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

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Abstract

The Black Death killed 40% of Europe's population between 1347-1352, making it one of the largest shocks in the history of mankind. Despite its historical importance, little is known about its spatial effects and the effects of pandemics more generally. Using a novel dataset that provides information on spatial variation in Plague mortality at the city level, as well as various identification strategies, we explore the short-run and long-run impacts of the Black Death on city growth. On average, cities recovered their pre-Plague populations within two centuries. In addition, aggregate convergence masked heterogeneity in urban recovery. We show that both of these facts are consistent with a Malthusian model in which population returns to high-mortality locations endowed with more rural and urban fixed factors of production. Land suitability and natural and historical trade networks played a vital role in urban recovery. Our study highlights the role played by pandemics in determining both the sizes and placements of populations.

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

Keywords: Pandemics; Black Death; Mortality; Path Dependence; Cities; Urbanization; Malthusian Theory; Migration; Growth; Europe

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The Black Death was the largest demographic shock in European history, killing approximately 40% of its population between 1347-1352. Some regions and cities were spared, others were severely hit: England, France, Italy and Spain lost as much as 50-60% of their population in just one or two years. This also makes the Black Death one of, if not the, largest shocks in the history of mankind. While the Black Death has been extensively studied by historians and social scientists, little is know about its spatial effects due to the lack of disaggregated data on mortality. Additionally, little is known about the economic effects of continent-wide pandemics due to their rarity. In this paper, we use spatially disaggregated data on mortality to study the long-run economic and spatial consequences of the Black Death across Western Europe.

We test whether cities and regions that had relatively high mortality rates were permanently affected. A priori, the spatial effects of mortality are ambiguous: (i) If there are strong agglomeration economies a large negative shock to a population could lead to a negative feedback cycle in which wages and population continue to decline; (ii) If the economy is "Malthusian", i.e. if production largely relies on fixed factors of production, such as land and other natural resources, wages go up after the shock, thus allowing population recovery by either increasing fertility and decreasing mortality in high-mortality areas or causing migration from low to high-mortality areas; alternatively (iii) If the population shock and the resulting higher wages permit investments in technology and physical or human capital or leads to institutional or demographic changes, high-mortality areas may gain a long-run productivity advantage and keep growing. The implications of each of these models for long-run city population growth are starkly different: relative decline (i), recovery (ii) or growth acceleration (iii).

The standard model of growth in the pre-industrial world is Malthusian (Galor and Weil, 2000; Ashraf and Galor, 2011; Galor, 2011). Within this literature, the Black Death is seen as a canonical test of the theory that a collapse in population raised income per capita (Clark, 2007). This effect was eventually negated in parts of Europe by increased fertility and lower mortality (Galor, 2005). In other parts of Europe, however, as Voigtländer and Voth (2013b) argue, urbanization accelerated since increases in per capita incomes increased consumption of urban goods. As cities had higher mortality rates than the countryside, increased urbanization checked population growth and led to permanently higher incomes, possibly consistent with

growth effects at the macroeconomic level. Historians, however, view the evidence as at odds with a Malthusian framework. For Hatcher and Bailey (2001, 57) "the case for a neo-Malthusian interpretation of this era is not as strong as it might appear to be", commenting on "the widening gap which has emerged between its general descriptions of events and the welter of *local* evidence uncovered in recent years". While existing economic studies have considerably improved our understanding of the Black Death, they use macroeconomic approaches instead of localized data on Black Death mortality and thus cannot test if the disaggregated spatial effects of the Black Death are indeed causal and consistent with the Malthusian recovery hypothesis ((ii) above).[1]

More generally, our setting is well suited to testing how localized shocks affect economic activity via agglomeration, Malthusian, or growth effects. The Black Death was a comparatively "pure" population shock. Unlike other shocks considered in the literature buildings and physical capital were not destroyed and the event itself did not target a particular social, age, gender, ethnic or skill group. Wars and bombings, as studied by Davis and Weinstein (2002), Bosker et al. (2008), and Glocker and Sturm (2014) killed people but also led to massive physical destruction and resulted in government reconstruction programs. Genocides, as studied by Acemoglu et al. (2011b) and Rogall and Yanagizawa-Drott (2013) killed a large number of people but also resulted in physical destruction and disproportionately targeted intellectual elites or minority groups. Famines, as studied by Meng et al. (2015), killed large numbers of people but the poor died at higher rates. Disasters such as earthquakes and fires, as studied by Hornbeck and Keniston (2017) and Ager et al. (2018), tend to kill far less people but also lead to massive physical destruction. Expulsions, as studied by Waldinger (2010, 2012), Chaney and Hornbeck (2015), and Johnson and Koyama (2017) targeted specific subpopulations. Finally, other diseases such as malaria, HIV, or the 1918 influenza pandemic, as studied by Bleakley (2010), Young (2005), and Almond (2006) and Beach et al. (2018), disproportionately kill subgroups of the population.[2]

Using data for 165 cities, comprising 60% of the total urban population of Western Europe, we find that between 1300 and 1400 a 10 percentage point higher Black Death

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

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

mortality rate was associated with a 8.7 percentage point fall in city population. After just one or two centuries the impact of mortality was close to zero. When we examine the spill-over and general equilibrium effects of the Black Death on city populations we similarly find negative effects in the short-run and mostly nil effects in the long-run. Cities and urban systems, on average, had relatively recovered to their pre-Plague population levels by the 16th century. Using data on deforestation, we then show that rural areas close to higher-mortality cities recovered their populations around a century after the recovery in urban populations. Next, we use data on deserted medieval villages in England to show that more settlements were abandoned in low, rather than high, mortality areas—especially farther away from cities. Therefore, recovery in high-mortality areas must have been accelerated by migration from low-mortality areas. In other words, recovery was not simply driven by the higher fertility and lower mortality effects described in the macrohistorical literature on Malthusian recovery.

We also show that urban recovery is entirely explained by the interacted effects of mortality with city characteristics that proxy for fixed factors of production: rural fixed factors related to better land suitability and urban fixed factors related to natural advantages (e.g., coastal access) or sunk man-made advantages (e.g., roads) favoring trade. We show that aggregate urban recovery hides permutations in the distribution of cities. Some cities permanently collapsed after the Black Death whereas other cities gained in the long run. We provide evidence that these permutations were associated with the presence of fixed factors.[3] Since permutations favored cities with better land and trade potential, urban systems may have become more productive.[4]

We provide evidence that these results are causal. First, we show that the virulence of the Plague was plausibly unrelated to factors related to future city growth. Second, the parallel trends assumption is verified as, prior to 1300, there was no difference in growth between areas most affected and those comparatively less affected by the Black Death. Third, results are robust to the inclusion of controls for city characteristics, region fixed effects, and contemporaneous events. Fourth, our results hold when we implement instrumental variables strategies premised on the facts that: (i) the Black

[3] In contrast, the seminal study on the non-effects of Allied bombing of Japanese cities on relative city sizes by Davis and Weinstein (2002) shows few permutations. Strikingly, Hiroshima and Nagasaki were the 8th and 12th largest cities in 1940 and 10th and 11th largest ones in 1960.

[4] This analysis echoes the work of Michaels and Rauch (2018). However, without estimating the causal effects of land suitability and trade potential on growth, we cannot directly identify this effect.

Death entered Europe through the Sicilian port of Messina and was more virulent in its earlier stages, for pathogenic reasons; (ii) the Black Death was more lethal in cities in which it reached its peak in the summer since the fleas that transmitted the disease were more active then; and (iii) it was connectedness to Messina and not connectedness to other important cities that mattered for plague virulence.

In contrast to previous studies, we: (i) Provide econometric evidence that the Black Death had strong spatial-economic effects in the short-run, but no such effects in the long-run. To our knowledge, we are the first paper to use city-level data on Black Death mortality to study its local effects using various identification strategies and to document that its spatial effects are consistent with a Malthusian model driven by the presence of fixed factors of production.[5] (ii) Our analysis focuses on cities, which are often neglected in the Malthusian literature.[6] The standard Malthusian model focuses on the agrarian economy. Cities simply proxy for productivity growth in the countryside. It is only when countries industrialize and emerge from the Malthusian trap that they significantly urbanize (Galor, 2005, p.191). It is also generally assumed that cities are less "Malthusian" than villages since urban production primarily relies on physical and human capital (Lucas, 2004; Moretti, 2004), two factors that are a priori not "fixed".[7] However, cities were important for trade in this period so high-mortality cities with natural or sunk man-made advantages that favored trade were more likely to attract labor after being hit. Their recovery was largely driven by migration instead of natural increase. Another study on the importance of natural transport routes in the Malthusian era is Barjamovic, Chaney, Coşar and Hortaçsu (2017). (iii) Since fixed factors related to land and trade played a role in city recovery, our analysis suggests that the Black Death led to a reset consistent with a potentially superior population distribution.[8]

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

[6] As discussed above, a notable exception is Voigtländer and Voth (2013b), although they study the aggregate urban sector rather than looking at cities. Boerner and Severgnini (2014) use localized data on the timing of the Black Death to estimate the determinants of medieval trade.

[7] Physical capital was largely' fixed" in this period. Buildings where city residents lived and worked were made of stone and brick in southern Europe and wood in northern Europe. To the extent that housing and work space was durable, cities experiencing negative population shocks could have attracted migrants through cheaper housing and workshops, as in Glaeser and Gyourko (2005).

[8] The Black Death may have had positive macroeconomic effects as in Voigtländer and Voth (2013b,a).

We also contribute to research on shocks and long-run persistence. Numerous studies have shown how path dependent spatial economic patterns are. Different causes have been advanced for this path dependence, including locational fundamentals (i.e. natural advantages), sunk investments (i.e. man-made advantages), agglomeration effects (i.e., the direct effect of population scale), or institutions (e.g., Davis and Weinstein, 2002; Bleakley and Lin, 2012; Duranton et al., eds, 2015; Dell and Olken, 2019). A number of studies have demonstrated that demographic shocks have long-run impacts (e.g., Almond, 2006). The Black Death differs from other shocks in its magnitude—an overall mortality rate of 40% is unprecedented.[9] Other demographic shocks were also not pure population shocks so it is difficult to assess the channels through which they affected long-run development. In this paper, we do not find evidence for strong agglomeration effects. We do not observe higher-mortality cities collapsing after the Black Death, nor do we find that the population in the aftermath of the Plague predicts recovery. What we do find is that the recovery of cities was mostly driven by locational fundamentals related to land suitability, coastal access, proximity to rivers, and sunk investments related to historical transportation and trade networks, hence "fixed factors".[10]

Finally, we add to the literature on the economics of pandemics. Various studies have focused on understanding why pandemics spread (e.g., Oster, 2005, 2012; Boerner and Severgnini, 2014; Greenwood et al., 2017). Most studies of their economic consequences use macroeconomic approaches (Young, 2005; Weil, 2010; Voigtländer and Voth, 2013b,a), notable exceptions being Almond (2006) and Beach et al. (2018) who study the 1918 influenza pandemic. There is also a recent literature on the effects of plague recurrences in Europe (Dittmar and Meisenzahl, 2019; Siuda and Sunde, 2017). However, these events were on average much less deadly than the Black Death and only affected a few areas at a time (Aberth, 2010, p.37). Likewise, there is a nascent literature on the effects of the West African Ebola epidemic (2013–2016) (e.g., Bowles et

[9]Other examples of shocks include the Irish Famine (10-12.5%), World War I (1.7-1.9% of the involved countries' population), the 1918 influenza pandemic (3-5% of the world's population), the Ukrainian Famine (10%), World War II (3.5% of the world's population), the Bengal Famine (6.6%), the Great Chinese Famine (2.5-6.8%), the Cambodian Genocide (21-24%) and the Rwandan Genocide (11%). The introduction of European diseases in the Americas after 1492 may have killed something between 75% and 90% of the population, but this was over a period of more than one century (Koch et al., 2019).

[10]Other historical events that may have shaped spatial economic patterns in pre-industrial Europe includes the foundation of Medieval universities (e.g., Cantoni and Yuchtman, 2014), the discovery of the Americas (e.g., Acemoglu et al., 2005a), the Reformation (e.g., Cantoni et al., 2018), the introduction of the potato (e.g., Nunn and Qian, 2011) and the French Revolution (e.g., Acemoglu et al., 2011a).

al., 2016). However, this disease has killed only about 10,000 people which is 0.003% of West Africa's population. Pandemics like the Black Death differ from epidemics in that they affect a very large number of areas and people, so their effects are likely to differ.

Understanding the economic effects of pandemics is all the more important given that their frequency and severity may increase with climate change. According to one recent broadcast by the BBC (2017), "Climate change is melting permafrost soils that have been frozen for thousands of years, and as the soils melt they are releasing ancient viruses and bacteria that, having lain dormant, are springing back to life." Likewise, Bill Gates argues that "given the continual emergence of new pathogens, the increasing risk of a bioterror attack, and the ever-increasing connectedness of our world, there is a significant probability that a large and lethal modern-day pandemic will occur in our lifetime" (Gates, 2018). An uncontrolled Ebola outbreak could have dramatic economic consequences in Africa (e.g. UNECA, 2015), but it is difficult to assess which consequences without empirical evidence from previous pandemics.[11]

While the Black Death took place centuries ago, we believe our study is relevant for policy makers today. We exploit city-level data along with a pandemic shock that was massive, highly variable, and plausibly random. The IV strategies we employ are based on pathogenic factors and market access. Our focus on an era when there was no international community able to intervene insulates us from an important source of endogeneity. Finally, our results are particularly relevant for the developing world today. Indeed, it can be argued that today's poor economies disproportionately rely on fixed factors of production similar to the ones we identify in our context. This could explain why mortality increases observed in the present day often have positive effects (Young, 2005; Rogall and Yanagizawa-Drott, 2013; Esteban et al., 2015) and why population increases may have negative effects (Acemoglu and Johnson, 2007; Ashraf et al., 2011). While this is likely true for rural areas, one could also argue that poor country cities are to some extent "Malthusian". Jedwab and Vollrath (2019) show how slums grow when megacities grow too fast due to constraints on formal sector development and inelastic housing supply. Gollin et al. (2016) show how many developing country cities have very low shares of skilled workers. Lastly, our context of decentralized polities with weak state capacity is similar to that observed in poor countries today.

