Pandemics and Cities: Evidence from the Black Death and the Long-Run

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Abstract

We ask what effects a high fatality rate pandemic could have on city growth. 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 explore the long-run impacts it had on city growth. On average, cities recovered their pre-Plague populations within two centuries. However, aggregate convergence masked heterogeneity in urban recovery. We show that both of these facts are consistent with populations returning to high-mortality locations endowed with more rural and urban fixed factors of production. Land suitability and natural and historical trade networks played a vital role in urban recovery. Our study thus highlights the role played by pandemics and physical and economic geography in determining the relative size of cities in less developed countries.

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

Keywords: Pandemics; Cities; Localized Shocks; Path Dependence

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Davis and Weinstein (2002) use a "spatial catastrophe"—the bombing of Japanese cities during World War II—to investigate the importance of various theories of urban location. We build on their work by studying the effects of the Black Death, the largest demographic shock in modern history. It killed 40% of Europe's population between 1347-1352. Some regions and cities were spared, others were devastated—England, France, Italy and Spain lost 50-60% of their populations. While the Black Death has been widely studied (notably by Voigtländer and Voth, 2013a,b), little is known about its effects on city growth. More generally, little is known about the long-run effects of pandemics on cities. It is particularly important to study how a population shock such as that caused by pandemics affect cities as agglomeration effects often play a crucial role in explaining the distribution of urban population. Fortunately, recent disease outbreaks have featured either low rates of contagion (e.g. Ebola) or relatively low fatality rates (e.g. COVID-19). But what if a pandemic that is both highly contagious and has a high fatality rate, like the Black Death (70%), was to spread?[1]

In this paper, we use city-level data on Black Death mortality to test whether cities that experience very high mortality shocks can be permanently affected, and if not, explore the mechanisms that lead them to recover. A priori, the city-level effects of mortality shocks are ambiguous: (i) If there are local increasing returns a large negative shock to a population could lead to a negative feedback cycle in which city wages and population continue to decline; (ii) If urban incomes rely largely on spatially fixed factors of production such as land and other natural resources such as bodies of water, i.e. locational fundamentals, or if there are historically sunk investments in housing and infrastructure, net wages go up due to labor scarcity, thus allowing population recovery by either causing migration from low to high-mortality areas or increasing net fertility in the latter; alternatively (iii) If the population shock and the resulting higher wages release financial constraints, thus permitting investments in capital,

[1]COVID-19 is highly contagious but its infection fatality rate is likely under 0.5% and it has killed 0.005% of the world's population so far. See https://www.who.int/emergencies/diseases/novel-coronavirus-2019 for more information. See Benedictow (2005, 2010) for the Plague.

or leads to beneficial institutional changes, high-mortality cities may gain a long-run productivity advantage and keep growing. The implications of each of these models for long-run city growth are different: relative *decline* (i), *recovery* (ii) or *growth acceleration* (iii). The relative importance of local increasing returns, locational fundamentals, historically sunk investments, and financial/institutional changes may then vary across contexts. For example, locational fundamentals might play a disproportionately bigger role in less developed economies, thus increasing the likelihood of the recovery scenario.

Unlike other shocks considered in the literature, our shock was exceptionally large. The Black Death was also a comparatively "pure" population shock. This makes our setting well suited to test for the existence of multiple equilibria in city sizes (see Bleakley and Lin (2015) for a survey). More precisely, buildings and equipment were not destroyed and the event itself did not directly target a particular demographic group. Wars and bombings, as studied by Davis and Weinstein (2002, 2008), Glocker and Sturm (2014) and Redding and Sturm (2016) killed people but also led to massive physical destruction and resulted in government reconstruction programs. Disasters such as floods and fires, as studied by Boustan et al. (2017) and Hornbeck and Keniston (2017) kill far less people but also lead to physical destruction.[2] Malaria, HIV, or the 1918 influenza pandemic, as studied by Bleakley (2010), Young (2005), Almond (2006), Donaldson and Keniston (2016), Beach et al. (2018), Barro et al. (n.d.) and Barro (2020) disproportionately kill subgroups of the population, much like COVID-19.[3] Finally, studies have examined the effects on cities of local changes in economic conditions (Glaeser and Gyourko, 2005; Glaeser et al., 2015), transportation technology (Redding et al., 2011; Bleakley and Lin, 2012) or political boundaries (Ahlfeldt, Redding, Sturm and Wolf, 2015).[4]

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

[3]Malaria tends to kill children whereas HIV kills working-age adults (Weil, 2010). The 1918 influenza killed the "very young, those around age 30, and the elderly" (Almond, 2006). COVID-19 disproportionately kills individuals above 60 or with pre-existing medical conditions.

[4]An overall mortality rate of 40% is unprecedented. Other examples of shocks include the

Using data for 165 cities, comprising 60% of the total urban population of Western Europe, we find that between 1300 and 1400 a 10 percentage point higher mortality rate was associated with a 8.7 percentage point fall in city population. After two centuries the impact of mortality was zero, in part due to migration from low-mortality areas. When we examine the spill-over and general equilibrium effects of the Black Death, we find similarly negative effects in the short-run and nil effects in the long-run, and likewise for rural areas. While rural recovery was to be expected, given how important (fixed) land is for agriculture, a surprising fact is that large cities recovered as well, consistent with scenario (ii) above. Thus, in less developed economies where locational fundamentals and sunk investments may play a relatively more important role for cities, pandemics might not durably affect, their relative city size distribution. Nonetheless, where populations were initially sub-optimally located, there was potential for populations to be reallocated to cities with better fixed factors.[5]

Consistent with that fact, we show that urban recovery is almost entirely explained by the interacted effects of mortality with city characteristics that proxy for fixed factors of production: rural fixed factors related to better land suitability, but also urban fixed factors related to natural advantages (e.g., coastal access and waterways) or historically sunk investments (e.g., roads and trade networks) favoring trade. We show that aggregate urban recovery hides permutations in the distribution of cities. Some cities permanently collapsed after the Black Death whereas other cities gained in the long run. We provide evidence that these permutations were associated with the presence of rural and urban fixed factors. Importantly, many prominent present-day cities would have fallen into oblivion absent fixed factors. Lastly, since permutations favored cities with better land and trade potential, urban systems may have become more productive.

1918 influenza (3-5% of the world's population), the Ukrainian Famine (10%), WWII (3.5% of the world's population), the Bengal Famine (6.6%), the Great Chinese Famine (2.5-6.8%), and the Rwandan Genocide (11%). The introduction of European diseases in the Americas killed between 75% and 90% of the population, but this was over the course of a century (Koch et al., 2019).

[5]The poorest countries on earth have similar income and urbanization levels as European countries in 1300 (sources: Bairoch (1988); Bolt and van Zanden (2014); United Nations (2018)).

These results are causal. First, the virulence of the Plague was unrelated to factors related to future city growth. Second, the parallel trends assumption is verified. Third, our results hold when we implement instrumental variables strategies premised on the facts that: (i) the Black Death entered Europe through the Sicilian port of Messina (largely by chance) and was more virulent in its earlier stages (for pathogenic reasons); (ii) it was 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 complement Davis and Weinstein (2002, 2008) in four ways. First, our shock is far larger. Mean Plague mortality was 40% and all cities were impacted, with mortality equal to 5% and 80% at the 1st and 99th percentiles. In contrast, 20% and 8.5% of the populations of Hiroshima and Nagasaki were killed during WWII, respectively, and 80% of Japan's cities were not targeted. Second, no buildings were destroyed and government assistance was non-existent in our context. Thus, we isolate the effects of mortality. Third, Davis and Weinstein explain that cities with strong defense capabilities, of historical significance, or with a specific topography, were spared by the bombings whereas Plague virulence was apparently exogenous. Fourth, their results imply that locational fundamentals explain urban recovery but they do not interact the bombings with the geographical characteristics of the cities to identify which locational fundamentals mattered for urban recovery and permutations. However, we do not have the richness of their population and industry data.

We also add to the literature on the economics of pandemics. Most pre-COVID studies of their economic consequences use macroeconomic approaches (Young, 2005; Weil, 2010; Voigtländer and Voth, 2013b; Barro et al., n.d.), notable exceptions being Almond (2006), Donaldson and Keniston (2016) and Beach et al. (2018).[6] Likewise, there is a limited literature on the effects of Ebola (Bowles et

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

al., 2016). Given its high case fatality rate (50%) an uncontrolled Ebola pandemic could have dramatic effects in Africa (UNECA, 2015; Gates, 2018).

Finally, we build on previous research on the Black Death (Voigtländer and Voth, 2013a,b).[7] Compared to prior studies, we examine its local effects, which are not the focus of the growth literature (Galor and Weil, 2000; Galor, 2005; Ashraf and Galor, 2011). In the standard model of the Malthusian World, any population decline temporarily increases income, until population re-increases and cities only proxy for income growth in the countryside. In an important contribution, Voigtländer and Voth (2013b), however, use a calibrated model to show how a large mortality shock can trigger a transition to a new steady state with higher incomes. When wages increase, non-homothetic preferences lead to higher demand for urban goods, thus spurring urbanization. Since cities were unhealthy then and because the plague returned frequently and conflict was endemic, death rates, and thus incomes, remained high.

We complement these macro theoretical analyses with an econometric analysis of the spatial effects of the Black Death by building on the work of Voigtländer and Voth (2013b). In their conclusion, they use aggregate data from 23 countries to show that those areas that experienced a bigger population decline in 1300-1400 urbanized faster. In contrast to this approach, we focus on cities, use data on city-level mortality, examine factors that led mortality to vary across space, estimate causal effects, provide evidence for city recovery, emphasize the importance of urban fixed factors and migration in that recovery process, document permutations in the ranking of cities, and identify which factors mattered for city recovery and the permutations.[8] More generally, understanding how the Plague interacted with locational fundamentals and sunk investments improves our understanding of how, in a Malthusian economy,

//voxeu.org/pages/covid-19-page for the very recent literature on the effects of COVID-19.

[7]Important contributions to the historical literature includes: Ziegler (1969); Brenner (1976); Gottfried (1983); Benedictow (2005); Pamuk (2007); Clark (2007, 2016); Campbell (2016).

[8]Voigtländer and Voth (2013a) shows that areas with more deserted villages specialized in pastoral farming. We complement this analysis by showing that low-mortality, not high-mortality, areas had more deserted villages, since wages increased in high-mortality regions. We also show how high-mortality areas suitable for pastoral farming saw their cities recover slower.

pandemics might lead to a beneficial spatial reallocation of economic activity.[9]

1. Data

See Web Appx. Sections 1.-3. for details and Table A.1 for summary statistics.

Mortality. Data on cumulative Black Death mortality for the period 1347-1352 come from Christakos et al. (2005, 117-122) who compile data on mortality rates based on information from a wide array of historical sources including ecclesiastical and parish records, testaments, tax records, court rolls, chroniclers' reports, donations to the church, financial transactions, mortality of famous people, letters, edicts, guild 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.[10] (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.[11] (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 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

[9] A related paper is Boerner and Severgnini (2014) who use data on the diffusion of the Black Death to estimate its speed of transmission between cities, and its determinants.

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

[11] 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.

inclusion in the dataset is a city population greater than 1,000 inhabitants. We update Bairoch where historians – Chandler (1974, 1987), Nicholas (1997), and Campbell (2008) – have revised estimates of the population of particular cities. 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).

