that migration, not fertility, was behind city population recovery.

5. Heterogeneity in City Recovery

In a Malthusian economy a pandemic might have no long-term spatial effects *on average*. Nonetheless, there could be still *permutations* among cities as some large cities become relatively smaller, and small cities, relatively larger. These permutations may in turn be affected by how mortality and the pre-pandemic characteristics of these cities interact (Hypothesis H4).

Permutations. Historical evidence suggests there was heterogeneity across cities in the response to the Black Death.[22] 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***. Hence, large cities tended to remain large and small cities tended to remain small. However, the R2 is 0.56, suggesting that aggregate recovery hides *permutations*. Figure 5(a) illustrates this, with many cities far from the forty-five degree line.

We test whether these permutations were associated with some of the characteristics considered in Table 2. We modify Eq. 1 by interacting mortality ($\text{Mort}_{i,1347-52}$) with selected city characteristics (Char_i) while controlling for the characteristics themselves and mortality:

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

Throughout, we focus on our main sample of 165 cities, for the period 1300-2015. For two cities experiencing the *same* mortality shock (e.g., 50%), the vector $\boldsymbol{\theta}$

[22] Campbell (2016, 365) notes that "towns competed with each other in an urban survival of the fittest." See Web Appx. Section 7. for a lengthier discussion of the permutations in the data.

captures the differential recovery effects of each characteristic.

Interpretation of θ. If labor becomes scarce in both cities but one city has important local factors complementary to labor, net wages should disproportionately increase there, attracting people. The city's population then recovers relative to lower-mortality cities. With high migration costs (including information costs), relative recovery will be slow enough that the negative effect of mortality may not have been fully offset by 1400 or even 1500. In the longer run, locations recovering faster thanks to advantageous characteristics may also gain a long-run productivity advantage and grow relatively faster. Thus, θ will be significant immediately after the shock and its magnitude may also increase in later periods.

An alternative scenario is when one highly impacted city has a local factor that is not valuable immediately after the Black Death shock (e.g., given the state of technology). Initially, the city will not recover faster than another equally impacted city. However, once the factor becomes valuable (which may occur a few centuries later), this helps the city escape the low-population equilibrium in which the Black Death shock put it. θ would then be small for some time before increasing in magnitude and possibly turning significant.

Factor Selection. With 165 cities, we cannot add all 27 variables of Table 2 and their interactions with mortality. Instead, we select those that proxy for: (i) *land quality*: the three agricultural suitability measures (cereal, potato, pastoral); and (ii) *access to markets and trade*: coastal and river dummies, Roman road or medieval land route intersections, and the Hanseatic League dummy. Coast and rivers lowered transportation costs. Roman roads remained the basis of the road network in the medieval era (Dalgaard et al., 2018b). Medieval trade routes reflected long-established trading linkages. We include factors proxying for (iii) *agglomeration effects* as the log of the estimated population of the city in 1353 (= pop._{1300} x (100-mort.)/100). Finally, we proxy for (iv) *institutions* using three dummies for whether the city was part of a monarchy, was a state capital, and whether it had a representative body (c. 1300).[23]

[23]Lasso regressions cannot be implemented because current Lasso programs do not allow for regression weights (we use as weights city population in 1300). In addition, one major reason to

Identification. Table 5 shows the 11 interacted effects, for 1300-1750 (col. (1)-(5)) and 1300-2015 (col. (6)). The 11 interacted effects are *simultaneously* included in the model and show the recovery effect associated with each factor *conditional* on the recovery effect associated with each other factor. 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 thus use 1300 as the start year instead of 1353 because we do not know the true population of each city in 1353.[24]

We do not use panel or IV regressions for this analysis as we have demonstrated above that these methods return results that are similar to OLS. We thus rely on the baseline OLS cross-sectional regression for its simplicity and transparency. However, since we cannot be entirely sure that mortality was indeed exogenous, the results in this section should be taken with caution.[25] For the sake of conciseness, and since what only matters are the interactions with mortality, we also do not report the independent effect of each factor.

Land Quality. The coefficient on mortality*cereal suitability is positive (but not significant) after 1400 (col. (2)). However, the implied economic impact is meaningful since the beta coefficient (henceforth, "beta") reaches 0.47 by 1600 and remains high thereafter (0.17 in 2015). The 1600 coefficient of 0.47 is half of the standardized effect for mortality. In other words, places with 1 SD higher cereal suitability recovered twice as fast as cities with poor cereal suitability experiencing the same mortality rate. Potato suitability may have also helped highly impacted cities escape their post-plague low-population equilibrium from the 17th century onwards (col. (4)). Nunn and Qian (2011) show that countries that were more suitable for potato cultivation urbanized faster after potato

use Lasso techniques is to make a model sparser in order to reduce muticollinearity. However, the 11 factors are only weakly correlated (mean correlation for the 11 x 10 ÷ 2 = 55 combinations ≈ 0.15). Lasso regressions also cannot tell us which other factors from Table 2 should be added.

[24]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 2015 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).

[25]We have 23 variables and 165 cities. Adding interactions of each factor with instruments would leave us with no variation, and mechanically creates multiple weak instruments.

cultivation diffused in Europe (the non-effects in col. (1)-(3) are reassuring).[26]

In high-mortality areas suitable for pastoral farming we find a negative effect in 1500-1600 (col. (3)) and no effects before (col. (1)-(2)). The effect in 1500-1600 is strong (beta = -0.64) and becomes weaker over time (beta = -0.25 in 2015). We believe this is caused by higher wages due to labor shortages that created incentives for landlords to specialize in pastoral agriculture, thus further reducing the need for labor (Voigtländer and Voth, 2013a, p. 2255).[27] As seen, the effect only became significant in the 16th century. Indeed, pastoral farming as a solution to labor scarcity and rising wages did not arise immediately or across Europe (Jedwab et al., 2021). This effect diminishes after 1750.[28]

Agglomeration Economies. The literature (e.g., Duranton and Puga, 2004) distinguishes economies of scale – in production, market places, and consumption – and agglomeration economies strictly defined, according to which a larger population increases productivity and wages, which should cause in-migration. In row 8 of Table 5 we interact estimated city population in the immediate aftermath of the Black Death with mortality to investigate if the population shock reduced agglomeration economies, thereby slowing recovery. We find no economically or statistically significant effects, suggesting that agglomeration economies did not play a major role in city growth at this time.

Access to Markets and Trade. The interacted effect for coastal proximity is one of the only two significant coefficients in 1300-1400 (col. (1)) along with the interacted effect for the Hanseatic league. While the coefficient of mortality is -3.9, the coefficient of mortality*coastal proximity is 1.2. Thus, relative to non-coastal cities, coastal cities recovered almost 33% faster by 1400. In 1500, the

[26]The country-level effects of Nunn and Qian (2011) appear in 1750, whereas our interacted effects 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).

[27]This effect is consistent with contemporary accounts. E.g. In 1516 Sir Thomas Moore wrote in *Utopia*, "Your sheep. . . that commonly are so meek and so little, now, as I hear, they have become so greedy and fierce that they devour men themselves."

[28]This may reflect the rise of labor-intensive proto-industry in rural areas, in particular textile production, which was associated with more rapid population growth (Mendels, 1972; Pfister, 1989). Wool was the most common textile used in making clothing.

interacted effect was strong enough that coastal high-mortality cities had on average fully recovered relative to low-mortality cities. 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 remained strong for most of the pre-industrial era (beta = 0.99 in 1500, slowly decreasing to 0.33 in 2015). By 2015, it is not significant, consistent with coastal proximity having become less of an advantage given advances in transportation technologies (e.g., railroads and roads) (Henderson et al., 2017b) and port concentration, for example due to containerization (Brooks et al., 2018).

The Hanseatic League had its origins in the 12th century. According to Dollinger (1970, viii), by the mid-13th century, Hansa merchants had a near monopoly of trade between Germany and England. By 1300 it comprised a network of cities across the North Sea, capable of boycotting trade with states that violated its rights (such as Norway between 1284-1285).[29] Hansa members negotiated lower taxes on trade for their merchants and provided them with protection from expropriation. As the network developed, cities were able to invest in infrastructure (e.g. warehouses) and armed guards for their merchants.

Our results suggest that following the Black Death cities that were part of the Hansa were well positioned to recover and this is consistent with qualitative evidence provided by historians suggesting that the period from the late 14th century to the mid-15th century was the zenith of the league. Specifically, the interacted Hansa effect is particularly strong (beta = 0.72) in 1300-1400. A coefficient of 2.9 compared to -3.9 for mortality implies that a Hansa town had already relatively recovered three fourths (2.9/3.9 = 0.74) of their population by 1400.[30] The interacted Hansa effect then remained significant until 1700 (col. (4)), by which time the league was in decline (Dollinger, 1970).

[29] Our source for this data is Dollinger (1970) and is the most comprehensive list of Hanseatic cities available. Unfortunately, Dollinger does not provide details on when each city became a member of the Hanseatic league. However, Wikipedia provides information on a subset of Hanseatic cities. Using this data, we estimate that approximately 75% of these cities were likely members of the league prior to the time of the Black Death, thus giving us confidence that our Hansa dummy mostly captures pre-plague conditions (data available on request).

[30] For a coastal Hansa town, the effect is 1.2 + 2.9 = 4.1, as much as the effect of mortality (-3.9).

Rivers exhibited positive and significant effects from the 17th century onwards (col. (4)), indicating that an increasing value of rivers might have eventually helped some highly impacted cities escape their post-plague low-population equilibrium. River transportation was important throughout the medieval period (Masschaele, 1993). But we find the interacted effects of mortality and rivers 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 might 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.[31]

Being at the intersection of two 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.[32]

The results in Table 5 suggest that cities with greater access to markets and with better geographic endowments recovered more quickly. By contrast, cities without these characteristics were more likely to stagnate in the aftermath of the Black Death—possibly taking centuries to regain their former populations. In Table 6 we provide further support for these findings by investigating whether cities with particularly strong access to these endowments exhibited even faster recovery. We also study alternative factors as robustness checks.

In regression 1 of Table 6 we examine whether specific coastal and riverine cities recovered faster. In our main analysis in Table 2 we consider cities within 10 km of the coast. 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

[31] In France, in the seventeenth century Jean-Baptiste 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 details).

[32] The effects of road intersections are stronger for Roman roads than for medieval roads (not shown). Roman roads were often stone-paved, and thus durable (Dalgaard et al., 2018b). We find insignificant negative or nil interacted effects for elevation and ruggedness (not shown).

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 interacted effects are stronger for truly coastal or estuarine locations, which we call the "best" coastal locations.

Regression 2 shows the results when we instead replace the coast dummy with two dummies for being one of the best Mediterranean coastal locations and for being one of the best Atlantic locations. We find strong recovery effects for the Mediterranean. Indeed, Mediterranean trade was particularly important around the time of the Black Death. We observe significant effects for Atlantic cities starting in the 17th century, consistent with Acemoglu et al. (2005) who show that Atlantic ports city growth began to increase around 1600. Thus, an increasing value of the Atlantic coast might have eventually helped some highly impacted cities escape their post-plague low-population equilibrium.

In regression 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 and riverine cities recovered faster when they were part of a relatively unified state (as proxied by monarchy), possibly because they served a larger market since within-state tariffs may have been lower than between-state tariffs (Cervellati et al., 2019). Overall, in regressions 1-3 we observe strong interacted effects for the best coastal locations.

In the right panel of Table 6, we further examine the role of agglomeration economies in urban recovery. To do this, we estimate a regression based on equation (3) but adding log population in 1300 and its interaction with mortality as explanatory variables. When we do this we find no effect (regression 4). We also find no effects when we proxy agglomeration using market access (regression 5) or overall state population size (regression 6).

We next examine whether factors related to potential economies of scale

played a role in recovery. We do not find positive significant effects when adding the following variables and their interaction with mortality: a dummy for the presence of craft guilds before the Black Death (regression 7; source: Ogilvie (2019)), a dummy for the presence of a market fair c. 1300 (regression 8), and log walled area c. 1300 (controlling for log city population size c. 1300) (regression 9). The presence of craft 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. City walls may also 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 examine whether a city being the seat of a bishopric or archbishopric c. 1300 (row 10) or if it was a university town c. 1300 (row 11) played a role in recovery. Both of these measures can be thought of as capturing economies of scale in human capital and institutional capacity. We only find a positive significant effect for the presence of a university in 1600. This is unsurprising as many European universities were largely training grounds for the Church and did not teach commercial or engineering skills (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 other parts of 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.). We find for the 16th and 17th centuries negative recovery effects in areas with a stronger attachment to serfdom (regression 12). The effect in 1750 is economically large (-10.9), about half the effect of mortality itself (about -21).

Country Fixed Effects. Are the interacted effects we have presented causal? In all our regressions, we *simultaneously* control for mortality, the individual effects of the 11 characteristics used for the interactions, and the 11 interactions. The effects also "activate" and "deactivate" when expected. In addition, most of the important effects we discuss in Table 5 remain strong and significant when including 13 modern country fixed effects (Web Appx. Table A.10). Identification then comes from comparing cities experiencing the same initial shock, having the same 11 characteristics, and belonging to the same entity. With 165 observations, 23 variables and 13 fixed effects, this makes our test more stringent.
Interpretation. Of our four categories of factors potentially contributing to city recovery, which of them explains the most variation? When we add the absolute values of the beta coefficients for the different types of factors shown in Table 5, land quality and trade potential are particularly important in the first centuries after the Black Death and their importance decreases over time (Fig. 6(a)). The other factors (institutions and agglomeration) are less important.[33]

Table 1 showed no long-term effects of the Black Death at the city level. In Table 5, the significant negative effects of mortality once we control for the factors and their interactions with mortality implies that any city without these 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 such factors, a large Black Death shock would likely have permanently reduced the size of the city relative to cities with better endowments.

Finally, for our 165 cities existing in 1300, we predict which high-mortality cities would have relatively suffered by 1750 or 2015 if they were, counterfactually, not endowed with good factors for recovery. We do this by, first, using the estimated models in Table 5 to predict the counterfactual population level of each city in 1750 or 2015 absent good endowments. In doing this, we account

[33] 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 identification strategy requires us to study the effects of pre-, not post-, plague institutions.

for the fact that predicted percentage population growth in 1300-1750 or 1300-2015 must be left-censored at -100 by estimating Tobit models. The effects on the latent variable are almost the same as with 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 (0.79) with the actual 1750 rank of each city among the 165 cities. For 2015, the correlation is weaker – at 0.61 – due to factors appearing in the Industrial Era. We then perform the same analysis to predict the 1750 or 2015 rank of each city had the interacted effects of mortality and the fixed factors been set to 0. Comparing the predicted ranks of each city excluding the recovery effects of the factors with their predicted ranks when the same effects are included, we identify cities that would have lost a lot had such factors not mattered.

As seen in Figure 5(b), examples of such cities include some of the largest cities in our sample of 165 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%) (see Web Appx. Fig. A.7 for 1750). What these cities all have in common is that they were hit hard by the plague and were either coastal, riverine, located on a road intersection, or part of the Hanseatic league.

Implications. Our results suggest that people disproportionately abandoned (or chose not to move to) cities with worse land quality or trade potential. Thus, the relative population decline of more poorly endowed areas suggests that the growth potential of Europe's urban population might have improved in the aggregate due to the Black Death. Indeed, if, among the 165 cities that existed in 1300, cities with better land or trade potential recovered faster, we should observe over time higher shares of the total population of these 165 cities living in locations with such advantages.

For example, high-mortality cities along a river recovered faster than non-river cities from the 17th century. As a result, an increasing share of the total

urban population must have resided along a river over time. Figure 6(b) shows that the unconditional population share of the 165 cities located along a river did increase from 1400-2015. It also reports the predicted increase in the population share of riverine cities based on their population in 1300 and the estimated contribution of the recovery effect of rivers to predicted city growth (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 and explains almost all of the unconditional share.

We do not further pursue the possibility that the Black Death led to a beneficial "urban reset" as this would require a theory of what a dynamically optimal distribution of population involves.[34] 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 emerged between Europe and the rest of the world and the Little Divergence that took place within Europe.

6. Conclusion

Our main findings can be broken down into short-run effects of the Black Death and the long-run effects of the Black Death. In the short-run, the Black Death had a massive impact on the population of cities. Between 1300 and 1400 a 10 percentage point higher mortality rate was associated with a 8.7 percentage point fall in city population. In the long-run, however, there was recovery and the aggregate effect of the Black Death on city population was zero.

We provide evidence that plague mortality was largely locally exogenous (Hypothesis H1) and that our results are plausibly causal (Hypothesis H2). First, we establish that the virulence of the Black Death was unrelated to factors related to future city growth. Second, we show that the parallel trends assumption is verified. Third, we use a panel regression accounting for city effects. Fourth, we employ several instrumental variables strategies premised on the facts that: (i) the Black Death entered Europe through the Sicilian port of Messina (largely by chance) and was more virulent in its earlier stages (for pathogenic reasons);

[34] This result echoes the work of Michaels and Rauch (2018).

(ii) it was specific connectedness to Messina and not connectedness to other important cities that mattered for plague virulence; and (iii) the Black Death was more lethal in cities in which it reached its peak in the summer since the fleas that transmitted the disease were more active then.