[11] See Gráda (2015) for a comparison between Ebola and the Black Death.

1. Data

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

Black Death Mortality. Data on cumulative Black Death mortality for the period 1347-1352 come from Christakos et al. (2005, 117-122) who compile data on mortality rates based on information from a wide array of historical sources including ecclesiastical and parish records, testaments, tax records, court rolls, chroniclers' reports, donations to the church, financial transactions, mortality of famous people, letters, edicts, guild records, hospital records, cemeteries and tombstones. Christakos et al. (2005) carefully examine each data point and arbitrate between conflicting estimates based on the best available information. We have checked these data using other sources including Ziegler (1969), Russell (1972), Gottfried (1983), and Benedictow (2005). Details on data construction and which mortality estimates were selected by Christakos et al. (2005) are provided in Web Appendix Section 1.. These data yield estimates of mortality for 274 localities which we map along with their mortality rates in Figure 1.

For 177 of these 274 cities we have a percentage estimate of the mortality rate. For example, Venice had a mortality rate of 60%. In other cases the sources report more qualitative estimates: (i) For 49 cities Christakos et al. (2005) provide a literary description of mortality. We rank these descriptions based on the implied magnitude of the shock and assign each one of them a numeric mortality rate.[12] (ii) For 19 cities we know the mortality rate of the clergy. Christakos et al. (2005) provide evidence that clergy mortality was about 8% higher than general mortality, so we divide the clergy mortality rates by 1.08.[13] (iii) For 29 cities we know the desertion rate, which includes both people who died and people who never came back. Following Christakos et al. (2005, 154-155), who show that desertion rates were 1.2 times higher than mortality rates, we divide desertion rates by 1.2. Since the data generated for (i)-(iii) require assumptions, we will show that our results hold when using only numerical estimates.

City Populations. Our main source of city population data is the Bairoch (1988)

[12] 5% for "spared"/"escaped", 10% for "partially spared"/"minimal", 20% for "low", 25% for "moderate", 50% for "high", 66% for "highly depopulated", and 80% for "close to being depopulated"/"decimated".

[13] This may contradict our claim that the Black Death did not target specific populations. Clergymen may have been more exposed simply because they lived communally. However, clergymen only comprised a tiny share of the population of cities, so this should not be consequential for our analysis.

dataset. This source reports population estimates for 1,726 cities between 800 and 1850. Observations are provided for every century up to 1700 and then for each fifty year interval up to 1850. The criterion for inclusion in the Bairoch dataset is a city population greater than 1,000 inhabitants at any point between 800 and 1850. This dataset has been widely used (De Long and Shleifer, 1993; Dittmar, 2011; Nunn and Qian, 2011).

We follow Bosker et al. (2013) and Voigtländer and Voth (2013b) in updating the Bairoch dataset where historians have revised estimates of the population of particular cities. We supplement the Bairoch (1988) dataset using several sources including Chandler (1974, 1987), Nicholas (1997), and Campbell (2008). We also add 76 cities that are mentioned as cities (i.e., localities of more than 1,000 inhabitants) in Christakos et al. (2005) but not in Bairoch (1988). In the end, we obtain 1,801 cities.[14]

Controls. Controls for *locational fundamentals* include mean growing season temperature in 1500-1600, 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.[15] To proxy for *agglomeration effects*, we control for population and market access in 1300. We calculate market access for every city in our main sample to the cities of the full sample for which we have populations in 1300. Market access for town i is defined as $MA_i = \Sigma_j \frac{L_j}{\tau_{ij}^{\sigma}}$, with L_j being the population of town $j \neq i$, τ_{ij} the travel time, i.e. network distance, 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 (see details below)—using the data from Boerner and Severgnini (2014) who estimate the speed at which the Plague traveled via each mode of transportation. To proxy for *sunk investments*, we control for the presence of major and minor Roman roads (and their intersections) using the data from McCormick et al. (2013), medieval trade routes (and their intersections) after digitizing a map from Shepherd (1923), and dummies capturing the presence of medieval market fairs, membership in the Hanseatic league (Dollinger, 1970), whether a city possessed a university (Bosker et al., 2013), and whether a city was within 10 km of a Roman aqueduct (Talbert, ed, 2000). To control for *institutions*, we distinguish between cities that were located in monarchies, self-governing cities, or whether the

[14]Details on the choices we make are confined to Web Appendix Section 2.. In our regressions we will use both the corrected dataset and the original Bairoch dataset.

[15]We use average values within a 10 km radius for some controls due to measurement error.

city was a state capital around 1300 (Bosker et al., 2013; Stasavage, 2014). We measure parliamentary activity during the 14th century using data from van Zanden et al. (2012) and control for whether a city was within 100 km of a battle in 1300-1350.

2. Historical Setting

2.1. The Epidemiological Shock

The Black Death arrived in Europe in October 1347. Over the next few years it spread across the continent killing between 30% and 50% of the population.[16] 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 swell painfully as they become buboes. Once infected, death occurred within ten days with 70% probability.[17]

While the vector for bubonic plague is infected fleas, they cannot spread the disease far in the absence of hosts such as rats. The spread of the Plague was rapid and its trajectory was largely determined by chance. For example, one important means of transmission depended on which ships became inhabited with infected fleas. It was largely coincidence that the Plague spread first from Kaffa in the Black Sea to Messina in Sicily in October 1347 as the ships carrying the Plague were originally bound to Genoa (but the ships could have been bound to other ports). Figure 1 shows the locations of Messina and Kaffa. Similarly, it was coincidental that the Plague spread from Messina to Marseilles rather than to, say, Barcelona, Lisbon, or Antwerp. From the various coasts where infected ships docked the Plague then spread inland along rivers and roads.[18]

Virulence was greater in cities affected earlier (Christakos et al., 2005, 212-213). Initially, epidemics spread exponentially and then, as they run out of victims, the

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

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

[18] The local spread of the Plague thus also depended on the local populations of black rats. Since black rats are territorial, their numbers were not correlated with population density (Benedictow, 2005). For example, similar death rates are recorded in urban and in rural areas (Herlihy, 1965).

disease mutates in favor of benign pathogens.[19] People also develop immunities and pathogen mutation increases individual immune responses due to "contacted individuals becoming infected only if they are exposed to strains that are significantly different from other strains in their memory repertoire" (Girvan et al., 2002). Thus, pathogen mutation and natural immunization eventually cause an epidemic to end.

Additionally, the Black Death was at its most virulent during the summer months (Benedictow, 2005, 233-235). Fleas become most active when it is fairly warm and humid (Gottfried, 1983, 9). Christakos et al. (2005, 230) notes that Black Death mortality displayed seasonal patterns with deaths diminishing with colder weather "without the epidemic coming to a complete halt". In our sample, using available data on the year and month of first and last infection for 61 out of the 165 towns, the average duration of the Black Death was 7 months (see Web Appendix Figure A.1). According to Christakos et al. (2005, 212-213), mortality on average peaked 3.5 months after the first infection. Therefore, cities that became infected in late fall escaped relatively unscathed compared to cities that were first infected during the spring.

Based on the facts described above, it is apparent that the local virulence, of the Plague had a significant random component (see Web Appendix Section 4. for more qualitative evidence). When studying variation in mortality rates across space, historical accounts have been unable to rationalize the patterns in the data (Ziegler, 1969; Gottfried, 1983; Theilmann and Cate, 2007; Cohn and Alfani, 2007). To illustrate, Venice had high mortality (60%) while Milan escaped comparatively unscathed (15% mortality). Highly urbanized Sicily suffered heavily from the Plague. Equally urbanized Flanders, however, had low death rates. Southern Europe and the Mediterranean were hit especially hard, but so were the British Isles and Scandinavia.[20] Likewise, Christakos et al. (2005, 150) explain that some scholars have "argued that Black Death hit harder the ports and large cities along trade routes" but that "the generalization is logically valid at a regional level at best" and that "examples and counterexamples abound, making it

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

[20] Variation in sanitation does not explain this pattern. Gottfried (1983, 69) notes "it would be a mistake to attribute too much to sanitation" given the "failure of Venice's excellent sanitation to stem the deadly effect of the plague".

impossible to reach any definite conclusion." Consistent with this, Figure 2(a) illustrates the lack of a relationship between mortality rates and city population in 1300 (Y = 42.5*** - 1.01 X; Obs. = 165;$R^2 = 0.00$). Likewise, Figure 2(b) shows that there is no relationship between mortality rates and city market access in 1300 (Y = 40.0*** -0.20 X; Obs. = 124; $R^2 = 0.00$).[21]

Subsequent outbreaks of bubonic plague reoccurred in Europe for two and a half centuries following the Black Death. Epidemiologists and historians have long noted the virulence, spread, and associated mortality of the Black Death differed from the pattern associated with later outbreaks of bubonic plague (see discussion in Web Appendix 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).[22] Though on occasion later outbreaks could devastate a city, in general mortality was significantly lower than in the initial pandemic (Aberth, 2010, 37). Europe-wide studies of later outbreaks focus on the extensive margin of the plague and not on the intensive margin for which comprehensive estimates only exist for the Black Death itself.

Finally, the Black Death affected all segments of the population, rulers and commoners, rich and poor, adults and children, men and women. Neither the medical profession nor authorities were able to respond effectively. Medical knowledge was rudimentary: Boccaccio (2005, 1371) wrote that "all the advice of physicians and all the power of medicine were profitless and unavailing". Individuals, regardless of wealth, were unable to protect themselves. Institutional measures of prevention were nonexistent: the practice of quarantine was not employed until 20 years later.[23]

The Black Death represents as close to a *pure* population shock as we are aware of in the literature. Death rates were similar across social, age, gender, ethnic or skill groups

[21] Random measurement error in dependent variables, here mortality, does not lead to bias, only to higher standard error (the R2 would still be 0.00). Random measurement error in market access then produces a downward bias. However, our measure follows a specification and uses an elasticity that are now standard (e.g., Donaldson, 2018). In addition, Web Appendix Table A.2 still shows no correlation when market access is computed using a lower elasticity or when relying on Euclidean distance instead of network distance in case the speed parameters are misestimated. Lastly, we will show that there is no positive significant effect of other measures of transportation and trade networks. Similarly, some scholars have argued that death rates were lower in mountainous regions, but mortality rates in mountainous Savoy were high whereas "despite Switzerland having the most rugged terrain in Europe, the Black Death reached almost every inhabited region of the country" (Christakos et al., 2005, 150). We also find no relationship with the density of population within walled cities (see Web Appx. Figure A.3).

[22] Other studies are: Dittmar and Meisenzahl (2019); Siuda and Sunde (2017); Alfani and Percoco (2019).

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

(see Web Appendix Section 5. for qualitative evidence). The available evidence suggests that the Black Death was "massive and indiscriminate, making no exception to factors such as personal hygiene, health, age, sex, or social class" (Christakos et al., 2005, 150). This is supported by recent studies of plague outbreaks (Alfani and Bonetti, 2018).[24]

2.2. Consequences of the Black Death

The Black Death caused immediate economic damage. In rural areas harvests went uncollected. In cities, trade was disrupted. As local economies collapsed, there were food shortages and inflation. Campbell (2016, 355) notes that "the simultaneous shock that plague then inflicted upon the supply of labour and the demand for goods and services set in train an immediate and enduring contraction in economic activity". Nominal wages increased immediately after the shock. Real wages, however, rose only gradually due to inflation (Munro, 2003). In England, for example, it was not until the 1380s that real wages reached their pre-Plague levels.[25] In the long run wages did increase but this rise was gradual—peaking in many parts of Europe in the mid-15th century before declining as a result of population growth after 1500 (see Web Appendix Section 6. for more qualitative evidence on wage patterns after the Black Death).

The demographic recovery was also slow. Figure 3(a) presents estimates of total population (source: McEvedy and Jones (1978)) and urbanization for the 16 (modern) countries which contain the cities of our full sample. Europe only regained its pre-Plague population by around 1600. Urbanization, here defined as the population share of localities above 1,000 inhabitants, rose in the aftermath of the Black Death from around 8% to 11% by 1400, and 13% in 1600. This increase was driven by urban areas fully recovering their population faster than rural areas (see Figure 3(b)). In particular, cities that already existed in 1300 recovered their population by 1500 while new cities also emerged after 1353 (see Figure 3(c)).

The demographic consequences of the Black Death varied across Europe. In aggregate the population eventually recovered. But the European Marriage Pattern (EMP) — a higher age of first marriage and high rates of female celibacy — characteristic

[24] Christakos et al. (2005, 150) note that the claim that the poor died more than the rich is a plausible sounding statement "rather than an independent scientific conclusion derived from the analysis of real Black Death data". For contrary evidence, see DeWitte and Wood (2008) who study London only.

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

of parts of Europe is thought to have emerged or at least strengthened after the Black Death (Hajnal, 1965; Moor and Zanden, 2010; Dennison and Ogilvie, 2014). This reduced rates of natural increase. Voigtländer and Voth (2013a) argue that the the Black Death had different demographic impacts where the land could be converted to pastoral agriculture compared to where the land was more suitable for arable farming. As wages increased, pastoral farms increased their demand for female labor, this generated a labor and marriage market equilibrium in which individuals married late and restricted fertility; a phenomenon that in turn raised per capita incomes. At the city level, however, post-Plague recovery was largely driven by migration from the countryside as urban mortality exceeded rural mortality.[26]

Migration occurred both from the countryside to the cities and within the countryside itself. Historians speculate that "the first few years after the epidemic witnessed especially high migration rates" (Poos, 1991, 108). Penn and Dyer (1990, 363) note that late medieval wage earners had a great "capacity for geographical mobility" evident "from the indirect testimony of locative surnames which reflect migration into towns, and the patterns of immigration and emigration". Cities attracted migrants from the nearby countryside to repopulate. In York the number of freeman admitted into the city increased by 365% in the year of the Plague (Dobson, 1973, 17). However, as the countryside was often as hard hit as the cities by the Plague, population took many decades, not years, to recover. London, for example, saw a "great concourse of aliens and denizens to the city and suburbs, now that the pestilence is stayed" but the city remained depopulated for many years (Sloane, 2011). Finally, as many peasants migrated to other rural or urban areas, some plots were converted from arable to pastoral farming, marginal lands were abandoned, and woodlands re-emerged (Campbell, 2016, 363) (see Web Appx. Sections 9.-10. for more qualitative evidence).[27]

3. Main Results: Black Death Shock and City Recovery

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

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

[26] See Voigtländer and Voth (2013a, 780). See Web Appendix Section 7. for more qualitative evidence.

