Bones, Bacteria and Break Points: The Heterogeneous Spatial Effects of the Black Death and Long-Run Growth

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Abstract

The Black Death killed about 40% of Europe's population between 1347-1352. Historical studies suggest that this mortality shock played a major role in shifting Europe onto a path to sustained economic growth. Using a novel dataset that provides information on spatial variation in plague mortality at the city level, as well as a range of controls and various identification strategies based on the spread of the epidemic, we explore the short-run and long-run impact of the Black Death on city growth. We find evidence for *aggregate convergence*. On average, cities recovered their pre-plague population within two centuries. However, there was considerable heterogeneity in the response to the shock, hence *local divergence*. The Black Death led to an *urban reset*: cities with better geographical and non-geographical endowments did relatively well, while other cities collapsed. In particular, our results emphasize the importance of trading networks in explaining urban recovery. Furthermore, the Black Death led to the creation of new cities in areas that were relatively less urbanized before it hit. Our analysis thus suggests that the Black Death may have permanently affected the spatial distribution and aggregate level of economic activity, potentially contributing to long-run growth in Europe.

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

Keywords: Urban Reset; Path Dependence; Multiple Equilibria; Urbanization; Cities; Black Death; Epidemics; Mortality; Little Divergence; Long-Run Growth

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1. Introduction

The Black Death was the greatest demographic shock in European history, and possibly world history: about 40% of Europe's population died in just 5 years. In recent accounts of the Great Divergence, the Black Death plays a crucial role in explaining the economic rise of western Europe. However, to our knowledge, no existing research makes use of city-level data on the demographic impact of the Black Death to study subsequent local economic development across all of western Europe. This is the objective of this paper.

We construct a novel dataset which combines estimates of Black Death mortality, city populations, and numerous geographic, economic and institutional variables to study both the short-run and long-run impact of the Black Death on local economic development. These data allow us to test several theories of the role the Black Death played in the subsequent spatial evolution of economic activity across time. Indeed, several hypotheses identify the Black Death as a pivotal moment in economic history:

Hypothesis 1: Great Divergence. Numerous scholars of the Great Divergence that took place between western Europe and the rest of world have traced the onset of this process to the Black Death (North and Thomas, 1973; Gottfried, 1983; Herlihy, 1997; Epstein, 2000; Pamuk, 2007; Acemoglu and Robinson, 2012).

Hypothesis 2: Structural Change. Recent research argues that the Black Death led to an increase in urbanization in Europe. Voigtländer and Voth (2013b) construct a model with multiple equilibria in which a shock that temporarily raises real wages like the Black Death could have moved Europe from a low to a high income equilibrium. In their model, higher real wages generate greater demand for manufactured goods produced in cities which contributes to higher levels of urbanization. As cities had higher death rates, this produced a 'horsemen effect'—an s-shaped income-death schedule—which enabled Europe to attain higher per capita income in the pre-industrial period.

Hypothesis 3: Institutional Change. Another hypothesis concerns whether the Black Death led to institutional changes. Scholars have argued that the demographic shock of 1347-1352 changed the configuration of de facto political power (North and Thomas, 1973; Brenner, 1976; Epstein, 2000; Cohn, 2003, 2010; Acemoglu and Robinson, 2012). In the wake of the Black Death, stronger states emerged in England, France and later Spain (Ertman, 1997; Gennaioli and Voth, 2015).

Hypothesis 4: Little Divergence. Building on the previous arguments, economic

historians also trace the onset of a Little Divergence within Europe to the Black Death. Scholars argue that as a consequence of Hypotheses 2 and 3, Northwestern Europe rose to economic prominence in the wake of plague (Pamuk, 2007; van Zanden, 2009; Moor and Zanden, 2010; de Pleijt and van Zanden, 2013; Voigtländer and Voth, 2013a). Furthermore, Voigtländer and Voth (2013a) argue that the demographic shock of the Black Death had a differential effect in northern Europe—where the land could be turned over to pastoral agriculture—than in southern Europe where the land was more suited to arable farming. As pastoral farming increased the demand for female labor, this contributed to a labor and marriage market equilibrium in which individuals married late and restricted fertility (the European marriage pattern or EMP); a phenomenon that in turn raised per capita incomes.[1] They argue that this helped give rise to the Little Divergence in incomes that took place between the north and the rest of Europe in the period between 1400 and 1800.

Hypothesis 5: Spatial Reset. We provide evidence that somewhat corroborates the previous hypotheses, but we also highlight new factors and mechanisms that played an important role in European growth after 1300. In particular, we find that there was *aggregate convergence* from the Black Death but *local divergence*. The effects of the Black Death were highly heterogeneous: while some cities did not recover their pre-Black Death populations until the 19th century, the epidemic stimulated city growth in areas that were previously predominantly rural. A fifth hypothesis is thus whether the Black Death led to a more optimal spatial allocation of resources across space.

More precisely, using data for our main sample of 140 cities, we find that between 1300 and 1400, a ten percent higher Black Death mortality rate was associated with a 8.4% fall in urban population. In the long-run, between 1300 and 1750, the overall impact of the Black Death was close to zero in the aggregate. These effects are causal. First, we provide evidence that the spread of the plague and its virulence was due to factors largely exogenous to city growth. Second, we show that the parallel trends assumption is verified in that prior to 1300, there was no difference in growth between cities most affected and those comparatively unaffected by the Black Death. Third, our results are robust to the inclusion of controls for locational fundamentals, increasing returns, institutions, market access, region fixed effects, and contemporaneous events.

[1]The EMP is studied by Hajnal (1965); Macfarlane (1986); Moor and Zanden (2010).

Lastly, several instrumental variable approaches based on the randomness of the timing of infection and distance to the point of first infection suggest that our baseline estimates indeed reflect the causal impact of the plague on urban development.

Although the aggregate impact of the Black Death had already disappeared by 1600, urban recovery was highly heterogeneous until 1750, before the Industrial Revolution. We find evidence that the Black Death stimulated urban development in areas which were previously less developed. This result is consistent with the theoretical predictions of Voigtländer and Voth (2013b) and suggests that a significant share of the impact of the Black Death on subsequent urban development occurred on the extensive margin.

Another major result we find is that many of the factors that were important for growth in the wake of the plague shock were linked to trade. We measure the shock to market access produced by the Black Death and show that this played an important role in mediating urban recovery. There were spillovers from the Black Death such that places that lost market access between 1347-52 recovered less quickly whereas gains to market access after 1352 were positively correlated with population growth. Furthermore, cities rebounded more strongly from the Black Death if they were located on the Atlantic, North Sea or Baltic coast. Cities located in the Hanseatic League grew especially fast after Plague shock. Being located on inland rivers or the Mediterranean coast conveyed less of an advantage. Likewise, cities located close to Roman roads recovered less quickly. Taken as a whole, these results suggest that the European urban network reset in the wake of the Black Death by shifting gradually from the south towards the north of the continent. Our analysis provides novel quantitative evidence that the Black Death was not just a one-off mortality shock: it changed the aggregate level, and spatial distribution, of economic activity in Europe.

In addition to the literature on the role of the Black Death in the Great Divergence and the Little Divergence, we contribute to the literature on shocks and long-run persistence in economic development. A number of important recent additions to this literature have demonstrated that demographic shocks can have long-run impacts (Young, 2005; Acemoglu and Johnson, 2007; Nunn, 2008; Bloom et al., 2014). The Black Death differs from the mortality shocks previously considered in the literature in its magnitude—an overall mortality rate of 40% dwarfs other morality shocks that we are aware of such as the 1919 influenza pandemic which killed between 3-5% of the world's

population or World War Two which killed approximately 3.7%.[2]

The Black Death was also different than other types of shocks associated with warfare or bombings because it was a purely demographic one that left man-made capital untouched. In this respect our setting has some resemblance to papers that study the impact of the Holocaust (Acemoglu et al., 2011) or large scale expulsions of populations. Acemoglu et al. (2011) find that the Holocaust had negative long-term economic effects as Jews comprised a large proportion of the middle class throughout the Soviet Union. Chaney and Hornbeck (2015) find that the expulsion of the Morisco's had a positive impact on per capita income because it made more land available. Similarly, Jebwab et al. (2015) do not find a negative effect associated with the expulsions of European settlers in Kenya.[3] Unlike the expulsion of Jewish or Muslim populations, however, the Black Death did not target a specific group, but affected the entire economy. This aspect of our analysis has important implications for considering the impact of modern pandemic diseases such Aids and malaria which have had a noticeable demographic impact in Africa (Piot et al., 2001; Sachs and Malaney, 2002; Young, 2005; Weil, 2014). It thus connects with a literature on the consequences of both recent diseases (Almond, 2006; Bleakley and Lange, 2009; Alsan, 2015; Gráda, 2015) and the historical impact of pandemic diseases (Findlay and Lundahl, 2006; Bosker et al., 2008b; Alfani, 2013).