We find that mortality rate in a city affected not just that city but also the city's "neighbors" (Hypothesis H3), suggesting that the Black Death led to market disintermediation and regional decline in the medium-run. This aggregate recovery, however, masks substantial heterogeneity. Cities did not recover their exact position in the urban hierarchy, as Davis and Weinstein (2002) find for Nagasaki and Hiroshima. Some cities permanently collapsed after the Black Death whereas other cities gained. This heterogeneous recovery is best explained by factors associated with land quality and trade potential (Hypothesis H4).

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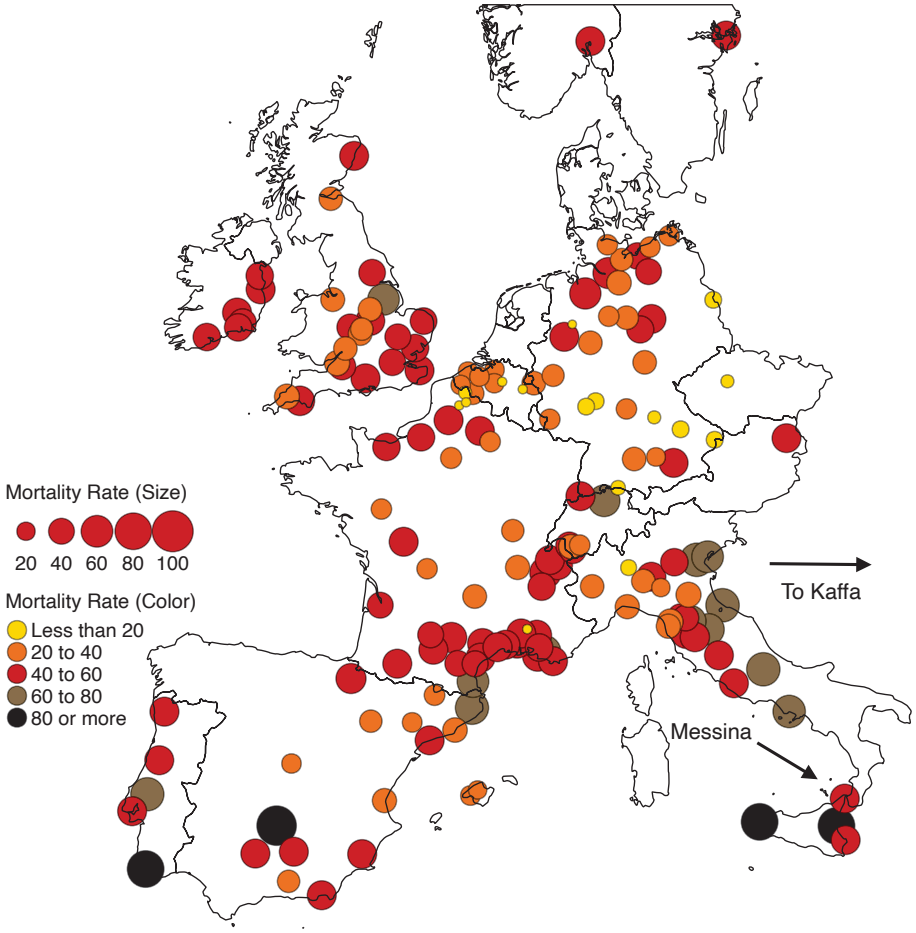
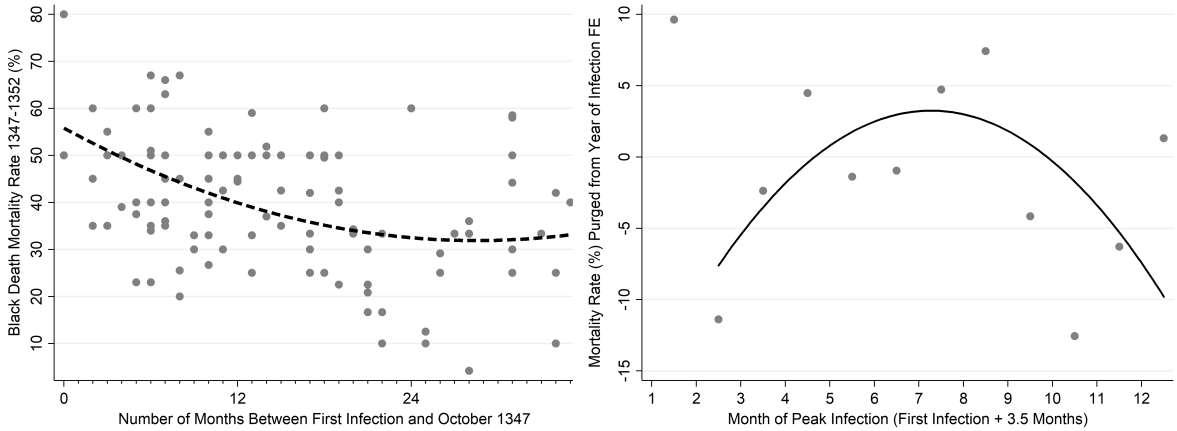
Figure 1: Black Death Mortality Rates (%) in 1347-1352



Notes: This map plots the location of all 165 existing cities (i.e. loc. $\geq$ 1,000 inh.) in 1300 for which we know their Black Death mortality rate (%) in 1347-52 and the modern boundaries of the 16 Western European countries of our analysis.

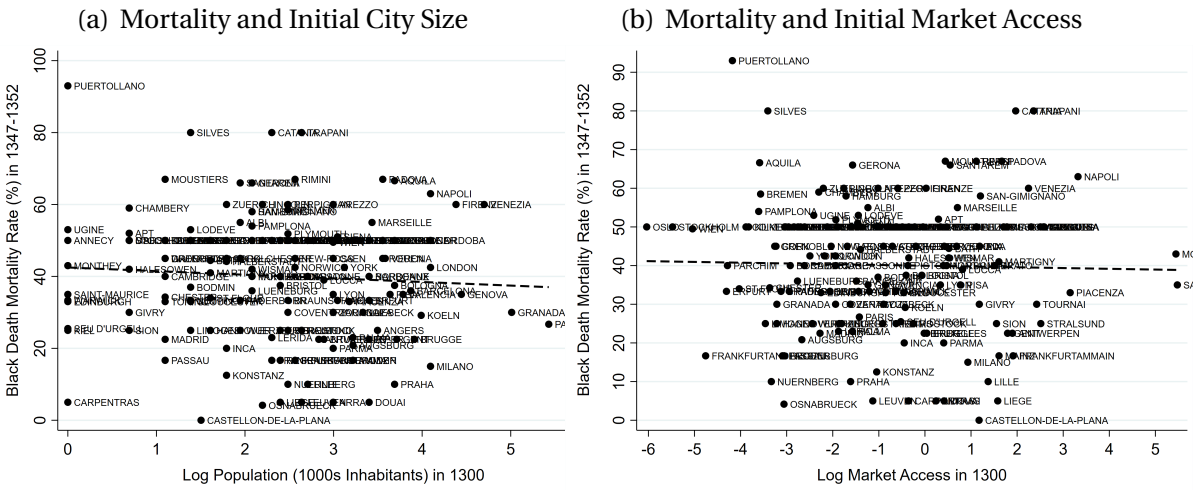
Figure 2: Timing of the Onset of the Black Death and Black Death Mortality

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



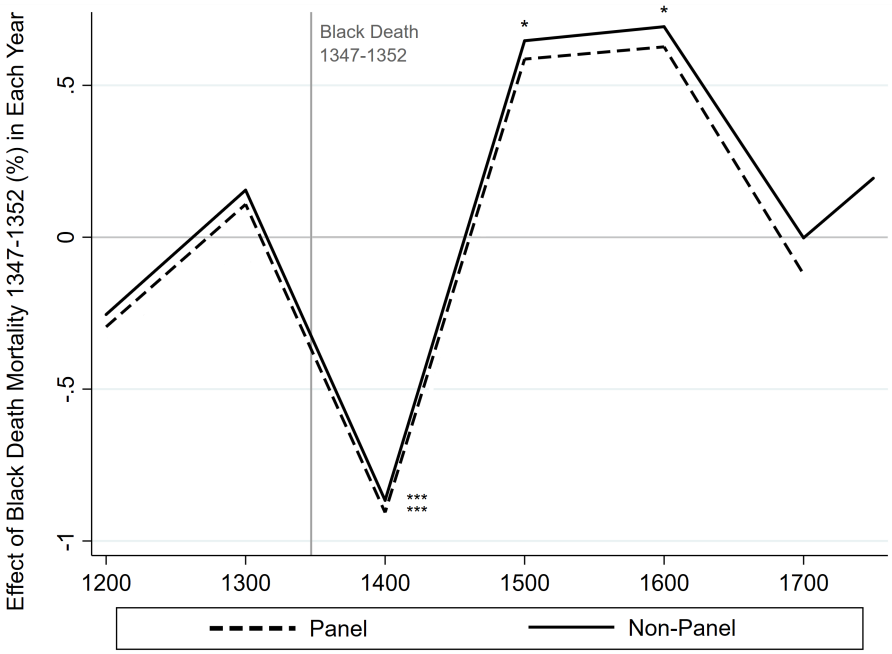
Notes: Subfig. 2(a) shows for 124 cities the relationship between mortality and the timing of the onset of the Black Death in the city. Number of months is measured since Oct. 1347, the date Messina – the port of entry of the Black Death in Europe – was infected by mischance. Subfig. 2(b) shows for 124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

Figure 3: Mortality Rates, City Size and City Market Access in 1300



Notes: Subfig. 3(a) shows the relationship between mortality (%, 1347-1352) and log city pop. in 1300 for our main sample of 165 cities (Y = 42.5*** -1.01 X; R2 = 0.00). Subfig. 3(b) shows for the same 165 cities the relationship between mortality (%, 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 pop. of town $j \neq i$, D_{ij} the travel time between city i and city j, and $\sigma = 3.8$. To obtain the travel times, we compute the least cost travel paths via four transportation modes — by sea, by river, by road and by walk — with the transportation speeds from Boerner and Severgnini (2014).

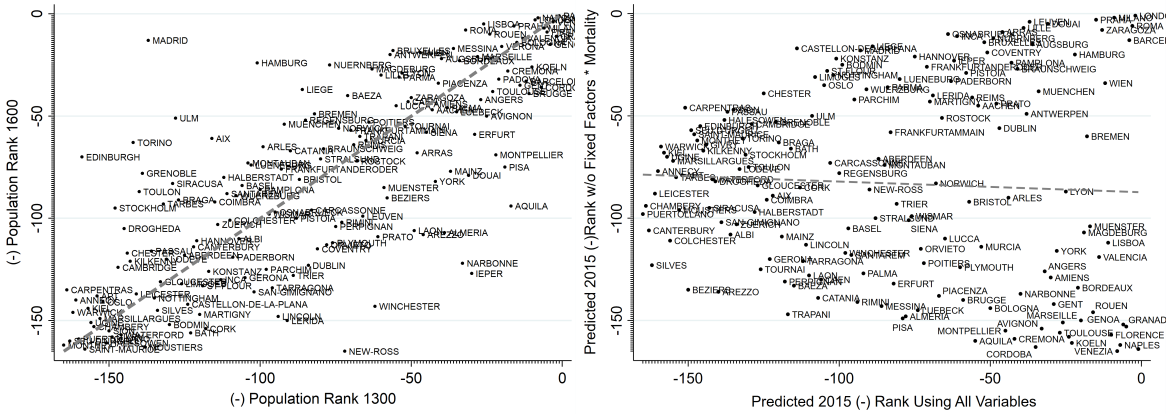
Figure 4: Yearly Effect of Black Death Mortality 1347-52 (%), Panel Regressions



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

Figure 5: Permutations in the Distribution of Cities and Fixed Factors

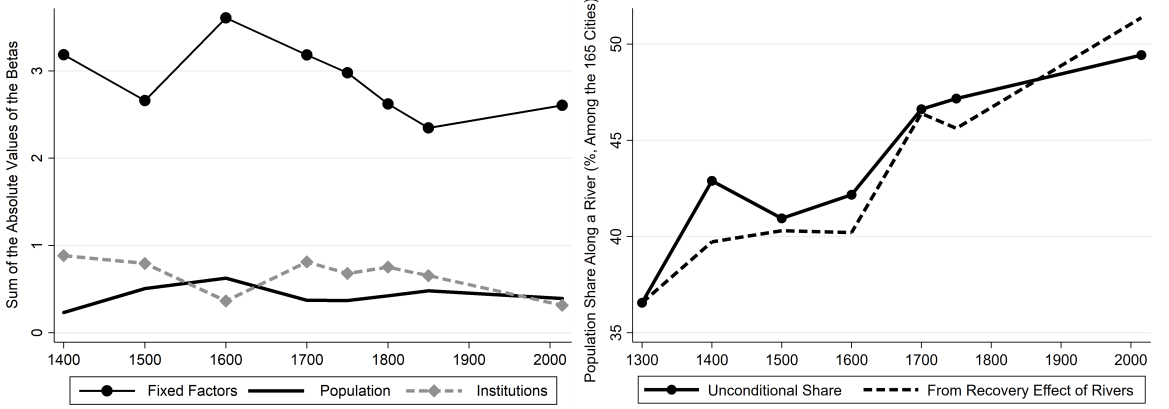
(a) Changes in Pop. Ranks Among the 165 Cities (b) Fixed Factors & Counterfactual Ranks 2015



Notes: Subfig. 5(a) shows for the 165 cities of the main sample the relationship between their inverted pop. rank in 1300 (among the 165 cities; 0 = largest city) and their inverted pop. rank in 1600 (among the 165 cities; 0 = largest city). Subfig. 5(b) shows the relationship between the predicted 2015 inverted pop. rank (among the 165 main cities; 0 = largest city) when using their respective populations in 1300 and the Tobit-estimated regression results of Table 5 (incl. the independent effects of the variables shown at left) and the predicted 2015 inverted pop. rank (0 = largest city) when ignoring the interacted effects of mortality with the fixed factors (cereal, potato, pastoral, coast, rivers, road, Hanseatic).

Figure 6: Total Contribution of the Fixed Factors to Aggregate Patterns

(a) Total Contribution of the Fixed Factors (b) Population Share Along a River



Notes: Subfig. 6(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). Subfig. 6(b) shows the percentage share of the total pop. of the 165 main cities that is located along a river in the raw population data ("unconditional") and based on 1300 population × the recovery effect of rivers to predicted city population growth ("from recovery effect of rivers").

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

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

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. 12 5x5 Degree Cell FE (for Cell >= 5 Cities)	-0.64**	[0.32]	140	-0.26	[0.63]	139
6. IV1: Timing w/ Controls (IV F-stat = 11.8)	-1.07**	[0.50]	124	0.05	[1.32]	124
7. IV2: Messina w/ Controls (IV F-stat = 22.6)	-1.20**	[0.56]	163	-0.68	[1.69]	163
8. IV3: Month w/ Controls (IV F-stat = 6.0)	-0.93***	[0.33]	124	-0.23	[0.58]	124
9. IV1+IV2+IV3 w/ Controls (IV F-stat = 6.8)	-1.29***	[0.39]	123	0.53	[0.58]	123

Notes: Row 2: Adding the controls of Table 2. Row 3: Adding 13 country FE. Rows 4-5: Adding 44 state FE or 26 cell FE but excluding states/cells with less than 5 cities. Row 6: IV = number of months between the city-specific date of first infection and October 1347. Row 7: IV = Euclidean distance to Messina, controlling for (population-weighted) average Euclidean distance to European and MENA cities in 1300 (Messina is dropped). Row 8: IV = 11 dummies for the month of peak infection (= month of onset (October is omitted) + 3.5). Robust SE's (clust. at state (1300) level in rows 6-9): * p<0.10, ** p<0.05, *** p<0.01.

Table 4: MORTALITY AND CITY GROWTH, AGGREGATE EFFECTS, 1300-1600

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel A: Dep. Var.:	% Change in Total City Population, 1300-1400			% Change City Pop., 1300-1400		Dummy if Exists 1400	Log City Pop. 1400
Mortality Rate (%)	-0.87***	-1.15***	-1.47**	-0.72**	-0.68**	-0.002***	-0.004***
	[0.28]	[0.40]	[0.57]	[0.31]	[0.33]	[0.001]	[0.001]
Mort. Other Cities (%)				-0.39	-0.57		
				[0.43]	[0.47]		
Sample	City	State	State	State	Dist. 10%	City	City
Population	Intensive	Intensive	Total	Other	Other	Extensive	Intensive
Observations	165	68	68	165	165	1,335	1,335
Panel B: Dep. Var.:	% Change in Total City Population, 1300-1600			% Change City Pop., 1300-1600		Dummy if Exists 1600	Log City Pop. 1600
Mortality Rate (%)	0.36	-1.49	-1.34	-0.08	0.16	-0.001	0.002
	[0.80]	[1.32]	[3.17]	[0.82]	[0.84]	[0.001]	[0.002]
Mort. Other Cities (%)				1.49	0.60		
				[1.50]	[1.78]		

Notes: (1) Baseline city-level regressions. (2)-(3) State-level regressions: We run the main regressions at the state (1300) level. Intensive / Total: The cities considered to construct total city pop. are the cities that already existed in 1300 / all cities. (4)-(5) Indirect mortality: State: Avg. mortality rate of other cities in the same state (1300). Indirect mortality: Dist. 10%: Avg. mortality rate of other cities within the bottom 10% of Euclidean distance to the city. (6) Extensive: We consider 1,335 cities that did not already exist in 1300 but existed at one point in Bairoch (1988) (800-1850). Robust SE's: * p<0.10, ** p<0.05, *** p<0.01.