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

where $\%\Delta \text{Pop}_{i,t}$ is the percentage population growth (%) in city i over period t-1 to t, and $\text{Mort}_{i,1347-52}$ is the city-level cumulative mortality rate (%) between 1347 and 1352. We weight observations by their initial population size in year t-1 to minimize issues arising from smaller cities mechanically experiencing larger percentage changes.[28]

3.1. Short-Run and Long-Run Effects

Short-Run. Column (1) of Table 1 measures the short-run impact of the Plague in 1300-1400. The coefficient is -0.87***. This should be interpreted relative to the immediate effect for the period 1347-1352 which is -1.00 by construction. The fact that the coefficient is not significantly different from -1.00 suggests that there was 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 deviation decrease in population growth.

Long-Run. The effect for the period 1300-1500 is negative (-0.28, col. (2)) but smaller in size compared to the effect in 1300-1400 and significantly different from -1. Columns (3)-(5) examine the cumulative effect up to 1750. The coefficient increases to 0.36, 0.47 and 0.85 by 1600, 1700 and 1750 respectively. However, the magnitudes are small: A one standard deviation increase in mortality is associated with a 0.02-0.03 standard deviation increase in population growth, implying total recovery. The effects are also not statistically significant. The estimate in Column (5) suggests that within the 95% confidence band some cities experienced no recovery at all (coefficient of -1.5) whereas others more than made up for the shock (coefficient of 3.2). This shows that the experience of European cities after the Black Death was heterogenous. For the following analysis, we focus on the period between 1300 and 1600 since cities recovered by the 16th century and to minimize contamination from potentially confounding events.[29]

3.2. Investigating Causality

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

[28] 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.

[29] Effects remain small and insignificant if we use 1800, 1850 or 2015 (not shown).

Biases. For the short-run a downward bias is more problematic than an upward bias as we then overestimate the effect of the Plague (the true effect in 1300-1400 must be higher than -0.87). The short-run effect could be downward biased if cities that were inherently growing slower (faster) were also affected by higher (lower) mortality rates. For the long-run, an upward bias is more problematic as we then overestimate how fast cities recover (the true effect in 1300-1600 is lower than 0.36). 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 city characteristics that could also have caused future city growth. We define city level characteristics according to whether they proxy for locational fundamentals (1), increasing returns (2) (i.e. agglomeration effects or sunk investments) or institutions (3). The only variables that have explanatory power are proximity to rivers and latitude (column (1)). Proximity to rivers is negatively correlated with mortality, which is inconsistent with the claim that trade routes were correlated with Plague virulence. Other measures of transportation and trade networks do not have economically or statistically significant effects. The coefficient on latitude reflects the fact that the Black Death hit southern Europe first and was more virulent in the early years of the epidemic. Finally, no effect is significant once all controls are included.[30]

Parallel Trends. Columns (6)-(7) of Table 1 show that prior to 1300, there is no difference in growth between cities most affected and those comparatively unaffected by the Black Death. The cities that were hit hardest and those that escaped comparatively unscathed were following similar development paths prior to the 14th century.[31]

Controls. To deal with potential sources of a downward bias for the short-run effect and an upward bias for the long-run effects, we show in rows 2-5 of Table 3 that our results are robust to the inclusion of all covariates in Table 2. For example, 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 or amenities driving future growth, this would be a source of upward bias. Likewise, if being on a trade route was positively correlated

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

[31]The parallel trends assumption is also verified if we test for the overall period 1100-1300 (N = 62) or the period 1000-1100 (N = 59) (results not shown, but available upon request).

with both mortality and a city's growth potential, this would lead to an upward bias. If anything, an upward bias would make us under-estimate the short-term negative effect of the plague. Table 2 shows that mortality was not significantly correlated with most observable characteristics. Nevertheless, we show results hold when we include the controls proxying for locational fundamentals (row 2), increasing returns (row 3) or institutions (row 4), or all of them simultaneously (row 5).

Outliers. In rows 6 and 7, we drop towns with the 5% or 10% highest and the 5% or 10% lowest mortality rates to ensure that our results are not driven by outliers. In general, no community was prepared to deal with the Black Death. It was attributed to the "vengeance of God" or the "'conjunction of certain stars and planets" (Horrox, ed, 1994, 48-49). Thus, there was little variation in a city's ability to deal with it. Historians report that some cities had either natural baths (Bath, Nuremberg) or tried to take action in response to the plague (Milan, Venice). Results hold when we drop these (row 8).

Spatial Fixed Effects. Results hold if we employ 13 modern country fixed effects (row 9), to control for regionally correlated unobservables (e.g., linguistic or genetic diversity). Modern country borders differ substantially, however, from the political units of the fourteenth century so in row 10 we assign a separate dummy variable to each of the independent polities that had at least 5 cities in our dataset.[32]

Correlated Shocks. The plague reoccurred following the Black Death. This could be a potential source of bias if subsequent plague outbreaks were correlated with the initial pandemic. In row 11 we use data from Biraben (1975) and show results hold if we include a dummy for plague recurrence and the number of recurrences within 50 km of the city in the period of interest 1353-1400 or 1353-1600, respectively.[33]

The immediate impact of the Black Death reduced the intensity and scale of conflict (in the Hundred Years War, for example, as documented by Sumpton (1999)). However, as Voigtländer and Voth (2013a) and Dincecco and Onorato (2015) argue, warfare ultimately intensified and contributed to increases in urbanization in late medieval and early modern Europe. Row 12 shows that results hold if we include a dummy for battle

[32]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).

[33]Subsequent plagues were not correlated with mortality (see Web Appendix Table A.3). These results on plague recurrences are also robust to using other distance thresholds (10, 25 or 100km, not shown). Later recurrences had a different epidemiology to the initial outbreak (see Web Appendix Section 4.).

ocurrence and the number of battles in the period of interest 1353-1400 or 1353-1600.[34]

In a related paper, Jebwab et al. (2018) show that lower-mortality cities were more likely to persecute their Jewish community, which then impacted their growth—especially if the persecution consisted of a pogrom as opposed to an expulsion. Our results hold if we add three dummies for whether the city had a Jewish community, a persecution took place, and the persecution took the form of a pogrom during the Black Death (row 13). In row 14, we show results hold if we drop any city with a persecution. Controlling for or omitting persecutions ensures that our population shock does not affect human capital. In addition, Jewish populations were small relative to the non-Jewish city populations, so their demographic contribution was marginal.

In Table 4, we implement three IV strategies. The first two strategies rely on the date of first infection in the city, which is only available for 124 cities. We thus verify in row 2 that OLS estimates are similar for these 124 cities than the ones for the main 165 cities (row 1). The effect in 1300-1600 is higher now (but not significantly so).

IV1: Timing. Our first source of exogenous variation in mortality is timing of first infection because there was a lot of randomness in the local patterns of the Plague, depending on where infected rats and fleas went. Figure 4(a) plots mortality rates against the *date* that the city was first infected (n. of months since October 1347). Cities infected later, indeed, had lower mortality. Using the number of months since October 1347 as an IV, the coefficient is -1.67** in 1300-1400 and -0.24 in 1300-1600 (row 3 of Table 4, IV F-stat.: 21.2)[35] This is precisely estimated; however, the short-term estimate is also larger (but not significantly so) than our OLS coefficient (-0.087***). The Black Death spread from the South and as the urban network of medieval Europe was centered around the Mediterranean, it is possible that the date of first infection was correlated with latitude, trade, and city size. Thus, we add the controls of Table 2 (incl. longitude and latitude) and the squares and cubes of longitude and latitude in order to better exploit the random component of the spread of the Plague (row 4). The IV F-stat decreases to 12.6, and the coefficients (-1.07** and 0.05) are close to our OLS estimates.[36]

[34]Relatedly, Cervellati et al. (2014) show that in the post-World War 2 period disease outbreaks are associated with an intensification of civil conflict. Arguably, the respective number of soldiers involved, and whether the cities were burned or sacked, may be a better proxy for the devastation associated with warfare. Our results hold if we use these controls (not shown but available upon request).

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

[36]In Web Appendix Table A.5, we show the reduced-form effect of the IV and verify that the IV is

IV2: Within-Year Month of First Infection. Our second IV makes use of the variation in mortality generated by differences in the *month* of first infection *within* a single year. Figure 4(b) suggests that this IV has explanatory power. For 124 cities of the main sample for which we have data on the onset of the Black Death, it shows the relationship between mortality rates and the month of peak infection in the city (= month of onset + 3.5 months). The Black Death was more virulent when peak mortality in the city occurred during summer (6-8). We report results using our second IV, twelve dummies for the month of peak infection, while simultaneously adding dummies for the year of first infection to control for the fact that cities infected in earlier years had higher mortality (row 5). We obtain qualitatively similar results, but the coefficient is now significantly higher than the OLS coefficient (-2.08***, IV F-stat.: 7.3). Conditioning our IV on the same controls used for IV1 allows us to better isolate the random component of the spread of the infection within a year. The short-run coefficient is close to our OLS estimate (-0.85**, see row 6). However, the F-statistic is low, at 3.3.

IV3: Proximity to Messina Market access to Messina should predict Plague virulence, since the Plague was more virulent initially, for pathogenic reasons. We construct an IV based on a city's log market access to Messina (see Figure 5(b)), *conditional* on a city's log market access to all 1,801 cities (see Figure 5(a)). Market access to Messina m for town i is defined as $MA_{im} = \Sigma(L_m \div \tau_{im}^{\sigma})$, with L_m being the population of Messina in 1300, τ_{im} the computed travel time between town i and Messina, and $\sigma = 3.8$. Controlling for market access 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.[37]

We report estimates using this instrument in rows 7-8 (Messina is dropped from the regressions). The short-run coefficients are similar to our OLS estimates, at -1.32*** and -0.90† (p-value of 0.126) once controls are added (IV F-stat of 29.0–9.9). However, the long-run effect becomes large and negative (-1.41), but not significant, when all controls are added. Given that the IV is identifying a LATE, one possibility is that it is being driven by the economic collapse of Sicily in the early modern period. Indeed, the IVs give more weight to the LATE compliers, here the cities close to Messina.

unrelated with city growth in the preceding centuries (1200-1300 and 1100-1200).

[37]The correlation between the two market access measures is indeed lower than 1, at -0.48, since Northern European cities also had access to large cities other than Messina.

The exclusion restriction is satisfied as long as connectedness to Messina is not correlated with factors that directly influenced city growth, conditional on connectedness overall and the controls. In Web Appendix Table A.5, we show the reduced-form effect of the IV, but then verify that the IV is unrelated with city growth in the preceding centuries (1200-1300; 1100-1200). Web Appendix Table A.6 shows results hold if we control for log market access to cities in the Middle-East and North Africa. A more realistic measure of market access would account for variation in tariffs and currencies. However, the majority of these are not recorded by historians. In any case, local tariffs were likely endogenous to city characteristics, so adding them to our analysis could introduce additional bias. Instead, Web Appendix Table A.6 shows results hold if we use an ahistorical measure of market access as our IV—the log Euclidean distance to Messina, conditional on average log Euclidean distance to all cities.[38] Finally, results hold when using the three IVs simultaneously (rows 9-10 of Table 4).[39]

Summary. Our analysis of city characteristics, parallel trends, outliers, controls, and IV strategies suggest that the impact of the Black Death was plausibly exogenous. The identification strategies suggest short- and long-run effects that are not significantly different from our baseline OLS effects of -0.87*** (1300-1400) and 0.36 (1300-1600). In the rest of the analysis we will thus employ OLS estimates as our baseline.

3.3. Robustness Checks

We now show that our results are robust to potential concerns about data measurement, sampling issues, and specification. Row 1 of Table 5 reports our baseline estimates (for both the short-run effect in column (1) and the long-run effect in column (2)).

Measurement Concerns. Classical measurement error in mortality should bias the short-run estimates towards zero. In that case, our effect is less negative than the true effect, which is less of an issue than a downward bias. Additionally, if measurement error is classical, the long-term effect should be as biased as the short-term effect, which would not change our results. However, measurement error could be non-classical, hence the need to examine how the effects vary depending on the sources of

[38] Results also hold if market access to all towns exclude Messina (Web Appendix Table A.6).

[39] One source of exogenous variation we do not pursue is the distribution of rats. According to the latest scientific research the Black Death was transmitted by several vectors. The most important of these were the fleas of the black rat. 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. However, little is known about the geographical distribution of black rats at the time of the Black Death.

mortality data. Our estimates do not systematically differ when: we (i) include dummies for different sources of mortality data (row 2);[40](ii) drop estimates based on literary descriptions (row 3); (iii) drop estimates based on desertion rates (row 4); (iv) drop estimates based on clergy mortality (row 5); and (v) only use numerical estimates (row 6), also excluding estimates that yield a round number such as 1/4, 1/3, 1/2 (row 7). Alternatively 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, city population growth, should increase standard errors. However, our results remain precise. We can employ alternative population estimates. Row 9 reports estimates using the uncorrected Bairoch data (N = 151-150). Row 10 reports results using only observations from the Chandler (1974, 1987) dataset (N = 59-60). Results hold.