Sample. Our sample consists of 165 cities existing 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 from 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 around 1300 (Bosker et al., 2013; Stasavage, 2014). We measure parliamentary activity during the 14th century (Zanden et al., 2012) and control for whether a city was within 100 km of a battle in 1300-50.

2. The Shock

Epidemiology. 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 shows the locations of Messina and Kaffa). Over the next three years it spread across Europe killing about 40% of the population (we calculate a 38.9% mortality rate for the 274 localities with mortality data). Recent discoveries in plague pits have corroborated the hypothesis that the Black Death was Bubonic plague. The bacterium *Yersinia Pestis* was transmitted by the fleas of the black rat. Infected fleas suffer from a blocked esophagus. These "blocked" fleas are unable to sate themselves and continue to bite rats or humans, regurgitating the bacterium into the bite wound thereby infecting rats or humans. Within less than a week, the bacteria is transmitted from the bite to the lymph nodes causing them to become buboes. Once infected, death occurred within ten days with 70% probability.[12]

One important question is to what extent the epidemiological characteristics of the Black Death made its spread endogenous to population density and trade. The Black Death was largely *bubonic plague* and in this sense differed from more recent diseases, such as the 1918 Influenza or COVID-19, which are *pneumonic*. Pneumonic diseases are spread from inhaling respiratory droplets of infected individuals who are in close contact and causes an infection in the lungs. The Black Death, by contrast, was caused by flea bites and caused an infection in the lymph nodes. Direct person-to-person transmission was rare (Campbell, 2016, 235), weakening the potential of density and trade to explain its spread.

However, fleas cannot spread the disease far in the absence of hosts. An infected rat 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.[13] Yet, dispersal occurs over long distances (10 km) if resources are scarce or for mate-searching. Thus, a rat may plausibly infect other rats 0.5-10 km away,

[12] See Benedictow (2005, 2010).

[13] "...dispersal of rats away from their natal site is generally over short distances [...] 496 m for black rats [...]" (Byers et al., 2019).

and in turn that population cluster could infect other rats 0.5-10 km away, etc. Once an infected 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. As they run out of victims, the disease mutates in favor of benign pathogens.[14] 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 eventually cause an epidemic to end.

This explains why Sicilian cities had abnormally high mortality rates (two thirds on average) rather than them being situated on the coast or correlated with other characteristics related to trade potential (Figure 1). Indeed, other coastal cities such as Barcelona, Bristol, Edinburgh and Rostock experienced much lower mortality rates. Likewise, this also explains why average mortality strongly decreased over time (see Figure 2(a)) and why the disease disappeared on its own after more than three years. 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.

This begs the question, what determined why some cities got infected

[14]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 [...]."

relatively earlier than others? Population density and trade could have mattered in theory, since the largest and most connected cities of Europe may have received infected people and cargoes before other cities. However, 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 was only the 55th largest city in Europe. While the exact origin of the Black Death is 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 here that the Genoese galleys with infected rats and humans on their voyage home stopped in Messina in October 1347. Two months later boats left for, and infected, Genoa. Other infected boats 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).

Once It Arrived in 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. 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. Indeed, while we find an aggregate mortality rate of 38.9%, the mortality rate for cities only was 38.8%.

Seasonal Patterns. Another important fact was that the Plague was at its 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 towns, 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.

Did Random Factors Compensate for Non-Random Factors? The local virulence of the Plague had a very 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 4. for more qualitative evidence). When studying variation in mortality rates across space, historical accounts have been unable to rationalize the patterns in the data (Ziegler, 1969; Gottfried, 1983; Theilmann and Cate, 2007; Cohn and Alfani, 2007). Venice had high mortality (60%) while Milan escaped comparatively unscathed (15%). Paris' mortality rate was almost 20 points lower than London's. Highly urbanized Sicily suffered heavily. Equally urbanized Flanders, however, had low death rates. Southern Europe and the Mediterranean were hit hard, but so were the British Isles and Scandinavia.[15] Likewise, Christakos et al. (2005, 150) explain that some scholars have "argued that Black Death hit harder the ports and large cities along trade routes" but that "the generalization is logically valid at a regional level at best" and that "examples and counterexamples abound."

Consistent with this, Figure 3(a) illustrates the lack of a relationship between mortality (1347-52) and log 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.3).

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

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. However, 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);[16] (ii) using alternative measures of transport costs or Euclidean distance;[17] (iii) including the city itself;[18] (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 (MENA).[19] Importantly, in 1300 Eastern Europe and the MENA had far fewer cities than our 16 countries. Eastern Europe was still relatively underdeveloped, with only 70 cities (vs. 466 for Western Europe). The Islamic Golden Age had already ended by 1347 and in the MENA there were almost four times fewer (10,000+) cities than Europe. As such, our cities' market access was driven by in-sample cities.[20] This is consistent with the findings of Bosker et al. (2013) that there was little trade contact between Europe and the MENA during this period.

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

Plague Recurrences. Subsequent outbreaks of bubonic plague reoccurred for two centuries following the Black Death. Epidemiologists and historians have long noted the virulence, spread, and associated mortality of the Black Death dramatically differed from the pattern associated with later outbreaks of bubonic

[16] 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).

[17] 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).

[18] 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.

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

[20] We also find very strong correlations (above 0.95) between our main measure of market access and measures of market access including Eastern Europe and the MENA.

plague (see Web Appx. Section 4.). These plague recurrences were caused either by local plague reservoirs or the repeated reintroduction of the bacteria from Asia (Schmid et al., 2015). In general, only a few cities at a time were affected, and mortality was much lower than in the initial pandemic (Aberth, 2010, 37). Europe-wide studies of later outbreaks also only focus on the extensive margin of the plague, since, unlike us, they do not have consistent data on the level of mortality experienced by each city during each Plague recurrence.

Composition Effects. Unlike the 1918 Influenza or COVID-19, death rates were similar across groups (see Web Appx. Section 5. for qualitative evidence). The Black Death was "massive and indiscriminate, making no exception to factors such as personal hygiene, health, age, sex, or social class" (Christakos et al., 2005, 150). This is supported by recent studies of plague outbreaks (Alfani and Bonetti, 2018). Christakos et al. (2005, 150) note that the claim that the poor died more than the rich is a plausible sounding statement "rather than an independent scientific conclusion derived from the analysis of real Black Death data".[21] Neither the medical profession nor authorities could respond. Medical knowledge was rudimentary: Boccaccio (2005, 1371) wrote that "all the advice of physicians and all the power of medicine were profitless and unavailing". Individuals, regardless of wealth, could not protect themselves. Prevention measures were nonexistent: the practice of quarantine was not employed until 1377.[22] Thus, even if the Black Death had *indirect* longer run effects on human capital, due to the economy changing in its aftermath (see Donaldson and Keniston (2016) for an example of how education responded in the aftermath of the 1918 Influenza), our shock did not *directly* impact human capital.

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

[21] For contrary evidence, see DeWitte and Wood (2008). However, they study one cemetery only.
[22] The term quarantine was first used in Ragusa in Croatia in 1377 (Gensini et al., 2004).

and enduring contraction in economic activity". Nominal wages increased immediately after the shock. Real wages, however, rose only gradually due to inflation (Munro, 2003). In England, it was not until the 1380s that real wages reached their pre-Plague levels. In the long run wages did increase—peaking in parts of Europe in the mid-15th century before declining as a result of population growth after 1500 (see Web Appx. Section 6. for more qualitative evidence).

The demographic recovery was slow. Web Appx. Fig. 4(a) presents estimates of total population and urbanization for the 16 countries which contain our cities. Europe regained its pre-Plague population by 1600. Urbanization, here defined as the population share of localities above 1,000 inhabitants, rose in the aftermath of the Black Death from 8% to 11% by 1400, and 13% in 1600. This was due to urban areas recovering faster than rural areas (see Web Appx. Fig. 4(b)).

3. Black Death Shock and City Recovery

To estimate the short- and long-run effects of Black Death mortality on (relative) city growth, in order to test whether cities that had relatively high mortality rates were permanently affected, we estimate a series of city-level regressions based on:

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

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.[23]

Col. (1) of Table 1 measures the short-run impact in 1300-1400. The coefficient, -0.87***, should be interpreted relative to the immediate effect in 1347-52, -1.00 by construction. The fact that the coefficient is not significantly different from -1.00 suggests little recovery in population in the decades directly following the onset of the Plague. In terms of magnitude, the effect is large: a one standard deviation increase in mortality is associated with a 0.31 standard

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

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 a 0.02-0.03 standard deviation increase in population growth, implying total recovery. The effects are also not significant. Likewise, if we use 1850 or 2015, we still find small and insignificant effects: the standardized effect is 0.02 and 0.00, respectively (not shown, but available upon request). Section 2. suggests Plague virulence was not well explained by city characteristics. We now provide further evidence that it was plausibly exogenous.

Biases. For the short-run a downward bias is more problematic than an upward bias as we then underestimate the negative effect of the Plague (the true effect in 1300-1400 is less negative than -0.87). The short-run effect is downward biased if cities that were inherently growing slower (faster) were also affected by higher (lower) mortality. For the long-run, an upward bias is more problematic as we then overestimate how fast cities recover (the true effect in 1300-1600 is less positive than 0.36). Thus, when comparing the short- and long-run effects, biases would have to work in opposite directions, a statistically unlikely occurrence, for our recovery hypothesis to be invalidated. We discuss these potential biases below and how our identification strategies minimize them.

Correlates of Mortality. In Table 2 we show that mortality rates were uncorrelated with various city characteristics proxying for locational fundamentals (1), increasing returns and sunk investments (2) or institutions (3). The only variables that have explanatory power are proximity to rivers and latitude (col. (1)). Proximity to rivers is negatively correlated with mortality, which is inconsistent with the claim that trade routes were correlated with Plague virulence. Other measures of transportation and trade networks do not have economically or statistically significant effects. The coefficient on latitude reflects the fact that the Black Death hit southern Europe first and was more virulent in the early years of

the epidemic. Finally, no effect is significant once all controls are included.[24]

Controls. If larger cities had higher mortality rates due to higher densities propagating the disease, and if larger cities were larger due to the presence of sectors driving future growth, this would be a source of upward bias. Likewise, if being on a trade route was positively correlated with both mortality and a city's growth potential, this would lead to an upward bias. If anything, an upward bias makes us under-estimate the short-term negative effect of the plague, which is inconsequential for our analysis. In row 2 of Table 3, we show results hold when we include all the controls of Table 2 simultaneously. The effect in 1300-1400 is now less negative. Indeed, we will show in Section 6. that city characteristics affected the recovery of higher-mortality cities in 1353-1400 and beyond. Over-controlling then leads us to under-estimate the negative short-run effects.

Spatial FE. Results hold if we employ 13 modern country fixed effects (FE) (row 3). Modern country borders differ from the political units of the fourteenth century so in row 4 we assign a separate dummy variable to each of the independent polities with at least 5 cities in our dataset.[25]

Outliers. In row 5, we drop towns with the 5% highest and the 5% lowest mortality rates. 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 (row 6).