Third, our analysis sheds light on the determinants of long-run urban development. This literature finds episodes where large shocks do not change the spatial distribution of economic activity and cases where shocks do lead to urban resets. Prominent theories in economic geography suggest that this depends on whether locational fundamentals (i.e. physical geography), increasing returns (i.e. economic geography) or institutions determine the location of cities. If locational fundamentals dominate then there is a unique equilibrium location for a given set of cities and economic activity will be centered around these locations irrespective of demographic shocks, no matter how

[2]Other major mortality shocks include the Ukrainian and Chinese famines of the mid-twentieth century. Recent estimates for mortality in the Ukrainian famine of 1931-1933 are around 10% of the population (Snyder, 2010). Estimates for the Chinese Great Famine range from 2.5 to 6.8% of the population (Meng et al., 2016). During the Bengal famine of 1943 about 6.6% of Bengal's population died. The worst genocides in recent history are responsible for comparable losses of life. About 11% of Rwanda's population was killed in 1994 and 25% of Cambodia's population during the rule of Pol Pot. Again, these death tolls are significantly smaller as a fraction of the population than the Black Death.

[3]Studies of the impact of expulsions of Jews from Nazi Germany include Akbulut-Yuksel and Yuksel (2015) on human capital; Waldinger (2010, 2012) on scientific output. These studies are less directly comparable to ours as the numbers involved were much smaller.

large they are. But, in the presence of increasing returns, multiple equilibria may exist and a large enough shock can cause the spatial system to reset (e.g. Krugman, 1991a,b).

Davis and Weinstein's (2002) seminal study of Japanese urban development in the aftermath of World War 2 provides evidence for the importance of locational fundamentals. In particular they show that after experiencing complete destruction, both Hiroshima and Nagasaki returned to the same relative positions in Japan's distribution of cities within 20 years. Similarly, Glocker and Sturm (2014) and Miguel and Roland (2011) study the wartime destruction of Germany and Vietnam respectively, to test how these shocks affected the relative ranking of cities. Miguel and Roland (2011) find rapid rates of recovery suggesting a high degree of persistence in urban location. Maloney and Caicedo (2015) also find a high degree of persistence in the location of economic activity in the New World between the precolonial period and today.

Other research, however, demonstrates the importance of increasing returns in enabling local shocks to have permanent spatial effects (Bosker et al., 2007, 2008a; Redding et al., 2011; Bleakley and Lin, 2012). For example, Rauch and Michaels (2013) contrast England, where the urban network was reset after the Fall of the Roman Empire, with France, where Roman city locations remained settled throughout the early middle ages. They argue that the locations of French cities may have been shaped by path dependence and hence impeded subsequent economic development. In England, however, the urban network was reset allowing English cities to relocate along coasts and rivers that were more beneficial for economic activity.

There is also evidence that institutions play a role in determining how shocks affect economic outcomes and particularly urban development. Acemoglu et al. (2005) show that the opening up of the Atlantic trade with the New World stimulated economic development in those countries where the power of the sovereign was initially weak (England, the Netherlands) but not where he was initially powerful (Spain and Portugal). Campante and Glaeser (2009) argue that the contrasting developmental paths of Buenos Aires and Chicago in the twentieth centuries can be partly explained in terms of differing political institutions. Nunn and Puga (2012) demonstrate that in the presence of an extractive institution like slavery, 'bad' geography—in this case ruggedness—could be beneficial in sub-Saharan Africa. Finally, Dincecco and Onorato (2015) study the effects of war in stimulating urban development in preindustrial Europe.

The structure of the remainder of the paper is as follows. Section 2. describes a conceptual framework of the effect of the Black Death on the aggregate level and spatial distribution of economic activity. Section 3. describes our data and provides background information concerning the Black Death. In Section 4. we report our baseline results. Sections 5. and 6. demonstrates that city-level recovery was highly heterogenous, explores what factors drove city growth in the aftermath of the Black Death, and provides evidence that there was a 'spatial reset'. Section 7. concludes.

2. Conceptual Framework

Set-up. To see how the plague could have modified the aggregate level and shaped the spatial distribution of economic activity, consider an economy that comprises three locations: $i \in \{A, B, C\}$ where A and B are urban locations (i.e. cities) while C is a rural location (i.e. a set of villages). Denote the total population of the country by P and the population in each location by P_i. The urbanization rate is defined as $(P_A + P_B)/P * 100$.

Malthusian dynamics The Black Death was a huge epidemiological shock to the economy. The overall mortality rate across Europe was 40%. Given the Malthusian nature of the economy, the population losses generated sharp rises in wages. High wages, and hence incomes for workers, increased the demand for manufactured goods[4]. Since manufacturing was centered in urban locations this increase in demand translated into higher urban wages which attracted migrants from the countryside. As a result, the Black Death shock led to higher rates of urbanization across Europe.

Spatial equilibrium. To determine where this aggregate increase in urbanization took place, and thus the increase in population in each urban location, we need to specify various characteristics of the urban locations. In equilibrium, individuals migrate between locations until utilities are equalized. We will only consider net urban wages in the utility function, and thus abstract from the contribution of urban amenities to utility and location decisions. Net wages are equal to marginal productivity in each location. The two urban locations differ in the following respects. Productivity (and thus the net wage) in city i is θ_i and is a function of city locational characteristics and city population. City productivity is increasing in city population up to a threshold $\overline{P}$.

[4]This is consistent with a model with a subsistence constraint or Stone-Geary preferences as in Voigtländer and Voth (2013b).

This captures the influence of net increasing returns on city wages and city growth. City locational characteristics are fixed and are denoted by F_i.

Suboptimal pre-plague urban equilibrium. We consider the case where pre-plague population in city A is greater than $\overline{P}$ but pre-plague population in city B is below $\overline{P}$. We assume that locational characteristics in location B are superior to those in location A so that $F_B > F_A$, but the strength of increasing returns is larger than those of locational fundamentals so that in the pre-plague equilibrium $P_A^* > P_B^*$.[5] Here, the distribution of urban population is suboptimal, since overall productivity would be higher if people moved from city A to city B. However, this suboptimal equilibrium is stable because no one in city A has an incentive to individually move to city B. Enough people would need to simultaneously move for the optimal equilibrium to be realized.

Post-plague urban equilibrium with symmetrical population shock. In the aftermath of the Black Death the populations of A, B, and C all fall dramatically. For illustrative purposes we focus on the case where the decline in population is similar across locations and that the severity of the shock is sufficient to reduce population in location A below $\overline{P}$. In the post-plague period population growth in locations A and B is driven by rural to urban migration rather than by natural increase.[6] Thus people from location C choose which urban location to migrate to. Previously, A could offer higher wages for a given level of population than location B due to its relatively higher level of population and thus increasing returns. The plague wiped out some of this advantage. Now city B can offer higher wages for a given level of population than city A because it is better located with respect to locational fundamentals. Hence as urbanization increases in the wake of the plague, we expect the location of economic activity to shift from city A to city B. This is what we mean by an urban reset. Therefore, the plague may have led to a spatial reset: (i) from villages to cities, and (ii) from some cities to other cities.

Comparisons with the existing literature. Our empirical framework will allow us to test both the Voigtländer and Voth (2013b) prediction concerning the level of urban

[5]The source of increasing returns might be due to being located within an *old* trade network or perhaps the city used to be a political capital. In such a situation, city A may remain an attractive urban center due to increasing returns even though the benefits from being in the old trade network or at one point being a political capital have long since disappeared. In contrast, city B does not benefit from these locked in advantages, but it is better located with respect to *current* trade networks.

[6]This is due to higher steady-state mortality rates in cities where sanitation and disease were always higher than in the countryside up until the epidemiological revolution of the late 19th century. We also assume that people did not significantly increase their fertility rates following the plague.

development and predictions about the relative importance of increasing returns and spatial amenities in determining the location of economic activity. Our historical setting and dataset confer several advantages with respect to the existing literature. One major contribution of our paper will be to show that the Black Death shock was exogenous at a local level. Furthermore, by studying a shock that killed people but did not destroy physical capital, we have an ideal framework to identify the impact of a demographic shock in a Malthusian environment. Finally, our analysis allows us to indirectly speak to the 'optimality' of urban reset. Rauch and Michaels (2013) argue that the persistence of the Roman network in France was inefficient. One problem with this claim is that there are costs of switching to a new urban network and these may be high. The old network may have been inferior relative to a possible new one but, in the presence of positive coordination costs, it may not have been efficient to change the location of cities. The Black Death, by contrast, was a sufficiently large shock that it could have led to a coordinated relocation of urban activity. And while there is no objective metric to characterize the optimality of an urban network, our evidence suggests that the new cities that emerged may have done so in better locations.