External Validity. There could be concerns about sample size. We employ as regression weights populations in 1300 ensuring less weight is placed on small towns. Given that our 165 cities are capturing 60% of the total urban population of Western Europe, having more cities would not dramatically change the results (unless they are very large). We also use several, albeit imperfect, methods to obtain mortality estimates for more cities. Results remain similar if we use: (i) the mortality of the closest neighboring city with data if this city is within 50 km (row 11, N = 290–286); (ii) the average mortality of the cities in the same state (row 12, N = 380–274, SEs clustered at the state level); and (iii) estimates of mortality based on spatial extrapolation (row 13, N = 464–457).[41]

Since the 165 cities represent 60% of the total population of the 466 existing cities in 1300, our sample is biased towards large cities. Kernel distributions of 1300 populations and Kolmogorov-Smirnov tests confirm that. In row 14 we reweigh observations to match the distribution of city populations in 1300.[42] We also drop cities located within

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

[41]We create a two-dimensional surface of predicted plague mortality using an inverse distance weighted function of known mortality rates for the full sample of 274 cities with mortality data. For every point on the surface a predicted mortality rate is then generated using the closest 15 cities within an approximately 1,000 km radius circle around the point. Details can be found in Web Appendix Section 1. (map of extrapolated mortality rates shown in Web Appendix Figure 2(b)).

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

different modern country borders (Web Appx. Table A.7): France, Germany, Italy, the United Kingdom and Spain. Other countries contribute few cities.

Specification. Our results are relatively unchanged when we control for past population trends, i.e. city population growth in 1200-1300 (row 15, N = 93) or in both 1200-1300 and 1100-1200 (row 16, N = 62). In row 17, we drop the population weights while simultaneously excluding the cities with fewer than 5,000 inhabitants as these cities have high growth rates simply because they are small initially.[43] In row 18, we employ a panel model with city fixed effects and year fixed effects for the period 1100-1750. We interact the mortality rate with dummies for the years 1300 and 1600. We find comparable coefficients to our baseline analysis. We also employ Conley (2008) standard errors with a radius of 100 km (row 19) and cluster standard errors at the state (1300) level (row 20) to account for spatial auto-correlation in the error term.

4. Mechanisms

The previous section suggests that high-mortality cities relatively recovered to their pre-Plague population levels by the 16th century, consistent with the Malthusian model. In this section, we provide evidence that: (a) urban systems and rural areas close to cities also relatively recovered to their pre-Plague population levels; (b) the recovery of high-mortality locations was driven by migration, not just natural increase; (c) urban recovery is entirely explained by the interacted effects of mortality with city characteristics that proxy for fixed factors of production; and (d) aggregate urban recovery hides permutations in the distribution of cities, and that these permutations are also explained by the presence or absence of fixed factors.

4.1. Effects on Urban Systems

Spillovers. Thus far, we have studied the effects of a city's mortality rate on its subsequent growth. However, we might expect cities to be affected by mortality in neighboring towns. Table 6 examines the effects of a city's own mortality rate and the population-weighted average mortality of nearby towns, defined in four ways:[44] (i) of the same state in 1300 (col. 1 and 5); (ii) of the same country in 2018 (col. 2 and 6); (iii)

[43] Results also hold if we use log city population growth or the absolute change in the number of city inhabitants instead of percentage city population growth (not shown but available upon request).

[44] We include all 1,801 towns, not just 466 cities already "existing" (population $\geq$ 1,000) by 1300. For the other towns, and in the absence of data on their population, we give them 500 inhabitants. We use the other towns so that the measures better capture overall mortality rather than city mortality only.

within the bottom 10% of Euclidean distance to the city (col. 3 and 7)[45] and (iv) or using all 1,801 cities but relying on the total change in market access between 1300 and 1353 (col. 4 and 8).[46] Here, since mortality is only available for a subset of cities (N = 274), we use spatially extrapolated mortality rates for cities without mortality data (N = 1,527).[47]

Cities that experienced a large direct shock did not always experience a large indirect shock. The correlations between city mortality and the four measures of indirect mortality are 0.43, 0.43, 0.51 and 0.49, respectively. As can be seen in columns (1)-(4), the short-run effect (1300-1400) of direct mortality is close to the baseline effect (-0.87***), at -0.68**/-0.79**. The indirect effect is negative, but not significant, around -0.16/-0.57. The combined direct and indirect effects are -0.96/-1.25, and significantly different from 0. By contrast, the long-run (1300-1600) direct and spillover effects are not significantly different from 0 (col. (5)-(8)). While some coefficients appear large, the beta coefficients are small or positive, about 0.04/0.14 for the direct effect and -0.03/0.17 for the indirect effect. Both direct and indirect effects thus disappeared by 1600.[48]

Aggregate Effects. In addition to the local and indirect effects, there may have been effects at the state level. In columns (1)-(5) of Table 7, 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.[49] Column (1) of Panel A replicates our baseline short-run effect (1300-1400) at the city level (-0.87***). Columns (2) and (4) show the effects at the state and country levels, also for cities that existed in 1300. The effect is now more negative, at -1.13/-1.15, close to the combined local and indirect effects (again not significantly different from the baseline effect). We interpret these estimates as telling us about the *intensive* margin—i.e. general equilibrium effects

[45]Results are robust to using the 5th or 25th percentiles, not shown but available upon request.

[46]To construct market access in 1353, we use the predicted population of the other towns in the aftermath of the Black Death (= pop. in 1300 x (100-mortality)/100).

[47]Note that for each of the 165 city-observations, the extrapolated mortality rates of the other towns are constructed excluding the mortality rate of the city-observation itself, so as to avoid any mechanical correlation between own mortality and the constructed neighbors mortality rate.

[48]Results hold if we use alternative indirect mortality rates (not shown, but available upon request): (i) based on extrapolated mortality for all 1,801 cities, thus including the city-observation itself; (ii) using only 274 cities with non-extrapolated mortality rates; (iii) excluding cities that did not already exist by 1300; and (iv) including the cities of 5 countries contiguous to our 16 countries (Finland, Hungary, Poland, Slovakia, Slovenia), using their country's mortality rate in the absence of localized mortality data.

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

of the Black Death on cities which already existed when the Plague hit.

In columns (3) and (5), we examine the state- and country-level effects on all cities that are in the dataset in 1400 (including cities not in the dataset in 1300). The effects are larger than before, at -1.27/-1.47 (however, still not significantly different from the baseline effect) implying that in high-mortality areas, fewer new cities emerged. We verify this in column (6)-(7) using 1,335 cities in the Bairoch data set that did not exist in 1300. These cities can be thought of as the universe of potential city locations. We test whether their emergence in 1400 — via a simple dummy — is related to the Black Death. Cities were less likely to emerge when their extrapolated mortality rate was high (col. 6). Likewise, we regress the log population of these 1,335 cities (using 500 for cities below 1,000) on mortality and find that fewer locations became urbanized in high-mortality areas (col. 7). Consistent with previous results, we find however that these negative effects of the Black Death disappeared by 1600 (Panel B).[50]

Timing. The direct, spillover and aggregate effects of the Black Death on cities disappeared by 1600. When running the same regressions for the period 1300-1500, we find negative coefficients, but these are not significantly different from 0.[51] Thus, the local recovery of cities and urban systems took place in the 16th century.

4.2. Effects on the Countryside

In the aftermath of the Black Death, contemporaries noted the phenomenon of *Wüstungen*, the depopulation of rural areas. Gottfried (1983, 135) observes that "However much plague depleted urban populations, there were always country folk ready to replace them. This migration to towns combined with the effects of disease in rural areas caused a pronounced shortage of agricultural workers".

While urban populations recovered by the 16th century, rural population recovered in the aggregate only by 1600 (see Figure 3(b)). Unfortunately, we do not have localized data on rural populations, and thus cannot test if rural population also recovered locally by 1600. Land use data, however, provides a proxy for rural population.

Land Use. The Black Death led to a period of massive reforestation in Europe as the need for land and wood dramatically declined and marginal soils were abandoned

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

[51] Not shown, but available upon request.

(Campbell, 2016) (see Web Appendix Section 10. for qualitative evidence). We use data on land use as a proxy for rural population. Kaplan et al. (2009) recreates localized data on land use from 1000 BCE to 1850 at the 5 by 5 minute (10 x 10 km) grid-cell level by combining information on country population, historical forest cover maps, and maps of soil suitability.[52] Using these data, we obtain the mean land use share (%) of the 16 modern-day countries covering our sample of cities. As shown in Figure 3(d), a greater share of land was used before the 14th century, reaching two thirds by 1300, and decreasing by 15 percentage points by 1400. Land use did not fully recover until 1800 (not shown). The delayed recovery of land use may reflect the employment of more capital per worker and the switch to pastoral farming. We obtain the mean land share within a 10 km radius of each of the 165 main cities and examine how land use varied.[53]

Table 8 shows that mortality led to a decline in land use close to cities, hence reforestation, in 1300-1400 (column (1)), which remained significant until 1500 (column (2)). The magnitude of the beta coefficient in 1300-1400 is lower than for the effect on city population but still high (-0.20 vs -0.35). No effects are found in 1600, 1700 and 1750 (col. 3-5). We also verify the parallel trends assumption—i.e. that there is no negative significant effect for the two centuries before the Black Death century (col. 6-7).[54]

Timing. Overall, cities recovered their populations by the 16th century and their rural areas, based on the results on land shares, recovered theirs by 1600. Hence cities recovered from the Black Death slightly before the countryside. Obviously, we expect cities and their rural areas to recover together: (i) As land is a fixed factor, rural wages increase when rural population declines, which, combined with non-homothetic preferences, increases the demand for urban goods, thus attracting people to surrounding cities (Voigtländer and Voth, 2013b); and (ii) Fixed factors in cities means

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

[53] Since country populations are one input used in the creation of these data, we verify that land use changes are not mechanically correlated with population changes. The correlation between the percentage change in land use within 10 km of a city and the percentage change in the population of that city is 0.14 in 1300-1400 and -0.03 in 1300-1600 (correlations are similar for larger circles). The correlations with the percentage change of that city's country population (source: McEvedy and Jones (1978)) are higher, but still low, at -0.30 and 0.44 respectively. Additionally, we control for the contemporaneous changes in both city population and country population in all land share regressions.

[54] Results generally hold if we use larger circles around cities or cluster observations at the country level (not shown, but available upon request). It is possible that land use is mis-measured. However, random measurement error in land use, the dependent variable, should if anything lower the precision of our estimates. The effects are precise for the periods 1300-1400 and 1400-1500, thus minimizing this concern.

that urban wages increase when urban population declines, thus raising the demand for food and attracting people to surrounding villages. However, cities recovered before land use, suggesting that either peasants were initially replaced by technology or livestock or that urban fixed factors played an important role in urban recovery.

If both cities and their immediate hinterlands recovered their populations by 1500-1600, population recovery in high-mortality areas must have been driven by either differentially increasing fertility and decreasing mortality in high-mortality areas or migration from low- to high-mortality areas. Since rural areas close to cities also recovered, migrants must have come *on net* from rural areas farther away.[55] This implies that potentially more marginal rural areas suffered relatively greater population losses following the Black Death. Indeed, while land use recovered locally by 1600 (see Table 8), it did not recover in the aggregate before the 18th century (not shown). Accordingly, using gridcell-level data on cereal suitability, cereal suitability decreases with distance to existing cities in 1300 (Web Appendix Figure A.4).[56]

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

Deserted Medieval Villages (DMVs). Historians document how the Black Death led to the desertion of villages in England (Beresford, 1954), France, and Germany (Braudel, ed, 1965; Pounds, 1974). The Black Death directly led to the disappearance of some rural communities. Others declined more gradually: "…'a steady hemorrhage of labor', whether through migration or early deaths, caused rural settlement to fall back. Some of the smaller villages were lost in these circumstances many more shrank dramatically in size…" (Platt, 1996, 16). In the longer run, since labor was in short supply and peasants demanded better pay, many landowners switched to sheep rearing, which required much less labor than arable farming. This gave rise to Thomas Moore's observation in the 16th century that sheep "devour men themselves". Peasants also abandoned their villages to seek newly available economic opportunities in high-mortality areas. This

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

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

immigration "topped up otherwise diminishing urban communities" (Platt, 1996, 20). But immigrants had to come from somewhere and it was often the countryside that lost population (see Web Appendix Section 11. for more qualitative evidence).

Unfortunately, data do not exist on the location of DMVs for all of Europe. But these data are available for all 41 English counties during the medieval period (Fenwick and Turner, 2015). For 28 of these counties, we also know from Shrewsbury (1970) and Scott and Duncan (2001) the mortality rate of the clergy, which we use as a proxy for the overall mortality rate. This allows us to study how the number of deserted villages varied with Black Death mortality, depending on their proximity to cities.

Since this sample is different from the main sample of 165 cities, we verify in columns (1)-(3) of Table 9 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 a insignificant negative effect, at -0.96 (col. (2)), but the effect is smaller than the short-run effect once standardized (beta coefficient of -0.10 vs. -0.35 in 1290-1377). When using 1801 (England's first census), the standardized effect is small (-0.08, not shown). We then check parallel trends and find no effect in 1086-1290 (0.05, beta coeff. of 0.00, see col. (3)).[57]

In columns (4) - (6) we use the log number of DMVs per 1000 sq. km's as the dependent variable. We also control for the county's log area and log population in 1290 since the density of DMVs depends on the pre-Plague human density of counties.[58] We find a negative effect of mortality, at -0.46*** (col. (4), beta coefficient of -0.51).[59] Low-mortality areas had more DMVs than high-mortality areas, which suggests people disproportionately left the relatively Plague-free rural areas. Table 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. This suggests that we should

[57] We weight observations by their population in 1290 and exclude Cornwall whose population in 1290 is severely underestimated due to the lack of reliable data on their large mining population (see Broadberry et al. (2010, , 14) for a discussion of this issue). Note that Middlesex—the county representing London in the medieval period—is not included in the analysis, due to the lack of data on its mortality. However, if we use London's estimate instead, results are similar. Likewise, if we impute overall mortality from other sources, results still hold (not shown but available upon request).

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

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

expect relatively more DMVs in low-mortality areas *beyond* 10 km from cities. For 39 counties, we obtain from Fenwick and Turner (2015) the precise location of each DMV in England and compute the minimal distance to an existing city in 1300. For each of the 28 counties, we then construct the number of DMVs (per 1000 sq km) both within and beyond 10 km from a city. We then verify in columns (5)-(6) that the loss of villages is driven by areas farther away from cities.[60]

In column (7) of Table 9, we regress the absolute change in the urban share (%) on mortality (using the cities from our city-level data set to estimate each county's urban population) and find a small and insignificant negative effect.[61] As discussed above, many DMVs were small. Therefore, in England the loss of villages in low-mortality areas may have not been large enough to affect urbanization patterns across counties. This also suggests that the repopulation of high-mortality areas was allowed by migration from both urban and rural areas in low-mortality areas.