Table 5: BLACK DEATH MORTALITY AND POPULATION RECOVERY, 1300-2015

Dependent Variable: Percentage Change in City Population (%) in Period 1300-t						
Period 1300-t:	1400	1500	1600	1700	1750	2015
All Variables Simultaneously Included:	(1)	(2)	(3)	(4)	(5)	(6)
Land Quality:						
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]
Access to Markets & Trade:						
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]
Institutions:						
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**	-374**
	[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 the 165 cities the effects of Black Death mortality (%) interacted with 11 selected factors (the 11 interacted effects are simultaneously included). We only show the interacted effects and the effect of mortality but the 11 factors are also used as controls. We use as weights city population in 1300. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01.

Table 6: BLACK DEATH MORTALITY AND CITY POPULATION RECOVERY: GEOGRAPHY, AGGLOMERATION EFFECTS, AND INSTITUTIONS

Dependent Variable: Percentage Change in City Population (%) in Period 1300-t									
Period 1300-t:	1500	1600	1700	1750		1500	1600	1700	1750
	(1)	(2)	(3)	(4)		(5)	(6)	(7)	(8)
Each row is for a separate regression and shows the effect of Mortality (%) $\times$ the variable shown at left									
1. Truly Coastal	2.8***	4.4**	6.7**	7.2*	4. 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**	5 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	6 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**	7. 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*	8. 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**	9. 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	10. 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*	11. 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	12. 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 is similar to Table 5 (where we include Black Death mortality, the 11 factors, and their interactions with Black Death mortality) except we also add the variable(s) shown at left and show the effect(s) of its (their) interaction(s) with Black Death mortality (%). We use as weights city population in 1300. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01.

WEB APPENDIX - NOT FOR PUBLICATION

Contents

1. Black Death Mortality Rates and Spread

Mortality Rates. Our data on cumulative Black Death mortality rates (%) for 274 localities for the period 1347-1352 are based on the estimates collected by Christakos et al. (2005) which come from a wide range of historical sources. We verify and supplement these where possible with data from other sources including Ziegler (1969), Russell (1972), Pounds (1973), Gottfried (1983), and Benedictow (2005). These localities belong to 16 countries in Western Europe using today's boundaries. We obtain percentage estimates for 177 of these 274 localities. For example, Cologne, Granada, and Zurich had estimated cumulative Black Death mortality rates of 35%, 30%, and 60% respectively.

For the 97 other localities the sources report more qualitative estimates: (i) For 49 towns Christakos et al. (2005) provide a literary description of mortality. We rank these descriptions based on the supposed magnitude of the shock and assign each one of them a numeric mortality rate: 5% for "spared" or "escaped", 10% for "partially spared" or "minimal", 20% for "low", 25% for "moderate", 50% for "high", 66% for "highly depopulated", and 80% if the town is "close to being depopulated" or "decimated"; (ii) For 19 towns, we know the mortality rate of the clergy. Christakos et al. (2005, p.138) cite Ziegler (1969), who argues that "it would be reasonable to state as a general rule that the proportion of benefited clergy who died in any given diocese could not possibly have been much smaller than the corresponding figure for the laity and is unlikely to have been very much bigger. Arbitrary limits of 10% less [mortality among benefited clergy] and 25% more [mortality among benefited clergy] seem to provide a reasonable bracket within which the correct figure must be encompassed." This suggests

that clergy mortality was only 8% higher than general mortality. We thus divide the clergy mortality rates by 1.08 to obtain mortality for these 19 towns; and (iii) For 29 towns we know the desertion rate which includes both people who died and people who never returned. Christakos et al. (2005, p.154-155), using data on both desertion rates and mortality rates available for 10 towns, show that the desertion rate is on average 1.2 times higher than the mortality rate. We thus divide the desertion rates by 1.2 to obtain the mortality rate of these 19 towns.[1]

How did Christakos et al. (2005) compile this information? First, they examine the nature of the information available for each location. Information on the intensity and timing of the plague comes from the following types of sources: (1) ecclesiastical records; (2) parish records; (3) testaments; (4) tax records; (5) court rolls; (6) chronicles by contemporaries; (7) donations to the church; (8) financial transactions; (9) deaths of famous individuals; (10) surviving letters; (11) edicts; (12) guild records; (13) hospital records; (14) new cemeteries; (15) tombstones; (16) abnormal increases in adoptions. For each type of data, Christakos et al. (2005) discuss issues of veracity or selectivity.

Second, each data point in our analysis has been examined by historians and by Christakos et al. (2005). Where possible, different sources of data are cross-tabulated. In particular, when the quality of the underlying information was suspect, they describe their method as follows:

> When systematic error is likely to be significant and the sources are contradictory, it becomes important to use some kind of logical cross-validation (using, e.g., the reasoning rules of Tables II.2 and II.3), check original sources, get a deeper understanding of the underlying assumptions, and investigate the guesswork behind some of the numbers. When gathering data for this study, we never ceased recording evidence at a given locality by assuming that there was enough information already. In this way, by the end of the information acquisition stage of the SEP method we collected approximately 2,500 typed literal transcriptions making more than 300 pages of text. When the time came to use the information bases, a systematic search for redundancies and logical inconsistencies was employed to discard questionable data and rigorously assess the reliability of the resulting mortality values. In the event of agreement among the produced numbers, our general tendency was to quote the original source, the first author to publish the original data, or the scholar who was most knowledgeable about the specific region [...]

Indeed, sometimes the estimates provided by medieval chroniclers are unreliable. For example, Christakos et al. (2005, 124) note that

> "In Lübeck (Germany) some chroniclers described a state of complete hysteria in the streets and about 90,000 casualties by the time the plague was over. Independent assessments give Lübeck a population of about 25,000 residents on the eve of the Black Death outbreak, and there are more credible but partial accounts of the passing away of 11 of the 30 city councilors, 2 out of the 5 town clerks and 27% of the property owners—roughly a death rate of 1 in 3. Therefore, most likely the actual casualties were in the order of 9,000 instead of 90,000."

In general, however, the assessment of modern historians is that the high estimates provided by contemporaries are often accurate. The modern historical and scientific consensus is that mortality rates during the Black Death were very high (around 33–50%). This vindicates contemporaries and runs against mid-20th century historians like Russell (1948) or Shrewsbury (1970) who suggested

[1] These data are the same as those used in Jebwab et al. (2018).

much lower numbers. Overall, there is considerable variation in mortality rates. It was estimated to be only 10% in Alsace, Lorraine, and Bohemia (Gottfried, 1983). However, Cambridgeshire had an estimated mortality rate of between 53-70%. Rural mortality rates were then at least as high as urban mortality rates. Benedictow (2005) provides a range of estimates for some but not all parts of Europe. Christakos et al. (2005) also report scattered estimates of rural mortality.

Spread of the Black Death. We use the data from Christakos et al. (2005) to obtain for 124 cities among the 165 cities of our main sample the year and month of first infection in the town (the day is almost never available). Information is sparser for the year-month of last infection and thus the duration of the epidemic in each city and only available for 61 cities among the 165 main cities. The distribution of the duration of the Black Death in each city is shown in Web Appendix Figure A.2.

Extrapolated Mortality Data. In order to extend our analysis to cities for which we don't have explicit mortality rates we use spatial analysis to impute the missing values. Our assumptions in doing this are that (1) there exist some underlying causes of mortality rates which are unobserved, (2) these causes have a large random component (i.e. are external to our model of subsequent city growth), (3) these causes are also spatially correlated. For example, it is widely acknowledged that fleas living off of rat populations were a primary vector for the plague. It is highly plausible that a latent variable measuring the suitability of a city's surrounding region for sustaining large rat populations satisfies the three criteria laid out above.

In order to impute the missing mortality rates we create a two-dimensional surface of predicted plague mortality using an inverse distance weighted function of known mortality rates (Shephard, 1968). We specifically use the IDW procedure contained in the gstat package for R, documentation for which can be downloaded from: https://cran.r-project.org/web/packages/gstat/gstat.pdf. For a point on the surface, x, with unknown mortality the influence of city, i, with known mortality diminishes with its distance from x according to the weight used. This weight is given by a parameter, $p \geq 0$, referred to as *power*. As the power decreases, the influence of more distant points increases. If $p = 0$, then all points receive equal weight in determining all other points on the map. The influence of more distant points decreases exponentially as p increases.

To create our mortality estimates we choose an optimal p using cross-validation techniques. The procedure begins by choosing some power, $\bar{p}$. Then, using the sample of n cities with known mortality rates, we create a predicted mortality rate surface using all of the cities except for city j. We then predict the mortality rate for city j as $\hat{m}_j$ using our mortality surface and create it's residual as $(\hat{m}_j - m_j)$ where m_j is the known mortality rate for city j. This procedure is then repeated to create predicted mortality rates and residuals for all the remaining cities. From this, we calculate the Root Mean Square Error (RMSE) of the residuals created as $\text{RMSE}(\bar{p}) = \sqrt{\frac{\sum_{i=1}^{n}(\hat{m}_i - m_i)^2}{n}}$, where i indexes the cities with known mortality rates. This procedure is repeated for a large number of choices of p and then the optimal power is chosen as the one which minimizes the RMSE.

We generate optimal mortality surfaces using several different city samples. Our baseline sample consists of cities for which mortality rates are reported in the historical literature. There are 274 of these cities and the cross-validation exercise chooses an optimal power for creating the mortality surface as 1.76 (RMSE=15.186). Note that for the regressions in Web Appendix Table 4 (the spillover regressions) we take the additional step of leaving out the "own" city when calculating the predicted mortality surface so as not to contaminate the calculated mortality "spillover" for city i with the variance in mortality from city i. As such, when creating these data we perform the above optimization procedure 165 times—once for each city in our main sample.

2. City Population Estimates

Our main source of data is the Bairoch (1988) dataset of city populations. For the 16 European countries of our full sample (Austria, Belgium, the Czech Republic, Denmark, France, Germany, Ireland, Italy, Luxembourg, Norway, Poland, Portugal, Spain, Sweden, Switzerland and the United Kingdom), the dataset reports estimates for 1,726 cities between 800 and 1850. The criterion for inclusion in the dataset is a population greater than 1,000 inhabitants.

This dataset has been widely used by a range of scholars. We follow Bosker et al. (2013) and Voigtländer and Voth (2013b) in updating the dataset where a consensus of historians have provided revised estimates of the population of a particular city, including Bruges, Paris, and London. Indeed, while the Bairoch data set is our main source of information, Chandler (1974, 1987) is more specific in the sources used to measure city population. In this way we can better assess the "true" population of each city in each year. For example, we prefer Chandler's population estimates for a range of cities including Granada, Paris, Venice, and Milan. We use this corrected dataset as our benchmark. We also employ the Bairoch dataset and the Chandler dataset in our robustness exercises.

The Bairoch dataset reports city populations every century more or less (800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1750, 1800, 1850). Therefore, we have population estimates for our entire sample both for 1300 and for 1400. This provides an important benchmark for our analysis but is far from ideal as population growth or shrinkage may have taken place between 1300 and 1347-1352 and population recovery may have occurred between 1347-1352 and 1400.

We then collected data on estimates of pre- and post-Plague populations for cities not in the Bairoch data set from a range of historical sources. Chandler (1974, 1987) provides alternative estimates on city populations including estimates for city sizes in other decades of the 14th century. Christakos et al. (2005) summarize a wide range of historical estimates of pre-Plague populations. We used their data and consulted several of the sources in the secondary literature to provide checks on their estimates of pre-Plague population including Ziegler (1969), Russell (1972), Pounds (1973), Gottfried (1983), Nicholas (1997) and Benedictow (2005). Doing so allows us to add 76 cities to our analysis. Our full sample then has 1,726 + 76 = 1,802 cities. However, we drop Ponte Delgada, in the Azores, since this city is too far away from the rest of sample. In the end, our full sample has 1,801 cities.

Of the 165 cities in our main sample one exits the dataset between 1400 and 1600. This in New Ross in Ireland; it reappears after 1600. Of the 466 cities in the dataset in 1300, only 9 exit the sample after 1400. We verify that these cities were amongst the smallest cities in the dataset in 1300. As very few city exit the sample, we are confident that our data captures the dynamics of Western Europe's urban development over this period. Finally, data on the modern (circa 2015) population of the 165 cities of our main sample is obtained from the Wikipedia webpage of each city. Wikipedia mostly reports census estimates for both the city itself and the agglomeration it may belong to. Since urban areas have grown a lot since 1850, and cities became agglomerations, we use modern population estimates for the city itself instead of the agglomeration.

3. Control Variables and Other Variables

Average Temperature 1500-1600. We use temperature data from Luterbacher et al. (2004). They reconstruct seasonal European temperatures (celsius degrees) since 1500 using proxy data from ice cores, tree rings, and written records. The data cover 0.5° x 0.5° grids which is approximately 50km x 50km at European latitudes. The data extend from 25° W to 40° E and 35° N to 70° N which includes all of the cities in our full sample. We extract the growing season (summer) temperature for each of

our cities during the 16th century as this is the closest century to the Black Death period for which we have data. No comparable data exist for earlier centuries.

Elevation. City elevation data come from Jarvis et al. (2008) which is available at http://srtm.csi.cgiar.org. These data report elevation in meters with a spatial resolution between 1 and 3 arc-seconds. Where there are missing data we have supplemented it using Wikipedia.

Cereal Suitability. Our soil suitability data are from the FAO Global Agro-Ecological Zones (GAEZ) dataset as described in Fischer et al. (2002). We use these in preference to Ramankutty et al. (2002) as the latter does not have full coverage for all of western Europe. We use the GAEZ's cereal suitability data assuming low inputs and rain-fed irrigation. We extract the average soil suitability within 10 km radius circles around each city. Overall, cereal suitability is scaled from 1-9 where 1 is best, 8 is unsuitable and 9 is water (seas and oceans are treated as missing values). In some regressions, we invert the measure so that positive values are associated with higher cereal suitability.

Potato Suitability. The potato suitabliity numbers are constructed using the Global Agro-Ecological Zones (GAEZ) data. We specifically use the data on white potatoes grown under conditions using low inputs and rain-fed irrigation for the baseline period 1961-1990. The raster file for the data along with support documentation are available for download from: http://www.fao.org/nr/gaez/newsevents/detail/en/c/141573/. These data are constructed in two stages. First the Food and Agriculture Organization (FAO) compiles information on the nutrients, soil, irrigation, and climatic conditions under which the potato grows best. Then the FAO compiles data on the physical environment for the entire world at a resolution of 5 arc minutes x 5 arc minutes ($\approx$ 10 x 10 Km). These characteristics include soil type, slope, average water availability, humidity, temperature, wind speed, etc. Then these two types of data are combined in order to create a value for "potential suitability for potato cultivation" for each raster cell. These values run from 1 to 9, where 1 is most suitable, 8 is least suitable, and 9 is water (or impossible to cultivate). See Monteduro (2012) for more details on the construction of the suitability raster. We use the GAEZ data to construct our city-level measures of potato suitability by extracting the average value of the raster cells within a 10 km radius of each city.

Pastoral Suitability. We control for the potential suitability of a region surrounding a city for pastoral farming with a variable measuring grazing suitability. This variable come from Erb et al. (2007) who create land use measures at a resolution of 5 arc minute cells ($\approx$ 10 km X 10 km). They record how land is used in each cell in 2000. The five categories they code for are: cropland, grazing, forest, urban, and areas without land use. Their grazing category is calculated as a residual after accounting for the percentage of area taken up by the other four uses. As part of this analysis they also generate a variable measuring the suitability of each cell for grazing (as opposed to actual present-day use). The suitability measure is created by first separating grazing land into three categories based on cover: 'high suitability of cultivated and managed areas, medium suitability of grazing land found under tree cover, and low suitability if shrub cover or sparse vegetation is detected in remote sensing' (Erb et al., 2007, 199). They then further subdivide the first two of these categories into areas with a net primary productivity of Carbon per meter squared is greater than 200 grams and those in which it is less than 200 grams. This results in five categories which they regroup into four categories with 1 = most suitable and 4 = least suitable. There is a fifth category which is 'no grazing' which we re-code as 5. We then create a dummy equal to 1 if the cell is most or moderately suitable. Finally, we extract the average suitability of the region around a city for grazing using circles of 10 km's.

Coasts and Rivers. We create variables to measure distances to the coast and major rivers using

ArcGIS. We base these distances on the 1300 shape file downloaded from Nussli (2011). We then create two dummies for whether each city is within 10 km from the coast or a river.

Market Access. Market access for city i in 1300/1353 is defined as $MA_i = \Sigma_j \frac{P_j}{D_{ij}^{\sigma}}$, with P_j being the population of the other 1801 - 1 = 1800 cities $j \neq i$ in 1300/1353, D_{ij} the travel time between city i and city j, and $\sigma = 3.8$. To obtain the travel times, we use the gdistance package in R to 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).[2] More on the gdistance package is available for download at this link: https://cran.r-project.org/web/packages/gdistance/vignettes/gdistance1.pdf. The predicted population of each city in the aftermath of the Black Death (1353) is constructed as = pop. in 1300 x (100-mortality)/100.