In addition to identifying the effect of the Black Death on the level and distribution of economic activity, we will also investigate whether the plague affected the pattern of urban activity via its effect on institutions. Acemoglu et al. (2005) show that the opening up of the Atlantic trade with the New World stimulated economic development in those countries where the power of the sovereign was initially weak (England, the Netherlands) but not where he was initially powerful (Spain and Portugal). Campante and Glaeser (2009) argue that the contrasting developmental paths of Buenos Aires and Chicago in the twentieth centuries can be partly explained in terms of differing political institutions. Nunn and Puga (2012) demonstrate that in the presence of an extractive institution like slavery, 'bad' geography—in this case ruggedness—could be beneficial in sub-Saharan Africa. Finally, Dincecco and Onorato (2015) study the effects of war in stimulating urban development in preindustrial Europe. Richardson and McBride (2009) show that the Black Death led to organizational and religious changes while Acemoglu and Robinson (2012) argue that the Black Death was a critical juncture that gave rise to institutional change in western Europe (see *Hypothesis* 3). In our regressions, we will control for various measures of political institutions in medieval

and early modern Europe including whether a city was located in a monarchy, republic and its level of parliamentary activity. We will test whether these institutional variables interacted with the plague shock to either facilitate or impeded urban development.

3. Data and Background

3.1. Data

Data on Black Death mortality come from Christakos et al. (2005) who compile information from a wide array of historical sources.[7] These data yield estimates of mortality for 263 localities. Of these, we can match 140 mortality rate locations to our city database. Therefore, we have 140 cities for which we have an estimate of plague mortality and population in 1300. We have a percentage estimate of the mortality rate for 89 of these 140 cities. For example, Florence had an estimated mortality rate of 60%. In other cases the sources report more qualitative estimates (e.g. that 'about half' or 'at least half' of the population died) in which case we code our estimate as 50% or that the city was 'desolated' or 'abandoned' in which case we attribute a mortality rate of 80%. Figure 1 shows the spatial distribution of the 140 cities in our main sample as well as their reported mortality rates. Further details on how these data were assembled are provided in the Web Appendix.

Our main source of urban population data is the Bairoch (1988) dataset of city populations. The Bairoch dataset reports estimates for 1,797 cities between 800 and 1850. It provides estimates for every century up to 1700 and then for each fifty year interval up to 1850. We use 1,792 of these cities as 5 cities, in northern Scandinavia, and in the remote West Atlantic, cannot be matched to the GIS data that we employ to create our controls. 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 by a range of scholars studying premodern urbanization and economic development (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 a consensus of historians have provided revised estimates of the

[7] We verify these data by consulting Ziegler (1969), Russell (1972), Pounds (1973), Gottfried (1983), and Benedictow (2005).

population of particular cities, including Bruges, Paris, and London. Specifically, we supplement the Bairoch (1988) dataset using several sources for pre-plague population including Chandler (1974, 1987), Nicholas (1997), and Campbell (2008).[8] In our regressions we will use both the corrected dataset and the original Bairoch dataset.

We group our control variables into three categories: (1) measures of locational fundamentals, (2) factors that might generate increasing returns, and (3) institutional variables. We consider locational fundamental to be primarily related to the surrounding physical geography of city that could affect its success. We create the following geographic variables: a dummy variable for whether a city is within 10 km of a coast or a major river; every city's elevation in meters; the suitability of the land surrounding a city for cereal or pastoral agriculture; longitude and latitude; and the average growing season temperature between 1500-1600 (the earliest data for which estimates of the level of temperature exist). Following Nunn and Qian (2011) we will also control for potato suitability in some regressions.

Increasing returns can stem from a variety of sources. First, there are preexisting trade routes. To capture the importance of these we create measures of market access (see Eaton and Kortum, 2002; Donaldson and Hornbeck, 2016; Storeygard, 2016). We will use the market access measures in two ways. First, we will show that Black Death mortality rates were unrelated to market potential. Second, we will investigate how the plague could affect a cities growth rate through the spillover effects it generated by altering overall market access. We also will use several other measures of trade potential including a dummy variable if a city belonged to the Hanseatic league (Dollinger, 1970) or was within 10 km of a medieval (land) trade route or trade route intersection (Shepherd, 1923). A second source of increasing returns stems from prior investments in physical capital which may generate agglomeration effects. Previous research has shown that the Roman road network remained the major road network throughout medieval Europe (Bosker et al., 2013). We include a dummy if a city was within 10 km of a major Roman road, any Roman road or an intersection of either a major or any Roman road (McCormick et al., 2013). Thirdly, human capital may be another

[8]The historical populations of Paris and London in 1300 are now considered to have been higher than Bairoch's estimates. We use the figure of 228,000 for Paris and 60,000 for London. On the other hand, the population of Bruges is now thought to be smaller than the number given by Bairoch. We assign it a population of 12,000 in 1000, 15,000 in 1100, 25,000 in 1200 and 35,000 in 1700. Further details are confined to the Web Appendix

source of agglomeration affects as demonstrated by Dittmar's (2011) study of the effect of the printing press on city growth rates. Therefore we include a dummy variable for whether a city had a medieval university (Shepherd, 1923). Lastly, we control for log city population in 1300 as this may also influence subsequent city growth. If there are agglomeration effects and these dominate congestion effects, then we would expect larger city cities to grow faster than smaller cities. If, on the contrary, urban growth is limited by a fixed factor of production as Dittmar (2015) suggests was the case then we would expect the coefficient on log population in 1300 to be negative.

Following De Long and Shleifer (1993), Acemoglu et al. (2005), and others we also expect institutional factors to affect city growth in preindustrial Europe. To measure institutional differences between European cities we assign each city to its political jurisdiction in 1300 based on the maps provided in Nussli (2011). We also use modern political boundary fixed effects in our analysis as a robustness check. Relying on coding by Stasavage (2014) and Bosker et al. (2013), we distinguish between cities that were located in monarchies such as England and France and autonomous cities which include either city republics such as Florence and Venice or cities which had de facto self-governance such as Lübeck. These were the major distinctions in types of polities in the medieval period. The historical literature suggests that autonomous cities adopted policies that were more conducive to trade and commerce (Stasavage, 2014). However, there is also evidence that city states like Venice and Florence imposed high taxes and extractive policies on the surrounding countryside (Epstein, 2000). In addition, since administrative centers may have followed different development paths, we code whether a city was a political capital in 1300 using Bosker et al. (2013).

Another source of potential heterogeneity for which we must control are major post-plague shocks to cities. While the Black Death of 1347-1352 was, by far, the most severe mortality shock, the plague did periodically return to cities up until the eighteenth century. As such, we control for 5,630 plague recurrences using data from Biraben (1975).[9] We also control for two instances in which a city was sacked or had a significant fire between 1300 and 1600 using data collected from Wikipedia. In addition to enduring the Black Death, the fourteenth century also witnessed an intensification in warfare which may have affected economic growth as suggested by Voigtländer and

[9]We extract a digitized version of this data data taken from Schmid et al. (2015).

Voth (2013b). As it is impossible to obtain precise numbers on mortality due to warfare for the medieval period, we follow recent scholarship in collecting data on the location of conflicts (e.g. Dincecco and Onorato, 2015; Iyigun et al., 2015). Our main source is Wikipedia's list of all battles that took place between 1300 and 1600. For each battle we assign a geo-coordinate based on either the location of the battle or the location of the nearest town or city mentioned in the entry. We exclude naval battles and conflicts which cannot be located (such battles were typically extremely minor). We then create a dummy variable that measures the distance to major battles between 1300-1350 and 1350-1600. This provides a proxy for both the disruption caused by warfare to nearby areas and for the 'safe-harbor' effect identified by Dincecco and Onorato (2015) that might have led to urban growth as rural citizens moved to cities for greater security.

Finally, in order to conduct robustness checks and to explore the impact of the plague on the emergence of new cities we predict mortality rates for those cities for which we have no explicit estimates based on spatial extrapolation techniques using the cities for which we do have data. These mortality extrapolations rely on the assumption that there existed spatially correlated unobservables which affected plague severity. Using this assumption we use cross-validation techniques to calibrate an inverse distance weighted function of known mortality rates to create a two-dimensional surface of predicted plague mortality rates. We then extract predicted mortality rates for all Bairoch cities from this surface. Details of the construction of these data are contained in the Web Appendix Section 1.4.