4.3. Natural Increase vs. Migration

Natural Increase. The relative recovery of high-mortality areas could have been due to higher wages there raising fertility and lowering mortality relative to low-mortality areas. While the population recovery of Europe's total population by 1600 (Fig. 3(a)) was only possible due to natural increase, it is less clear whether natural increase was also responsible for local recovery. The literature on the European Marriage Pattern (EMP) — a higher age of first marriage and high rates of female celibacy — shows how the Black Death may have reduced fertility, moderating the rate of natural increase (Hajnal, 1965; Voigtländer and Voth, 2013a). The EMP was stronger in Northern than in Southern Europe (Moor and Zanden, 2010; Dennison and Ogilvie, 2014). The EMP also functioned at the macro level, affecting social norms about the appropriate age of first marriage that were common across entire regions, rather than varying at the local level. Thus, natural increase likely only played a minor role in *local* recovery. In this case, migration must have been the source, especially for cities (see Section 2.2.).

Migration. Overall mortality rates in cities in the post-Plague period were much

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

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

higher than in the countryside (Woods, 2003). The rate of urban recovery we observe, moreover, can only have occurred via migration. High-mortality cities relatively recovered by the 16th century and various cities had already recovered before 1400. In particular, 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 fully explain recovery. These rates were unheard of until the 20th century, particularly in preindustrial cities where such rates were typically nil or negative (Woods, 2003; Voigtländer and Voth, 2013b; Jedwab and Vollrath, 2019).

North/EMP Effects. In addition, we test if the speed at which high-mortality cities relatively recover depends on whether the cities belong to a Northern Europe region or a region characterized by the European Marriage Pattern (EMP). We classify our cities into North/Strong EMP vs. South/Weak EMP, based on modern country boundaries and data from Dennison and Ogilvie (2014) on the average age at first marriage and the female celibacy rate (%) at the country or regional level (e.g., Southern France). For the 165 cities, we use the same specification as before but interact mortality with a North/Strong EMP dummy and test if the interacted is negative and significant. Indeed, if natural increase was important for local urban recovery, we should expect high-mortality cities in North/Strong EMP regions to recover relatively slower than high-mortality cities in South/Weak EMP regions, because North/Strong EMP cities were more likely to recover solely through migration whereas South/Weak-EMP cities were more likely to experience both migration *and* natural increase. A lack of any significant difference between North/Strong EMP and South/Weak EMP cities implies that natural increase was not significant in either Southern cities or cities where the EMP was weaker.

Table 10 shows the effects of mortality, the North/Strong EMP dummy and their interaction for the long-run period 1300-1600. Column 1 replicates the baseline effect (0.36). The North dummy is equal to 1 for: (i) 114 cities in 9 Northern European countries (col. 2, see notes under the table for details); (ii) 75 cities in the same 9 countries except France since it could be viewed as either Northern or Southern (col. 3). The Strong EMP dummy is equal to 1 for: (i) 88 and 63 cities in countries or regions with an age at first marriage above the mean or median in the sample (col. 4-5); and (ii) 30 and 77 cities in countries or regions with a female celibacy rate above the mean

or median in the sample (col. 6-7). Note that the dummies are not highly correlated (not shown) so they capture different aspects of the EMP. Nevertheless, the interacted effect shows that North/Strong EMP cities did not recover relatively slower, since the coefficients are not negative and significant, thus suggesting that migration was the main driver behind local urban recovery.[62]

4.4. Heterogeneity in Population Recovery

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

We indeed find that aggregate urban recovery hides changes in the ranking of cities. 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. The fact that mortality had nil effects by 1600 implies that the Black Death did not *on average* alter the ranking of cities. However, this hides *permutations* in that the R2 of the rank regression is 0.56. There are thus changes that are unexplained by average patterns. Web Appendix Figure 5(a) illustrates this, with many cities far from the regression line, having permanently declined or grown relative to other cities.[63] 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, as with the Black Death, but a R2 also close to 1.00, unlike in our context.

We now test whether the observed permutations were associated with the presence

[62] Results hold if we also reclassify Austria and Switzerland as "Southern" or only consider French cities above Bordeaux or Paris as "Northern" (not shown but available upon request).

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

or absence of fixed factors. We expect high-mortality cities with fixed factors raising the marginal product of labor to recover faster (and to potentially become larger in the long-run). Indeed, when mortality is high and labor becomes scarce, if there are fixed factors complementary to labor, wages should increase, attracting people. 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. We modify Equation 1 by interacting mortality ($\text{Mort}_{i,1347-52}$) with selected fixed factors (FixFact_i) while simultaneously controlling for the fixed factors themselves and mortality:

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

For cities experiencing the same mortality shock, the vector $\boldsymbol{\theta}$ captures the differential effects of each fix factor. Throughout, we focus on our main sample of 165 cities, for the period 1300-1750 which we then extend to 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 intersections, medieval land trade route intersections, and the Hanseatic League dummy.[64] While the coast and rivers are by construction "fixed", the main advantage that they conferred was that they lowered transportation costs. Hence, coastal and riverine cities were more likely to develop trading and manufacturing sectors. Roman roads and the Hanseatic league can be treated as fixed factors, since they represent past sunk investments in transportation and trade networks. Roman roads remained the basis of the road network in the medieval era (Dalgaard et al., 2018). Medieval trade routes reflected long-established trading linkages. The origins of the Hanseatic league go back to its establishment in Lübeck in 1159 and it rose to prominence in the century before the Black Death (Dollinger, 1970, xviii).

Finally, we include factors proxying for agglomeration effects and institutions, both as controls and to show that only the fixed factors described above attracted populations to high-mortality cities. These variable factors include the log of the estimated population of the city in 1353 (= pop. in 1300 x (100-mortality)/100), since larger cities in the aftermath of the Black Death may have recovered faster if there were agglomeration effects. We also include three dummies for whether the city was part of

[64] Since we have 165 cities, we do not add all 27 variables of Table 2 and their interactions with mortality.

a kingdom, was a state capital, and whether it had a representative body (ca. 1300).

Rural Fixed Factors. Table 11 shows the interacted effects, for 1300-1750 (col. (1)-(5)) and 1300-2015 (col. (6)).[65] The coefficients of cereal suitability becomes positive (but are not significant) after 1400 (col. (2)). However, the effect is meaningful since the beta coefficient reached 0.47 by 1600 and remained high after (e.g., 0.17 in 2015). Potato suitability also helped cities recover from the 17th century onwards (col. (4)). This effect is consistent with Nunn and Qian (2011) who shows 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).[66] Finally, in high-mortality areas that were also suitable for pastoral farming we find a negative interacted effect in 1500-1600 (col. (3)) and no effects before (col. (1)-(2)). The effect in 1500-1600 is strong (beta coefficient of -0.64) and becomes weaker over time (beta coefficient of -0.25 in 2015). We believe this is caused by higher wages from the 16th century due to labor shortages creating incentives for landlords to specialize in sheep rearing, for which only a few shepherds are needed, thus reducing the need for labor.[67]

Urban Fixed Factors. We find strong recovery effects for coastal cities. The interacted effect for coastal proximity is one of the only two significant coefficients in 1300-1400 (col. (1)), along with the interacted effect for the Hanseatic league. This suggests that these cities were in prime location, and that their economic sectors could afford the high wages to attract new residents. Examples of rapid recovery include Barcelona (mortality of 36%; full recovery by 1355) and Venice (60%; 1375) for coastal cities and Lübeck (30%; 1360) as an example of a major Hansa town. The coastal effect is strong throughout the period, with the beta coefficient reaching 0.99 in 1500 and then slowly decreasing to 0.33 in 2015.[68] The Hanseatic league effect is strong (beta coefficient of 0.72) in

[65] One issue with 2015 is that population does not come from the Bairoch data set which stops in 1850. Cities have grown dramatically since 1850, becoming large multi-city agglomerations, thus creating consistency issues. For each of the 165 cities, we read the webpage of each city in Wikipedia and selected the population of the city itself rather than the population of the agglomeration.

[66] Note that their direct effects become significant in 1850, whereas our interacted effects with mortality are significant as early as 1600-1700, perhaps because our analysis is at a more granular level and focused on the city level. Our effect is still very large, and significant, in 2015 (beta coefficient of 1.06).

[67] Note that we use the suitability indexes of the pixel that the city belongs to in our GIS maps of suitability. Results are robust if we use larger circles around cities (not shown, but available upon request). Also, the suitability measures combine information on soils and the climate. Lastly, we also examine the effect of being in the top 25% of elevation, thus testing whether geographical isolation matters for recovery. We find a negative effect that is only significant at 15% in 1300-1500 (not shown).

[68] Decomposing the coastal dummy into three dummies for the Mediterranean Sea, the Atlantic Ocean

1300-1400 and significant until 1700 (col. (4)), by which time the Hanseatic league was in decline (Dollinger, 1970). Rivers have positive and significant effects from the 17th century onwards (col. (4)). The effect in 1300-1700 is fairly strong – beta coefficient of 0.40 – and remained so up to the present day—beta coefficient of 0.45. Finally, being at the intersection of two or more roads/trade routes has a positive, significant, and economically large effect in 1300-1500 (col. (2), beta coeff. of 0.51). The effect is not significant and weaker in later years (beta coefficient of 0.15 in 2015).[69]

Interpretation. We do not find any significant effects of initial population or institutions.[70] Adding the absolute values of the beta coefficients for the different types of factors (see Web Appendix Figure 5(b)), we find that urban fixed factors were particularly important in the first centuries after the Black Death and that their importance decreases over time, probably as other transportation technologies and networks emerged. Rural fixed factors become important by the 16th century, perhaps as Europe's population increased and growing cities needed more food. Then their importance decreases to the same extent as urban fixed factors. Other factors such as initial population and institutions are less important overall and their relevance declines over time, probably as population levels and institutions changed over time.[71]

Finally, Table 1 showed no long-term effects of the Black Death at the city level. In Table 11, the significant negative effects of mortality once we control for the fixed factors and their interactions with mortality implies that any city without agriculture-related rural fixed factors and trade-related urban fixed factors would have literally collapsed (the point estimate becomes more negative but the beta coefficient remains similar across years, at -1.55 in 1300-1400 and -1.26 in 1300-2015). In other words, had European cities not had rural and urban fixed factors, the Black Death would have had permanent, and very negative, localized effects. This analysis shows how important both fixed factors have been for the spatial recovery of Europe after the Black Death.

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and the North-Baltic Sea, we find very strong recovery effects for the Mediterranean (not shown, but available upon request). The beta coefficient reaches 1.07 in 1300-1500, then slowly decreasing to 0.34 in 2015. Indeed, while Mediterranean trade was particularly important around the time of the Black Death, it eventually declined and trade with the Americas took off (Acemoglu et al., 2005b).

[69] We find no effect for cities along single roads (not shown, but available upon request).

[70] We also find no effects if we try more factors from Table 2, such as markets and fairs and universities (not shown, but available upon request). Additionally, we find the results on rural and urban fixed factors generally hold if we add the other controls from Table 2 or country fixed effects (not shown).

[71] It is also likely that institutions changed endogenously as a result of the Black Death (Brenner, 1976; Acemoglu and Robinson, 2012). However, this is not the focus of this paper.

Implications. Our results suggest that people disproportionately left marginal rural areas as well as cities with "worse" rural and urban factors. Thus, the relative population decline of "worse" areas suggests that the growth potential of Europe's distribution of population may have also improved in the aggregate. We do not pursue this analysis further as it would require a causal analysis of the effects of these fixed factors on long-run economic development, and a theory-grounded analysis of what a dynamically optimal distribution of population involves. But it is important to note that the impact of the Black Death on Europe's spatial distribution of economic activity may have been one factor contributing to both the Great Divergence that opened up between Europe and the rest of the world after 1700 and the Little Divergence that took place within Europe itself (Pamuk, 2007; Voigtländer and Voth, 2013b).

5. Conclusion

The Black Death killed 40% of Europe's population between 1347-1352. We shed light on its spatial effects and the effects of pandemics more generally. Using a novel dataset that provides information on spatial variation in Plague mortality at the city level, as well as various identification strategies, we explore the short-run and long-run impacts of the Black Death on city growth. On average, cities recovered their pre-Plague populations within two centuries. Aggregate convergence masked heterogeneity in recovery. We show that both of these facts are consistent with a Malthusian model in which population returns to high-mortality locations endowed with better rural and urban fixed factors of production. Land suitability and natural and historical trade networks played a vital role in urban recovery. Our study highlights the role played by pandemics in determining both the levels and placements of populations.