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. As can be seen in the lower sample sizes,

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

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

many cities also did not exist (i.e. were below 1,000) 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.[26]

Panel. We restrict the sample to the 165 cities, focusing on the years 1100, 1200, 1300, 1400, 1500, 1600, 1700 and 1750. We run a panel regression where the dependent variable is the percentage change in population between t-1 and t (1100 is dropped), where city fixed effects and year fixed effects are included, and where the variables of interest are Black Death mortality in 1347-1352 interacted with the year effects (1750 is the omitted year). We use as weights city population in t-1 and cluster standard errors at the city level. Figure 4 shows: (i) The interacted effects ("Panel"); (ii) The corresponding effects when running the cross-sectional regression for each year one by one ("Non-Panel"). For the period 1400-1750, these differ from col. (2)-(5) in Table 1 which showed long-difference effects with 1300 as the start year; and (iii) The panel effects when the dependent variable is the change in log population size between t-1 and t ("Panel w/ Log"). The negative effects in 1300-1400 ("1400"; -0.87***, -0.91*** and -0.94***, respectively) are offset by positive effects in 1400-1500 ("1500") and 1500-1600 ("1600"). Results are very similar across the three specifications (coefficients shown in Web Appx. Table A.5), implying city effects do not matter.

Results hold if we control for log population size in t-1 (not shown). This is unsurprising as mortality was uncorrelated with population. As population is a "bad" control due to dynamic panel bias (Nickell, 1981), we ignore these results.

Given how insensitive our results are to the inclusion of city effects, we use long-difference regressions for the rest of the analysis. Furthermore, the IV strategies developed below do not suit a panel framework. Indeed, the instruments would need to be interacted with the year fixed effects, thus creating multiple first-stage regressions and generating multiple weak instruments.[27]

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

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

Correlated Shocks. The plague reoccurred following the Black Death. This could be a source of bias if subsequent outbreaks were correlated with the initial pandemic. In row 7 of Table 3 we use data from Biraben (1975) and show results hold if we include a dummy for plague recurrence and the number of recurrences within 50 km in the period of interest.[28] The Plague initially reduced the intensity conflict (see Sumpton (1999)). However, warfare ultimately intensified and led to urbanization (Voigtländer and Voth, 2013a). Row 8 shows results hold if we include a dummy for battle occurrence and the number of battles in the period of interest.[29] Results hold if we control for the number of famines experienced by the city's region or country in 1300-1400 or 1300-1600 (row 9). Jebwab et al. (2018) show that higher-mortality cities persecuted Jewish communities less and that Jews were relatively skilled. Results nonetheless hold if we add dummies for whether the city had a Jewish community, a persecution took place, and the persecution took the form of a pogrom during the Black Death (row 10). Row 11 shows results hold if we drop any city with a persecution. Jewish populations were very small relative to the non-Jewish city populations. As such, these pogroms were unlikely to affect human capital in the short to medium terms.

In Table 3, we implement three IV strategies: IV1, IV2 and IV3. IV1 and IV3 rely on the date of first infection in the city, which is available for 124 cities.[30] 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. The first IV exploits the fact that there was a lot of randomness in Plague diffusion depending on where infected rats and fleas went. We thus create a variable for date of first infection for a city. 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. Using the number of months since October 1347 as an IV, and adding the controls of Table 2 (incl. longitude

[28] Subsequent plagues were not correlated with mortality (Web Appx. Table A.4). Later recurrences also had a different epidemiology to the initial outbreak (Web Appx. Section 4.).

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

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

and latitude) and the squares and cubes of longitude and latitude to exploit the random component of the spread of the Plague, we find coefficients similar to our OLS estimates (row 12 of Table 3; -1.07** and 0.05; IV-F stat = 11.8).

IV2. Proximity to Messina should predict Plague mortality, since the port of entry happened to be Messina (due to mischance) and the Plague was more virulent initially (for pathogenic reasons). 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 MENA (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 (then possibly endogenous) speeds of Plague transmission. We add the same controls as for IV1, including the controls for the various possible means of transportation. Controlling for longitude and latitude and their squares and cubes is then particularly important because it will capture any South vs. North and East vs. West effects. Lastly, Messina is dropped from the regressions. We report the IV estimates in row 13. 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 almost twice lower than the short-run effect and not significant.[31]

IV3. 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)

[31]For this IV, we use cities above 1,000 in Europe and cities above 10,000 in the MENA (population estimates not available below). Next, what could matter for overall trade to influence mortality could be proximity to large cities only, or proximity to many cities, even if small. We take an intermediary approach and use as weights the log of city 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 city population – giving more weight to the largest cities – or no weights – making a high spatial density of cities relatively more important – when computing the average distance to all cities (not shown, but available upon request).

(the quadratic fit omits January (1), which has abnormally high mortality rates due to October being the month of onset of the plague in Europe). We report the results using our third IV, dummies for the month of peak infection, while simultaneously adding the controls used for IV1 and dummies for the year of first infection to control for the fact that cities infected in earlier years had higher mortality. We obtain similar results (row 14; -0.93*** and -0.23; IV-F stat = 6.0).

Lastly, results hold when using the three IVs simultaneously (row 15). The IV-F-statistic is low, however, which is mechanical as one includes more controls.[32]

Summary. The identification strategies return effects that are not significantly different from our OLS effects, implying that Plague mortality was indeed locally exogenous, consistent with random factors more than offsetting non-random factors. In the rest of the analysis we can thus only employ OLS estimates.

Results are also robust to potential concerns about specification, data measurement, and sampling. Row 1 of Table 4 reports our baseline estimates.

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

Measurement Concerns. Classical measurement error in mortality should bias the short-run estimates towards zero. In that case, our effect is less negative than the true effect, which is less of an issue than a downward bias. Additionally, if measurement error is classical, the long-term effect should be as biased as the short-term effect, which would not change our results. However, measurement error could be non-classical. Yet, our estimates do not systematically differ when: we (i) include dummies for different sources of mortality data (row 6);[33] (ii) drop estimates based on literary descriptions (row 7); (iii) drop estimates based on

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

[33] These are: population records, literary descriptions, desertion rates, and clergy mortality.

desertion rates (row 8); (iv) drop estimates based on clergy mortality (row 9); and (v) only use the raw numerical mortality estimates directly provided by Christakos et al. (2005) (row 10), thus omitting the 25 description-based mortality estimates. Among these, "high" (assigned 50%) appears 15 times, "spared" (5%) 3 times, and the other seven descriptions only once. We verify that results hold if we use a different rate for "high" (e.g., 60 or 40%) and spared (e.g., 10 or 0%), or drop each description one by one (not shown). Finally, in row 11 we focus on cities that are either in the bottom 10% of least affected cities or in the top 10% of most affected cities, since measurement errors in mortality rates are more likely when comparing cities with relatively similar estimated rates.

Classical measurement error in the dependent variable should increase standard errors. However, our results remain precise. We also employ alternative population estimates. Row 12 reports estimates using the uncorrected Bairoch data. Row 13 reports results using only observations from Chandler (1974, 1987).

External Validity. There could be concerns about sample size. We employ as regression weights populations in 1300 ensuring less weight is placed on small towns. Our 165 cities capture 60% of the total population of the 466 existing cities in 1300 (Web Appx. Fig. A.5 shows a map), so having more cities should not change the results. We also use several methods to obtain more mortality estimates. Results hold if we use: (i) the mortality of the closest city with data if this city is within 50 km (row 14, N = 290–286); (ii) the mean mortality of the cities in the same state (row 15, 380–274, SEs clustered at the state level); and (iii) spatially extrapolated mortality rates (row 16, 464–457).[34] Lastly, we study for all 466 cities in 1300 the reduced-form effect of the inverse of log distance to Messina, conditional on average log distance to all cities and the controls used for IV3. The short-term effect remains negative and significant (not shown).

Our sample is biased towards large cities. In row 17 we reweight observations

[34]We create a two-dimensional surface of predicted mortality using an inverse distance weighted function of known mortality rates for the sample of 274 cities with mortality data. For every point on the surface a predicted mortality rate is generated using the closest 15 cities within an approximately 1,000 km radius circle around the point (details in Web Appx. Section 1.).

to match the distribution of city populations in 1300.[35] Initial population size is not the only characteristic in which our sample is selected. For the 466 cities, we regress a dummy equal to one if mortality data is available on the controls of Table 2. We find significant differences for temperature, monarchy, capital cities, and representative cities. If we reweight observations to match the distribution of each characteristic one by one, results are unchanged (not shown). Finally, we drop cities located within France, Germany, Italy, the United Kingdom or Spain (Web Appx. Table A.7). Other countries contribute few cities.

4. The Black Death as a Trade Shock

So far, we only studied 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 Black Death was a massive trade shock, in that it reduced market potential by killing people in many locations.

We find that urban systems relatively recovered to their pre-Plague population levels. This echoes the work of Voigtländer and Voth (2013b), whose Figure 10 uses aggregate data from 23 countries to show that those areas that experienced a bigger population decline in 1300-1400 urbanized faster. We improve upon their mostly graphical analysis by using data on city-level mortality and growth, examining both spillover and general equilibrium effects, and distinguishing the extensive and intensive margins of city growth. To our knowledge, the results that we now discuss have never been econometrically established before.

Cities could be affected by mortality in nearby towns. Table 5 studies the effects of a city's own mortality and the population-weighted average mortality of neighboring towns, defined in four ways:[36] (i) of the same state (1300); (ii) of the same country (2020); (iii) within the bottom 10% of distance to the city; and

[35] 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.

[36] We include all 1,801 Western European towns. We give 500 inhabitants to the towns with less than 1,000 in 1300. Note that we do not include cities in Eastern European or MENA countries for this exercise because we do not have local mortality estimates available in these.

(iv) or using all 1,801 cities but relying on the change in market access between 1300 and 1353.[37] Cities that experienced a large direct shock did not always experience a large indirect shock (correlation of 0.43-0.51). The indirect effect is always negative, but not significant. The combined direct and indirect effects are about -1.00 to -1.25, and significant. By contrast, the long-run direct and spillover effects are not significantly different from 0 (coefficients appear large but the beta coefficients are very small, at -0.02/0.02).

There may also have been effects at the state level. In col. (1)-(5) of Table 6, we estimate the effects of population-weighted average mortality at the state/country level on the percentage change in urban population at the state/country level.[38] Col. (1) of Panel A replicates our baseline short-run effect at the city level (-0.87***). Col. (2) and (4) show the effects at the state and country levels, for cities that existed in 1300. The effect is now more negative (ca. -1.15). Note that we cannot directly compare our results with the Figure 10 of Voigtländer and Voth (2013b) because their left and right hand side variables are different. For example, they do not use data on Black Death mortality.

In col. (3) and (5), we examine the state- and country-level effects on all cities that are in the dataset in 1400 (incl. cities not in the dataset in 1300). The effects are larger than before, at -1.27 to -1.47 implying that in high-mortality areas, fewer new cities emerged. We verify this in col. (6)-(7) using 1,335 cities in the Bairoch data set that did not exist in 1300. These cities can be thought of as the universe of potential city locations. We test whether their emergence in 1400 — via a simple dummy — is related to the Black Death. Cities were less likely to emerge when their extrapolated mortality rate was high (col. 6). Likewise, we regress the log population of these 1,335 cities (using 500 for cities below 1,000) on mortality and find that fewer locations became urbanized in high-

[37] To construct market access in 1353, we use the predicted population of the other towns in 1353 ($= \text{pop}_{1300}$ x (100-mort.)/100). 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.

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

mortality areas (col. 7). Consistent with previous results, we find however that these negative effects of the Black Death disappeared by 1600 (Panel B).[39]

Thus, the Black Death did not delay the transition of villages into towns. Small towns exist because they serve the needs of villages. As such, their "recovery" disproportionately relies on fixed geographical factors that are particularly important in the rural sector, whose own recovery effects we now discuss.