3.2. Epidemiological Background

The Black Death arrived in Europe in 1347. Over the next five years it spread across the continent killing between 30% and 50% of the population.[10] Death rates were

[10]Conventionally the death rate was estimated at 1/3 of Europe's population. More recent studies suggest that the overall death rate was considerably higher than this. Benedictow (2005) argues for a mortality rate as high as 60%. This higher estimate, though controversial, has not been rejected by other scholars. One reviewer notes that 'Benedictow's mortality estimates may eventually come to be regarded as the standard, in spite of readers' doubts that the remarkably similar die-off across regions is due in part to rejecting data indicating lower figures through source criticism. The estimates are internally consistent with his assessments of plague case-fatality (circa 80 percent, p. 350) and prevalence. If plague lethality is over 50 percent in modern populations, then 80 percent is not implausible for medieval times, considering the nutritional stresses of the fourteenth century' (Noymer, 2007, 624). Recent research by Lewis (2016) confirms a high estimate for plague mortality in England.

comparable in the cities and in rural areas and, in general, historical accounts are unable to explain variation in mortality rates (Ziegler, 1969; Gottfried, 1983; Theilmann and Cate, 2007; Cohn and Alfani, 2007).[11] Black Death mortality was not correlated with city size or population density. To illustrate, Venice had extremely high mortality (60%) while Milan escaped comparatively unscathed (15% mortality).[12] Highly urbanized Sicily suffered heavily from the plague. However, equally urbanized Flanders (modern-day Belgium) had relatively low death rates, while the more rural northern Netherlands was devastated. Southern Europe and the Mediterranean was hit especially hard, but so were the British Isles and Scandinavia. Consistent with these stylized facts, Figure 2(a) illustrates the lack of a relationship between Black Death mortality rates and city population in 1300.

The spread of the plague was rapid and its precise trajectory was largely determined by chance. As we confirm in our formal analysis, the only systematic predictor of plague virulence was latitude, as the plague spread from southern to northern Europe. In many respects, however, the spread of the plague had a considerable random component. For example, it was largely coincidence that the plague spread first from Kaffa in the Black Sea to Messina in Sicily rather than elsewhere as the ships carrying the plague could have stopped at other ports in the Mediterranean. A ship from Kaffa did arrive in Genoa but as all onboard had already died, it was not a direct source of infection. Similarly, it was partly coincidental that the plague spread first from Messina to Marseilles. The early arrival of the plague in Marseilles ensured its speedy transmission through much of western Europe in the year 1348.[13] When it arrived in a country it moved quickly. For instance, it arrived in southern England in June 1348 in Dorset in the southwest of the country. It reached London by November. It hit the north of England by early 1349, peaking in that part of the country in the summer of 1349 (Theilmann and Cate, 2007).

[11] Prior to the Black Death there had been no major outbreak of epidemic disease for several centuries and as a result neither the medical profession nor political authorities were able to respond effectively.

[12] There is no indication that variation in sanitation or hygiene explains this pattern. Gottfried notes 'it would be a mistake to attribute too much to sanitation. The failure of Venice's excellent sanitation to stem the deadly effect of the plague has been discussed' (Gottfried, 1983, 69).

[13] For example, Benedictow notes that 'The surprisingly early invasion of Marseilles by the Black Death may have affected profoundly its pattern of strategic advance in Southern and Western Europe, and also, as we shall see, the invasion of the British Isles. It was another momentous event in the sinister history of the Black Death ... From this commercial centre, communication lines radiated both by sea and by land. This city was an excellent bridgehead for the Black Death's offensive against south-western and Western Europe' (Benedictow, 2005, 73).

The Black Death affected all segments of the population.[14] Medical knowledge was rudimentary and ineffective: Boccaccio, for instance, wrote that 'all the advice of physicians and all the power of medicine were profitless and unavailing' (Boccaccio, 2005, 1371). Individuals, regardless of wealth, were largely unable to protect themselves from the disease. Institutional measures of prevention were nonexistent: the practice of quarantine was not employed until later in the fourteenth century.[15]

3.3. Historical Background

Figure 3(a) presents estimates of total population and urbanization for the 17 (modern) countries which contain the cities we use in our main analysis. Europe only regained its pre-plague population by around 1600. Urbanization, in contrast, rose in the aftermath of the Black Death from around 7% to 10% by 1400, and 12% in 1600. The source of the increase in urbanization is evident in Figure 3(b) which depicts the evolution of Europe's urban population between 1100 and 1600. Approximately 50% of the acceleration in urban growth after 1353 is attributable to the growth of cities that either did not exist or were below the threshold of 1,000 inhabitants in 1300. These aggregate statistics suggest that the emergence of new urban centers is important in explaining the growth in urban population relative to total population between 1353 and 1600.

Figure 4 illustrates another important stylized fact concerning urban recovery from the Black Death which is that it was highly heterogenous. Some cites recovered quickly and even surpassed their previous population rank in the urban network whereas others stagnated or simply disappeared altogether. The Figure illustrates the distribution of city sizes at four different times relative to 1300. We normalize all city populations to 100 in 1300. The distribution for 1353, immediately after the main plague shock, shows the average population of cities declined by about 40%. By 1400, on average, the urban population of Europe was already well on its way to recovery, however, the variance in the distribution suggests that some cities recovered much more quickly than others. The distributions for 1500 and 1600 only reinforce these trends. By 1600, average city size has

[14] It is commonly asserted that the Black Death killed indiscriminately. Recent research examining the skeletons from plague pits in London, however, suggests that on average plague victims were more likely to be older, frailer, or less well-nourished (DeWitte and Wood, 2008).

[15] The term quarantine was first used in the city of Ragusa, part of the Venetian empire in 1377. It was adopted as a standard policy by Venice in 1423 (Gensini et al., 2004, 257).

completely recovered, however, many cities had not recovered their 1300 populations by 1600 while other cities rapidly expanded in these centuries.

4. Aggregate Convergence and Local Divergence

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

$$\%\Delta \text{Pop}_{i,t} = \alpha + \beta_t D_{i,1347-52} + \mathbf{X}_i'\delta + \gamma_c + \epsilon_{i,t} \tag{1}$$

where $\%\Delta \text{Pop}_{i,t}$ is the percentage population growth (%) in city i over period t-1 to t, and $D_{i,1347-52}$ is a measure of the city-level cumulative mortality rate of the Bleak Death (%) between 1347 and 1352. $\mathbf{X}_i$ is a vector of city-specific controls.

4.1. Short-Run and Long-Run Effects: 1300-1750

Short-Run (1300-1400). Column (1) of Table 1 measures the short-run impact of the plague in 1300-1400. The coefficient from our baseline OLS regression is -0.84***. This should be interpreted relative to the immediate effect for the period 1347-1352 which is -1 by construction. A value greater than -1 in magnitude might occur if, in addition to the death caused by the plague, people disproportionately left cities between 1353-1400 that were struck harder by the disease. The coefficient that we obtain suggests that a 10% higher mortality rate was associated with an 8.4% smaller population growth rate between 1300 and 1400. This suggests that there was little recovery in population in the decades directly following the onset of the plague. This finding is also consistent with the observations of historians like Nicholas (1999, 99) and Hohenberg (2004, 14) who write of an 'urban crisis' in the wake of the Black Death.

Long-Run (1300-1750). Columns (2)-(6) examine the impact of the Black Death in subsequent centuries. There was recovery during the periods 1400-1500 and 1500-1600 (columns (2)-(3)). The coefficient we obtain in Column (4) of -0.13 is not significantly different from zero and demonstrates that by 1600 there was complete convergence to 1300 populations in the aggregate. The coefficient then slightly increased to 0.09 and 0.44 by 1700 and 1750 respectively (columns (5)-(6)). In most of the following analysis we focus on the period between 1300 and 1600 to minimize contamination

from potentially confounding events such as the discovery of the Americas (Acemoglu et al., 2005) or the introduction of the potato (Nunn and Qian, 2011).

In their analysis of Japanese city growth, Davis and Weinstein (2002) find full convergence from the shock of World War 2 by 1960. Their coefficient of interest is precisely estimated and the standard errors associated with their estimates are small suggesting that convergence occurred at the city level. For example, Hiroshima and Nagasaki were ranked 7 and 11 respectively in the distribution of Japanese cities in 1940. They fell to 26 and 25 in 1945. By 1960 their relative ranking was again 6 and 11.

In contrast, we find long-run convergence in the aggregate but we do not obtain convergence at the city level. The coefficient we obtain in Column (6) is not precisely estimated. It is not just that the coefficient we obtain is small, but that the confidence intervals for Column (6) and associated standard errors are very wide. The estimate in Column (4), for example, suggests that within the 95% confidence band some cities experienced no recovery at all (coefficient of -1.1) whereas others more than made up for the shock (coefficient of 0.9). Overall, Columns (2)–(6) of Table 1 suggest that the experience of European cities after the Black Death was highly heterogenous.