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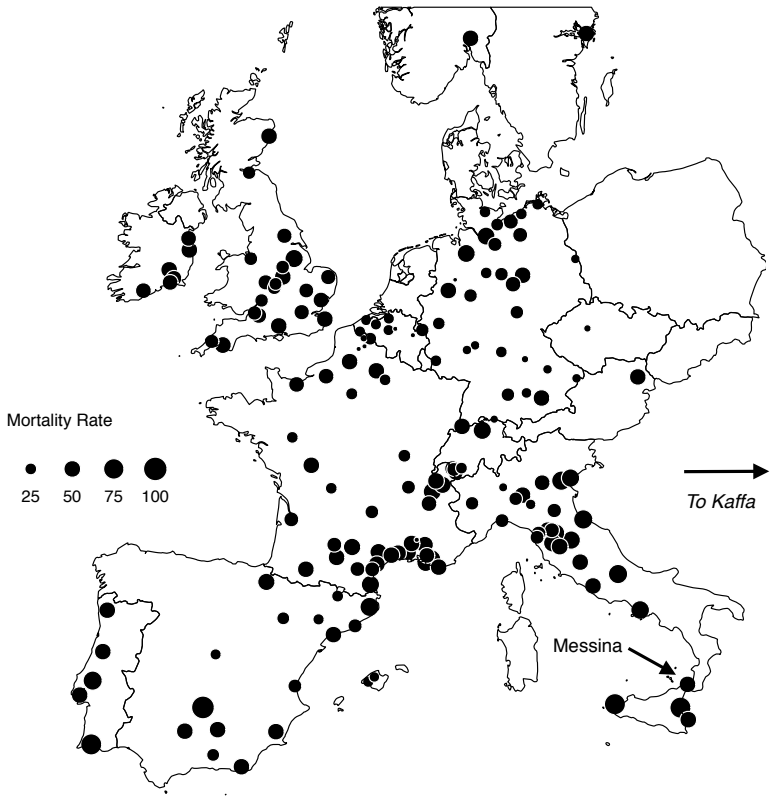
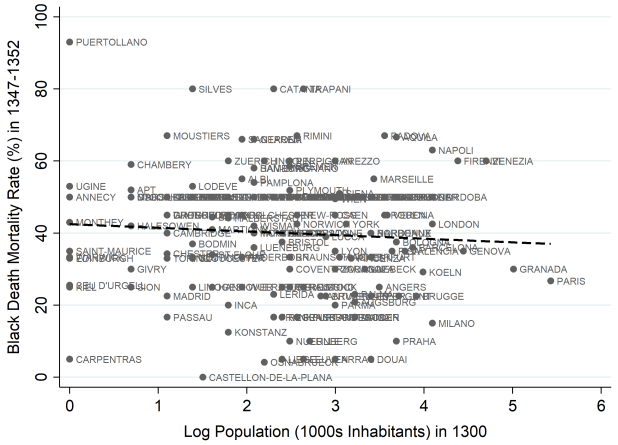
Figure 1: Black Death Mortality Rates (%) in 1347-1352



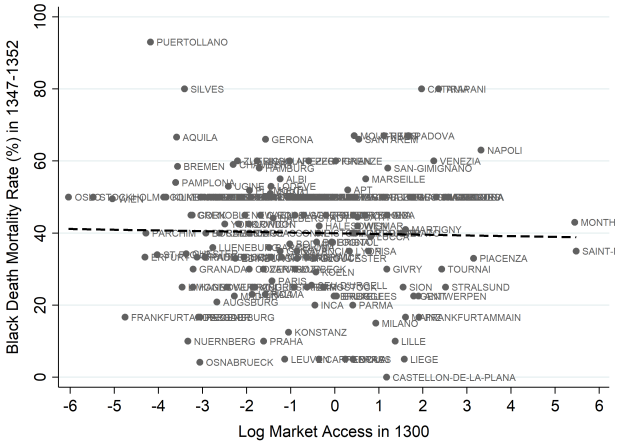
Notes: This map plots the location of all 165 existing cities (i.e. localities $\geq$ 1,000 inhabitants) in 1300 for which we know their Black Death mortality rate (%) in 1347-1352 as well as the modern boundaries of the 16 Western European countries of our main analysis (Austria, Belgium, Czech Republic,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

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

(a) Mortality and Initial City Size

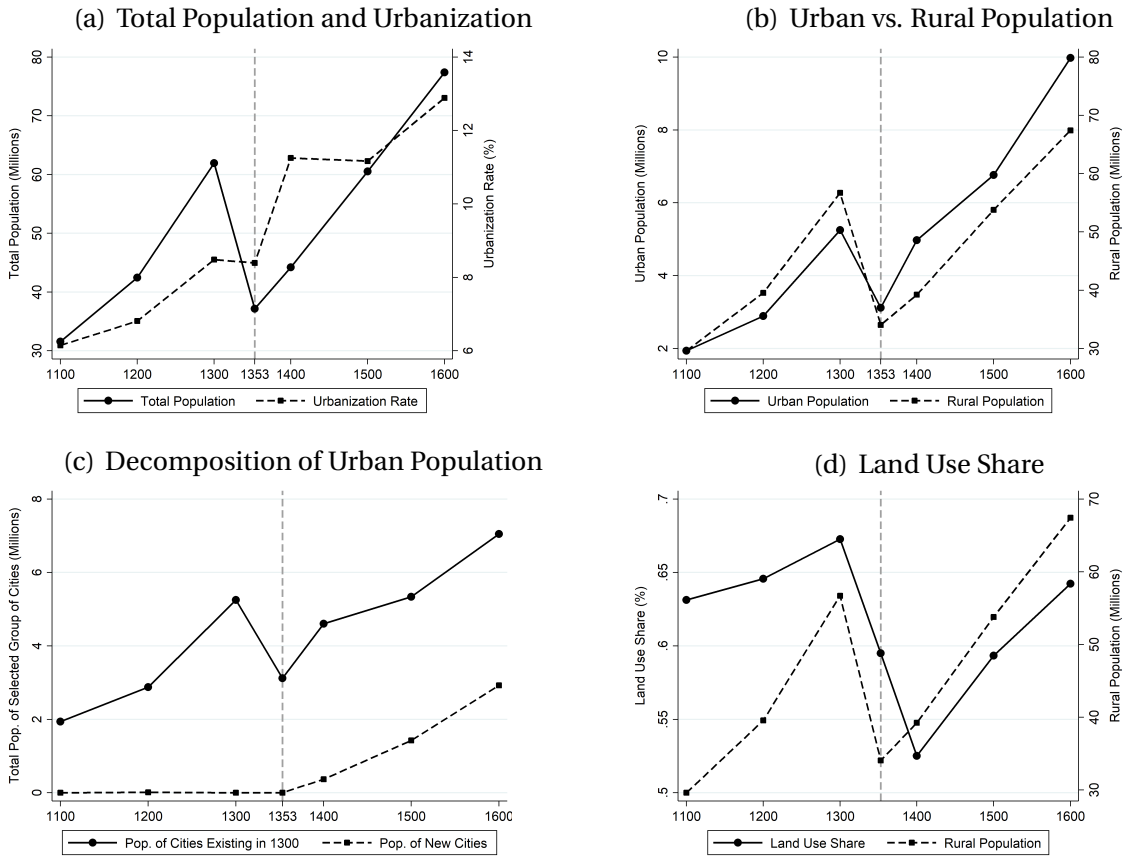


(b) Mortality and Initial Market Access



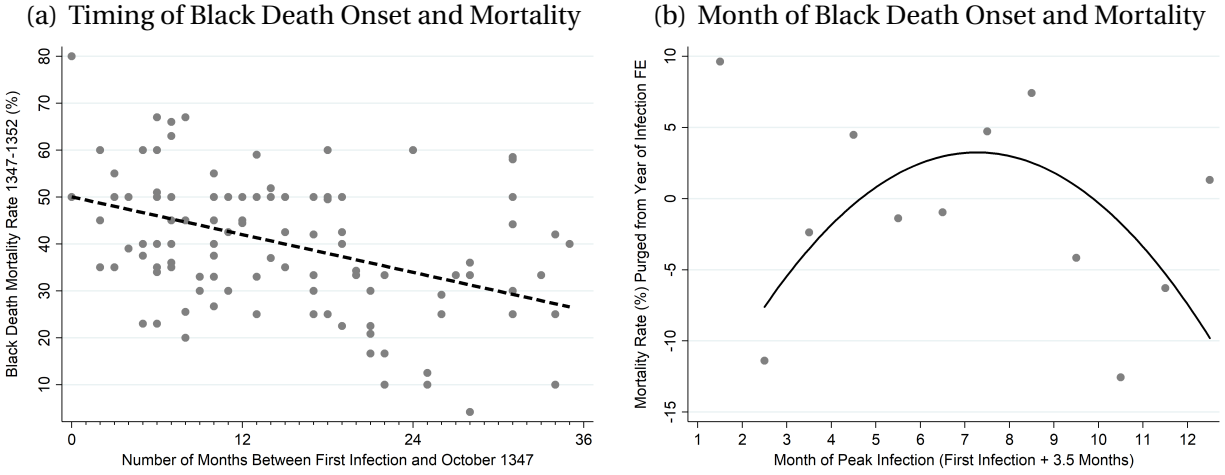
Notes: Subfigure 2(a) shows the relationship between mortality rates (%, 1347-1352) and log city population in 1300 for our main sample of 165 cities (Y = 42.5*** -1.01 X; R2 = 0.00). Subfigure 2(b) shows for the same 165 cities the relationship between mortality rates (%, 1347-1352) and log market access to all 1,801 cities in 1300 (Y = 40.0*** -0.20 X; R2 = 0.00). Market access for city i is defined as $MA_i = \Sigma_j \frac{P_j}{D_{ij}^{\sigma}}$, with P_j being the population of town $j \neq i$, D_{ij} the travel time between city i and city j, and $\sigma = 3.8$. To obtain the travel times, we compute the least cost travel paths via four transportation modes — by sea, by river, by road and by walk — with the transportation speeds from Boerner and Severgnini (2014). See Web Appendix for data sources.

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



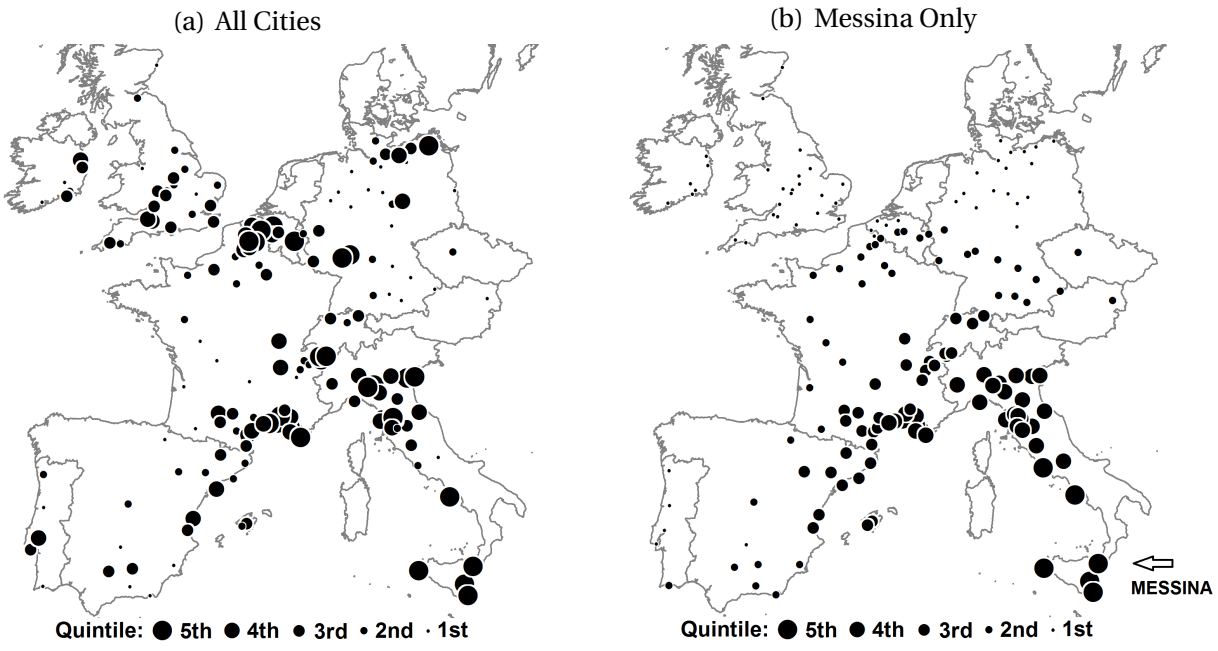
Notes: The subfigures show the respective evolutions of the total population (millions) and urbanization rate (%) (Subfigure 3(a)), the total urban and rural populations (Subfigure 3(b)), the population of cities that existed in 1300 and the population of cities that did not exist in 1300 (Subfigure 3(c)), and the land use share (%) (Subfigure 3(d)) of the 16 European countries in 1100-1600. Total (urban) population in 1353 is proxied by the total (urban) population in 1300 times the population-weighted average total (urban) mortality rate in 1347-1352 (40% and 38.8%). Rural populations are estimated residually. See Web Appendix for data sources.

Figure 4: Timing of the Onset of the Black Death and Black Death Mortality



Notes: Subfigure 4(a) shows for 165 cities the relationship between mortality (%) and the timing of the onset of the Black Death in the city (Y = 50.0*** - 0.67***; R2 = 0.19). Number of months is measured since October 1347, the date Messina – the port of entry of the Black Death in Europe – was infected. Subfigure 4(b) shows for the same 165 cities and for each month of peak infection (month of first infection + 3.5 months) the average mortality rate (%) purged of year of infection fixed effects. The quadratic fit shows that mortality was the highest when peak mortality was in the summer and the lowest in the winter. The quadratic fit omits October, which has high mortality rates due to being the month of onset of the Black Death in Europe. See Web Appendix for data sources.

Figure 5: Market Access to All Cities vs. Market Access to Messina Only, 1300.



Notes: Subfigure 5(a) shows for 165 cities their log market access to all 1,801 cities in 1300. Subfigure 5(b) shows for the same cities their log market access to Messina in 1300. See notes under figure 2(b) for details on how market access is calculated. We use as an instrument log market access to Messina, *conditional* on log market access to all cities. See Web Appendix for data sources.

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

	Dependent Variable: Percentage Change in City Population (%) 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	-0.87***	-0.28	0.36	0.47	0.85	-0.25	0.16
	[0.28]	[0.38]	[0.80]	[1.00]	[1.17]	[0.34]	[0.59]
	[-1.4 - -0.3]	[-1.0 - 0.5]	[-1.2 - 1.9]	[-1.5 - 2.4]	[-1.5 - 3.2]	[-0.9 - 0.4]	[-1.0 - 1.3]
Obs.	165	164	164	164	164	62	93
R^2	0.12	0.01	0.00	0.00	0.00	0.01	0.00

Notes: This table shows the effect β_t of the mortality rate (%) in 1347-1352 on the percentage change in city population (%) for each period t. The main sample consists of 165 cities (i.e. loc. $\geq$ 1,000 inh.) that existed in 1300 and for which mortality is available. We use city population in the initial year of period t as regression weights. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. The 95% confidence level intervals are shown into brackets below the SEs. See Web Appendix for data sources.

Table 2: CITY CHARACTERISTICS AND BLACK DEATH MORTALITY RATES

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

Notes: This table shows the effects of city characteristics on mortality (%, 1347-1352). See the text for a description of the variables. We use the main sample of 165 cities. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.