5. The Countryside and Mechanisms of City Recovery

5.1. 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) (see Web Appx. Sections 9.-10.). Kaplan et al. (2009) recreate localized data on land use from 1100 to 1850 at the 5 by 5 minute (10 x 10 km) cell level by combining information on country population, historical forest cover maps, and soil suitability.[40] Using these data, we obtain the

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

[40] Other sources have used data on tree rings or fossil pollen for plants used by humans, but for

land use share of the in-sample countries. The share was two thirds by 1300 and decreased by 15 percentage points by 1400. Land use recovered by 1800.

We obtain the mean land share within a 10 km radius of each of the 165 cities and examine how land use varied.[41] Web Appx. Table A.8 shows that mortality led to reforestation in 1300-1400, which remained significant until 1500. No effect is found after 1600.[42] Overall, cities thus recovered their populations by 1500 and their rural areas recovered theirs by 1600 (Fig. 4(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 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.

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"

few regions (van Hoof et al., 2006). As such, we cannot use this data for our analysis.

[41] 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. Additionally, we always control for the contemporaneous changes in both city population and country population.

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

(Platt, 1996, 20) (see Web Appx. Section 11.). 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 7 that mortality had a negative effect in the short run and no effect in the long run. For the same 28 counties, we obtained population in 1086, 1290, 1377, 1756 and 1801 (data unavailable ca. 1600). For the period 1290-1377, we find a negative effect, at -0.64** (col. (1)). For the period 1290-1756, we find an insignificant effect, at -0.96 (col. (2)), but the effect is smaller than the short-run effect once standardized (beta 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.[43]

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.[44] We find a negative

[43]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

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

effect of mortality, at -0.46*** (col. (4), beta coef. of -0.51).[45] Low-mortality areas had *more* DMVs than high-mortality areas. Therefore, people disproportionately left the relatively Plague-free rural areas. Web Appx. Table A.8 showed that rural areas in the vicinity of cities—within 10 km—were not affected by the Black Death in the long-run. By 1600 they were completely repopulated. We should thus expect relatively more DMVs in low-mortality areas *beyond* 10 km from cities. For 39 counties, we obtain from Fenwick and Turner (2015) the location of each DMV in England and compute the minimal distance to a 1300 city. For each of the 28 counties, we construct the number of DMVs (per 1000 sq km) both within and beyond 10 km from a city. We then verify in col. (5)-(6) that the loss of villages is driven by areas farther away from cities.[46] In col. (7), we regress the absolute change in the urban share (%) on mortality and find a small and insignificant negative effect.[47] 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.

5.2. 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.[48] Instead, Web Appx. Section 6. 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

[45] 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).

[46] 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).

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

[48] See, for example, data sets inspired by Robert Allen's work: http://www.iisg.nl/hpw/data.php.

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 actually 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 7.).

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). 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).[49]

EMP. The EMP was stronger in Northern than in Southern Europe (Dennison and Ogilvie, 2014; Voigtländer and Voth, 2013a). In other words, for a same mortality shock, we should expect fertility to decrease faster in a Northern Europe region or a region characterized by the EMP. We thus test if the speed at which high-mortality cities relatively recover depends on where the cities were located. We

[49] 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. We discuss how the decline of serfdom was related to the Black Death in Web Appx. Section 8.. As shown by Wolitzky and Acemoglu (2011) the effects of labor scarcity on the use of labor coercion are ambiguous.

classify our cities into North/Strong EMP vs. South/Weak EMP, based on data from Dennison and Ogilvie (2014) on the average age at first marriage and the female celibacy rate (%) at the country or regional level. For the 165 cities, we use the same specification as before but interact mortality with a North/Strong EMP dummy and test if the interacted is negative and significant. Indeed, if natural increase was important for local recovery, we should expect high-mortality cities in North/Strong EMP regions to recover relatively slower than high-mortality cities in South/Weak EMP regions, because North/Strong EMP cities were more likely to recover solely through migration whereas South/Weak-EMP cities were more likely to experience both migration *and* natural increase.

Web Appx. Table A.9 shows the effects of mortality, the North/Strong EMP dummy and their interaction 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 North/Strong EMP cities did not recover relatively slower, since the coefficients are not negative and significant, thus suggesting that migration was likely the main driver behind recovery. However, two caveats with this analysis is that the EMP measures from Dennison and Ogilvie (2014) are estimated for the post-Black Death period. In addition, while the EMP probably already existed before the Black Death (Laslett and Wall, 1972), Voigtländer and Voth (2013a) specifically showed how the EMP was shaped by the Black Death.

6. Heterogeneity in City Recovery and Fixed Factors

While, in a Malthusian economy, a pandemic might have no long-term spatial effects *on average*, in the sense that high-mortality cities fully recover relative to low-mortality cities, there could be still *permutations* between cities, as some large cities become relatively smaller, and small cities, relatively larger. These permutations may in turn be affected by mortality and the characteristics of these cities interact, which we now investigate. Now, if permutations are explained by the fact that some cities have "better" characteristics than others,

then a pandemic might lead to a beneficial spatial reallocation of population.

Permutations. Historical evidence suggests there was heterogeneity across cities in the response to the Black Death.[50] For our sample of 165 cities, we regress the rank of each city in 1600 on its rank in 1300 and find a slope of 0.86***. Therefore, large cities tended to remain large cities and small cities tended to remain small cities after the Black Death. However, aggregate urban recovery hides *permutations* in that the R2 of the regression is 0.56. Figure 5(a) illustrates this, with many cities far from the regression line. After World War II, Hiroshima, Nagasaki and other Japanese cities regained almost their exact pre-war rank. This would imply a slope coefficient close to 1.00 but an R2 also closer to 1.00.

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

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

For cities experiencing the *same* mortality shock, the vector $\boldsymbol{\theta}$ captures the differential recovery effects of each factor. Throughout, we focus on our main sample of 165 cities, for the period 1300-2015. From the variables in Table 2, we select those that proxy for: (i) *rural fixed factors*: the three agricultural suitability measures (cereal, potato, pastoral); and (ii) *urban fixed factors*: coastal and river dummies, Roman road or medieval land route intersections, and the Hanseatic League dummy.[51] While the coast and rivers are by construction "fixed", they lowered transportation costs. Hence, coastal and riverine cities were more likely to develop a trading sector. Roads and the Hanseatic league can be treated

[50] Campbell (2016, 365) notes that "as demand subsided and markets shrank, towns competed with each other in an urban survival of the fittest. Those able to secure a new commercial niche [...] fared well and grew, but 'success' for most meant the avoidance of decline." See Web Appx. Section 12. for a lengthier discussion of the permutations in the data.

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

as fixed factors, since they represent past sunk investments in transportation and trade networks. Roman roads remained the basis of the road network in the medieval era (Dalgaard et al., 2018). Medieval trade routes reflected long-established trading linkages. The origins of the Hanseatic league go back to its establishment in Lübeck in 1159 and it rose to prominence in the century before the Black Death (Dollinger, 1970, xviii). Lastly, we include factors proxying for agglomeration effects and institutions: (i) The log of the estimated population of the city in 1353 (= pop._{1300} x (100-mort.)/100), since larger cities in the aftermath of the Black Death may have recovered faster due to agglomeration effects; and (ii) Three dummies for whether the city was part of a monarchy, was a state capital, and whether it had a representative body (ca. 1300).

Table 8 shows the 11 interacted effects, for 1300-1750 (col. (1)-(5)) and 1300-2015 (col. (6)). The 11 interacted effects being *simultaneously* included, they show the recovery effect associated with each factor *conditional* on the recovery effect associated with each other factor. With 165 observations and 23 variables, this makes our test particularly stringent. Note that we show the interacted effects for the period 1300-1400 because cities started recovering in 1353-1400. We then use 1300 as the start year instead of 1353 because we do not know the true population of each city in 1353. Finally, we predict that the recovery effects of the fixed factors increase with their economic value, which may exogenously change over time, for example with new technologies. As such, since the factors we consider are likely to affect nominal wages much more than housing and non-housing prices and amenities, especially as pre-modern construction technologies were not particularly advanced or distinct across European cities, we interpret these recovery effects as reflecting nominal wage changes.[52]

We do not use panel or IV regressions for these regressions. We showed they returned results that were not different from the OLS, implying exogeneity of

[52]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).

mortality. We thus rely on OLS, given its simplicity and transparency.[53] 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.

Rural Fixed Factors. The coefficients of mortality*cereal suitability becomes positive (but are not significant) after 1400 (col. (2)). However, the effect is meaningful since the beta coefficient (henceforth, "beta") reached 0.47 by 1600 and remained high after (0.17 in 2015). Potato suitability also helped cities recover from the 17th century onwards (col. (4)). Nunn and Qian (2011) show that countries that were relatively more suitable for potato cultivation urbanized faster after potato cultivation diffused in Europe (the non-effects in col. (1)-(3) are reassuring). Their country-level effects 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). Next, 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). This effect diminishes after 1750. This may reflect the rise of proto-industry in rural areas, in particular textile production, which was associated with more rapid population growth (Mendels, 1972; Pfister, 1989). Wool was indeed the most common textile used in making clothing.

Urban Fixed Factors. The interacted effect for coastal proximity is one of the only two significant coefficients in 1300-1400 (col. (1) of Table 8), along with the interacted effect for the Hanseatic league. These factors can thus help account for the rapid relative recovery of some cities in 1353-1400. Stark examples include Barcelona (mortality of 36%; full recovery by 1355) and Venice (60%; 1375) for

[53] 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.

coastal cities and Lübeck (30%; 1360) as an example of a major Hansa town. The coastal effect is strong throughout (beta = 0.99 in 1500, slowly decreasing to 0.33 in 2015). The Hansa effect is strong (beta = 0.72) in 1300-1400 and significant until 1700 (col. (4)), by which time the league was in decline (Dollinger, 1970).

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

Next, 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.[54]

In rows 1-3 of Table 9, we examine whether specific coastal and riverine cities recovered faster. In our main analysis we consider cities within 10 km of the coast. However, cities located *directly* on the coast may have recovered faster than those several kilometers inland. A 10 km band also does not include some estuarine cities with a free connection to the open sea (e.g., Bordeaux). We replace the coast dummy and its interaction with mortality by three dummies for being located directly on the coast, for being within 50 km from the coast and its estuaries, and for being within 50 km of the coast and on a river, and their respective interactions with mortality (the river dummy is adjusted accordingly

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

to exclude such cities). As seen in row 1, the effects are stronger for truly coastal or estuarine locations, which we call the "best" coastal locations.

Row 2 shows the results when we instead replace the coast dummy and its interaction with mortality with two dummies for being one of the best Mediterranean coastal locations and for being one of the best Atlantic locations.[55] We find strong recovery effects for the Mediterranean. Indeed, Mediterranean trade was particularly important around the time of the Black Death. We also observe significant recovery effects for Atlantic cities starting in the 17th century. This is consistent with Acemoglu et al. (2005) who show that the Atlantic trade led to Atlantic ports with good institutions over-taking Mediterranean ports starting around 1600. However, in their analysis, identification comes from comparing the growth of Atlantic and non-Atlantic cities over time, whereas in our case it comes from the triple interaction between mortality, the coastal locations considered optimal, *and* the timing of the boom in Atlantic trade. As such, it is reassuring that we find no recovery effect in the earlier centuries.