4.2. Investigating Causality

To investigate the causal relation between Black Death mortality rates and city growth we introduce a range of identification strategies.

Biases. For the short-run, a downward bias is more problematical than an upward bias as we then overestimate the effect of the plague (the true effect in 1300-1400 must be higher than -0.84). 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 problematical as we then overestimate how fast cities recover (the true effect in 1300-1600 is lower than -0.13). We discuss below various of these potential biases and how our identification strategies minimize them.

Exogeneity Assumptions. As discussed in Section 3.2., there is evidence that the spread of the plague and its virulence was due to factors largely exogenous to city growth. The Black Death could have followed a very different path over the course of five years, and its overall virulence could have been higher or lower as a result. The

particular path taken by the plague from Kaffa to Messina and then to Marseille was contingent on the fact that the ship carrying infected rats from Kaffa could have first stopped in Cyprus, Brindisi or Venice. Similarly, had the plague not arrived in Marseille when it did, the diffusion of the plague to the rest of Europe would have been delayed and the resulting mortality may have been reduced. These facts give us ex ante reasons to view plague mortality as exogenous. We now test this more systematically.

Parallel Trends. The parallel trends assumption is satisfied. Columns (7)-(8) of Table 1 indicate that prior to 1300, there is no difference in growth between cities most affected and those comparatively unaffected by the Black Death. The cities in our sample that were hit hard by the Black Death and those that escaped comparatively unscathed were following similar development paths prior to the 14th century.[16]

City Characteristics and Mortality. In Table 2 we show that Black Death mortality is uncorrelated with observable city-level characteristics. Variation in mortality rates cannot be explained in terms of locational fundamentals (see Column (1)), nor are they accounted for by our measures of increasing returns (Column (2)), or by institutional factors (Column 3). Nor are these controls jointly significant (Column (4)). The only variable that has explanatory power is latitude, which has a significant negative relationship with mortality. This reflects the fact that the Black Death hit southern Europe first, and was, in general, more virulent in the early years of the epidemic.[17]

Market Access and Mortality. Table 2 suggests that proxies for trade networks were not associated with Black Death mortality. To ensure that our regressions are not picking up a potentially spurious relationship between the Black Death and subsequent city growth based on access to trade, we construct a measure of market access for the year 1300. We then test whether market access mattered for the intensity of the plague.[18]

Following Donaldson and Hornbeck (2016), we calculate log market access for city i as $log(\text{MA}_i) = log(\sum_j \text{N}_j \tau_{ij}^{-\sigma})$, where N_j is the population of city j in 1300 (using the 473

[16]Note, the parallel trends assumption is also verified if we: (i) test the effect for the overall period 1100-1300 (N = 59), or (ii) test the effect for the period 1000-1100 (N = 56).

[17]Note that if city level characteristics were entirely uncorrelated with the Black Death, then the coefficient of determination would be zero. The R-squared in Column (1) is higher than this. However, it falls to 0.07 when we exclude latitude and temperature (whose correlation with latitude is 0.89). If we re-run the specification in Column (4) while dropping latitude and temperature the coefficients of the other controls also remain insignificant and the R-squared decreases to 0.16. The R-squared does not entirely decrease to 0 because some of the remaining variables are still somewhat correlated with latitude.

[18]However, if cities trading more grew inherently faster, and were also infected earlier and more virulently by the Black Death, this should only lead to an upward bias of the short-run effect.

existing cities then) and τ_{ij} is the travel time from city i to city j in 1300. This travel time should reflect the time it took for merchants and other travels to move from city i to city j (and hence the potential time it took for infected rats, fleas and human bodies to be transported from city i to city j). There were four transportation technologies then, and we use the travel speeds estimated specifically for the spread of the Black Death by Boerner and Severgnini (2014) (*Speeds 1*): porters (speed normalized to 1), roman or medieval roads (0.50), rivers (0.24) and seas (0.18). To calculate τ, we divide Europe into 5x5 km cells and then assign the least cost of transport to each of these cells. We then apply Djikstra's algorithm to calculate the cumulative cost of taking the least cost travel path between each city dyad. Lastly, we set $\sigma = 1$. We provide further details on the construction of the market access variable in the Web Data Appendix.

Table 3 reports the results of regressing market access in 1300 on plague mortality rates. We include a city's own population (divided by 5 km and using the speed of porters within the city to account for city congestion) in our calculation of market access. Otherwise, small towns in the outskirts of large cities like London and Paris receive much larger market access values than do the large cities themselves. In addition, what matters for receiving the plague before other cities is to what extent the city is "connected" to other cities, so the size of the city itself proxies for the number of merchants and other travels that may come to the city.[19]. Row 1 of Table 3 shows that market access in 1300 is unrelated to mortality rates during the Black Death. This remains the case when we include all other controls of Table 2 (row 2); differentially weight the population of a city's own population using the speed of roads rather the speed of porters (row 3); exclude a city's own population from our calculation of market access (row 4); use $\sigma = 3.8$ or $\sigma = 2$ (rows 5-6); and use different measures of travel speeds (*Speeds 2*, from McCormick (2001), row 7) or transport costs rather than travel speeds (*Costs 1*, from Bairoch (1990), row 8). Transport costs reflect the time it took to move grain and other bulky goods from city to city (see details below Table 3).

Controls. Table 4 further investigates the causal effect of the Black Death by introducing control variables. Row 1 presents our baseline estimates for comparison (the short-run effect in 1300-1400 in column (1) and the long-run effect in 1300-1600 in

[19]Note that the correlation between log market access including own city size and log city size in 1300 is only 0.42. The market access variable thus captures something different than purely city size.

column (2)). In row 2 we drop cities that are in the top or bottom 10% of the mortality distribution to ensure that our results are not driven by outliers that may have had very high or very low mortality rates for specific reasons related to future city growth. Likewise, row 3 excludes cities that are said by historians to have adopted extraordinary measures to prevent the spread of the plague such as Milan (Ziegler, 1969) or cities that are reported as possessing natural springs and baths such as Bath and Nuremberg.[20]

To deal with potential sources of a downwards bias for the short-run effect and an upward bias for the long-run effects, we show in rows 4-7 of Table 4 that our results are robust to the inclusion of all the controls listed in Table 2. For example, (i) cities with limited access to food supply (whether due to poor soils or bad transportation networks) grow slower. Then, their residents being malnourished, the Black Death kills more; (ii) Cities with more physicians and better trained bureaucrats grow faster. These cities are also better able to contain the plague; and (iii) Cities caught in a war grow less. If residents are already injured and malnourished, the Black Death kills more. That is why it may be important to control for (i) cereal suitability, potato suitability and whether the city is along the coast, a river, a Roman road, a medieval land route or belongs to the Hanseatic League, (ii) whether the city has an university, and (iii) the number of battles that took place within 100 km of the city in 1300-1350, respectively.[21]

Results hold when adding extra controls (see rows 8-13). In row 8, we additionally control for market access as defined in Table 3. Another factor influencing both the spread of disease and subsequent recovery is access to sea-based trade. Since the Black Death spread out from the Mediterranean, we distinguish between three coastlines in the regression results we report in row 9: the Atlantic, the Baltic and the Mediterranean.

The plague reoccurred throughout the centuries following the Black Death. This could be a potential source of bias if subsequent plague outbreaks were correlated with the initial pandemic. In Row 10 we use data from Biraben (1975) to create a dummy for plague reoccurrence in the period of interest 1350-1400 or 1350-1600, respectively.[22]

[20] Note that we will also control for whether a city was within 10km of an aqueduct (rows 5 and 7).

[21] We decided not to list all potential sources of downward biases for the short-run effects and upward biases for the long-run effects. Note again that sources of upward biases for the short-run and downward biases for the long-run are less consequential for our analysis. Here, mortality could be higher in warmer areas, less isolated areas and bigger cities, since these characteristics could affect the distribution of humans, rats, fleas and ultimately bacteria. However, we do not find any significant correlation with mortality rates (see Table 2). Likewise, we control for these factors in rows 4-7 of Table 4.

[22] Results hold if we drop the cities with the highest number of plague reoccurrences or directly control

We control for military conflict in rows 11-12. Historians note that 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)) but, at the same time, in the longer-run as Voigtländer and Voth (2013a) and Dincecco and Onorato (2015) argue, warfare intensified and became a factor contributing to increases in urbanization in late medieval and early modern Europe.[23] Cities that were sacked or burned might experience a fall in population that could have had lasting consequences. On the other hand, cities that were burned could be rebuilt along more modern lines, which could have facilitated future growth. We thus collected data on these variables, and show in rows 11-12 that results hold when using them as controls.