Roman Roads. Data on Roman roads is provided by the *Digital Atlas of Roman and Medieval Civilizations*. We use this shape file to create two distances: (1) distance to all Roman roads and (2) distance to 'major' Roman roads. Since major settlements often formed along intersections of the road network, we also create variables for distances to Roman road intersection. We then create four dummies if the city is within 10 from any Roman road, a major Roman road, any Roman road intersection, or a major Roman road intersection.

Medieval Trade Routes. We use Shepherd (1923) to create a map of major medieval land trade routes. We create a GIS file that allows us to measure the distance to major medieval land trade routes or the intersection of two of them. We then create dummy variables that take the value of 1 if a city is within 10 kilometers of a trade route or an intersection of two of them.

Market Fairs. We obtain data on the location of important medieval fairs from two sources. The main source is Shepherd (1923). The second source we use is the *Digital Atlas of Roman and Medieval Civilizations*. The original source for this information is: Ditchburn, David and MacLean, Simon (eds.) 2007, Atlas of Medieval Europe, 2nd edn, London and New York, p. 158. We drop fairs that they cannot be matched with cities in the Bairoch dataset.

Hanseatic League. We document whether or not a city was a member of the Hanseatic League. We do this by matching where possible the city data with available lists of cities which belonged to the League. We include only cities which were members of the League and do not include cities with Hansa trading posts or communities. The source we use is from Dollinger (1970) and is the most comprehensive list of Hanseatic cities available. Unfortunately, Dollinger does not provide details on when each city became a member of the Hanseatic league. However Wikipedia provides information on a subset of Hanseatic cities. Using this data, we estimate that approximately 75% of these cities were likely members of the league prior to the time of the Black Death, thus giving us confidence that our Hansa dummy mostly captures pre-plague conditions.[3]

Aqueducts. We use GIS to create a shape file for whether or not a town was within 10 km from a Roman aqueduct using the map provided by Talbert, ed (2000) as well as information from two Wikipedia webpages: https://en.wikipedia.org/wiki/List_of_aqueducts_in_the_Roman_Empire and https://fr.wikipedia.org/wiki/Liste_des_aqueducs_romains.

Medieval Universities. Bosker et al. (2013) provides data on the presence of medieval universities for European cities with populations greater than 10,000 (at some point between 800 and 1800).

[2] Normalizing the speed to porters to 1, this assigns a travel cost of 0.5 to roads and rivers and 0.18 to seas.
[3] Data available on request.

We consulted Wikipedia and other sources to find evidence of medieval universities with smaller populations. There are five medieval universities missing from the list in Bosker et al. (2013): Angers, Greifswald, Ingolstadt, Tuebingen, and Uppsala. However, as none of these were established prior to the Black Death we do not include them in our analysis.

Monarchy in 1300. We construct information on whether or not a city was ruled by a major kingdom using the shape files provided by Nussli (2011) who report political boundaries in Europe for every century. We then assign each city to its political boundary in 1300 by hand. We assign a city as belonging to a monarchy in 1300 if it belonged to the Kingdom of Bohemia, the Kingdom of Denmark, the Crown of Castile, the Kingdom of France, the Kingdom of Norway, the Kingdom of England, the Kingdom of Sicily in Naples, the Kingdom of Granada, the Kingdom of Scotland, the Kingdom of Hungary, the Kingdom of Sicily, the Kingdom of Galicia-Volhynia, the Crown of Aragon, the Kingdom of Portugal, the Kingdom of Majorca, the Kingdom of Sweden.

State Capital in 1300. We use the data provided by Bosker et al. (2013) who collect data on capital cities from McEvedy and Jones (1978).

Representative Body in 1300. Bosker et al. (2013) provide information on the existence of communes for a subset of the cities in the Bairoch dataset. Bosker et al. (2013) create a variable"commune" that takes a value of 1 if there is indication of the presence of a local urban participative organization that decided on local urban affairs. Stasavage (2014) provides data on 169 cities that were autonomous at some point between 1000 and 1800. We use the variable for 1300-1400. Stasavage (2014) defines autonomous cities in the following terms:

> 'I have defined an "autonomous city" as being one in which there is clear evidence that such institutions of self-governance existed, and in addition there is also clear evidence of exercise of prerogatives in at least one of the policy areas referred to above. In the absence of such evidence the default is to code a city as non-autonomous (6).'

As Stasavage (2014) notes, his definition of city autonomy is stricter than the definition of commune used by Bosker et al. (2013). We create a dummy equal to one if the city is a commune in the Bosker et al. (2013) data set or a self-governing city according to Stasavage (2014).

Parliamentary Activity and Distance to Parliament 1300-1400. Our data on parliamentary activity is from van Zanden et al. (2012). This measures the number of times that Parliaments met at a regional level in 1300–1400. We create a dummy variable based on whether or not a town is in a region/country which had above the median number of parliamentary meetings. We also obtain a list of whether the parliaments were held for each region/country. We then use GIS to compute for each city the minimal Euclidean distance to a parliament.

Battles. As our main source we use Wikipedia's list of all battles that took place between 1300 and 1600. https://en.wikipedia.org/wiki/List_of_battles_1301-1800. This is a highly reliable source for the most important battles of the period. We are not concerned about sample selection here as Wikipedia's coverage of European history is extensive; battles not listed on Wikipedia are likely to have been extremely small. For each battle we assign geo-coordinates based on either the location of the battle or the location of the nearest city mentioned in the entry. Note that we exclude naval battles.

States 1300. We know from Nussli (2011) which state each city belonged to circa 1300.

Plague Recurrences. We use the data from Schmid et al. (2015) based on Biraben (1975) to obtain the location of Plague recurrences. We then use GIS to obtain the Euclidean distance from each city in our

sample to each plague recurrence, which allows to recreate various controls.

Jewish Presence, Persecutions, Pogroms 1347-1352. Data on whether a Jewish community was present at the onset of the Black Death (1347) and whether a persecution, and a pogrom in particular, took place during the Black Death (1347-52) come from Jebwab et al. (2018).

Land Use. Kaplan et al. (2009) construct localized data on land use from 1000 BCE to 1850 at the 5 by 5 minute (i.e., 10 x 10 km) grid-cell level by combining information on country population, historical forest cover maps, and maps of soil suitability. We then obtain the mean land use share (%) for all gridcells within 10 km from each of the 165 cities.

Mortality, Population, and Deserted Villages for English Counties. England had 41 counties during the medieval period. For 28 of these we know from Shrewsbury (1970) and Scott and Duncan (2001) the mortality rate of the clergy which we use as a proxy for overall mortality. From Fenwick and Turner (2015), we know the total number of deserted medieval villages (DMVs) in each county. For 39 of them we also know the precise location of each DMV. Finally, we obtain from the same sources as before the population of each county in 1086, 1290, 1377, 1756 and 1801.

North-EMP. We use 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., Northern, Central and Southern France) to classify our 165 cities as "Strong EMP" or "Weak-EMP". We also classify the countries that the 165 cities belong to into "North" vs. "South". "North" includes Austria, Belgium, France, Germany, Ireland, Norway, Sweden, Switzerland, and the United Kingdom.

Guilds Our data on guilds is from Ogilvie (2019).[4] We use "Qualitative Database" which contains 12,051 observations on guild activities across 23 countries between 1095 and 1862.

We construct three variables based on the database. "Guild 1" is equal to 1 if there is mention of a guild engaged in some activity in the city before the Black Death (1347) and 0 otherwise. "'Guild " is equal to a 1 if there is mention of Guild activity either before the Black Death or sometime during the 14th century. "Guild 3" is equal to 1 if there is mention of guild activity any time after the Black Death.

Serfdom We code whether or not serfdom was present in 1800 based on Acemoglu and Robinson (2012, p. 276). This is an extremely crude proxy, as it is based on modern country borders, rather than on historical borders. But for our purposes, matters is simply the nearby presence of coercive labor institutions that could retard labor mobility. Therefore the population growth of a city might still be affected by the presence of serfdom in nearby regions.

Descriptive Statistics. The summary statistics for the variables of Tables 1-9 are shown in Web Appendix Table A.1 (the variables are shown as they appear in the paper).

4. Spread of the Black Death and Exogeneity

Web Appx. Fig. 3(a)-Web Appx. Fig. 3(d) show the spread of the Black Death in the first year of the pandemic (October 1347-September 1348; for 124 cities for which data is available).

Web Appx. Fig. 3(a) depicts the first cities affected by the plague. That is, the cities hit during first three months (months 0-2) that the plague was in Europe. These included Sicilian cities and Genoa, Marseille, and Venice. Marseille was hit before other larger Mediterranean coastal cities such as Naples, Barcelona and Valencia (about 35,000 vs. 60,000, 50,000 and 45,000 c. 1300).

[4]This data is available at: http://www.econ.cam.ac.uk/people/faculty/sco2/projects/ogilvie-guilds-databases

Web Appx. Fig. 3(b) shows the cities that were infected by plague in months 3-5. In Italy, this includes inland cities such as Arezzo and Siena (20,000) that were impacted before larger cites such as (coastal) Naples (60,000) and (inland) Rome (30,000). In Spain, this includes (inland) Lerida and (coastal) Palma that were impacted before larger cites such as (coastal) Barcelona (50,000) and Valencia (45,000).

Web Appx. Fig. 3(c) shows the cities that were newly infected in months 6-8. The inland city of Rouen (35,000) in Northern France was hit before other large cities "along a path" from the Mediterranean basin to Rouen: Cordoba (60,000), Libson (35,000), Bordeaux (30,000), and London (60,000).

Web Appx. Fig. 3(d) shows the newly infected cities in months 9-11. Paris, the largest city in Europe, and London were finally impacted, but other cities among the largest in Europe had still not been impacted by then (e.g., Cordoba and Cologne). Some cities were then impacted earlier than other cities due to idiosyncrasies. For example, Bristol was infected but Plymouth, better located, was not.

5. The Black Death as Bubonic Plague

Traditional histories relied on general estimates of the mortality associated with the Black Death such as Boccaccio's estimate that a third of humanity died during the plague years. However, since the 1960s micro studies by historical demographers have provided a range of city or region-specific mortality rates that we employ for our analysis.

Importantly for our study, mortality rates during the Black Death period were unrelated to population size and density. Benedictow notes that 'it is a unique feature of' the Bubonic plague 'that the densities of rats and rat fleas overrule the effects of the density of the susceptible human population that is the decisive factor for the dynamics of epidemic spread in the case of all diseases that spread directly between human beings by cross infection' (Benedictow, 2005, 284).

The strength of this assertion is based on evidence from outbreaks of modern Bubonic plagues in the late 19th and early 20th centuries. Modern bubonic plague was spread by the fleas that live on black rats. These fleas only target humans when their hosts are dead. Rats are territorial animals. In rural areas a single rat colony may cohabit with a single household. However, in urban areas people live closer together and the ratio between rats and humans tends to be lower. As Benedictow argues:

> 'This epidemiological model provides a basic explanation for how plague may wreak havoc after having arrived at some small-scale residential unit, and why, in the case of plague, severity of impact on human population does not increase with mounting density of human settlement' (Benedictow, 2005, 33)

This argument rests on the identification of the Black Death with modern bubonic plague. However, for a long time this was contested by scholars. Twigg (1984) was the first to question this identification arguing that plague diffusion was too fast for bubonic plague and pointing to an absence of references to rats dying in medieval sources. Similarly, Cohn (2003) argued that the seasonal timing of mortality during the Black Death was incompatible with that disease being the bubonic plague.

However, modern scientific research has identified the DNA of skeletons from mass graves associated with the Black Death and demonstrated that they were positive for *Yersinia pestis* (Haensch et al., 2010; Bos et al., 2011; Schuenemann et al., 2011). This decisive evidence means that we are confident in following Benedictow (2005) in arguing that the Black Death was bubonic plague on the basis of what we know about outbreaks of bubonic plague in the modern period.

Following the Black Death (1347-1352) bubonic plague remained endemic in Europe for the next 250 years (Biraben, 1975; Alfani and Murphy, 2017). These outbreaks are studied by Biraben (1975) and

recently by Siuda and Sunde (2017) and Dittmar and Meisenzahl (2019).

Epidemiologists, scientists, and historians dispute why the plague returned over and over again. The older position was that subsequent plague outbreaks were caused by reinfection from local plague spores. It is possible, as recent work indicates, that later plague outbreaks may have been due to the reintroduction of the bacterium from Asia.

Note that the Black Death pandemic differed significantly from reoccurrences of the plague in later years. After 1400 the plague tended to affect only urban areas. In general, subsequent outbreaks of the plague were less virulent than the initial outbreak in 1348-1353 (Aberth, 2010, 37), though the outbreak of bubonic plague in Italy in the 1630s was unusually damaging (Alfani, 2013). Moreover, until the first pandemic they were mostly localized.

6. The Countryside and Mechanisms of City Recovery

6.1. Effects on the Countryside

The Countryside. Urban areas differ from rural areas in that their production process is disproportionately less dependent on fixed geographical factors, in particular land. In the Malthusian model, any decline in labor-to-land ratios temporarily increases agricultural incomes, until population increases, mostly as a result of higher net fertility (Galor, 2005, 2011). As the demand for urban products increases, the urban sector expands, due to rural-to-urban migration. In their calibrated model, Voigtländer and Voth (2013b) assumes that "migration into cities was unconstrained [after the Black Death] because many city dwellers had died" and that "city population can reach its pre-plague level immediately." In their appendix, they also show simulation results assuming a slower transition. More generally, in the macroeconomic and historical literatures, the urban sector recovers because rural areas recover. However, there is, to our knowledge, little spatial econometric evidence on the latter, which is what focus on now.

Land use data provides a proxy for rural population. Indeed, the Plague led to reforestation as the need for land and wood declined and marginal soils were abandoned (Campbell, 2016, 363) (Web Appx. Sections 11.-12.). Kaplan et al. (2009) recreate data on land use from 1100 to 1850 at the 5 by 5 minute (10x10km) cell level by combining information on country population, historical forest cover maps, and soil suitability. Using these data, we obtain the land use share of the in-sample countries, i.e. the share of the land used for crops vs. naturally forested (forests were not managed). The share was two thirds by 1300 and decreased by 15 percentage points by 1400. Land use recovered by 1800.[5]

We obtain the mean land share within a 10 km radius of each of the 165 cities and examine how land use varied.[6] Web Appx. Table A.12 shows that mortality led to reforestation in 1300-1400, which remained significant until 1500. No effect is found after 1600.[7] Overall, cities thus recovered their populations by 1500 and their rural areas recovered theirs by 1600 (Fig. 9(b)). This is consistent with the mechanism emphasized by Voigtländer and Voth (2013b): higher incomes led to cities recovering faster than the countryside. One can also envision different mechanisms: trade-related fixed factors

[5]Total population data comes from McEvedy and Jones (1978) who rely on fiscal records and ecclesiastical registers at the state level. It is thus not constructed using city populations.

[6]Since country populations are one input used in the creation of these data, we verify land use changes are not mechanically correlated with population changes. The correlation between the change in land use within 10 km of a city and the change in the population of that city is 0.14 in 1300-1400 and -0.03 in 1300-1600. The correlations with the change of that city's country population are higher, but still low, at -0.30 and 0.44 respectively. Indeed, changes in the forest maps also cause local land use changes. Lastly, results hold whether we control (Panel A) or not (Panel B) for the contemporaneous changes in both city population and country population.

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

in cities – e.g., a good location on the coast – means that urban wages increase when urban population declines, thus raising the demand for food and driving rural recovery *locally*.

Next, recovery in high-mortality areas must have been driven by either differentially increasing fertility and decreasing mortality in high-mortality areas or migration from low- to high-mortality areas. Since rural areas close to cities also recovered, migrants must have come *on net* from rural areas farther away. Thus, marginal rural areas suffered relatively greater population losses following the Black Death. 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, which we test now.

Deserted Medieval Villages (DMVs). Historians document how the Plague led to the desertion of villages (Beresford, 1954; Braudel, ed, 1965). Peasants left their villages to seek newly available economic opportunities in high-mortality cities. This immigration "topped up otherwise diminishing urban communities" (Platt, 1996, 20) (see Web Appx. Section 13.). Since labor was in short supply and peasants demanded better pay, many landowners switched to sheep rearing, which required less labor than arable farming (Voigtländer and Voth, 2013a).

Data on the location of DMVs exist for all 41 English counties during the medieval era (Fenwick and Turner, 2015). For 28 of these, we know from Shrewsbury (1970) and Scott and Duncan (2001) clergy mortality, which we use as a proxy for overall mortality. This allows us to study how the number of DMVs varied with mortality, *depending* on their proximity to cities. Note that Voigtländer and Voth (2013a) use the same data to show that areas with more DMVs specialized in pastoral farming, a different question to ours.