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

Agglomeration Economies. The literature suggests that a larger population should increase productivity and wages, which may cause in-migration. The literature (e.g., Duranton and Puga, 2004) distinguishes economies of scale – in production, market places, and consumption – and agglomeration economies strictly defined, which include: (i) *sharing* the gains from access to varied inputs, a deepening of the division of labor, and risk pooling across sectors; (ii) *matching*,

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

between firms and suppliers and between employers and employees; and (iii) *learning*, i.e. the creation or diffusion of knowledge. Table 8 shows no significant recovery effect for cities with a larger residual population in 1353.

In the right panel of Table 9, we further examine the role of agglomeration economies in urban recovery following the Black Death. Using equation 2 but adding log population in 1300 and its interaction with mortality, we find no effect (row 4). It is possible that cities that were larger before the Black Death had more durable structures and infrastructure by 1353 and thus recovered faster. The city's historical "reputation" may also have mattered. But we find no evidence for either of these effects. We also find no positive, significant recovery effect of estimated market access (row 5) or estimated state population size (row 6) in 1353. Taken together these results suggest that agglomeration economies were not a significant factor in the post-Black Death recovery.

Turning to the economies of scale mechanisms, we do not find positive significant effects when adding the following variables and their interaction with mortality: a dummy for guild presence before the Black Death (row 7; source: Ogilvie (2019)), a dummy for the presence of a market fair circa 1300 (row 8), and log walled area circa 1300 (controlling for log city population size circa 1300) (row 9). Guilds reflect whether there was an industry large enough to organize and hence can proxy for economies of scale in production. However, guilds also reflected the political power of craftsmen and artisans (Ogilvie, 2019). The presence of a market fair can proxy for economies of scale in market places. The extent of a city's walls may proxy for economies of scale in production, market places, and consumption. Due to warfare between European states, walled cities offered protections to craftsmen, merchants, and more broadly residents, which Dincecco and Onorato (2017) and others have labeled the "safe harbor" effect. As Weber (1958, p.77) put it, the medieval city was a "fusion of fortress and market."

We also include a dummy if there was any bishopric or archbishopric circa 1300 (row 10) and if there was a university circa 1300 (row 11). 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 an 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). This may have been less true in Germany where after the Papal schism of 1386, universities started providing training in Roman and canon law, which emphasized contracts and property. This eventually led to city growth (Cantoni and Yuchtman, 2014). Unfortunately, we do not have enough German cities in our sample to test whether the mortality $\times$ university effect was stronger there.[56]

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 affected by serfdom (row 12).

Interpretation and Country Fixed Effects. Are the interacted effects causal? In all regressions presented here, we *simultaneously* control for mortality, the individual effects of the 11 characteristics used for the interactions, and the 11 interactions. The effects also only "activate" when expected. In addition, most important effects of Table 8 remain strong and significant when including 13 modern country fixed effects (Web Appx. Table A.10). Identification then comes from comparing cities experiencing the same exogenous 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.

We find few significant effects of population or institutions. Adding the absolute values of the beta coefficients for the different types of factors shown in Table 8, fixed factors are particularly important in the first centuries after the Black Death and their importance decreases over time (Fig. 6(a)). Adding

[56] We find a smaller and less significant effect in 1600 (3.2* vs. 3.5**) if we exclude Germany.

the beta coefficients also helps taking into account substitution effects possibly due to the interacted effects capturing each other's effect. Other factors such as population and institutions are less important. However, one caveat is that local institutions likely changed as a result of the Black Death (Brenner, 1976; Acemoglu and Robinson, 2012). This is not the focus of this paper, however, and our test imposes that we study the effects of pre-, not post-, Plague institutions.[57]

Table 1 showed no long-term effects of the Black Death at the city level. In Table 8, the significant negative effects of mortality once we control for the fixed factors and their interactions with mortality implies that any city without fixed factors would have possibly remained small (the point estimate becomes more negative but the beta coefficient remains similar across years, at -1.5 in 1300-1400 and -1.1 in 1300-2015). In other words, for cities without important fixed factors, a large Black Death shock would have had permanent, negative local effects.

Next, for our 165 cities existing in 1300, we predict which high-mortality cities would have relatively lost out by 1750 or 2015 had they not had fixed factors. First, to use Table 8 to predict the counterfactual population level of each city in 1750 or 2015 absent the recovery effects related to fixed factors, one must account for the fact that predicted percentage population growth in 1300-1750 or 1300-2015 must be left-censored at -100. We thus re-run the regression but with a Tobit model. The effects on the latent variable are almost the same as with the OLS model (not shown). We also verify that the predicted 1750 rank of each city among the 165 cities – based on predicted 1750 population levels calculated using 1300 populations and the predicted percentage change in 1300-1750 – is strongly correlated (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 fixed factors with their predicted ranks when the same effects are included, we

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

identify cities that would have lost a lot otherwise.

As seen in Figure 5(b), examples of such cities in 2015 include some of the largest cities in 1300: Venice (3rd largest, mortality of about 60%), Florence (5th, 60%), Cordoba (8th, 50%), Naples (9th, 65%), Cologne (10th, 30%), Cordoba (8th, 50%), Pisa (19th, 35%), Toulouse (23st, 50%), Rouen (24th, 45%), and Marseille (28th, 55%).[58] These cities have in common to have been hit hard by the Plague and either coastal, riverine, on a road intersection and/or part of the Hansa league. Some of these cities have become relatively smaller over time, due to other factors. But in the absence of important fixed factors aiding their recovery, they could have been at the bottom of the list of the 165 cities in 1750 and/or 2015. Thus, our analysis suggests that, absent the presence of important fixed factors, these cities would not have recovered, and subsequently grown, following the Black Death, for example during the Renaissance.

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

This is what we find. For example, high-mortality cities along a river started growing significantly faster than other cities from the 17th century. As a result, an increasing share of the total urban population of Europe must have resided along a river over time. Figure 6(b) shows that the unconditional population share of the 165 cities located along a river did increase from 1400-2015. Figure 6(b) also reports the predicted increase in the population share of riverine cities based on their population in 1300 and the estimated contribution of the recovery effect of rivers to predicted city population growth (i.e. the interacted effect of mortality and the river dummy times mortality times the river dummy). As can be seen,

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

the conditional population share along rivers has increased over time, close to the unconditional share. We find similar patterns when we perform the same analysis for the other fixed factors (not shown, but available upon request).

We do not pursue this analysis further as it would require a theory-grounded analysis of what a dynamically optimal distribution of population involves. But we note that the impact of the Black Death on Europe's spatial distribution of population might have been one factor contributing to both the Great Divergence that opened up between Europe and the rest of the world after 1700 and the Little Divergence that took place within Europe itself.

7. Conclusion

We asked what effects a high case fatality rate pandemic could have on city growth. 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 explored the long-run impacts it had on city growth. On average, cities recovered their pre-Plague populations within two centuries. However, aggregate convergence masked heterogeneity in urban recovery. We showed that both of these facts are consistent with populations returning to high-mortality locations endowed with more fixed factors of production. Land suitability and natural and historical trade networks played a vital role in urban recovery. Our study highlights the role played by pandemics and physical and economic geography in determining the relative size of cities in poorer countries more dependent on fixed factors.

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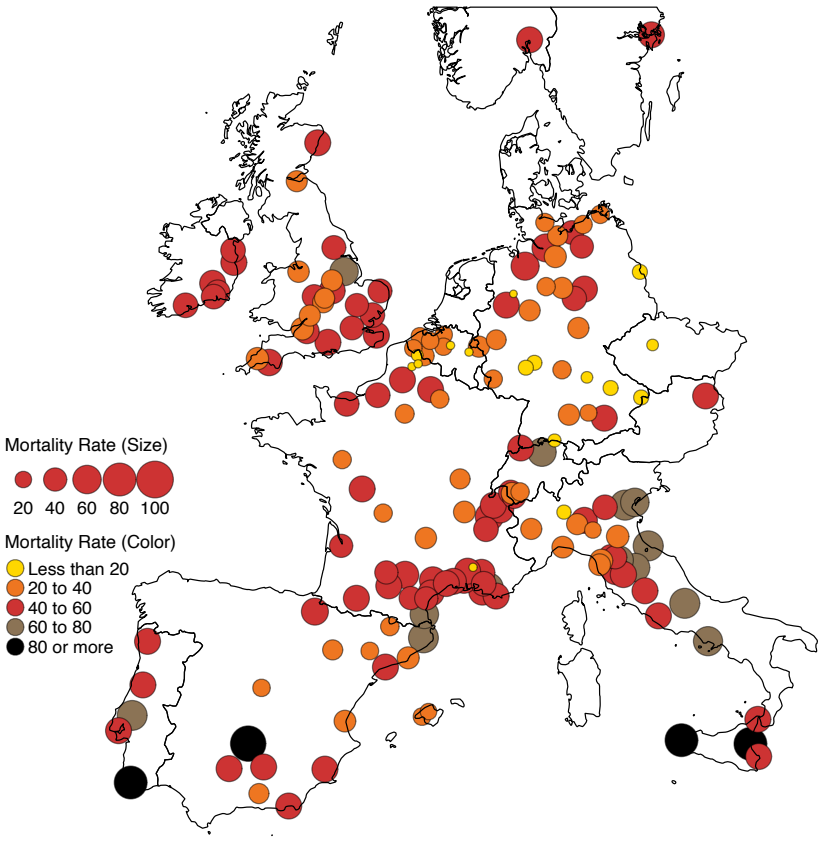
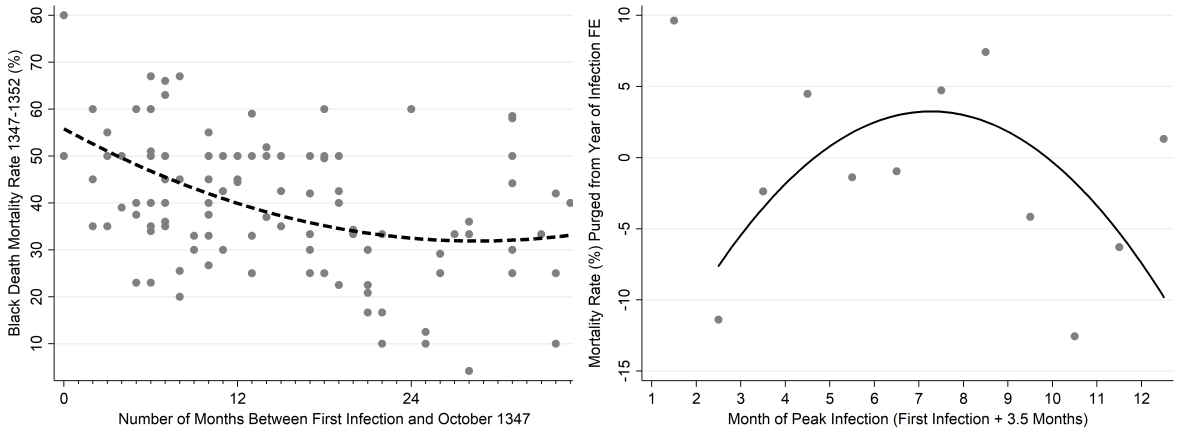
Figure 1: Black Death Mortality Rates (%) in 1347-1352



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.