For the period 1300-1400, our estimates might be downward biased if higher plague mortality was associated with widespread violence against Jews (Nohl, 1924; Cohn, 2007; Voigtländer and Voth, 2012; Finley and Koyama, 2016). Other research, however, indicates that presence of a Jewish community was positively associated with city growth (Johnson and Koyama, 2016; Pascali, 2016). If, as some historians have suggested, Jews had better hygiene practices than Christians, it is possible that mortality rates might be lower in cities with large Jewish communities. However, results are unchanged when we include dummy variables for whether a city had a Jewish community between 1300 and 1350 and whether there was either a pogrom or expulsion of a Jewish community in the city between 1347 and 1352 (row 13).

Spatial Fixed Effects. Another approach to control for the presence of unobservables is to use fixed effects. In row 14 we employ 17 modern country fixed effects 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 15 we assign a separate dummy variable to each of the 57 independent polities in our dataset using the information on borders contained in Nussli (2011). The sheer number of state raises a potential problem for our analysis, however, as many of these polities were city states or small principalities with only a single major city.

for the number of reoccurrences (see Web Appx. Table A.1 rows 2-3). We also find that subsequent plagues were not correlated with Black Death intensity (results not shown but available upon request).

[23] Relatedly Cervellati et al. (2014) show that in the postwar period disease outbreaks are associated with an intensification of civil conflict. Arguably the respective numbers of battles and soldiers involved in these battles may be a better proxy for the devastation associated with warfare. Our results are unchanged if we also use these controls (see Web Appx. Table A.1 rows 4-5).

As a result, there is insufficient variation and our estimates become less precise. The effect we obtain is almost significant at the 10% level (p-value = 0.107) and similar in magnitude. Our preferred fixed effects estimator is reported in row 16 where we restrict the sample to states with strictly more than 3 cities in our main dataset.[24]

IV1: Months of First Infection. Next we implement three IV strategies. Note that the IVs estimate local average treatment effects (LATE) and must be taken with caution. The first IV exploits the timing of the Black Death. Timing provides exogenous variation in mortality as there is evidence that plague virulence was correlated with time. Cities that were affected earlier, all else equal, tended to experience higher death rates. According to Berngruber et al. (2013): "Theory predicts that selection for pathogen virulence and horizontal transmission is highest at the onset of an epidemic 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 and may reverse the selection on virulence. This may favor benign pathogens after the acute phase of the epidemic." Figure 5 provides support for this IV strategy. For 109 cities of the main sample for which we have data on the onset of the Black Death, it plots mortality rates against the date that the city was first infected (here, the number of months since October 1347, when Messina was first infected). Cities infected later had indeed lower mortality rates.

Using the number of months since October 1347 as an IV we obtain a coefficient of -1.63** (row 17, F-statistic of 18.4). This is precisely estimated, however, this unconditional IV estimate is also significantly larger than our OLS coefficient (-0.84). The Black Death spread from the south and as the urban network of medieval Europe was still centered around the Mediterranean Sea it is likely that month of first infection is partially correlated with latitude, trade and city size. We therefore employ month of first infection conditional on our control variables as our preferred IV specification. By including the controls of Table 2, as well as a quartic in latitude and a quartic in longitude to control for the geographical origins of the plague, we hope to be able to isolate the random component of the spread of the infection. Our conditional IV yields

[24]These are the Kingdom of Aragon (N = 7), England (23), France (26), The Papal States (5), Portugal (5), Sicily-Naples (4) and Sicily-Trinacria (4).

a coefficient that is close to our OLS estimate at -0.85** (row 18, F-statistic of 21.3). This result is consistent with our baseline OLS estimates and with our hypothesis that the spread of the Black Death was largely exogenous to city-level characteristics.

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. The Black Death was at its most virulent during the summer months (Benedictow, 2005, 233-235). Rat fleas become most active when it is fairly warm and humid while the cold limits their activity (Gottfried, 1983, 9). The average duration of the Black Death in each city was 7 months (see Figure 6(a)). According to Christakos et al. (2005), mortality on average peaked 3.5 months after the first infection. Therefore, cities that became infected close to the winter escaped relatively unscathed compared to cities that were first infected during the spring, before the summer. The analysis we present in Figure 6(b) suggests that this IV has some explanatory power. For 109 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 onset + 3.5 months. The Black Death was more virulent when peak mortality in the city was reached during the summer months (6-8) and less virulent when peak mortality was reached in the months leading to winter (10-12).

In row 20 we report the results of our analysis using our second IV, twelve dummies for month of first infection, while simultaneously adding dummies for year of first infection to control for the fact that cities infected in earlier years had higher mortality rates (see IV1). We obtain qualitatively similar results, but there is some concern as coefficient is now around twice the size of the OLS coefficient (row 19, F-statistic of 9.2). We therefore condition our IV on our all of our baseline controls as well as a quartic in latitude and a quartic in longitude to control for the geographical origins of the plague (row 21). Doing so, we hope to be able to isolate the random component of the spread of the infection within a year. The coefficient we then obtain is very close to our OLS estimates and highly significant (row 20). However, the F-statistic is low, at 4.7.[25]

IV3: Travel Cost to Messina. The third instrument we use is the log of the travel cost to Messina. Messina was the point of entry for the Black Death into Europe. Therefore

[25]Results hold for both IV1 and IV2 if we use imputed data on the timing of the Black Death so as to include all 139 cities of the main sample rather than 109 cities only (Web Appx. Table A.1 rows 6-9).

the least travel cost to Messina should predict the spread and virulence of the Black Death. As we also control for overall market access to all cities (not just Messina) in these regressions, the cost of travel to Messina should not affect city growth except via the spread of the Black Death. We report estimates using this instrument in rows 21 and 22. In row 22, we also condition our IV on our all of our baseline controls as well as a quartic in latitude and a quartic in longitude. The short-run coefficients we obtain are somewhat in line with our OLS estimates (F-statistics of 30.0-12.0). However, the long-run effects are large and negative. 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.

The particular ship that spread the plague to Messina was initially bound for Genoa. Therefore, to further isolate the exogenous element of the plague spread, in row 10 of Web Appx. Table A.1, we also control for the least cost travel cost to Genoa. Furthermore, to ensure that our IV is not picking up travel costs to the Mediterranean basin in general, in row 11 of Web Appx. Table A.1, we also control for market access to the four largest cities in the Middle East and North Africa (Cairo, Constantinople, Fez and Tunis). In rows 12-15 of Web Appx. Table A.1, we show that results are unchanged when using various combinations of our instruments (IV1 + IV2 and IV1 + IV2 + IV3).

Together these identification strategies appear to confirm the exogeneity of Black Death mortality. Therefore, we will rely on OLS regressions for the rest of the analysis.

4.3. Robustness Checks

In this section, we demonstrate that our baseline results are robust to potential concerns about data measurement, specification, and sampling issues.

Measurement. The first row of Table 5 reports our baseline estimates (for both the short-run effect in 1300-1400 in column (1) and the long-run effect in 1300-1600 in column (2)). Rows 2-9 report robustness checks concerning the nature of our city population data and our mortality data. Historical data of the kind we employ are noisy. The Bairoch (1988) dataset is widely used by economists and economic historians but it also has well known issues and subsequent scholars have corrected it for certain cities (see the discussion in the Web Appendix). Row 2 reports estimates using the

uncorrected Bairoch data. In row 3, we show our estimates are unchanged when we include dummies for the different sources of our mortality data: raw number (N = 89), literary description (N=25), desertion rates (N=21) and clergy mortality rates (N=5). In rows 4 to 6 we systematically drop observations that are based on verbal descriptions of the death rates, desertion rates, and clergy death rates and in row 7 we only use raw mortality estimates. Our coefficient estimates are consistent across specifications.

Our main sample consists of 140 cities that existed in 1300 and for which we know the mortality rate. However, there were 473 existing cities in 1300 (and 467 cities existing in 1300 and 1400 or 1600). For the 473 - 140 = 333 cities for which we do not know the mortality rate, we use instead the spatially extrapolated mortality rates from our core sample of 263 (urban but also rural) localities for which we have mortality data in the Christakos et al. (2005) data set (see the Web Appendix for details). Indeed, if the mortality rate is not correlated with locality size as shown above, using the full mortality data set can only bring more valuable spatial information. Using extrapolated mortality rates allows us to ensure that the results are consistent across both the cities for which we have historical estimates for the mortality rate and other cities in the Bairoch database. In row 8, we show that results are similar when using the full data set of Bairoch cities in 1300. In row 9, we use the extrapolated mortality rates from our main sample of 140 cities instead, and results are again unchanged.