Table 3: MORTALITY AND CITY GROWTH, INVESTIGATION OF CAUSALITY

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

Regression:	(1) t = 1300-1400			(2) t = 1300-1600		
1. Baseline (See Columns (1) and (3) of Table 1)	-0.87***	[0.28]	165	0.36	[0.80]	164
2. Controls: Locational Fundamentals	-0.79***	[0.26]	165	0.24	[0.64]	164
3. Controls: Increasing Returns	-0.68***	[0.24]	165	-0.03	[0.55]	164
4. Controls: Institutions	-0.61**	[0.23]	165	0.66	[0.75]	164
5. Controls: All	-0.59***	[0.21]	165	-0.37	[0.70]	164
6. Dropping Top and Bottom 5% in Mortality	-0.67**	[0.29]	145	1.36	[1.12]	144
7. Dropping Top and Bottom 10% in Mortality	-0.67*	[0.38]	136	1.10	[0.92]	135
8. Dropping More Hygienic Cities	-0.89***	[0.30]	161	0.59	[0.90]	160
9. 13 Country (2018) FE	-0.62**	[0.26]	165	0.03	[0.76]	164
10. 7 States (1300) FE (for States >= 5 Cities)	-0.82**	[0.35]	105	-0.29	[0.68]	104
11. Ctrls for Dummy & Num. Plague Recur. 50km	-0.87***	[0.27]	165	0.41	[0.81]	164
12. Ctrls for Dummy & Num. Battles 50km	-0.84***	[0.28]	165	0.77	[0.78]	164
13. Ctrls for Jewish Pres., Pers., Pogr. 1347-1352	-0.83***	[0.30]	165	0.28	[0.81]	164
14. Drop if Jewish Persecution 1347-1352	-0.71***	[0.30]	115	0.67	[0.96]	114

Notes: This table shows the effect β_t of the mortality rate (%) on the percentage change in city population (%) for period t. Rows 2-5: Adding the controls of Table 2. Rows 6-7: Dropping the top and bottom 5% or 10% mortality rates. Row 8: Dropping cities with a better hygiene system. Row 9: Adding 13 country FE. Rows 10: Adding 44 state FE but excl. states with less than 5 cities in our sample. Rows 11-12: Adding a dummy if there was a plague recurrence/battle and the number of recurrences/battles in 1353-1400 or 1353-1600. Row 13: Adding dummies if Jews were present, and if a persecution, and a pogrom in particular, took place in 1347-1352. Row 14: Dropping cities with Jewish persecutions in 1347-1352. Robust SE's: † p=0.17, * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.

Table 4: MORTALITY AND CITY GROWTH, IV REGRESSIONS

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

Regression:	(1) t = 1300-1400			(2) t = 1300-1600		
1. Baseline (See Columns (1) and (3) of Table 1)	-0.87***	[0.28]	165	0.36	[0.80]	164
2. Baseline for IV1-IV2 (Sample of 124 Cities)	-0.90***	[0.35]	124	0.87	[0.99]	124
3. IV1: Timing w/o Controls (IV F-stat = 21.2; 21.2)	-1.67**	[0.67]	124	-0.24	[1.32]	124
4. IV1: Timing w/ Controls (IV F-stat = 12.6; 12.6)	-1.07**	[0.48]	124	0.05	[1.69]	124
5. IV2: Month w/o Controls (IV F-stat = 7.3; 7.3)	-2.08***	[0.55]	124	-1.52	[1.32]	124
6. IV2: Month w/ Controls (IV F-stat = 3.3; 3.3)	-0.85**	[0.43]	124	-0.39	[1.00]	124
7. IV3: Messina w/o Controls (IV F-stat = 29.0; 29.3)	-1.32***	[0.44]	164	1.10	[2.14]	163
8. IV3: Messina w/ Controls (IV F-stat = 9.9; 10.5)	-0.90†	[0.59]	164	-1.41	[2.07]	163
9. IV1 + IV2 + IV3 (IV F-stat = 7.3; 7.3)	-1.87***	[0.49]	123	-0.21	[1.22]	123
10. IV1 + IV2 + IV3 w/ Controls (IV F-stat = 3.2; 3.2)	-0.95**	[0.44]	123	-0.26	[0.98]	123

Notes: Robust SE's: † p<0.15, * p<0.10, ** p<0.05, *** p<0.01. Rows 3-4: Instrumenting by the number of months between the city-specific date of first infection and Oct 1347. Rows 5-6: Instrumenting by twelve dummies for the month of peak infection (month of first infection + 3.5 months) while adding dummies for the year of infection. Rows 7-8: Instrumenting by log market access to Messina in 1300 while controlling for log market access to all 1,801 towns in 1300 (Messina is mechanically dropped from the regression). In rows 4, 6 and 8, we add all the controls of Column (4) in Table 2 as well as latitude and longitude and their squares and cubes to flexibly control for Plague diffusion coming from the South and from the East. See Web Appendix for data sources.

Table 5: MORTALITY AND CITY GROWTH, ROBUSTNESS CHECKS

Dependent Variable: Percentage Change in City Population (%) in Period t						
Regression:	(1) t = 1300-1400			(2) t = 1300-1600		
1. Baseline (See Columns (1) and (3) of Table 1)	-0.87***	[0.28]	165	0.36	[0.80]	164
2. Dummies for Type of Mortality Data	-0.94***	[0.30]	165	0.27	[0.84]	164
3. Excl. Description-Based Mortality Data (N = 25)	-0.83***	[0.32]	140	0.37	[0.97]	139
4. Excl. Desertion-Based Mortality Data (N = 21)	-0.98***	[0.31]	144	0.31	[0.86]	143
5. Excl. Clergy-Based Mortality Data (N = 5)	-0.86***	[0.28]	160	0.38	[0.80]	158
6. Use Only Number-Based Mortality Data	-0.96***	[0.36]	114	0.35	[1.06]	113
7. Row 6 Excl. Mort. = {1/4, 1/3, 1/2, 2/3, 3/4}	-1.01**	[0.39]	88	0.71	[1.21]	87
8. Keeping Top and Bottom 10% in Mortality	-0.99**	[0.39]	34	-0.21	[1.01]	34
9. City Population Data: Bairoch Only	-0.78*	[0.41]	151	0.47	[0.80]	150
10. City Population Data: Chandler Only	-0.85**	[0.36]	59	1.37	[1.10]	60
11. Use Mortality of Nearest Avail. City w/i 50 km	-0.60***	[0.22]	290	0.41	[0.59]	286
12. Use Mortality of Other Cities in Same State	-0.70***	[0.17]	380	0.33	[0.63]	374
13. Extrapolated Rates Based on 274 Cities in 1300	-0.68***	[0.21]	464	0.43	[0.54]	457
14. Reweighting to Match Full City Size Dist.	-0.74***	[0.24]	165	-0.06	[0.65]	164
15. Add Pct. Change in Pop. 1200-1300	-1.07***	[0.36]	93	0.83	[1.04]	93
16. Add Pct. Change in Pop. 1200-1300, 1100-1200	-1.17**	[0.47]	62	1.12	[1.35]	62
17. No Weights & Excl. Cities $<$ 5,000 in 1300	-0.79***	[0.29]	123	-0.03	[0.54]	122
18. Panel Model with City FE, Year FE, 1100-1750	-1.16**	[0.52]	1021	0.18	[0.46]	1021
19. Conley Standard Errors (100 km)	-0.87***	[0.28]	165	0.36	[0.80]	164
20. SEs Clustered at State ca 1300 Level (N = 64)	-0.87***	[0.27]	165	0.36	[0.68]	164

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

Table 6: MORTALITY AND CITY GROWTH, 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	-0.4	-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
R^2	0.12	0.13	0.14	0.12	0.02	0.01	0.00	0.01

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: Percentage change in market access between 1300 and 1353. To construct market access in 1353, we use the predicted population 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. See Web Appendix for data sources.

Table 7: MORTALITY AND CITY GROWTH, AGGREGATE EFFECTS, 1300-1600

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

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

Table 8: BLACK DEATH MORTALITY RATES AND LAND USE, 1100-1750

Dependent Variable: Percentage Change in Land Use Share (%) in Period t

t:	1300-1400 (1)	1300-1500 (2)	1300-1600 (3)	1300-1700 (4)	1300-1750 (5)	1100-1200 (6)	1200-1300 (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 9: BLACK DEATH MORTALITY AND DESERTED VILLAGES, ENGLAND

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

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

Table 10: 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 11: BLACK DEATH MORTALITY RATES AND POPULATION RECOVERY, 1300-2015

Dependent Variable: Percentage Change in City Population (%) in Period t						
Period t:	1300-1400 (1)	1300-1500 (2)	1300-1600 (3)	1300-1700 (4)	1300-1750 (5)	1300-2015 (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 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.

WEB APPENDIX - NOT FOR PUBLICATION

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 13 countries in Western Europe using today's boundaries: Austria, Belgium, the Czech Republic, France, Germany, Ireland, Italy, Norway, Portugal, Spain, Sweden, Switzerland, and the United Kingdom. We 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, Christakos et al. (2005) examine the nature of the information available for each location. Information on the intensity, timing, and duration 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 potential 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

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

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

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 is shown in Web Appendix Figure A.1.

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

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

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

We generate optimal mortality surfaces using several different city samples. Our baseline sample consists of cities for which mortality rates are reported in the historical literature. There are 274 of these cities and the cross-validation exercise chooses an optimal power for creating the mortality surface as 1.76 (RMSE=15.186). Figure 2(a) shows the relationship between the measured and predicted mortality rates for this 274 city sample. Figure 2(b) shows the location of the 274 cities and the extrapolated mortality rates using the optimal power. Note that for the regressions in Table 6 (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, the capital of the Azores archipelago of Portugal 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

[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.

the road network, we also create variables for distances to Roman road intersection. We then create four dummies if the city is within 10 from any Roman road, a major Roman road, any Roman road intersection, or a major Roman road intersection.

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

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

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.

Descriptive Statistics. The summary statistics for the variables of Tables 1-11 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 the spread of the Black Death was too fast for bubonic plague and pointing to an absence of references to rat dying in medieval sources. Similarly, Cohn (2003) forcefully argued that the seasonal timing of mortality during the Black Death was incompatible with that disease being the bubonic plague.

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

what we know about outbreaks of bubonic plague in the modern period.

Following the Black Death (1347-1352) bubonic plague remained endemic in Europe for the next 250 years (Biraben, 1975; Alfani and Murphy, 2017). These outbreaks are studied by Biraben (1975) and recently by Siuda and Sunde (2017) and Dittmar and Meisenzahl (2019).

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

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

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 2.2.. 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 rates during the plague and economic recovery was mediated by institutions. Areas that were 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.

In general, 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 2.2. and 4.4. 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 2.2. 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). We examine this in Section 4.2..

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 also increased the demand of wood— to be used as a fuel—which led to a market for wood in Europe. 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 of 11 and mode of 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 4.4., 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, 26 in 1500, and only 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%.[3] 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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[3] Ziegler (1969, 86) estimates the death toll in Hamburg as between a half and two-thirds.

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Table A.1: DESCRIPTIVE STATISTICS FOR THE VARIABLES OF TABLES 1-11

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
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
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
TABLE 4					
Num. Mths 1st Inf. Since Oct47	124	14.4	9.5	0.0	35.0
Log MA Messina 1300	164	4.0	0.6	1.0	4.8
Log MA All Cities 1300	165	-0.9	2.0	-6.0	5.5

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Table A.2: DESCRIPTIVE STATISTICS FOR THE VARIABLES OF TABLES 1-11 - CONTINUED

Variable	Obs	Mean	Std.Dev.	Min	Max
TABLE 5					
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
Pct.Chg.Pop. 1200-1300	93	59	102	-70	483
Pct.Chg.Pop. 1100-1200	62	27	38	-30	200
TABLE 6					
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. MA	165	40.1	11.7	9.3	70.5
TABLE 7					
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 8					
Pct.Chg.LandUseSh. 1100-1200	58	2.5	4.1	-2.5	24.3
Pct.Chg.LandUseSh. 1200-1300	89	4.7	5.1	0.2	23.7
Pct.Chg.LandUseSh. 1300-1400	160	-20.4	20.6	-66.9	0.0
Pct.Chg.LandUseSh. 1300-1500	159	-12.0	15.9	-52.8	0.0
Pct.Chg.LandUseSh. 1300-1600	159	-5.0	8.2	-26.5	10.6
Pct.Chg.LandUseSh. 1300-1700	159	-6.6	13.7	-44.1	5.3
Pct.Chg.LandUseSh. 1300-1750	159	-6.6	13.7	-44.1	5.3
TABLE 9					
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 10					
North Dummy	165	0.7	0.5	0.0	1.0
North Excl. France Dummy	165	0.5	0.5	0.0	1.0
Abov. Mean Age 1st Marriage	165	0.5	0.5	0.0	1.0
Abov. Median Age 1st Marriage	165	0.4	0.5	0.0	1.0
Abov. Mean Female Celib. Sh.	165	0.2	0.4	0.0	1.0
Abov. Median Female Celib. Sh.	165	0.5	0.5	0.0	1.0

Table A.2: MARKET ACCESS AND MORTALITY RATES, ROBUSTNESS CHECKS

Dependent Variable: Black Death Mortality Rate (%, 1347-1352):				
	Effect of Log Market Access in 1300:			
1. Unconditional (See Figure 2(b))	-0.20	-0.78	-5.62	-0.03
	[0.67]	[1.87]	[5.33]	[0.81]
2. Including All Controls from Table 2 Column (4)	-0.34	-1.12	-6.87	0.13
	[0.82]	[2.47]	[7.60]	[0.83]
Sigma	3.8	2	1	3.8
Distance	Network	Network	Network	Euclidean

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

Table A.3: 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.4: FIRST STAGE OF THE INSTRUMENTAL VARIABLES REGRESSIONS

Dependent Variable:	Black Death Mortality Rate (%, 1347-1352):					
	IV1		IV2		IV3	
	(1)	(2)	(3)	(4)	(5)	(6)
♯ Months btw Oct 1347 & 1st Infec.	-0.78***	-1.22***				
	[0.17]	[0.34]				
Month Peak Infect.: Jan			26.62***	29.40***		
			[4.55]	[6.24]		
Month of Peak Infect.: Mar			15.21***	17.56***		
			[4.03]	[5.90]		
Month of Peak Infect.: Apr			23.38***	32.31***		
			[4.46]	[5.91]		
Month of Peak Infect.: May			17.67***	27.68***		
			[5.22]	[6.56]		
Month of Peak Infect.: Jun			24.06***	26.78***		
			[5.78]	[5.04]		
Month of Peak Infect.: Jul			27.01***	27.37***		
			[4.52]	[5.28]		
Month of Peak Infect.: Aug			21.70***	25.51***		
			[4.56]	[4.98]		
Month of Peak Infect.: Sep			16.99**	24.80***		
			[7.76]	[7.97]		
Month of Peak Infect.: Oct			8.69***	18.85***		
			[2.95]	[5.25]		
Month of Peak Infect.: Nov			6.36	11.90**		
			[3.96]	[4.93]		
Month of Peak Infect.: Dec			18.33***	9.18*		
			[3.98]	[5.54]		
Log Market Access to Messina					-14.52***	-15.93***
					[2.67]	[4.53]
Controls of Column (4) Table 2	N	Y	N	Y	N	Y
Year of First Infection FE	N	N	Y	Y	N	N
Log Market Access to All Cities	N	N	N	N	Y	Y
Observations	124	124	124	124	164	164

Notes: Col. (3)-(4): February is the omitted month. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01.