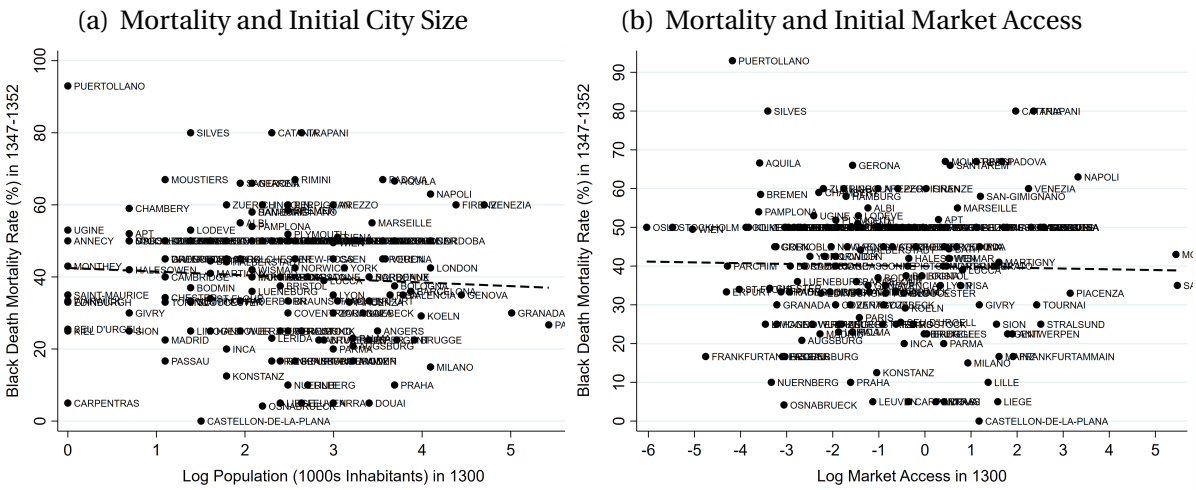
Figure 2: Timing of the Onset of the Black Death and Black Death Mortality

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



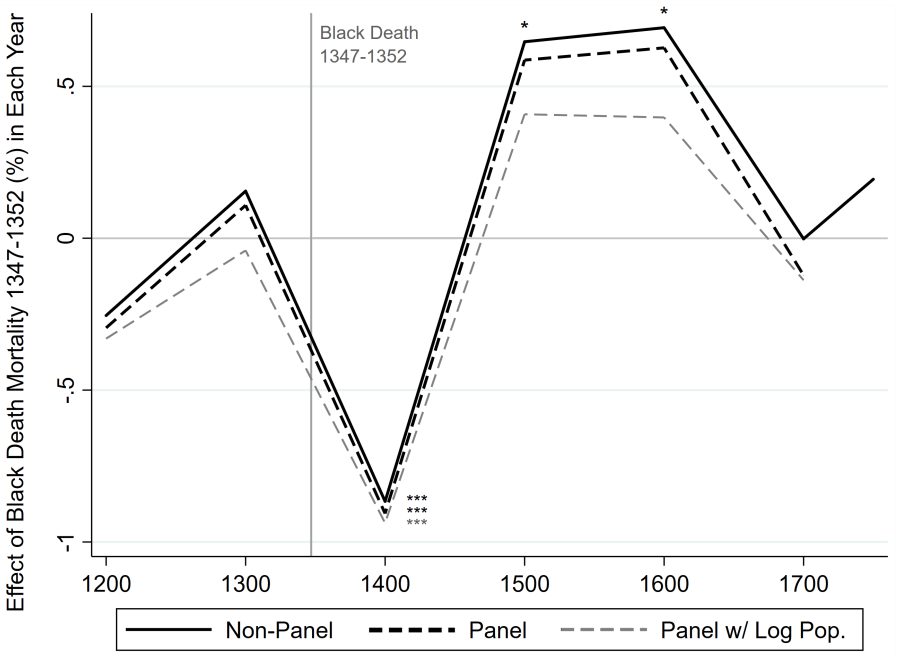
Notes: Subfig. 2(a) shows for 124 cities the relationship between mortality and the timing of the onset of the Black Death in the city. Number of months is measured since Oct. 1347, the date Messina – the port of entry of the Black Death in Europe – was infected by mischance. Subfig. 2(b) shows for 124 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).

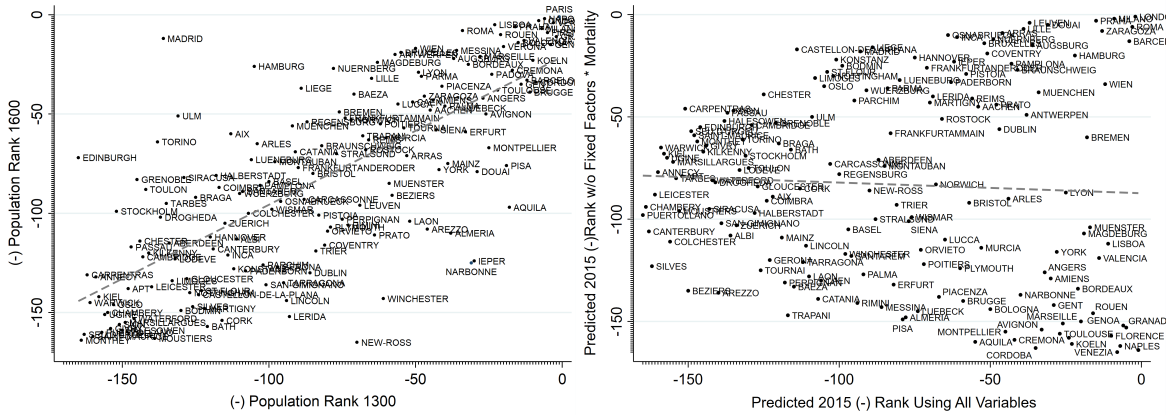
Figure 4: Yearly Effect of Black Death Mortality 1347-52 (%), Panel Regressions



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

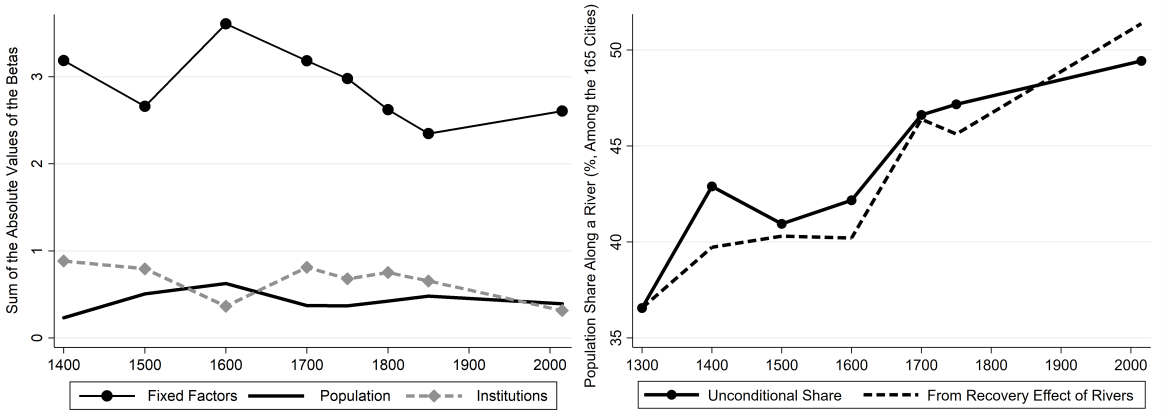
(a) Changes in Pop. Ranks Among the 165 Cities (b) Fixed Factors & Counterfactual Ranks 2015



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

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

Table 3: MORTALITY AND CITY GROWTH, INVESTIGATION OF CAUSALITY

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

Regression:	(1) t = 1300-1400			(2) t = 1300-1600		
1. Baseline (See Columns (1) and (3) of Table 1)	-0.87***	[0.28]	165	0.36	[0.80]	164
2. Controls: All	-0.59***	[0.21]	165	-0.37	[0.70]	164
3. 13 Country (2018) FE	-0.62**	[0.26]	165	0.03	[0.76]	164
4. 7 States (1300) FE (for States >= 5 Cities)	-0.82**	[0.35]	105	-0.29	[0.68]	104
5. Dropping Top and Bottom 5% in Mortality	-0.67**	[0.29]	145	1.36	[1.12]	144
6. Dropping More Hygienic Cities	-0.89***	[0.30]	161	0.59	[0.90]	160
7. Ctrls for Dummy & Num. Plague Recur. 50km	-0.87***	[0.27]	165	0.41	[0.81]	164
8. Ctrls for Dummy & Num. Battles 50km	-0.84***	[0.28]	165	0.77	[0.78]	164
9. Ctrl for Num. of Famines in Region/Country	-0.80***	[0.29]	165	0.45	[0.77]	164
10. Ctrls for Jewish Pres., Pers., Pogr. 1347-1352	-0.83***	[0.30]	165	0.28	[0.81]	164
11. Drop if Jewish Persecution 1347-1352	-0.71***	[0.30]	115	0.67	[0.96]	114
12. IV1: Timing w/ Controls (IV F-stat = 11.8)	-1.07**	[0.50]	124	0.05	[1.32]	124
13. IV2: Messina w/ Controls (IV F-stat = 22.6)	-1.20**	[0.56]	163	-0.68	[1.69]	163
14. IV3: Month w/ Controls (IV F-stat = 6.0)	-0.93***	[0.33]	124	-0.23	[0.58]	124
15. 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. Row 4: Adding 44 state FE but excl. states with less than 5 cities in our sample. Row 5: Dropping top and bottom 5% mort. rates. Row 6: Dropping cities with a better hygiene system. Rows 7-8: Adding a dummy if there was a plague recurrence/battle and their numbers in 1353-1400 or 1353-1600. Row 9: Adding the number of famines experienced by the city in t. Row 10: Adding dummies if Jews were present, and if a persecution, and a pogrom in particular, took place. Row 11: Excl. cities with Jewish persecutions. Row 12: IV = number of months between the city-specific date of first infection and Oct. 1347. Row 13: IV = Euclid. dist. to Messina, controlling for (pop.-weighted) avg. Euclid. dist. to European and MENA cities in 1300 (Messina is dropped). 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. Rows 12-15: 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 12-15): † p=0.17, * p<0.10, ** p<0.05, *** p<0.01.

Table 4: 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.	-0.74***	[0.24]	165	-0.06	[0.65]	164

Notes: See text for details on each robustness check. Robust SE's: † p=0.17, * p<0.10, ** p<0.05, *** p<0.01.

Table 5: MORTALITY AND CITY GROWTH, SPILLOVER EFFECTS, 1300-1600

	Dependent Variable: Percentage Change in City Population (%) in							
	(1)-(4) Period 1300-1400				(5)-(8) Period 1300-1600			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mortality Rate (%)	-0.72**	-0.75**	-0.68**	-0.79**	-0.08	0.05	0.16	-0.12
	[0.31]	[0.31]	[0.33]	[0.34]	[0.82]	[0.82]	[0.84]	[0.84]
Indirect Mort. (%)	-0.39	-0.40	-0.57	-0.16	1.49	1.08	0.60	1.07
	[0.43]	[0.47]	[0.47]	[0.39]	[1.50]	[1.18]	[1.78]	[1.16]
Definition Indirect	State	Country	Dist10%	MAshock	State	Country	Dist10%	MAshock
Observations	160	165	165	165	159	164	164	164

Notes: State/Country: Avg. mortality rate of other cities in the same state (1300) / country (2018). Dist10%: Avg. mortality rate of other cities within the bottom 10% of Euclidean distance to the city. MAshock: Pct. change in market access between 1300 and 1353. To construct market access in 1353, we use the predicted pop. of the other cities in the aftermath of the Black Death (= pop. in 1300 x (100-mortality)/100). Since mortality is only available for a subset of the other cities, we use spatially extrapolated mortality rates for cities without mortality data. Note that for each of the 165 city-observations, the extrapolated mortality rates are constructed excluding the mortality rate of the city itself. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01.