Specification. We show that our results remain unchanged when employ Conley (2008) standard errors with a radius of 100 km in order to account for spatial auto-correlation in the error term (row 10). We also control for percentage population growth in the previous century (1200-1300) in row 11, and use a Log-Log specification where the dependent variable is the log difference in city size and the main variable of interest is the log of the mortality rate in row 12. Row 13 estimates the relationship between population in either 1400 or 1600 on estimated plague mortality, also in absolute numbers, and initial population in 1300. In row 14 we employ a panel model with both city fixed effects and year fixed effects for the whole period 1200-1600. We interact the mortality rate with year dummies, and show the interacted effects for the years 1400 and 1600 (which are defined relative to the interacted effect of the year dummy in 1300). Overall, we find comparable coefficients to our baseline analysis.

Sampling. We show that the results are robust when we omit the five modern

countries with the largest numbers of observations in our dataset: France, Germany, Italy, the United Kingdom and Spain (rows 15-19). The results do not change across these regressions implying that our findings are generalizable to Europe as a whole.[26]

5. The Black Death and New City Creation

Table 6 analyzes the impact of the Black Death on on the rise of new cities after 1300. As already shown in 3(b), a significant share of the impact of the Black Death on subsequent urban development occurred on the extensive margin. While , We find evidence that areas where we estimate that the Black Death was more severe were more likely to be locations of city creation in the post-plague period, especially in areas that were less urbanized initially.

Among the sample of 1,797 cities in the Bairoch (1988) data set between 800 and 1850, 476 of them had already emerged (i.e. passed the 1,000 threshold) by 1300. 1,321 emerged later on, between 1400 and 1850. Panel A of Table 6 reports for various years t the effect of extrapolated plague mortality (based on 263 localities) on a dummy variable equal to one if the city had already emerged by year t.[27] In the absence of any control variables, we find a negative relationship between Black Death mortality and the emergence of new cities in every time period (row 1). When we include the baseline controls of Table 2 (row 2) and spatial fixed effects for modern country boundaries (row 3) however, the sign flips and becomes positive for 1600, 1700, and 1750. This suggests that plague mortality did stimulate the emergence of new urban centers, but not right away in 1300-1400. When including 228 spatial fixed effects for the existing states in 1300, the effect is considerably reduced and not significantly different from 0. However, including that many fixed effects significantly decrease the number of degrees

[26]Results hold when (see Web Appx. Table A.1): (i) We use the Chandler (1974, 1987) data set only (row 16), (ii) Drop the cities with a mortality rate of 25% and 50% (row 17), as the mortality data are heaped at these values, (iii) Use the extrapolated mortality rates based on 186, 166, 110 and 89 localities respectively (rows 18-21), (iv) Cluster standard errors at the country (2015) or state (1300) level (rows 22-23), (v) Additionally control for the percentage change in city population in both 1100-1200 and 1200-1300 (row 24), (vi) Use a Log-Log specification while simultaneously controlling for log city population in 1300 in order to account for mean reversion in city growth (row 25), (vii) Dropping the population weights while simultaneously excluding the cities with fewer than 5,000 inh. as these cities have high growth rates simply because they are small initially, and (viii) Dropping the top 10% outliers in population growth.

[27]By construction, estimates of Black Death death mortality rates do not exist for these cities so we use our extrapolated mortality rates described briefly in Section 3.1. and in detail in the Web Appendix.

of freedom.

To further explore the stimulative effects of plague mortality on the extensive margin of city growth we interact the mortality rate with a dummy equal to one if the city is farther than the median distance to an existing city in 1300 (here, 34.4 km). Row 5 reports the coefficient of this interaction which suggests that areas that were farther from existing cities in 1300 which also experienced higher predicted Black Death mortality urbanized more quickly in the period leading up to the industrial revolution (the non-interacted effects of the mortality rate and the distance dummy are not shown).[28]

Panel B of Table 6 studies the impact of plague mortality on the intensive margin of new city growth. For our dependent variable we use the log of the population of the new cities that enter the data set in 1400, 1600, 1700, and 1750 as this allows us to study the increase in the population of the new cities that enter our dataset after the Black Death. The overall story is the same as that of Panel A: we find a positive long-run effect of plague mortality on the size of new cities (see rows 3 and 4 of Panel B) and, particularly after 1700, this effect is large for those new cities that were further away from the existing cities in 1300 (row 5 of Panel B). These results remain robust to the inclusion of additional controls and a range of different estimates for our extrapolated mortality variables (See Web Appendix Table A.2).[29]

6. Heterogeneity in Recovery and Growth

Historical evidence suggests there was a great deal of heterogeneity across regions in the response to the Black Death. For example, consider the fate of two cities: Hamburg and Montpellier. Hamburg had a population of about 8,000 individuals in 1300. It was struck

[28] Importantly, these results are not driven by cities disappearing from the sample. Only one city from our main sample of 140 exits the sample in the post-Black Death period and this is New-Ross in Ireland. Only 7 cities from the total sample of 469 cities that existed in 1300 leave the sample by 1750 and these cities were small in 1300 and their disappearance was not associated with the Black Death. Results excluding these cities from the analysis are available upon request.

[29] Specifically in Web Appendix Table A.2 we show that these results hold if we: (i) add extra controls for sea-based trade (see row 9 of table 4), plague reoccurences (row 10), conflict (rows 11-12) and Jewish presence and persections (row 13), as can be seen in rows 2 of the Web Appendix Table, (ii) using the log of the mortality rate (+ 1 when it is equal to 0) rather than the mortality rate, especially as the dependent variable is in logs in Panel B, and (iii) using estimates of extrapolated mortality based based 186, 166, 140, 110 and 89 cities to show that our results are not sensitive to how we create this variable.

severly by Black Death, experiencing a mortality rate of approximately 58%.[30] However, it recovered and indeed boomed in the subsequent half-century, growing so rapidly that it had a population of 22,000 by 1400. Like the rapid recovery of Hiroshima and Nagasaki after World War 2, such impressive growth in the wake of a major demographic shock is in line with theories that emphasize the role locational fundamentals play in determining urban location and development (Davis and Weinstein, 2002). By the seventeenth century, Hamburg was a major center of international trade (Lindberg, 2008).

In contrast, for other cities the demographic shock of the Black Death appears to have led to a period of prolonged decline. Montpellier, for instance, had a population of 35,000 in 1300. Like Hamburg it was struck hard by the plague, experiencing a 50% mortality rate. However, unlike Hamburg, it did not recover. Its population in 1400 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. The city did not exceed its 1300 population until 1850. Relative to Hamburg, the fate of Montpellier is more consistent with models of multiple equilibria in urban location in which a large enough shock can cause the urban system to reset (e.g. Krugman, 1991b). Furthermore, the experience of Montpellier calls into question theories that emphasize locational fundamentals as the most important factors in driving city growth as the same fundamentals present before 1300 evidently could not ensure economic success in the post-Black Death period. We ask which experience—that of Hamburg's or that of Montpellier's—was more representative of Europe as a whole?

6.1. City Characteristics and Urban Growth in Preindustrial Europe

Table A.3 and A.4 in the Web Appendix explore the factors associated with preindustrial city growth on the intensive and extensive margins respectively. Columns (1) and (2) of Table A.1 report estimates for the period 1300-1700 and 1300-1750 for the sample of 139 cities for which we have plague mortality estimates while Columns (3) -(4) report estimates for the entire sample of cities existing in 1300.[31]

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

[31] Note that the two samples are different as the cities in our core sample of 139 tend to be larger than the 462 cities due to selection. Hence we do not expect the same factors to be necessarily correlated

It is evident from Table A.1 that coasts and rivers are associated with faster city growth across specifications. In contrast proxies for Roman or medieval trade networks are not related to city growth. When we investigate the importance of costal access for city growth, we find that access to the North Sea, Baltic and, especially the Atlantic were associated with faster growth rather than access to the Mediterranean. Unsurprisingly cities with state capitals grew rapidly in this period. Most other factors, however, are not associated with faster growth. The negative coefficient on log city population in 1300 suggests that there was convergence across city sizes and is consistent with a quasi-fixed factor of production constraining the growth of large cities (**?**).

Table A.2 examines the factors associated with growth on the extensive margin. Columns (1) and (2) employ a dummy for the emergence of a new city as the dependent variable The rise of new cities was more common in areas suitable for growing potatoes and with warmer temperatures. Unsurprisingly, since cities along the coast or on rivers tended to be important by 1300, coastal and riverine locations are not associated with the emergence of new cities. Institutional variables also matter for the emergence of new cities: parliamentary activity at the country or regional level is positively associated with the emergence of new cities while areas that were ruled by monarchies in 1400 saw few new cities emerge.