Since this sample differs from the sample of 165 cities, we verify in col. (1)-(3) of Table A.11 that mortality had a negative effect in the short run and no effect in the long run. For the same 28 counties, we obtained population in 1086, 1290, 1377, 1756 and 1801 (data unavailable ca. 1600). For the period 1290-1377, we find a negative effect, at -0.64** (col. (1)). For the period 1290-1756, we find an insignificant effect, at -0.96 (col. (2)), but the effect is smaller than the short-run effect once standardized (beta coef. of -0.10 vs. -0.35 in 1290-1377). When using 1801 (first census), the standardized effect is small (-0.08, not shown). We check parallel trends and find no effect in 1086-1290 (0.05, beta coef. of 0.00, col. (3)). This result implies local exogeneity of the Plague for total population levels, which were then mostly explained by rural population levels given England's low urbanization rate. We also discussed in Section 2. why the epidemiological characteristics of the Black Death – e.g., the fact it was Bubonic plague and black rats mattered – meant that rural mortality did not differ from urban mortality.[8]

In col. (4)-(6) the dependent variable is the log number of DMVs per 1000 sq. km. We control for the county's log area and log population in 1290 since the density of DMVs depends on pre-Plague human density.[9] We find a negative effect of mortality, at -0.46*** (col. (4), beta coef. of -0.51).[10] Low-mortality areas had *more* DMVs than high-mortality areas. Therefore, people disproportionately left the relatively Plague-free rural areas. Web Appx. Table A.12 showed that rural areas in the vicinity

[8]We weight observations by their population in 1290 and exclude Cornwall whose population in 1290 is underestimated due to the lack of data on their large mining population (see Broadberry et al. (2010, 14)). Middlesex is not included in the analysis, due to the lack of data on its mortality

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

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

of cities—within 10 km—were not affected by the Black Death in the long-run. By 1600 they were completely repopulated. We should thus expect relatively more DMVs in low-mortality areas *beyond* 10 km from cities. For 39 counties, we obtain from Fenwick and Turner (2015) the location of each DMV in England and compute the minimal distance to a 1300 city. For each of the 28 counties, we construct the number of DMVs (per 1000 sq km) both within and beyond 10 km from a city. We verify in col. (5)-(6) that the loss of villages is driven by areas farther away from cities.[11] In col. (7), we regress the absolute change in the urban share (%) on mortality and find a small and insignificant negative effect. DMVs were small. Thus, the loss of villages in low-mortality areas may have not been large enough to affect urbanization patterns across counties. This also suggests that the repopulation of high-mortality areas was allowed by migration from both urban and rural areas in low-mortality areas.

6.2. Urban Wages and Migration

Wages. Data on real wages 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. Instead, Web Appx. Section 9. provides qualitative evidence on wage patterns after the Black Death. Overall, the historical literature has consistently found that in cities where mortality was high living standards on average significantly 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 reduced fertility (Voigtländer and Voth, 2013a). Thus, natural increase likely played a minor role in *local* recovery. Migration must have been the source (Web Appx. Section 10.).

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; Voigtländer and Voth, 2013b; 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". Likewise, the number of freeman admitted into York increased by 365% in the year of the Plague (Dobson, 1973, 17). London saw a "great concourse of aliens and denizens to the city and suburbs, now that the pestilence is stayed" (Sloane, 2011).[12]

Fertility. For a same mortality shock, we should expect fertility to be lower in a Northern Europe region or a region characterized by the EMP (Dennison and Ogilvie, 2014; Voigtländer and Voth, 2013a). We thus test if the speed at which high-mortality cities relatively recover depends on where the cities were

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

[12] Migration was caused by both an improved bargaining position of peasants *and* an increase in labor coercion in some areas forcing peasants to escape these (Web Appx. Section 8.).

located. We classify our cities into North vs. South or Strong EMP vs. Weak EMP (based on historical data from Dennison and Ogilvie (2014) on the average age at first marriage and the female celibacy rate (%) at the country or regional level).

For the 165 cities, we use the same specification as before but interact mortality with a North dummy (col. (2)-(3)) or a Strong EMP dummy (col. (4)-(7)) and test if the interacted effect is negative. Indeed, if natural increase was important for local recovery, we should expect high-mortality cities in North or Strong EMP regions to recover slower than high-mortality cities in South or Weak EMP regions, because North or Strong EMP cities were more likely to recover solely through migration whereas South or Weak-EMP cities were more likely to experience both migration *and* natural increase.

However, three important caveats with the EMP analysis ((4)-(7)) is that the EMP measures from Dennison and Ogilvie (2014) are estimated for the post-Black Death period (1500-1900). In addition, while the EMP may have already existed before the Black Death (Laslett and Wall, 1972), Voigtländer and Voth (2013a) showed how the EMP was affected by the Black Death. Lastly, the EMP had a particular impact in rural areas (Voigtländer and Voth, 2013a) and it is unclear how much it could also capture urban fertility differentials. As such, the results focusing on the simple North/South distinction ((2)-(3)) are more reliable.

Web Appx. Table A.13 shows the interacted effects in 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/regions with an age at first marriage or a female celibacy rate above the mean or median in the sample. The interacted effects show that Northern cities did not recover relatively slower, since the coefficients are positive, *not* negative, thus suggesting that migration was likely the main driver behind recovery. We then find overall similar results for the EMP dummy. However, given the data limitations, we acknowledge that our econometric evidence is limited.

7. Rising and Declining Cities After the Black Death

As we discuss in Section 6.2., the Black Death led to various permutations in the distribution of city sizes. Some cities declined following the Plague, others rose to prominence.

In England there are two well-known instances of cities that declined after the Black Death: Winchester and York. Winchester was the old capital of Anglo-Saxon England and already in decline before 1348. The pace of its decline increased after the Black Death. This can be seen in the number of churches which fell from 57 in 1300, to 52 in 1348, and then 33 in 1400 and 12 in 1600 (Platt, 1996).

York, in contrast, was prospering before the Black Death and initially continued to do so for a few decades afterwards before declining in the 15th century (Kermode, 2000a). By the 1520s, the population of York was smaller than it had been prior to 1348. Similarly, the town of Grimsby in northeastern England lost 30% of its population in 1349. However, between 1377 and 1524 its population fell by a further 40% (Platt, 1996).

York had been a military, religious, and governmental capital for hundreds of years. Its economy was diversified and it was the center of the medieval textiles industry. But in the 15th century it declined as a mercantile center. Competition from rural industry in the countryside and the rise to prominence of London and the Hansa cities meant that York was marginalized and its industrial base collapsed. By 1548 its population was only 8,000 (Kermode, 2000b, 677).

In France, Montpellier had a population of 35,000 in 1300. It was struck hard by the plague, experiencing a 50% mortality rate. Its population did not recover: in 1400 it was 17,000, a 45% decline.

It fell from being the 4th largest French city to being the 20th. Moreover, the decline of Montpellier continued for centuries. By 1480, the population had fallen further to 13,000 (Nicholas, 1997). The city did not exceed its population in 1300 until 1850.

Other cities recovered rapidly and then boomed following the Black Death. Hamburg had a population of about 8,000 individuals in 1300. It was struck severely by the Black Death, experiencing a mortality rate of approximately 58%.[13] However, in the subsequent half-century it was growing so rapidly that it had a population of 22,000 by 1400. By the seventeenth century, Hamburg was a major center of international trade (Lindberg, 2008).

8. The Weakening of Serfdom

Serfdom weakened in Western Europe following the Black Death. For instance, in England prior to the Black Death around 50% of the population were serfs of some kind. Their mobility was restricted and lords were able to extract the entire surplus beyond that required for subsistence in the form of labor dues or fines. The number of serfs fell after the Black Death from 2 million to around 1 million or 35% of the population in 1400 and to just a few thousands (or a minuscule percent of the total population) by 1500 (Bailey, 2014, 4)

But while serfdom disappeared in Western Europe, it did not disappear in Eastern Europe. This observation has puzzled numerous scholars. Domar (1970) argued that serfdom emerged where and when labor was scarce. But others pointed out that scarcity increased the bargaining power of workers.

Postan (1972) argued that the demographic shock of the Black Death improved the bargaining powers of workers while reducing the value of land and that this enabled laborers to bargain for better conditions, thereby eroding the entire institution of serfdom. Historians influenced by Marxism emphasized class conflict more directly. They stressed the power of either laborers or landlords as the crucial factor (Hilton, 1969; Brenner, 1976; Hilton, ed, 1976). According to this set of arguments, institutional factors were more important than demographics and relative factor prices and a period of seigniorial reaction was able to prolong serfdom for decades after the Black Death (this debate is surveyed by Bailey (2014)). For Brenner (1976), a purely demographic model was insufficient to explain the *decline* of serfdom in western Europe following the Black Death; this phenomenon required studying political power and class relations. According to this argument, labor scarcity increased the bargaining power of laborers contributing to a crisis of surplus extraction. This crisis, in turn, brought about a switch from serfdom to rental contracts and wage labor.

Theoretically, Wolitzky and Acemoglu (2011) show one can reconcile both the demographic and the class-based arguments. Wolitzky and Acemoglu (2011) build a principal-agent model to study the relationship between labor scarcity, outside options, and labor coercion. In this framework, coercion and effort are complements. Hence, when labor is scarce, there is a stronger incentive to employ coercion. However, conditions of labor scarcity also improve workers' outside options, which reduces the incentive to use coercion.

Empirically, many historians have claimed that the Black Death had little immediate impact on social institutions such as serfdom but this is not supported by the most up-to-date and rigorous analysis. Bailey (2014) systematically studied a selection of 28 English manors from East Anglia and Oxfordshire/Buckinghamshire. He concludes that serfdom was in sharp decline from the 1350s onwards. This evidence suggests that labor scarcity and failing land values were critical to the decline

[13] Ziegler (1969, 86) estimates the death toll in Hamburg as between a half and two-thirds.

in serfdom rather than other factors such as manumission or peasant resistance. On the manors studied by Bailey there was no attempt to reimpose serfdom after the Black Death.

9. Real Wages Following the Black Death

We discuss how real wages changed after the Black Death in Section 2.. Consistent with the standard Malthusian model, in the long-run real wages rose following the Black Death shock. However, it is important to note that the initial period following the pandemic was one of economic crisis and that in the immediate aftermath of the Black Death real wages fell.

Trade routes were disrupted. The deaths of a large proportion of the population meant that food was left rotting in the fields and crops were not harvested. These shortages resulted in inflation. As Robbins observes: "The high degree of mortality had as an indirect result a serious effect on money and prices. The grain rotted in the fields for want of men to harvest it. The price of food was doubled" (Robbins, 1928). In France the disruption of the Plague occurred simultaneously with the devastation of the Hundred Years War and the devaluation of the currency so the economic collapse was prolonged and scholars are unsure when real wages began to rise (Perroy, 1955).

The best real wage data we have is from England. Nominal wages increased rapidly. Wages for laborers went from 12d before the Plague to 28d in the 1350s (Routt, 2018). Real wages, however, only increased in the 1380s as Munro (2003) demonstrates. The reason was both inflation and that during the 1350s and 1360s landlords were able to limit wage demands and labor mobility as they "benefited from substandard harvests during the 1350s and 1360s, which both buttressed prices for grain and denied the peasants full benefit of the newfound demographic advantage by containing real wages even as nominal wages climbed" (Routt, 2013, 476).

The historical literature emphasizes that the relationship between mortality and economic recovery was mediated by institutions. Areas hit hard by the plague could either decline or thrive afterwards; similarly some areas that were largely spared such as Bohemia did not benefit economically.

One reason why wages did not immediately respond to the scarcity of labor following the Black Death was wage restraint. Political elites across Europe tried to prevent wages from rising. The Statute of Laborers passed in 1349 sought to limit nominal wages. In France a comparable statute was passed in 1351 to regulate wages, prices, and to regulate guilds admittances. In Florence wages were permitted to rise for urban workers but not for rural labors who faced particularly stringent regulations and saw their real wages fall as they had to purchase basic commodities at "hyper-inflated prices" (Cohn, 2007, 468). Individuals who left their farms to seek new work were fined.

However, many of the regulations designed to prevent wages from rising proved impossible to enforce:

> 'Judging from the records in accounts of the universal payment of wages above the limits set in the Statute of Labourers, the law was broken each year by hundreds of thousands of workers, but after a very large number of cases were brought in some areas in the early 1350s, the justices in normal years dealt with only a few hundred offenders in each county for which we have information' (Penn and Dyer, 1990, 359).

Perroy noted that

> "it still remains unknown how far the legal rates of wages were actually enforced. In general they seem to have been rarely observed. If labour was plentiful, employers would pay less than the rate, using subterfuges for not openly breaking the law" (Perroy, 1955, 236).

Rising nominal wages may disguise the actual increase in the price of labor. Historians observe that

workers after the post-Black Death period were often paid in kind. For example, Penn and Dyer (1990, 371) notes that

> "An Essex ploughman in 1378, for instance, was offered a new tunic, and the use of the lord's plough on his own land as well as 20S. in cash and 43 quarters of grain per annum. Others were tempted with extra gratuities in cash, and concessions that would not be apparent in the manorial accounts. In 1372 a canon of Sempringham (Lincolnshire) offered a shepherd both an illegal extra 2S. per annum in cash, and the right to keep on the lord's pasture four more sheep of his own than he had been allowed by his previous employer."

In the longer-run, the effect on real wages was very significant. Real wages in England continued to rise during the 15th century peaking in the 1450s. Pamuk (2007, 292) observes that:

> "Even a cursory look at real wage series makes clear that modern economic growth and the Black Death are the two events that led to the most significant changes in wages and incomes during the last millennium".

10. Demographic Change After the Black Death

Sections 2. and 6.2. discusses demographic changes after the Black Death. In general demographic trends in the preindustrial period were affected more dramatically by death rates than by birth rates. Birth rates fluctuated between around 25 and 35 per thousand. Death rates went from around 25 per thousand to as high as 400 per thousand during the Black Death.

Demographers have long argued that the Black Death set in motion a series of changes that led to a distinctive European Marriage Pattern (EMP) (Hajnal, 1965). This was a unique low pressure demographic regime. Many scholars argue that the EMP was an important factor in the economic rise of Northwestern Europe (Moor and Zanden, 2010; Foreman-Peck, 2011; Voigtländer and Voth, 2013a).

There are several reasons for this. The EMP is thought to be characterized by greater balance between marriage partners, less parental authority, and nuclear households. Moor and Zanden (2010) argue that the EMP encouraged female autonomy, women's participation in labor markets and greater investment in human capital. In particular, the EMP was associated with the rise of animal husbandry and the practice of young people working as servants in other households prior to marriage, developments that are seen as crucial in the emergence of more "capitalist" or market-orientated practices in northwestern Europe.

To measure the intensity of the EMP we draw on Dennison and Ogilvie (2014), who compile a dataset of 4,705 observations drawn from 365 research studies in European demography. This provides them with 2,622 observations of female age of first marriage which we use as our primary measure of the EMP. They also have 1,172 observations of female lifetime celibacy which we use as a secondary measure of the intensity of the EMP. In contrast to earlier studies which identify England and the Netherlands as the nexus of the EMP, the evidence collected by Dennison and Ogilvie (2014) suggests that many European societies had elements of the EMP and that England and the Netherlands were not the "purest" manifestations of this system of household formation. The most extreme forms of the EMP were to be found in Scandinavia and Germany.

11. Rural to Urban Migration After the Black Death

In Section 2. we discuss the role played by migration in repopulating Europe's cities following the Black Death. We note in the main text, the historical literature suggests that the recovery of European cities was largely driven by migration from the countryside.

The initial impact of the plague was to generate an "urban crisis" (Nicholas (1999, 99) and Hohenberg (2004, 14)). Following the Plague numerous cities encouraged migration. For example, Orvieto gave immigrants automatic citizenship rights with no taxes or requirement to join the army for 10 years (Cohn, 2007). Tax exemptions are also recorded in Moravia and elsewhere. Summarizing, Bryne notes that "Immediate citizenship, tax and service exemptions, free housing, high wages, business subsidies, and immediate guild membership were among the perks offered (Byrne, 2012, 313-314).

In Suffolk, Bailey (2007, 182) reports that the

> "new economic conditions increased both the opportunity and the ability of individuals and whole families to move in search of land and/or work. The under-supply of tenants, lower rents, and easier land tenures all encouraged mobility, yet the incentive for Suffolk residents to migrate was enhanced by the rapid growth of new industrial activities."

There were attempts to regulate labor movement in order to keep wages low, the Statute of Laborers discussed above. But workers still were able to move and to find better work. Hence the numerous fines levied on workers for breaking contracts and moving testify to the ineffectiveness of laws designed to keep them in place: workers "calculated that it was more profitable to risk low fines in order to make much more lucrative new contracts" (Cohn, 2007, 470).

12. Land Use Change After the Black Death

Another widely studied consequence of the Black Death was the reforestation of Europe (van Hoof et al., 2006; Yeloff and Geel, 2007; Skog and Hauska, 2013).

In the immediate aftermath of the Black Death, land was often just abandoned. In Suffolk, Bailey (2007, 180-181) reports that

> "The majority of the land abandoned by the victims of the Black Death remained unwanted for most of the year in Brandon and Timworth; 55 per cent of abandoned land parcels were still untenanted in September 1349 at Horham; and by November heirs been admitted into only 18 per cent of such land parcels in Cornard Parva — although heirs had been identified and ordered to take up their inheritances in 36 per cent of cases, no heirs or tenants could be found for the remainder"

It took years for the land market to recover. When it did so land use changed. Grain farming became less profitable. Landlords had an incentive to shift towards pastoral agriculture where possible and to abandon marginal plots of farmland where it was not.

Reforestation was a consequence of this retreat of the "agricultural frontier" (Bavel and van Zanden, 2004, 516). There is evidence for particularly strong reforestation in central Europe. Elsewhere the reforestation may have been more modest. Data collected by Poos (1991) for medieval Essex suggested a significant decrease in the mean acreage of arable farmland and a small increase in land use for pasture and for woodland between the early 14th century and the late 14th century.