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

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

Notes: State/Country: We run the main regression at the state (1300)/country (2018) level. Intensive/Total: The cities considered to construct total city pop. are the cities that already existed in 1300 / all cities. Extensive: We consider cities that did not already exist in 1300 but existed at one point in Bairoch (1988) (800-1850). Robust SE's: * p<0.10, ** p<0.05, *** p<0.01.

Table 7: BLACK DEATH MORTALITY AND DESERTED VILLAGES, ENGLAND

Dep. Var.:	Percentage Change in Population (%) in Period t			Number of DMVs per 1000 Sq Km			Abs. Change Urban Share
	1290-1377	1290-1756	1086-1290	All	$\leq$10Km	>10Km	1290-1756
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
β_t	-0.64**	-0.96	0.05	-0.46***	-0.04	-0.41***	-0.16
	[0.31]	[2.06]	[2.77]	[0.33]	[0.10]	[0.30]	[0.21]
Obs.	27	27	27	28	28	28	27
R2	0.13	0.01	0.00	0.31	0.06	0.35	0.02

Notes: 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 know. 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 8: BLACK DEATH MORTALITY AND POPULATION RECOVERY, 1300-2015

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

Period 1300-t:	1400 (1)	1500 (2)	1600 (3)	1700 (4)	1750 (5)	2015 (6)
Mort.*Cereal Suitability Index	-0.1	0.2	0.6	0.9	1.0	8.8
	[0.3]	[0.3]	[0.7]	[1.5]	[1.7]	[33.0]
Mort.*Potato Suitability Index	0.3	-0.2	0.9	2.6**	3.0**	51.6**
	[0.3]	[0.4]	[0.6]	[1.3]	[1.5]	[23.9]
Mort.*Pastoral Suitability Index	0.6	-0.3	-4.1*	-3.2	-5.7*	-63.7
	[0.7]	[1.1]	[2.1]	[2.8]	[3.2]	[61.4]
Mort.*Coast 10 Km Dummy	1.2**	2.9***	4.8***	7.3*	7.6*	72.6
	[0.5]	[0.7]	[1.8]	[3.7]	[4.3]	[79.3]
Mort.*Rivers 10 Km Dummy	-0.5	0.3	1.7	5.0**	6.0**	112.4***
	[0.5]	[0.7]	[1.1]	[2.1]	[2.4]	[39.7]
Mort.*Road Intersection 10 Km Dummy	0.6	1.5*	1.3	2.2	2.7	31.3
	[0.6]	[0.8]	[1.5]	[2.7]	[3.1]	[57.7]
Mort.*Hanseatic League Dummy	2.9***	2.3*	4.1*	7.3*	8.5	92.2
	[0.9]	[1.2]	[2.4]	[4.4]	[5.8]	[104.2]
Mort.*Log Est .City Population 1353	-0.2	0.6	1.4	1.7	2.2	35.7
	[0.2]	[0.4]	[1.0]	[2.0]	[2.3]	[42.0]
Mort.*Monarchy 1300 Dummy	-0.2	0.7	1.2	2.3	1.9	-26.4
	[0.5]	[0.6]	[1.2]	[2.1]	[2.4]	[43.0]
Mort.*State Capital 1300 Dummy	-0.6	-1.5	-0.3	4.7	4.7	-19.1
	[0.8]	[1.3]	[2.5]	[4.3]	[5.3]	[87.1]
Mort.*Representative Body 1300 Dummy	0.8	-0.2	-0.5	-2.1	-3.1	-22.7
	[0.6]	[0.7]	[1.1]	[2.0]	[2.4]	[39.5]
Mortality	-3.9***	-1.8	-6.1*	-19.6**	-20.7**	-373.8**
	[1.4]	[1.9]	[3.5]	[9.2]	[10.2]	[156.9]
Observations	165	164	164	164	164	165
R-squared	0.45	0.29	0.39	0.35	0.35	0.25

Notes: This table shows for 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 factors are used as controls. We use as weights city population in 1300. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01.

Table 9: MORTALITY AND POPULATION RECOVERY, OTHER RESULTS

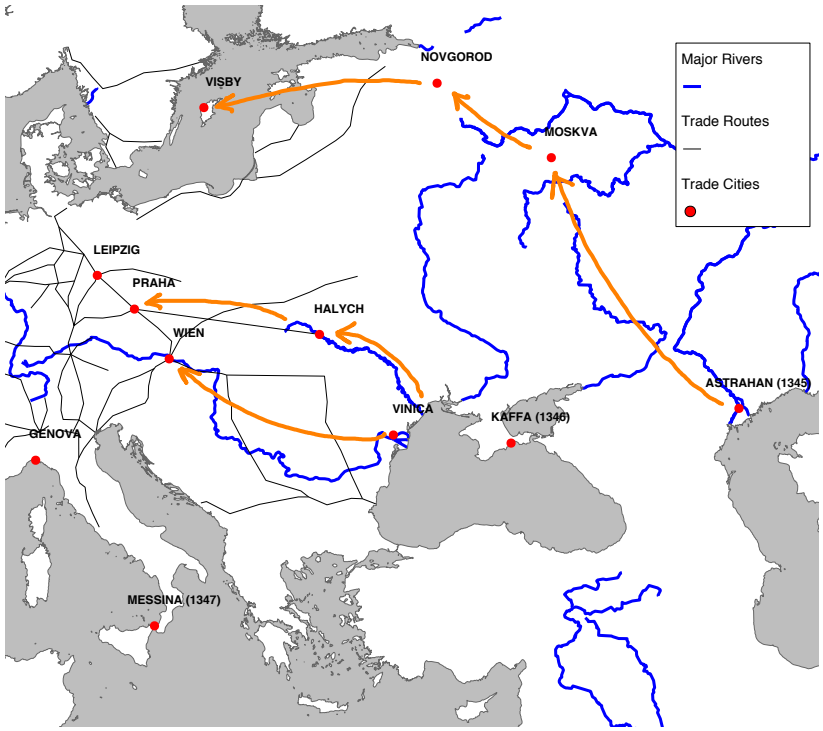
Dependent Variable: Percentage Change in City Population (%) in Period 1300-t

Period 1300-t:	1500 (1)	1600 (2)	1700 (3)	1750 (4)		1500 (5)	1600 (6)	1700 (7)	1750 (8)
Each row is for a separate regression and shows the effect of Mortality (%) × the variable shown at left									
1. 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 8 except we add the variable shown at left and show the effect of its interaction with Black Death mortality (%). We use as weights city population in 1300. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01.

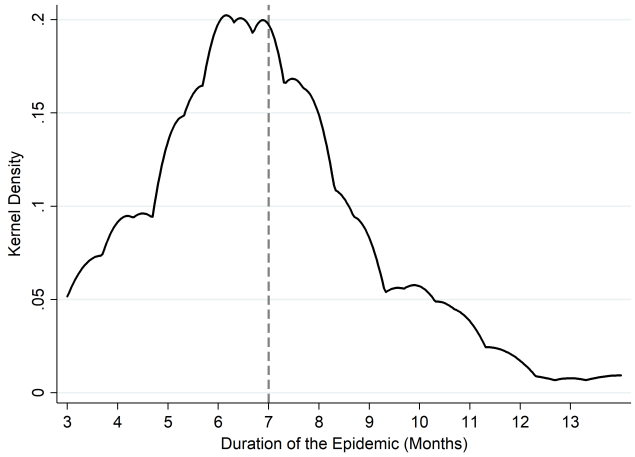
WEB APPENDIX - NOT FOR PUBLICATION

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



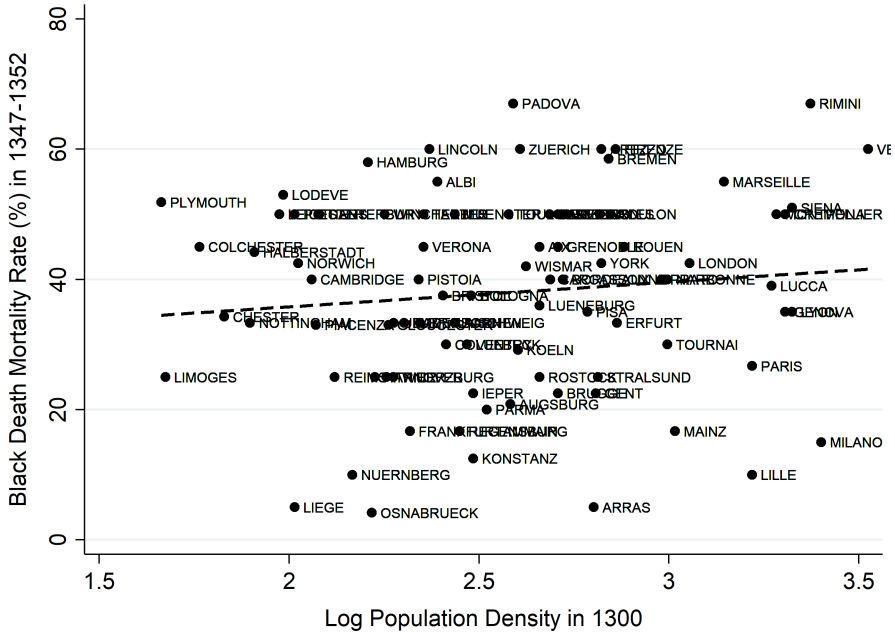
Notes: This map plots the location of the main port of entry in Eastern Europe, Astrakhan (Ukraine) in 1345. The Plague went from Astrakhan to the Genoese colony of Kaffa (Ukraine) in 1346. From Kaffa, it was carried to Messina (Sicily) in 1347. The map also shows various counterfactual ports of entry 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). See Web Appendix for more details on data sources.

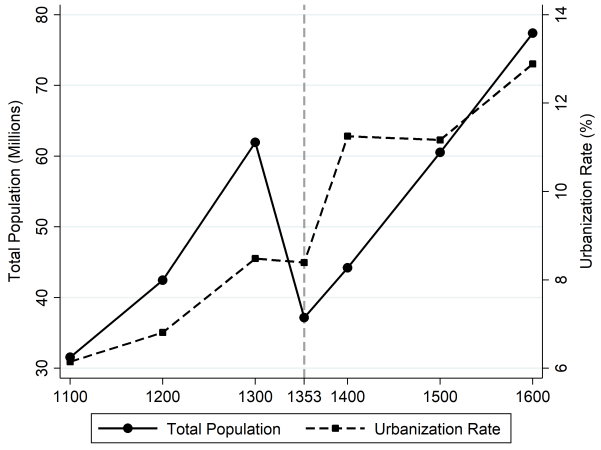
Figure A.3: Black Death Mortality and City Population Density circa 1300.