Table A.5: IV1, IV3 AND CITY GROWTH IN PREVIOUS CENTURIES

Dependent Variable:	Percentage Change in City Population in Period t:					
Period t:	1300-1400		1200-1300		1200-1100	
	(1)	(2)	(3)	(4)	(5)	(6)
IV1: ♯Months btw Oct. 1347 & 1st Infect.	1.30**	1.31*	-1.54	1.24	-0.92	0.46
	[0.52]	[0.77]	[1.01]	[2.29]	[0.61]	[2.04]
Observations	124	124	78	78	54	54
IV3: Log Market Access to Messina	19.22***	14.32†	-14.44	24.58	2.25	29.14
	[6.23]	[9.93]	[19.56]	[30.93]	[9.79]	[35.40]
Observations	164	164	92	92	61	61
Ctrls + Sq. & Cub. Lon. & Lat.	N	Y	N	Y	N	Y
Ctrl for Log Market Access to All Cities	N	N	N	Y	Y	Y

Notes: For our main sample of 165 towns, we regress the percentage change in city population in 1300-1400, 1200-1300 and 1100-1200 on the number of months between Oct. 1347 and the month of first infection in the town (IV1) or log market access to Messina while controlling for market access to all 1,801 cities (IV3). Col. (2), (4) & (6): Controls from col. (4) in Table 2 + squares and cubes of longitude and latitude. Robust SE's: † p<0.15, * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.

Table A.6: ROBUSTNESS CHECKS FOR THE IV3 STRATEGY

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

Regression for Period t:	(1) 1300-1400		(2) 1300-1600	
1. IV3: Messina w/o Controls (IV F = 29; 29)	-1.32***	[0.44]	1.10	[2.14]
2. IV3: Messina w/ Controls (IV F = 10; 11)	-0.90†	[0.59]	-1.41	[2.07]
3. Row 1, Also Controlling for Log MA MENA (IV F = 25; 10)	-1.24**	[0.51]	1.11	[2.40]
4. Row 2, Also Controlling for Log MA MENA (IV F = 25; 2)	-0.91†	[0.59]	-1.38	[2.03]
5. Row 1, Excl. Messina in Log MA to All Cities (IV F = 29; 30)	-1.25***	[0.43]	1.42	[2.31]
6. Row 2, Excl. Messina in Log MA to All Cities (IV F = 8; 9)	-1.00†	[0.65]	-1.52	[2.27]
7. Row 1 But Log Dist. Messina & Avg. Log Dist. All Cities (IV F = 37; 36)	-0.61†	[0.38]	2.23	[2.70]
8. Row 2 But Log Dist. Messina & Avg. Log Dist. All Cities (IV F = 17; 17)	-0.93*	[0.50]	-1.67	[1.42]

Notes: Rows 3-4: Instrumenting by log MA to Messina, controlling for log MA to all 1,801 cities (IV3) and log MA to Middle-East and North Africa (MENA). MA for city i is defined as $MA_i = \Sigma_j P_j / D_{ij}^{\sigma}$, with P_j being the population of town $j \neq i$, D_{ij} the travel time between city i and city j, and $\sigma = 3.8$. Cities j are the largest cities of the MENA in 1300 according to Chandler (1974, 1987): Cairo (450,000) whose port was Damietta, Damietta itself (90,000), Fez (200,000) and Marrakech (100,000) whose port was Ceuta, Istanbul (100,000) which was its own port, and Tunis (75,000) which was also its own port. To obtain travel times, we compute the least cost travel paths to the ports of Damietta, Ceuta, Istanbul and Tunis, respectively, via four transportation modes — by sea, by river, by road and by walking — with the speeds from Boerner & Severgnini (2014). Rows 5-6: We exclude Messina when constructing market access to all cities. Rows 7-8: The instrument is log Euclidean distance to Messina and simultaneously control for average log Euclidean distance to all 1,801 cities. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.

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

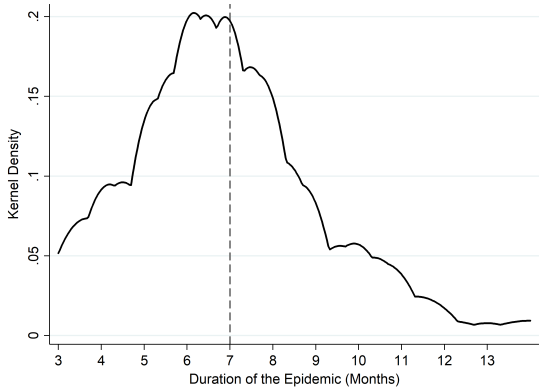
Notes: In rows 2-6, we drop specific countries. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01.

Table A.8: BLACK DEATH MORTALITY AND COUNTRY URBANIZATION, 1100-1850

Dep.Var.:	Absolute Change in Urban Share (%) in Period t:								
	1100-1200	1200-1300	1300-1400	1300-1500	1300-1600	1300-1700	1300-1750	1300-1800	1300-1850
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
β_t	-0.02	-0.01	-0.09	-0.05	0.09	0.09	0.16*	0.25**	0.32**
	[0.05]	[0.02]	[0.07]	[0.05]	[0.05]	[0.07]	[0.09]	[0.10]	[0.13]
Obs.	15	15	15	15	15	15	15	15	15
R2	0.02	0.03	0.15	0.06	0.12	0.10	0.21	0.26	0.30

Notes: This table shows for 15 countries the effect β_t of mortality (%) on the absolute change in the country's urban share (%) for different periods. For each year, the urban share is the population share of all cities above 1,000 inhabitants. The source for the total population data is McEvedy and Jones (1978). The sources for mortality at the country level are mostly Ziegler (1969), Gottfried (1983) and Benedictow (2005). We use as weights country populations 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.

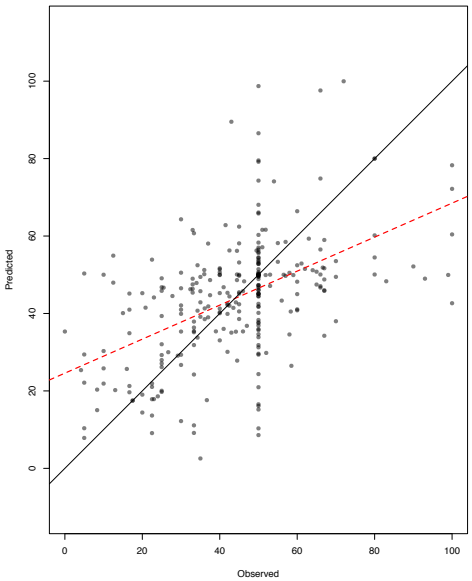
Figure A.1: Distribution of the Duration of the Black Death in Each City.



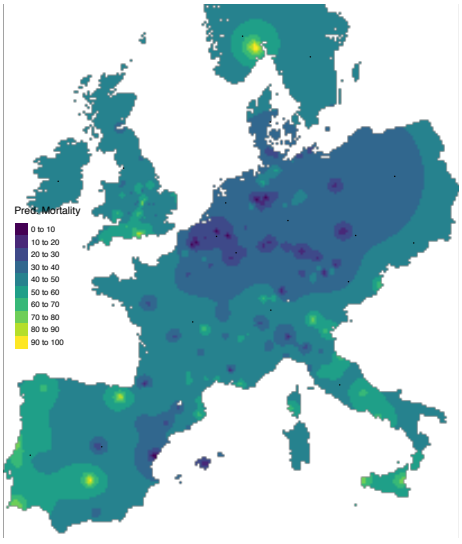
Notes: This figure shows the Kernel distribution of the duration of the Black Death in each city—i.e. the time difference between the year-month of the first infection in the city and the year-month of the last infection in the city (N = 61; mean and median = 7). See Web Appendix for more details on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

Figure A.2: Extrapolated Black Death Mortality Rates

(a) Predicted vs. Measured Mortality

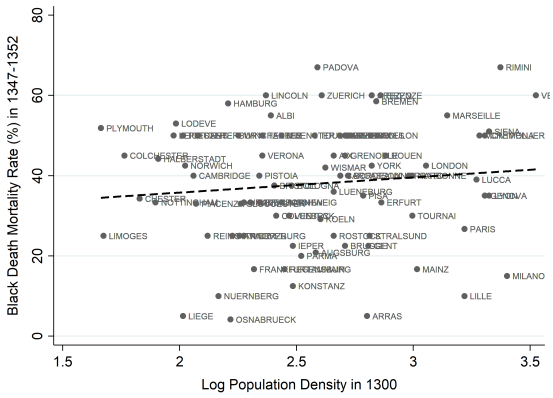


(b) Extrapolated Mortality Rates



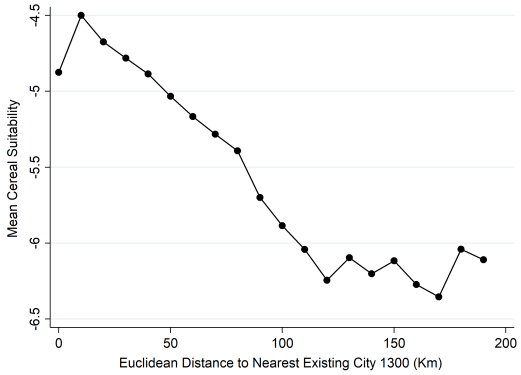
Notes: Figure 2(a) shows for the 274 cities with mortality data the relationship between true mortality and predicted mortality using spatial extrapolation. Figure 2(b) shows the 274 cities and surfaces of extrapolated mortality rates for the rest of Western Europe. See Web Appendix for more details on data sources.

Figure A.3: Black Death Mortality and Walled Density 1300.



Notes: Data on walled density (population ÷ walled area) for 88 cities comes from (Cesaretti et al., 2016). The figure shows there is no relationship between mortality and log walled density for these 88 cities (mort. = 28.2*** + 3.79 log.wall.dens.; R2 = 0.01).

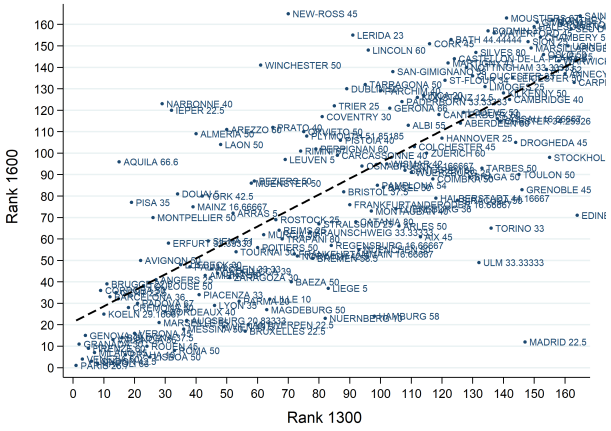
Figure A.4: Cereal Suitability and Distance to Existing Cities in 1300.



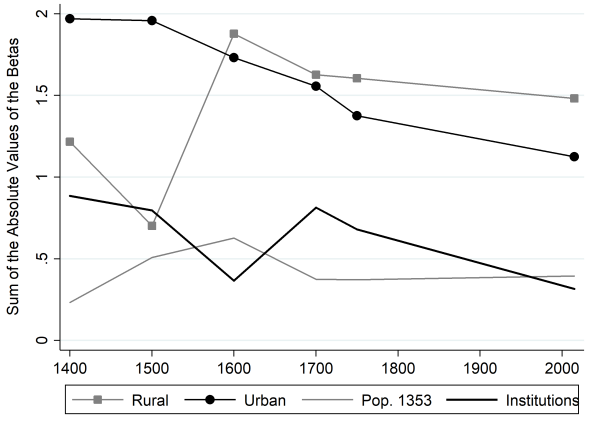
Notes: This figure shows for 200 distance bins of 10 km the relationship between mean cereal suitability (more positive values indicate higher land suitability) and Euclidean distance to the nearest existing city in 1300 (N = 466). Mean cereal suitability is obtained by averaging for each bin cereal suitability available for 15,754 gridcells in the 16 Western European countries of our full sample. See Web Appendix for details on data sources.

Figure A.5: Permutations and Contributions of Fixed Factors to These Permutations

(a) Rank Regressions



(b) Contributions of Fixed Factors



Notes: Figure 5(a) shows for the main 165 cities the relationship between their rank in 1300 (among the 165 cities) and their rank in 1600 (ditto). Figure 5(b) shows for each period from 1300-1400 ("1400") to 1300-2015 ("2015") the sum of the absolute values of the beta coefficients for urban fixed factors (coast, rivers, road intersections, Hanseatic league), rural fixed factors (cereal, potato, pastoral), agglomeration effects (pop. 1353) and institutions (monarchy, state capital, representative body). See Web Appendix for details on data sources.