Another factor in reforestation was that the rise of cities increased the demand of wood— to be used as a fuel—. Population pressure before the plague led to a short cropping cycle (with a mean and mode of 7 years). But after the Black Death this cropping cycle became extended (mean = 11; mode = 8). This less intensive system of woodland management can help to explain the recovery of woodlands that occurred in the late Middle Ages (Galloway et al., 1996, 454-455).

13. Deserted Medieval Villages

Migrants came from the countryside. The consequence was the desertion of many villages and rural areas. Thousands of villages disappeared across late medieval Europe. For example:

> "The village of Elkington in Northamptonshire, for example, contained thirty taxpayers in 1377 but by 1412 seems to have been almost depopulated. In the early sixteenth century all the village's arable was used for pasture" (Dodds, 2008, 75).

Some villages were suffering from depopulation even before the Black Death. The Black Death then delivered a major demographic blow. As Dyer (2002, 23) notes the mortality associated with the Black Death was unlikely to directly cause an entire village to be deserted but "it could have weakened settlements and created opportunities for migration". He gives the example of Tusmore in Oxford which was deserted by 1357. Other desertions occurred more gradually in the 1360s and 1370s and later and people gradually moved away in search of better economic activities. This process could take place over a century or more. These desertions were closely linked to changing land use, notably the shift from arable to pastoral farming.

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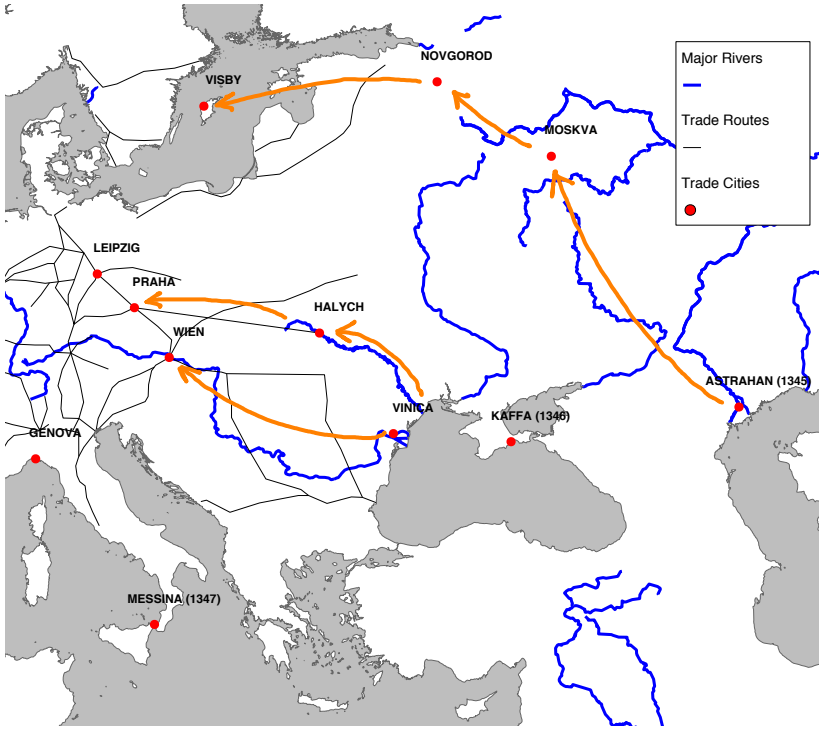
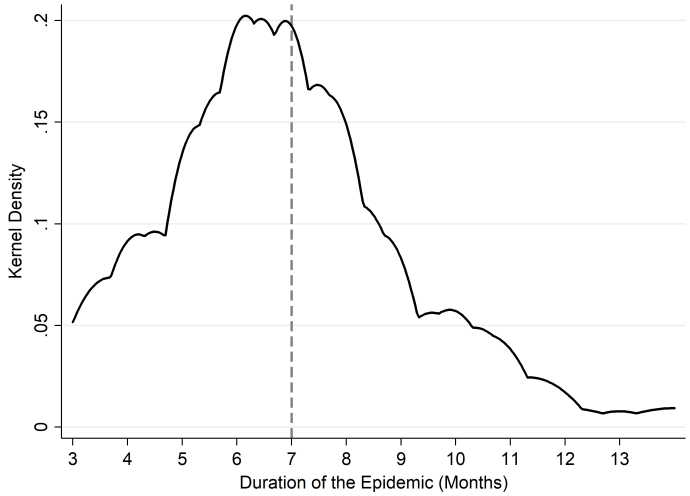
WEB APPENDIX - NOT FOR PUBLICATION

Figure A.1: Actual and Counterfactual Ports of Entry in Eastern and Western Europe



Notes: This map plots the location of the main port of entry in Eastern Europe, Astrakhan (Ukraine) in 1345. The Plague went from Astrakhan to the Genoese colony of Kaffa (Ukraine) in 1346. From Kaffa, it was carried to Messina (Sicily) in 1347. The map also shows various counterfactual ports of entry in our sample of 16 countries: Genoa (since the boat that stopped in Messina was initially supposed to go to Genoa), Vienna, Prague, Leipzig and Visby (Sweden). We argue that the Plague could have spread from Kaffa (i) to the Genoese colony of Vinica and then Vienna via the Danube; or (ii) to the Geneose colony along the Dniester river and then Halych (Ukraine) all the way to Prague and Leipzig (via the Via Regia, an important trade route then). Alternatively, it could have spread from Astrakhan to Moscow (via the Volga) and then Novgorod (via minor rivers not shown in the map) and then the Gulf of Finland, in particular Visby (Sweden).

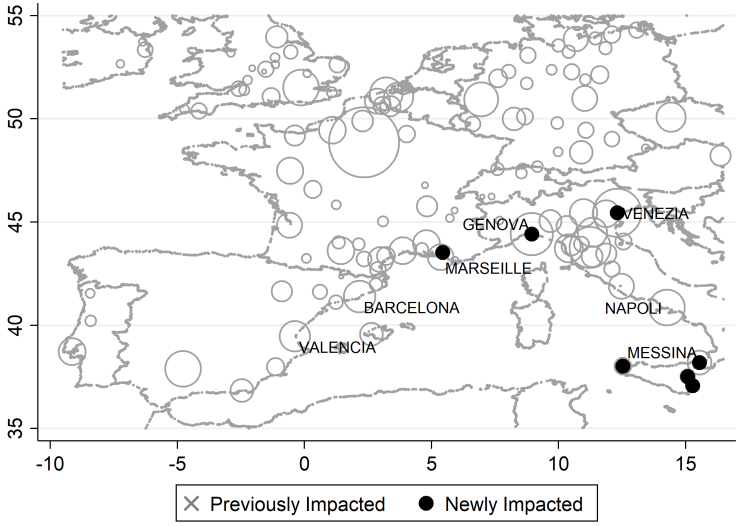
Figure A.2: Distribution of the Duration of the Black Death in Each City.



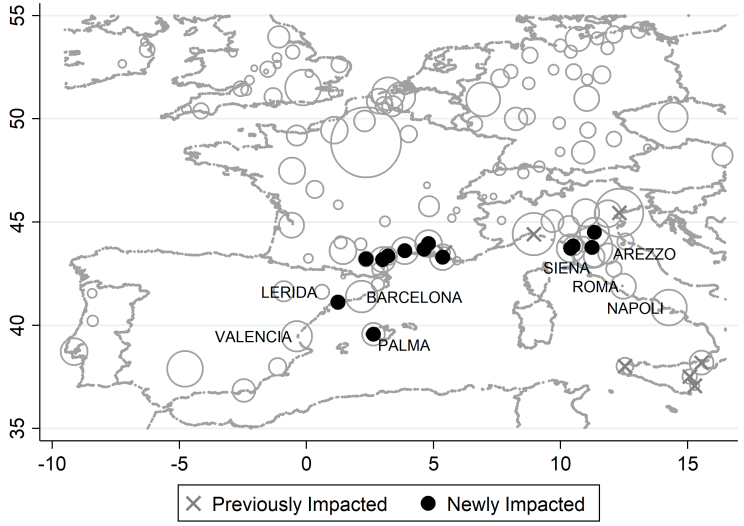
Notes: This figure shows the Kernel distribution of the duration of the Black Death in each city—i.e. the time difference between the year-month of the first infection in the city and the year-month of the last infection in the city (N = 61; mean = 7; median = 7). The source is Christakos et al. (2005). See Web Appendix Section 1. for more details on data sources.

Figure A.3: Spread of the Black Death in the First Year of the Pandemic

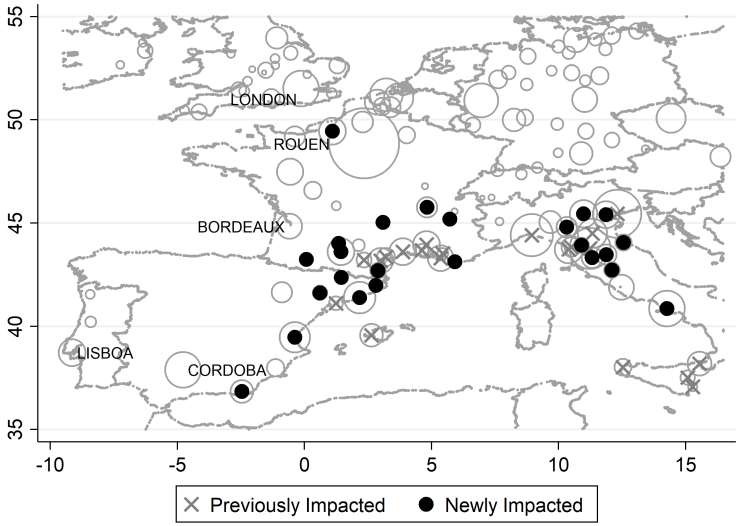
(a) Num. of Months Since Oct. 1347 = 0-2



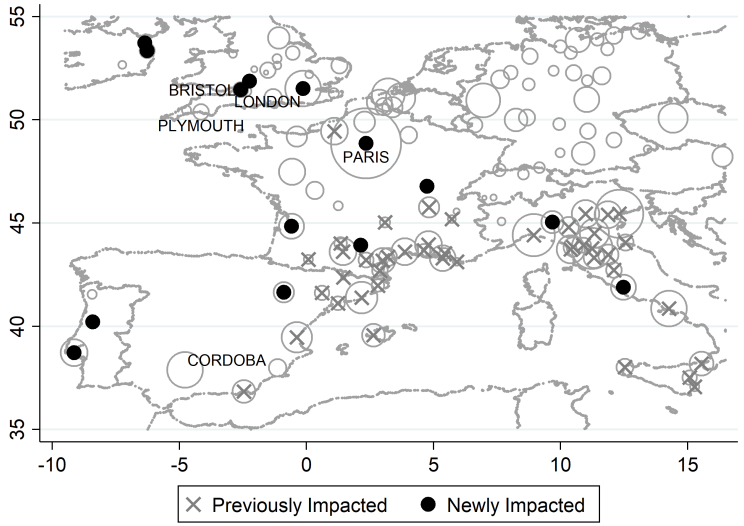
(b) Num. of Months Since Oct. 1347 = 3-5



(c) Num. of Months Since Oct. 1347 = 6-8

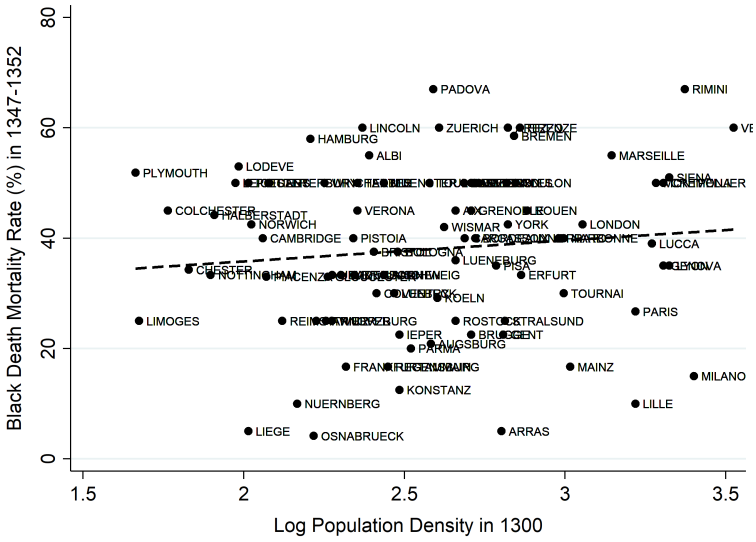


(d) Num. of Months Since Oct. 1347 = 9-11



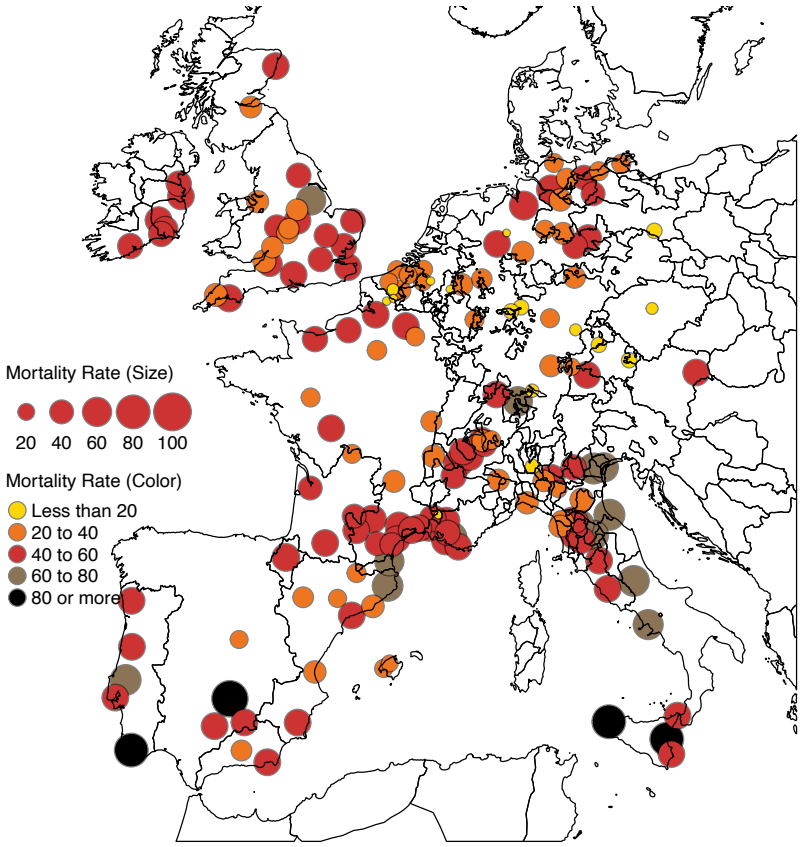
Notes: This figure shows the spread of the Black Death in the first year of the pandemic (October 1347-September 1348). We show the cities where the Black Death has already hit (grey X symbols) and the cities that were newly hit (black circles). We also show selected cities that we discuss in Web Appx. Section 4..

Figure A.4: Black Death Mortality and City Population Density circa 1300.



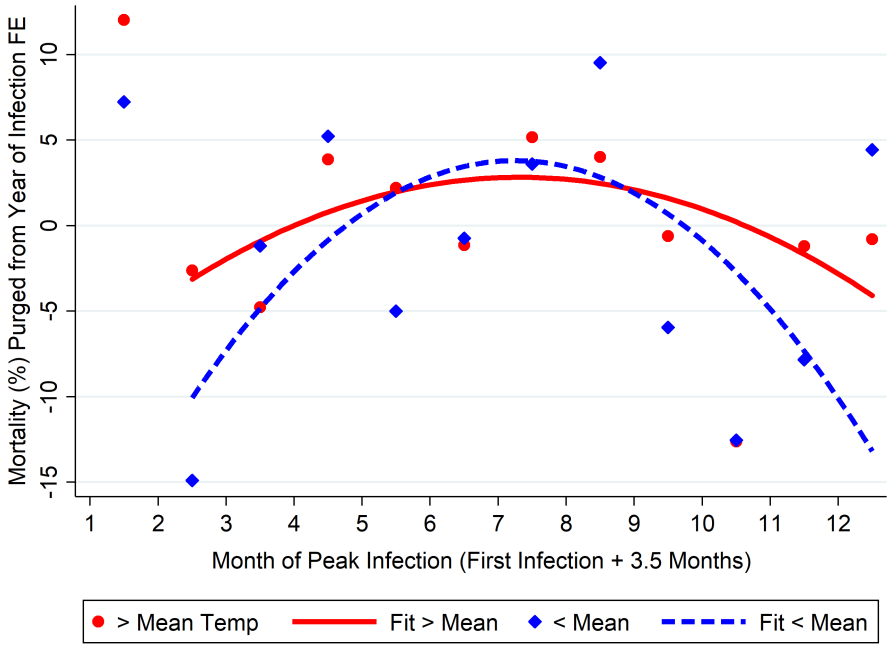
Notes: The figure shows the relationship between Black Death mortality (%; 1347-1352) and city population density defined as population divided by walled land area (Y = 28.2*** + 3.79 X; Obs. = 88; R2 = 0.01). Walled land area and city population density circa 1300 are available for only 88 out of the 165 cities in our sample (source: Cesaretti et al. (2016)). Total city land area does not exist. The main source for the Black Death mortality data is Christakos et al. (2005) (see Web Appendix Section 1. for more details on the data sources used to obtain the Black Death mortality data).

Figure A.5: Black Death Mortality Rates (%) in 1347-1352 and State Boundaries c. 1300.