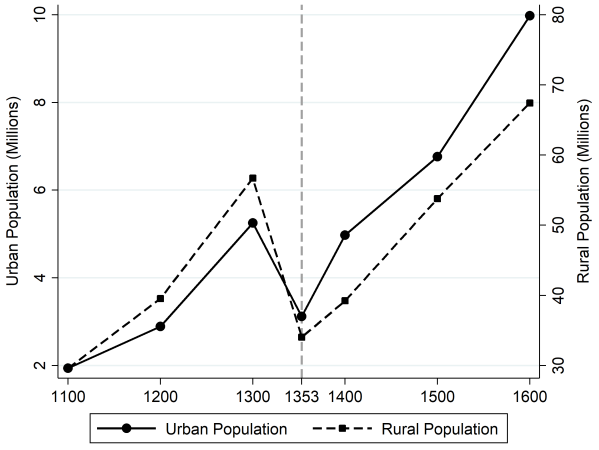
Notes: The figure shows the relationship between Black Death mortality (%; 1347-1352) and city population density defined as population divided by walled land area (Y = 28.2*** + 3.79 X; Obs. = 88; R2 = 0.01). Walled land area is available for only 88 out of the 165 cities in our sample. Total land area does not exist. See Web Appendix for data sources.

Figure A.4: Evolution of Western Europe's Total, Urban and Rural Populations, 1100-1600

(a) Total Population and Urbanization

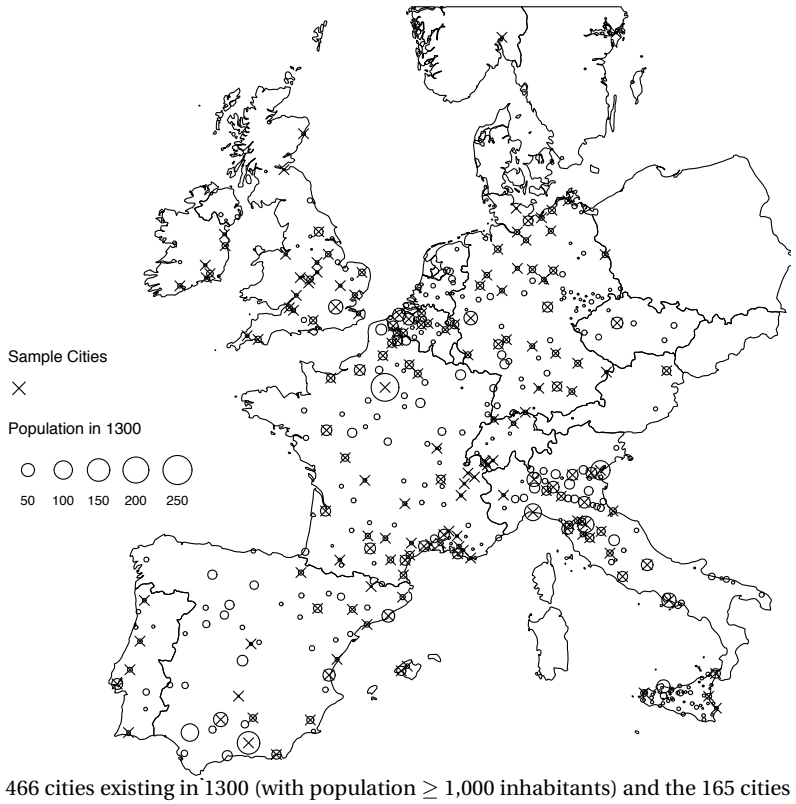


(b) Urban vs. Rural Population



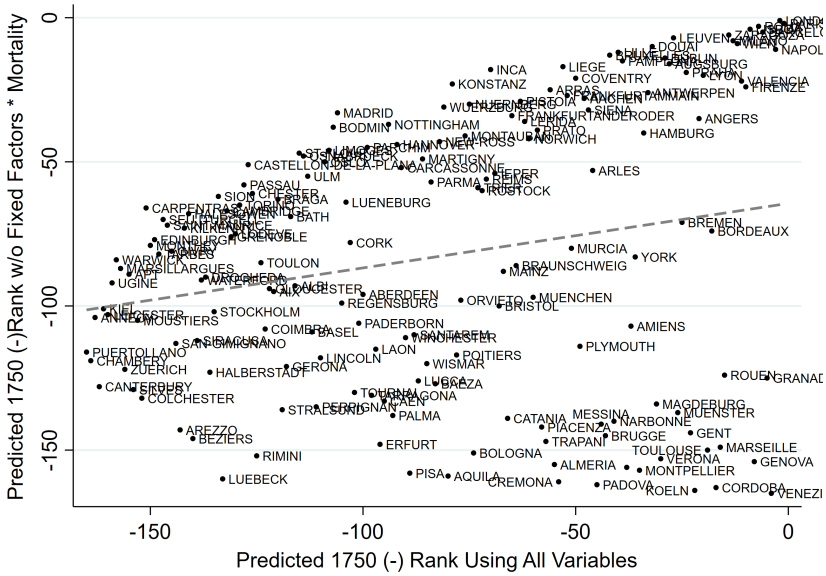
Notes: The subfigures show the respective evolutions of the total population (millions) and urbanization rate (%) (Subfigure 4(a)) and the total urban and rural populations (Subfigure 4(b)) of the 16 countries in 1100-1600. Total (urban) population in 1353 is proxied by the total (urban) population in 1300 times the pop.-weighted average total (urban) mortality rate in 1347-1352 (38.9% and 38.8%). Rural populations are estimated residually. See Web Appendix for data sources.

Figure A.5: 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.6: 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 8 (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).

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

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
TABLE 3					
Hygienic City	165	0.0	0.2	0.0	1.0
Plague Rec. 50Km 1400 Dummy	165	0.5	0.5	0.0	1.0
Plague Rec. 50Km 1400 Number	165	3.1	4.6	0.0	18.0
Plague Rec. 50Km 1600 Dummy	165	0.9	0.3	0.0	1.0
Plague Rec. 50Km 1600 Number	165	24.0	26.5	0.0	123.0
Battle 50 Km 1400 Dummy	165	0.1	0.3	0.0	1.0
Battle 50 Km 1400 Number	165	0.1	0.4	0.0	2.0
Battle 50 Km 1600 Dummy	165	0.4	0.5	0.0	1.0
Battle 50 Km 1600 Number	165	0.8	1.2	0.0	6.0
Famine 1300-1400 Number	165	11.3	7.9	3	23
Famine 1300-1600 Number	165	35.3	13.7	21	61
Jewish Presence 1347-1352	165	0.7	0.5	0.0	1.0
Jewish Persecution 1347-1352	165	0.3	0.5	0.0	1.0
Jewish Pogrom 1347-1352	165	0.3	0.4	0.0	1.0
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

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 4					
Pct.Chg.Pop. 1200-1300	93	59	102	-70	483
Abs.Chg.Pop. (000s) 1300-1400	162	-1.78	10.1	-50.0	55
Abs.Chg.Pop. (000s) 1300-1600	161	5.7	27.3	-81.0	215
Abs.Mort. (000s) 1347-1352	165	7.28	10.0	0.0	66.0
Pct.Chg.Pop. 1300-1400 Bairoch	151	-5	62	-93	400
Pct.Chg.Pop. 1300-1600 Bairoch	150	74	228	-83	2067
Pct.Chg.Pop. 1300-1400 Chandler	59	3	50	-97	175
Pct.Chg.Pop. 1300-1600 Chandler	61	52	117	-84	500
Mortality Nearest City 50 Km	291	42.0	18.1	0.0	100.0
Mortality Other Cities State	382	43.1	16.4	0.0	93.0
Extrapolated Mortality Rate	466	42.4	15.0	0.0	93.0
TABLE 5					
Indirect Mort. State	160	40.7	10.3	16.2	68.3
Indirect Mort. Country	165	40.4	9.1	19.5	61.0
Indirect Mort. Dist10%	165	39.9	9.1	22.1	66.9
Indirect Mort. MAshock	165	40.1	11.7	9.3	70.5
TABLE 6					
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
Pct.Chg.Pop. 1400 Country Int.	15	13.4	57.1	-33.3	200.0
Pct.Chg.Pop. 1400 Country Tot.	15	24.6	57.8	-33.3	200.0
Mortality Country	15	40.3	11.7	20.5	56.7
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 7					
Mortality County	28	43.7	7.8	33.0	58.0
Pct.Chg.Pop. 1086-1290	38	244	227	18	1104
Pct.Chg.Pop. 1290-1377	41	-43	24	-79	77
Pct.Chg.Pop. 1290-1756	41	56	126	-55	606
Pct.Chg.Pop. 1290-1801	41	124	229	-46	1004
Num. DMVs Per 1000 Sq Km	41	7.2	6.0	0.0	21.7
Num. DMVs within 10 Km	39	0.5	1.2	0.0	6.8
Num. DMVs beyond 10 Km	39	6.6	5.2	0.3	20.9
Abs. Chg. Urb. Sh. 1290-1756	41	10.7	9.6	-9.9	34.9
TABLE 8 (See Table 2)					
TABLE 9					
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: 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.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: 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]
Month of Peak Infect.: Dec			-
			-
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.7: 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.8: BLACK DEATH MORTALITY RATES AND LAND USE, 1100-1750

Dependent Variable: Percentage Change in Land Use Share (%) in Period t							
t:	1300-1400	1300-1500	1300-1600	1300-1700	1300-1750	1100-1200	1200-1300
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
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
R2	0.28	0.65	0.20	0.38	0.27	0.13	0.14

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 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). 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 Appendix for data sources.

Table A.9: BLACK DEATH MORTALITY, NATURAL INCREASE AND RECOVERY

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

Notes: This table shows for 165 cities the effects of mortality, the North/EMP dummy and their interaction. 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.

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.

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

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

> 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 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 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 5 (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

[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.

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. Our main source is Dollinger (1970).

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). 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.

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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).[3] 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. 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. Rates 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

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

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.

5. Non-Selectivity of Mortality During the Black Death

As discussed in the main text, all contemporary historical accounts emphasize that the initial pandemic that began in 1347 was remarkably non-selective. For instance, Getz (1991, 273) cites James of Agramont's tract on the plague as the first detailed description of the effects of the Black Death [In the region of Catalonia, Spain] where he confirms that it affected "wealthy and poor alike"

> 'James called attention to one of the most marvelous properties of plague: it killed master and servant alike, and even physician and confessor; this distinguished it from other diseases, which were peculiar to individual' (Getz, 1991, 273).

This observation was made by numerous contemporaries and it is this fact that distinguishes the Black Death from later outbreaks of bubonic plague. Urban and rural areas were both hit extremely hard and no class of individual was spared. Edward III's daughter princess Joan, for instance, died in 1348 on her way to marrying to King of Castile. Her death is illustrative because while the rich were able to flee in subsequent plague outbreaks, they did not know to do so in 1348: it is reported that her and her entourage did not flee from Bordeaux when the plague began because they were unaware of how dangerous it was. In this respect, we believe that the Black Death represents a uniquely "pure" shock to population that was remarkably unselective.

Nevertheless, it may well have been the case that the poor were more vulnerable. Christakos et al. (2005, 150) note that the claim that the poor died more than the rich is a plausible sounding statement "rather than an independent scientific conclusion derived from the analysis of real Black Death data". The current consensus is that the Black Death was "a universal killer" (Alfani and Murphy, 2017, 326). Although later plague recurrences did strike the poor more than the rich, this does not seem to the be case in the initial epidemic. The Black Death also affected male and female alike. Alfani and Murphy (2017) note that whether there was a differential impact on age is ambiguous at present but that the evidence does suggest that it did not differentially target the old and weak.

6. 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".

7. Demographic Change After the Black Death

Sections 2. and 6. 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.

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 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. 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).

10. 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).

11. 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.

12. Rising and Declining Cities After the Black Death

As we discuss in Section 6., 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%.[4] 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).

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

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