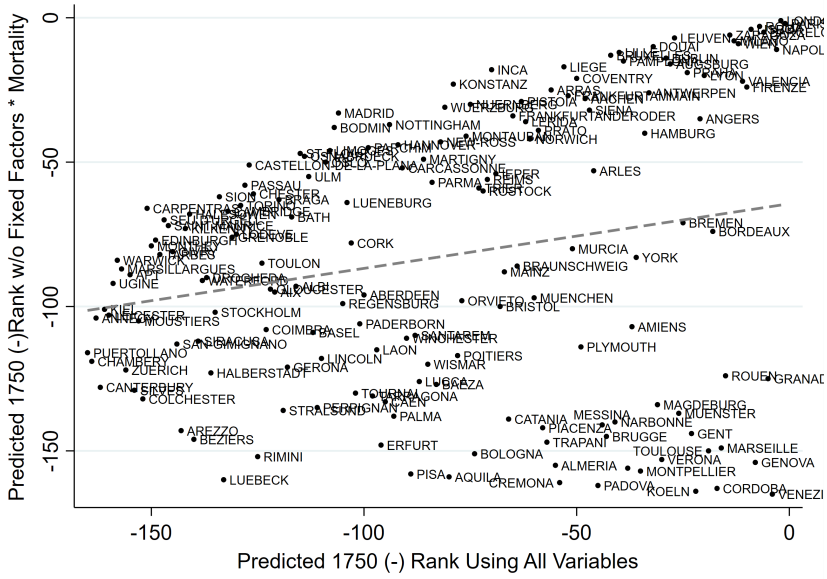
Notes: This map plots the location of all 165 cities (i.e. loc. $\geq$ 1,000 inh.) in 1300 for which we know their Black Death mortality rate (%) in 1347-52 and the historical state boundaries (c. 1300) used in our analysis (source: Nussli (2011)).

Figure A.6: Month of Black Death, Warmer vs. Colder Regions and Mortality.



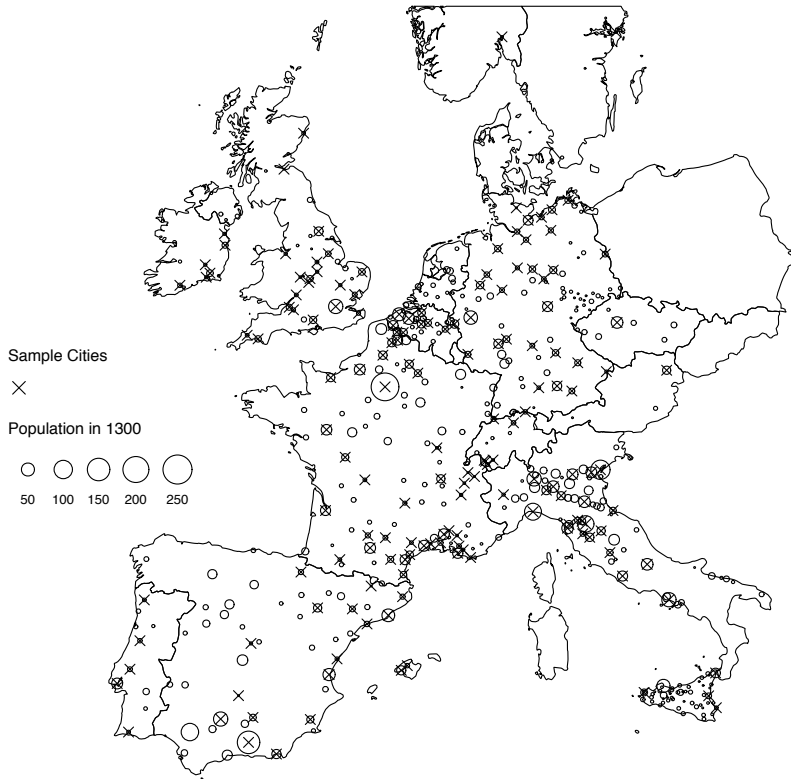
Notes: The figure shows for 124 cities and for each month of peak infection (month of first infection + 3.5) and warmer cities vs. colder cities the average mortality rate purged of year of infection fixed effects. Warmer (colder) cities are cities with an average temperature above (below) the mean in the sample (18° degrees). The quadratic fits show that mortality was the highest when peak mortality was in the summer and the lowest in the winter. As expected, mortality was even more reduced when the month of peak infection was in the winter and the city was located in a colder region (differences not significant). Note that the quadratic fits omit October, which has high mortality rates due to pathogenic reasons and being the month of onset of the Black Death in Europe.

Figure A.7: Fixed Factors and Counterfactual Population Ranks, 1750



Notes: This figure shows the relationship between the predicted 1750 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 5 (including the independent effects of the variables shown at left) and the predicted 1750 inverted population rank (0 = largest city) when ignoring the interacted effects of mortality with the fixed factors (cereal, potato, pastoral, coast, rivers, road, Hanseatic).

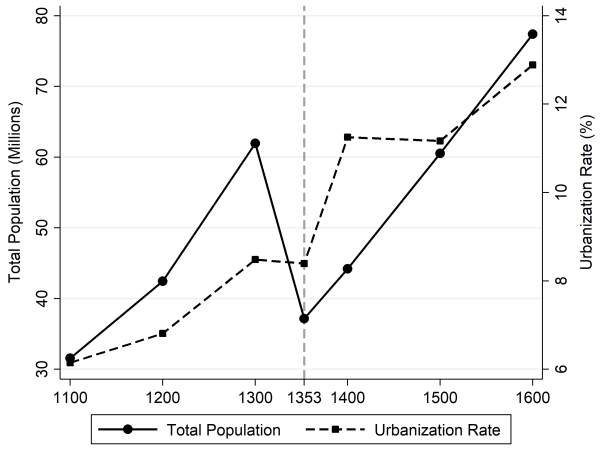
Figure A.8: Main Sample of 165 Cities vs. Location of the 466 Cities in 1300.



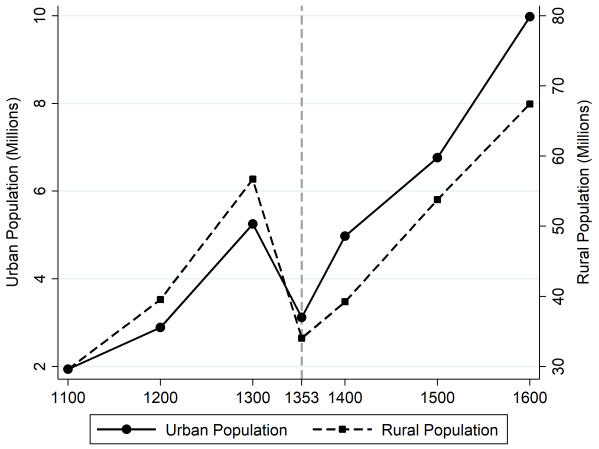
Notes: This figure shows the 466 cities existing in 1300 (with population $\geq$ 1,000 inhabitants) and the 165 cities of the main sample, i.e. the 165 cities among the 466 cities for which we know the Black Death mortality rate in 1347-1352.

Figure A.9: Evolution of Western Europe's Total, Urban and Rural Populations, 1100-1600

(a) Total Population and Urbanization



(b) Urban vs. Rural Population



Notes: The subfigures show the respective evolutions of the total population (millions) and urbanization rate (%) (Subfigure 9(a)) and the total urban and rural populations (Subfigure 9(b)) of the 16 countries in 1100-1600. Total (urban) population in 1353 is proxied by the total (urban) population in 1300 times the pop.-weighted average total (urban) mortality rate in 1347-1352 (38.9% and 38.8%). Rural populations are estimated residually by substracting total urban population levels from the total population levels obtained in McEvedy and Jones (1978). See Web Appendix for data sources.

Table A.1: DESCRIPTIVE STATISTICS FOR THE VARIABLES OF TABLES 1-6

Variable	Obs	Mean	Std.Dev.	Min	Max
TABLE 1					
Mortality	165	40.1	16.6	0.0	93.0
Pct. Chg. Pop. 1300-1400	165	-12	46	-100	175
Pct. Chg. Pop. 1300-1500	164	18	152	-100	1,700
Pct. Chg. Pop. 1300-1600	164	61	218	-100	2,067
Pct. Chg. Pop. 1300-1700	164	142	498	-100	4,567
Pct. Chg. Pop. 1300-1750	164	209	688	-100	5,600
Pct. Chg. Pop. 1300-1800	164	283	868	-100	8,200
Pct. Chg. Pop. 1300-1850	165	547	1,771	-68	19,300
Pct. Chg. Pop. 1300-2015	165	3,757	10,421	-77	104,633
City Pop. $\geq$ 1K 1100-1200	165	0.19	0.39	0	1
City Pop. $\geq$ 1K 1200-1300	165	0.35	0.48	0	1
TABLE 2					
Av. Temperature 1500-1600	165	18.0	3.2	9.3	24.4
Elevation	165	149	228	1	1999
Cereal Suitability	165	4.9	1.3	2.0	8.0
Potato Suitability	165	5.7	1.1	3.0	8.0
Pastoral Suitability	165	0.9	0.3	0.0	1.0
Coastal	165	0.2	0.4	0.0	1.0
Rivers	165	0.3	0.5	0.0	1.0
Longitude	165	4.6	6.2	-9.1	18.1
Latitude	165	47.1	5.1	36.8	59.9
Log Population	165	2.3	1.1	0.0	5.4
Log Market Access 1300	165	-0.9	2.0	-6.0	5.5
Maj.Roman Rd (MRR) 10 Km	165	0.6	0.5	0.0	1.0
Any Roman Rd (ARR) 10 Km	165	0.7	0.4	0.0	1.0
MRR Intersect. 10 Km	165	0.4	0.5	0.0	1.0
ARR Intersect. 10 Km	165	0.5	0.5	0.0	1.0
Medieval Route (MR) 10 Km	165	0.3	0.5	0.0	1.0
MR Intersect. 10 Km	165	0.1	0.3	0.0	1.0
Market and Fair Dummy	165	0.2	0.4	0.0	1.0
Hanseatic League Dummy	165	0.1	0.3	0.0	1.0
Aqueduct 10 Km Dummy	165	0.1	0.3	0.0	1.0
University Dummy	165	0.1	0.3	0.0	1.0
Monarchy 1300 Dummy	165	0.6	0.5	0.0	1.0
State Capital 1300 Dummy	165	0.1	0.3	0.0	1.0
Repr. Body 1300 Dummy	165	0.3	0.4	0.0	1.0
Paliament. Activ. 1300-1400	165	0.5	0.5	0.0	1.0
Log Dist. Parliam. 1300	165	4.3	3.3	-11.8	6.7
Battle 100 Km 1300-1350	165	0.4	0.5	0.0	1.0

CONTINUED ON THE NEXT PAGE

Table A.1: DESCRIPTIVE STATISTICS FOR THE VARIABLES OF TABLES 1-9 - CONTINUED

Variable	Obs	Mean	Std.Dev.	Min	Max
TABLE 3					
Num. Mths 1st Inf. Since Oct47	124	14.4	9.5	0.0	35.0
Dist. Messina (Km) 1300	163	1345	464	87	2473
Dist. All Cities (Km) 1300	163	1236	224	1014	2008
TABLE 4					
Pct.Chg.Pop. 1400 State Int.	68	-10.9	34.5	-80.0	150.0
Pct.Chg.Pop. 1400 State Tot.	68	1.5	50.8	-80.0	300.0
Mortality State	68	38.5	12.1	12.0	64.2
Indirect Mort. State	160	40.7	10.3	16.2	68.3
Indirect Mort. Dist10%	165	39.9	9.1	22.1	66.9
City Dummy 1400	1,335	0.1	0.3	0.0	1.0
City Dummy 1600	1,335	0.4	0.5	0.0	1.0
Log Pop. 1400 for New Cities	1,335	-0.6	0.5	-0.7	2.5
Log Pop. 1600 for New Cities	1,335	0.2	1.2	-0.7	3.7
TABLE 5 (See Table 2)					
TABLE 6					
Truly Coastal	165	0.13	0.34	0.00	1.00
50KmCoast Estuary	165	0.07	0.26	0.00	1.00
50KmCoast Ot.River	165	0.28	0.45	0.00	1.00
BestCoast Medi.	165	0.09	0.29	0.00	1.00
BestCoast Atlantic	165	0.12	0.32	0.00	1.00
BestCoast Mon	165	0.16	0.37	0.00	1.00
BestCoast No-Mon	165	0.05	0.22	0.00	1.00
Ot.River Mon	165	0.12	0.33	0.00	1.00
Ot.River No-Mon	165	0.16	0.37	0.00	1.00
Log Pop 1300	165	2.33	1.13	0.00	5.43
Log Market Access 1353	165	-1.39	2.03	-6.45	5.01
State Pop. Size	165	5.2	1.4	2.1	7.0
Guild	165	0.46	0.50	0.00	1.00
Market Fair	165	0.21	0.41	0.00	1.00
Log Wall Area	88	0.19	0.71	-1.71	2.08
(Arch)Bishopric	165	0.57	0.50	0.0	1.00
University	165	0.08	0.28	0.00	1.00
Serfdom	165	0.19	0.40	0.0	1.0

Table A.2: WESTERN EUROPEAN COUNTERFACTUAL PORTS OF ENTRY AND MORTALITY

Dependent Variable: Black Death Mortality Rate (%, 1347-1352):						
	Effect of Euclidean Distance to City ...:					
	(1) Messina	(2) Genoa	(3) Vienna	(4) Prague	(5) Leipzig	(6) Visby
	-0.06***	-0.02	0.02	0.08***	0.11***	0.17***
	[0.01]	[0.03]	[0.03]	[0.02]	[0.02]	[0.03]
Observations	164	163	163	163	164	164
R-squared	0.56	0.52	0.50	0.53	0.56	0.57

Notes: For our main sample of 165 cities, we sequentially regress the Black Death mortality rate on the Euclidean distances to Messina and each of the alternative ports of entry in our in-sample countries. Note that we exclude Messina and the port of entry itself. As can be seen, we only find a significant negative effect for Messina. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01.

Table A.3: MARKET ACCESS AND MORTALITY RATES, ROBUSTNESS CHECKS

Dependent Variable: Black Death Mortality Rate (%, 1347-1352):								
	Effect of Log Market Access in 1300:							
Test:	(1) Baseline (1.4; 2.9; 5.7) $\sigma = 3.8$	(2) Other Trade Elasticity $\sigma = 2$	(3) $\sigma = 1$	(4) Other Speeds (3.8; 3.8; 7.7)	(5) Costs = Euclid. Dist.	(6) Incl. Own Pop.	(7) Including Cities of East Euro.	(8) East & MENA
1. No Ctrls	-0.20	-0.78	-5.62	0.98	-0.03	-1.02	-0.19	-0.10
	[0.67]	[1.87]	[5.33]	[0.67]	[0.81]	[1.19]	[0.69]	[0.69]
R-squared	0.00	0.00	0.01	0.02	0.00	0.00	0.00	0.00
2. Incl. All Ctrls	-0.34	-1.12	-6.87	0.28	0.13	1.83	-0.22	-0.22
	[0.82]	[2.47]	[7.60]	[0.78]	[0.83]	[29.23]	[0.83]	[0.84]
R-squared	0.23	0.23	0.23	0.23	0.23	0.23	0.23	0.23

Notes: Main sample of 165 observations. Row 1: No controls are included. Row 2: We include the controls of column (4) in Table 2. Col. (1): We use the baseline market access specification. Col. (2)-(3): We use lower trade elasticities. Col. (4): We use a different set of speeds (sources: Pryor (1992); McCormick (2001)). Instead of traveling by sea being 1.4, 2.9 and 5.7 times faster than traveling by river, road or on a path (col. (1)), respectively, it is now 3.8, 3.8 and 5.7 faster, respectively. Col. (5): We use as the travel cost between city i and city j the Euclidean distance between city i and city j, which allows us to not have to rely on technology-specific cost parameters. Col. (6): We include the population of city i in the calculations of market access. To avoid a zero trade cost, we use the travel cost between Paris and Saint-Denis, two localities 7 km away from each other (Saint-Denis is now part of Paris). Col. (7): We include 70 mostly Eastern European cities existing (i.e., that were above 1,000 inhabitants) in 1300 and that are not located in European countries in our sample of 16 countries. Col. (8): In addition to Eastern European cities, we include 52 Middle East and North African (MENA) cities existing in 1300. The source for the population of MENA cites is Bosker et al. (2013), who only report population estimates for cities above 10,000. We thus verify results hold if we construct market access using only European and MENA cities above 10,000 inhabitants (not shown, but available upon request). Robust SE's: * p<0.10, ** p<0.05, *** p<0.01.

Table A.4: FIRST STAGE OF THE INSTRUMENTAL VARIABLES REGRESSIONS

Dependent Variable:	Black Death Mortality Rate (%, 1347-1352):		
	IV1 (1)	IV2 (2)	IV3 (3)
Number of Months btw Oct 1347 & 1st Infection	-1.22***		
	[0.36]		
Euclidean Dist. to Messina		-0.07***	
		[0.01]	
Month Peak Infect.: Jan. (Onset in Europe)			20.2**
			[9.1]
Month Peak Infect.: Feb.			-9.2
			[6.2]
Month of Peak Infect.: Mar			8.4
			[5.5]
Month of Peak Infect.: Apr			23.1***
			[6.6]
Month of Peak Infect.: May			18.5**
			[8.1]
Month of Peak Infect.: Jun			17.6**
			[7.8]
Month of Peak Infect.: Jul			18.2**
			[7.0]
Month of Peak Infect.: Aug			16.3***
			[5.1]
Month of Peak Infect.: Sep			15.6
			[11.0]
Month of Peak Infect.: Oct			9.7
			[6.9]
Month of Peak Infect.: Nov			2.7
			[3.7]
Controls of Column (4) Table 2	Y	Y	Y
Sq. & Cube of Long. & Lat.	Y	Y	Y
Year of First Infection FE	N	N	Y
Avg. Euclidean Distance to All Cities	N	Y	N
Observations	124	163	124

Notes: Col. (3): December is the omitted month. Robust SE's clustered at the state level (N = 53): * p<0.10, ** p<0.05, *** p<0.01.

Table A.5: EFFECT OF BLACK DEATH MORTALITY, PANEL REGRESSIONS

Dependent Variable:	Percentage Change in City Pop. in $[t-1;t]$:				Δ Log City Pop. in $[t-1;t]$:	
	Non-Panel		Panel		Panel w/ Log Pop.	
	(1)	(2)	(3)	(4)	(5)	(6)
Black Death Mortality*1200	-0.25	[0.34]	-0.29	[0.39]	-0.33	[0.32]
Black Death Mortality*1300	0.16	[0.59]	0.11	[0.68]	-0.04	[0.47]
Black Death Mortality*1400	-0.87***	[0.28]	-0.91***	[0.34]	-0.94***	[0.31]
Black Death Mortality*1500	0.65*	[0.34]	0.59	[0.46]	0.41	[0.36]
Black Death Mortality*1600	0.69*	[0.38]	0.63	[0.45]	0.40	[0.33]
Black Death Mortality*1700	-0.00	[0.41]	-0.12	[0.48]	-0.14	[0.42]
City FE	N		Y		Y	
Year FE	Y		Y		Y	
Observations	962		962		956	

Notes: Panel regressions for our main sample of 165 cities, focusing on the years 1100, 1200, 1300, 1400, 1500, 1600, 1700 and 1750. 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. *Non-Panel*: City FE are omitted. SE's clustered at the city level: * p<0.10, ** p<0.05, *** p<0.01.

Table A.6: MORTALITY AND CITY GROWTH, INVESTIGATION OF CAUSALITY, OTHER RESULTS

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. Dropping Top and Bottom 5% in Mortality	-0.67**	[0.29]	145	1.36	[1.12]	144
3. Dropping More Hygienic Cities	-0.89***	[0.30]	161	0.59	[0.90]	160
4. Ctrls for Dummy & Num. Plague Recur. 50km	-0.87***	[0.27]	165	0.41	[0.81]	164
5. Ctrls Dummy & Num. Battles 50km	-0.84***	[0.28]	165	0.77	[0.78]	164
6. Ctrls Dummy & Num. Battles 50km (Incl. 1300-52)	-0.83**	[0.32]	165	0.50	[0.84]	164
7. Ctrls Dummy & Num. 100 Yrs' War 1337-52 Battles 50km	-0.86***	[0.29]	165	0.24	[0.80]	164
8. Ctrl Num. of Famines in Region/Country	-0.87***	[0.28]	165	0.28	[0.74]	164
9. Ctrl Num. of Famines in Region/Country (Incl. 1300-52)	-0.77***	[0.29]	165	0.29	[0.71]	164
10. Ctrl for Avg Extra Wetness 1314-1316	-0.89***	[0.30]	165	0.36	[0.81]	164
11. Ctrl for Dummies Unusually Moist & Very Moist 1314-16	-0.92***	[0.31]	165	0.22	[0.81]	164
12. Ctrls for Jewish Pres., Pers., Pogr. 1347-1352	-0.83***	[0.30]	165	0.28	[0.81]	164
13. Drop if Jewish Persecution 1347-1352	-0.71***	[0.30]	115	0.67	[0.96]	114
14. IV3: Month w/ Controls (IV F-stat = 6.0)	-0.93***	[0.33]	124	-0.23	[0.58]	124
15. IV3: Month*Log(Temp) w/ Controls (IV F-stat = 7.2)	-0.80***	[0.30]	124	-0.24	[0.59]	124

Notes: Row 2: Adding the controls of Table 2. Row 3: Dropping cities with a better hygiene system. Historians report that some cities had either natural baths (Bath, Nuremberg) or tried to take action in response to the plague (Milan, Venice). Row 4: Adding a dummy if there was a plague recurrence in 1353-1400 or 1353-1600. Row 5: Adding a dummy if there was a battle and the number of battles in 1353-1400 or 1353-1600. Row 6: Row 5 also adding a dummy if there was a battle and the number of battles in 1300-1352. Row 7: Adding a dummy if there was a battle and the number of battles related to the Hundred Years' War in 1337-1352. More precisely, using the same battle data as for the robustness checks with the number of battles in 1300-1352 and 1353-1400 or 1353-1600, we identify battles that took place as a result of the Hundred Years' War. The Hundred Years' War began in 1337 and because the location of battles after the Black Death might be endogenous to the intensity of the plague, we only focus on events before or during the Black Death (that is, 1337-1352). We then show that results hold when controlling for the occurrence of such battles. Row 8: Adding the number of famines experienced by the city in 1353-1400 or 1353-1600. Row 9: Row 8 also adding the number of famines in 1300-1352. Rows 10-11: Baek et al. (2020) use "tree-ring based Old World Drought Atlas (OWDA) to show that the average of each growing season preceding the Great Famine years (1314–1316) was the fifth wettest over Europe from 1300 to 2012 C.E." Using their data, we obtain the average Palmer Drought Severity Index (PDSI) of each city in 1314-1316. A city is then defined as experiencing an unusually moist spell if the value is above 2. We thus measure wetness as 0 if PDSI is below 2 and (PDSI – 2) if it is above 2. This way we measure how unusually moist or wet a city was, and thus the potential magnitude of the Great Famine there. As seen in row 10, the baseline coefficients remain unchanged when we control for this measure. Alternatively, we show results hold if we include dummies for whether the city experienced an "unusually moist spell" (value between 2 and 3) or a "very moist spell" (value above 3) in 1314-1316 (row 11). Row 12: Adding dummies if Jews were present, and if a persecution, and a pogrom in particular, took place. Row 13: Excluding cities with Jewish persecutions. Row 14: IV = 11 dummies for the month of peak infection (= month of onset (Oct. is omitted) + 3.5). We add dummies for the year of infection. Row 15: We use as an IV dummies for the month of peak infection interacted with the log of a city location's average temperature (1500-1600; data unavailable before). Rows 14-15: We add the controls of Table 2 and the squares and cubes of longitude and latitude. Robust SE's (clust. at state (1300) level in rows 14-15).

Table A.7: BLACK DEATH MORTALITY AND PLAGUE RECURRENCES, 1353-1600

Dependent Variable:	Black Death Mortality Rate (1347-1352, %)			
Period t:	(1) 1353-1400		(2) 1353-1600	
Dummy Plague Recurrence within 50km in t	-0.69	[4.54]	1.02	[9.62]
Number Plague Recurrences within 50km in t	0.10	[0.56]	-0.04	[0.07]
R-Squared; Obs.	0.00; 165		0.01; 165	

Notes: Main sample of 165 towns. Robust SE's: † p<0.15, * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.

Table A.8: 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
6. Dummies for Type of Mortality Data	-0.94***	[0.30]	165	0.27	[0.84]	164
7. Excl. Description-Based Mortality Data (N = 25)	-0.83***	[0.32]	140	0.37	[0.97]	139
8. Excl. Desertion-Based Mortality Data (N = 21)	-0.98***	[0.31]	144	0.31	[0.86]	143
9. Excl. Clergy-Based Mortality Data (N = 5)	-0.86***	[0.28]	160	0.38	[0.80]	158
10. Use Only Number-Based Mortality Data	-0.96***	[0.36]	114	0.35	[1.06]	113
11. Keeping Top and Bottom 10% in Mortality	-0.99**	[0.39]	34	-0.21	[1.01]	34
12. City Population Data: Bairoch Only	-0.78*	[0.41]	151	0.47	[0.80]	150
13. City Population Data: Chandler Only	-0.85**	[0.36]	59	1.37	[1.10]	60
14. Use Mortality of Nearest Avail. City w/i 50 km	-0.60***	[0.22]	290	0.41	[0.59]	286
15. Use Mortality of Other Cities in Same State	-0.70***	[0.17]	380	0.33	[0.63]	374
16. Extrapolated Rates Based on 274 Cities in 1300	-0.68***	[0.21]	464	0.43	[0.54]	457
17. Reweighting to Match Full City Size Dist. in 1300	-0.74***	[0.24]	165	-0.06	[0.65]	164

Notes: (2) We control for past population trends, i.e. city population growth in 1200-1300. (3) We use as the dependent variable the absolute change in population and as the variable of interest the number of deaths. (4) We cluster standard errors at the state (1300) level. (5) We employ Conley (2008) (500 km) standard errors. (6) We include dummies for different sources of mortality data. These are: population records, literary descriptions, desertion rates, and clergy mortality. (7) We drop estimates based on literary descriptions. (8) We drop estimates based on desertion rates. (9) We drop estimates based on clergy mortality. (10) We only use the raw numerical mortality estimates directly provided by Christakos et al. (2005), 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). (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. (12) We report estimates using the uncorrected Bairoch data. (13) We report results using only observations from Chandler (1974, 1987). (14) We use the mortality of the closest city with data if this city is within 50 km (N = 290–286). (15) We use the mean mortality of the cities in the same state (380–274, SEs clustered at the state level). (16) We use spatially extrapolated mortality rates (464–457). For this analysis, we create a two-dimensional surface of predicted mortality using an inverse distance weighted function of known mortality rates for 274 localities (details in Web Appx. Section 1.). (17) We reweight observations to match the distribution of city populations in 1300. More precisely, 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 confirm using Kolmogorov-Smirnov tests. Robust SE's: † p=0.17, * p<0.10, ** p<0.05, *** p<0.01.

Table A.9: MORTALITY AND CITY GROWTH, DROPPING SELECTED OBSERVATIONS

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. Drop if France (N = 39)	-0.77**	[0.33]	126	0.42	[0.98]	125
3. Drop if Germany (N = 31)	-1.06***	[0.32]	134	0.27	[0.88]	133
4. Drop if Italy (N = 28)	-1.15***	[0.40]	138	0.05	[0.77]	137
5. Drop if United Kingdom (N = 21)	-0.84***	[0.28]	144	0.38	[0.82]	143
6. Drop if Spain (N = 18)	-0.95***	[0.29]	147	0.42	[0.82]	146

Notes: In rows 2-6, we drop specific countries. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01.

Table A.10: BLACK DEATH MORTALITY RATES AND POPULATION RECOVERY, 1300-2015

Dependent Variable: Percentage Change in City Population (%) in Period 1300-t						
Period 1300-t:	1400	1500	1600	1700	1750	2015
	(1)	(2)	(3)	(4)	(5)	(6)
Mort.*Cereal Suitability Index	0.1	0.5	1.3	1.3	1.4	11.5
	[0.2]	[0.3]	[0.9]	[1.9]	[2.2]	[42.1]
Mort.*Pastoral Suitability Index	0.6	-0.1	-3.2	-3.0	-5.9	-70.8
	[0.6]	[1.1]	[2.0]	[3.4]	[3.8]	[71.4]
Mort.*Potato Suitability Index	0.4	-0.1	1.3**	2.7**	3.0**	43.3*
	[0.3]	[0.4]	[0.6]	[1.3]	[1.5]	[24.2]
Mort.*Coast 10 Km Dummy	0.8*	2.4***	4.1*	6.1	6.7	91.2
	[0.5]	[0.7]	[2.1]	[4.7]	[5.4]	[101.7]
Mort.*Rivers 10 Km Dummy	0.4	1.5**	3.5***	5.7**	5.8**	133.5***
	[0.5]	[0.7]	[1.1]	[2.4]	[2.7]	[42.1]
Mort.*Road Intersection 10 Km Dummy	0.5	1.3*	1.6	1.8	2.6	25.2
	[0.6]	[0.7]	[1.5]	[3.1]	[3.5]	[63.4]
Mort.*Hanseatic League Dummy	2.6***	2.1*	5.7**	7.3	7.6	33.2
	[1.0]	[1.2]	[2.5]	[4.6]	[5.5]	[109.5]
Mort.*Log Est .City Population 1353	-0.1	0.6	1.1	1.8	2.4	28.0
	[0.2]	[0.4]	[0.9]	[2.0]	[2.4]	[41.6]
Mort.*Monarchy 1300 Dummy	0.1	1.2*	2.4**	3.1	2.5	-25.9
	[0.5]	[0.6]	[1.1]	[2.0]	[2.3]	[41.2]
Mort.*State Capital 1300 Dummy	0.1	-0.2	3.1	5.7	4.7	6.4
	[0.7]	[1.5]	[2.1]	[4.4]	[5.6]	[78.4]
Mort.*Representative Body 1300 Dummy	1.0**	-0.3	-0.5	-2.0	-2.6	-16.3
	[0.5]	[0.7]	[1.2]	[2.3]	[2.6]	[43.2]
Mortality	-3.8***	-2.1	-7.1**	-19.3**	-20.3**	-286.6**
	[1.3]	[1.8]	[3.0]	[7.4]	[8.2]	[121.1]
Country FE	Y	Y	Y	Y	Y	Y
Observations	165	164	164	164	164	165
R-squared	0.45	0.29	0.39	0.35	0.35	0.25

Notes: This table shows for the 165 cities of the main sample the effects of mortality (%) interacted with selected characteristics of Table 2. The table only shows the interacted effects and the effect of mortality but the characteristics are included as controls. 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.

Table A.11: 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	≤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: We show for 27-28 counties the effect β_t of mortality (%) on: (1)-(3) the pct change in total pop. (%) in different periods; (4)-(6) the number of DMVs per 1000 sq km (col. (5): Within 10 km from an existing city in 1300; col. (6): Beyond 10 km). We use county pop. in the initial years of the period as weights. Col. (1)-(3) and (7): We exclude Cornwall whose pop. in 1290 is not known. Col. (4)-(6): We control for log pop. in 1290 and log area. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01.

Table A.12: BLACK DEATH MORTALITY RATES AND LAND USE SHARE, 1100-1750

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

t:	1300-1400	1300-1500	1300-1600	1300-1700	1300-1750	1100-1200	1200-1300
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel A:	Controlling for the contemporaneous changes in both city population and country population						
β_t	-0.28**	-0.21**	-0.01	-0.01	0.02	0.06*	0.04
	[0.12]	[0.08]	[0.05]	[0.07]	[0.09]	[0.04]	[0.05]
Obs.	160	159	159	159	159	58	89
Panel B:	Not controlling for the contemporaneous changes in both city population and country population						
β_t	-0.37***	-0.09	-0.01	-0.00	0.00	0.03*	0.02
	[0.13]	[0.11]	[0.06]	[0.10]	[0.10]	[0.01]	[0.03]
Obs.	160	160	160	160	160	58	89

Notes: This table shows for the 165 cities the effect β_t of mortality (%) on the percentage change in the mean land use share (%) within 10 km for each period t. The land use share is the share of the land that is used for crops instead of being naturally forested (managed forests were very uncommon then). **Panel A:** The percentage changes in city population (%) and country population (%) in period t are added as controls (we lose 5 cities due to missing country population data). **Panel B:** We do not include the total and city population growth controls. In both panels, we use as weights populations of the cities in the initial year of the period. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. See Web Appx. for data sources.

Table A.13: BLACK DEATH MORTALITY, NATURAL INCREASE AND RECOVERY

Dep. Var.:	Percentage Change in City Population (%) in 1300-1600							
	(1)	(2)	(3)		(4)	(5)	(6)	(7)
"North" Definition	_	Northern Cntries	Excl. France	**"Strong EMP" Definition**	> Mean Age Mar.	> Med. Age Mar.	> Mean Celib.	> Med. Celib.
Mort.	0.36	0.20	0.23	Mort.	0.58	0.02	0.48	0.38
	[0.68]	[1.16]	[0.86]		[1.18]	[1.06]	[0.72]	[0.49]
North		-14.5	-22.5	Strong EMP	14.3	-37.6	31.2	29.7
		[54.6]	[34.9]		[57.0]	[47.7]	[69.0]	[36.7]
Mort.*North		0.24	1.01	Mort.*Strong EMP	0.49	1.14	0.43	-0.33
		[1.31]	[0.94]		[1.37]	[1.13]	[1.75]	[1.04]
Obs.	164	164	164	Obs.	164	164	164	164
R-squared	0.00	0.00	0.01	R-squared	0.02	0.01	0.02	0.01

Notes: This table shows for 165 cities the effects of mortality, the North dummy or EMP dummy and their interaction with mortality. Col. 2: North includes 114 cities in 9 Northern European countries (Austria, Belgium, France, Germany, Ireland, Norway, Sweden, Switzerland, the United Kingdom). Col. 3: North includes 75 cities in 8 of the 9 countries (we exclude France). Col. 4-5: EMP includes 88 and 63 cities in countries or regions with an age at first marriage above the mean or median in the sample. Col. 6-7: Strong EMP includes 30 and 77 cities in countries or regions with a female celibacy rate (%) above the mean or median in the sample. Robust SE's clustered at the country/region level: * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.