6.2. The Impact of the Plague and Heterogeneous Recovery

Table 7 studies the heterogeneous effects of the Black Death on city growth. Our interest is in how geographical, economic and institutional factors mediated the effect of Black Death mortality on urban recovery. In Columns (1)-(3) we report the interaction effects for each factor with Black Death mortality for our main sample of 140 cities. In Columns (4)-(6) we do the same using the sample of 462 cities for which we have interpolated Black Death mortality rates.

Columns (1) and (4) study the recovery during the period 1300-1600. Columns (2), (5), (3) and (6) extend the analysis out to 1700 and 1750 respectively to see whether the factors affecting population recovery had a differential impact in the period leading up to the Industrial Revolution.[32]

across samples.

[32]Note that the two samples: the main sample comprising 140 cities and the full sample of 462 are

Table 7 Panel A focuses on non-linearities in the Black Death shock. In the main sample we do not find statistically significance results when we interact Black Death mortality with various measures designed to capture non-linearities in the severity of the plague. However, when we turn to the extended sample of 462 cities, we find a positive interaction between larger cities and the size of the Black Shock. Cities that had reached a threshold size in the extended sample were able to recovery faster for a given level of plague mortality. This consistent with an account where increasing returns play a role in determining the location of urban activity.

Panel B of Table 7 studies the factors associated with the relative growth of northwestern Europe. First we show that median average temperature is negatively related with subsequent growth (row 7) This finding, however, is fully explained by latitude. Row 8 shows that cities with above mean latitude grew faster by 1750 in both the main and the extended sample.

Economic historians have focused on the rise of northwestern Europe following the plague (*Hypothesis 4*) (van Zanden, 2009; de Pleijt and van Zanden, 2013). In rows 10-12 we provide evidence supporting this hypothesis using several definitions of northwestern Europe. We employ a standard definition that includes all cities in the British Isles, Low Countries, Scandinavia, Germany and in northern France which is defined by 45th parallel. In row 11 we only include cities in France above the 48th parallel and in row 12 we exclude French cities east of 2.4 degrees longitude.

It is also evident from Panel B that Southern Europe suffered more from the severity of the plague shock. Being located close to the Mediterranean coast conferred some advantage to cities in the period 1300-1600 but that fades in magnitude and significance once we consider the entire period 1300-1750 (column 3). The Roman road network was most developed in southern Europe and proximity to this network associated with slower growth after the plague (rows 14-15).

Consistent with the rise of northern Europe, we also find a very robust relationship between a city being in the Hanseatic League in northern Germany and recovery from the plague. The coefficient reported in Row 18, Column 4 suggests that a one standard deviation increase in Black Death mortality was associated with approximately 64 %

different so there is not reason to expect the same factors would have the same interaction affects across samples. In particular, the main sample consists of cities that were significantly larger in 1300.

($\approx 4 \times 0.16$) faster growth for a Hansa city than for a non-Hanseatic city between 1300 and 1600. This is unsurprising as the Hanseatic league was at its peak as a commercial trading network during the fourteenth and fifteenth centuries.[33]

The rise of the Hanseatic cities reflects changing patterns of trade following the Black Death. Eastern Europe was hit less severely by the plague than was western Europe. The increase in wages and urbanization in western Europe generated new demands grain from Poland and the Baltic region and it was on the basis of these new trade networks that the Hanseatic cities grew in the period after 1400.

In contrast, institutional factors and political factors do not seem to have interacted with the plague shock in a consistent way. There is some evidence that autonomous cities recovered faster from the plague in the period 1300-1600 but this effect did not persist after 1600. And there is evidence in the extended sample that cities in monarchies grew faster in response to the Black Death. Similarly, our measures of locational fundamentals such as cereal suitability, grazing suitability and elevation were not associated with faster recovery from the Black Death.

6.3. Market Access and City Recovery

In addition to its direct impact on urban populations, the Black Death also represented a tremendous indirect shock to market access. In this section we consider the implications of this shock. Through its effect on the surrounding trade network, the Black Death could affect the growth of a city even if that city's own loss of population was moderate.

We use our market access variable in conjunction with our mortality data to estimate the negative shock to market access caused by the plague. Figures 7 and 8 show how this shock to market access was distributed across Europe. It is evident that the size of markets shrank most severely in southern Europe, particularly, in southern France, northern Italy, and Sicily, whereas in northern Europe the loss of market access was less severe.

[33] See Dollinger (1970). The political decline of the Hanseatic league occurred in the fifteenth century (see Rotz, 1977). However, they remained economically successful until the end of the sixteenth century when 'Amsterdam emerged as northern Europe's economic center, and Dutch control of the Baltic spice trade at this time marked the end of the dominant role of the Hanseatic cities within northern European trade' (Lindberg, 2008, 647).

In Table 8 we investigate the impact of this shock to market access on city growth in the immediate post-plague period and in subsequent centuries.[34] Columns (1)-(4) report the impact on the main sample of 140 cities. When we regress the mortality rate associated with the plague controlling for the loss of market access between 1300 and 1353 we find that the loss of market access had a direct impact on city populations in 1400 and that it accounts for about 50% of the direct of effect of a city's mortality rate on urban population in 1400.[35] This is unsurprising as plague mortality and the loss of market access have a correlation of 0.5. Loss of market access between 1300 and 1353 also predicts lower city growth by 1600 while the recovery of market access between 1353 and 1400 is positively associated with city growth up to 1600.

Taken together, these results suggest the impact of the market access shock caused by the plague lasted more than two centuries, but that after 1600 neither loss nor recovery of market access following the Black Death are associated with city growth (Columns (3)-(4)). In Columns (5) to (8) we investigate the impact of the market access shock using all the cities in our dataset and find comparable results to the base sample. Furthermore, combined with what we know concerning the spatial distribution of the Black Death shock to market access, we can confidently say that the plague caused a negative shock to market access that was particularly severe in southern Europe and that this shock can partly account for the shift in urban activity to northern Europe that we observe after the Black Death.

7. Conclusion

Numerous scholars have argued that the Black Death marks a watershed in European history, after which it is possible to detect trends that would eventually lead to the onset of sustained economic growth (Gottfried, 1983; Herlihy, 1997; Epstein, 2000; Haddock

[34]Note that we always use all the data in our sample to construct our market access variable. Therefore market access in 1300 is based on the 473 cities in the dataset while market access in later centuries is based on a lager number of cities as these enter our data.

[35]That is the coefficient on the plague mortality decreases in absolute magnitude from 0.84 to 0.42. Note, the coefficients on the change in market access and on the mortality rate are not directly comparable as our mortality variable ranges from 0 to 80% while the range of the loss of market access variable is more compressed. A one standard deviation increase in plague mortality is associated with 0.15 standard deviation decrease in population while a one standard deviation reduction in market access between 1300-1400 is associated with 0.18 standard deviation decrease in population.

and Kiesling, 2002; Pamuk, 2007; Acemoglu and Robinson, 2012). Existing accounts have focused on the fertility response to the Black Death (Moor and Zanden, 2010; Voigtländer and Voth, 2013a), its effect on real wages and GDP (Allen, 2001; Broadberry, 2013), or its impact on political institutions (Epstein, 2000; Acemoglu and Robinson, 2012). Recent work has focused on the effect of the Black Death on urbanization in Europe in comparison to China (Voigtländer and Voth, 2013b).

Our unique dataset of urban mortality allows us to explore the spatial dimension of the impact of the Black Death on urban development in preindustrial Europe. Using a range of identification strategies involving three IV strategies we find that the Black Death had a large impact on urban population in the short-run. On average cities recovered on from the Black Death within two centuries.

We use city-level data to show that areas of Europe that experienced higher Black Death mortality saw the emergence of new urban centers. This result is consistent with arguments by Voigtländer and Voth (2013b) and others which suggests the plague may have played a role in the precocious urbanization of northern Europe in the years leading up to the Little Divergence.

The aggregate convergence of city population that we identify masked a great deal of divergence at a local level. Some cities recovered rapidly while others declined. We find some evidence that cities recovered fastest from the Black Death if they had reached a threshold size by 1300 which is consistent with an account where increasing returns play a role in determining the location of urban activity. Locational characteristics such as the quality of agricultural land do not explain the differential patterns of recovery that we observe. Access to trade does predict city growth in the post-plague era. In particular, cities located on the Atlantic and Baltic coasts grew rapidly in the period 1400-1700. Furthermore, the Black Death reduced market access more in the south of Europe than in the North and this generated negative and positive spillovers for urban growth respectively. The only institutional factors we find that matter are those that may also be related to the growing importance of trade and commerce. Cities that belonged to the Hanseatic league grew rapidly in the wake of the plague and, consistent with recent accounts by economic historians, there is some evidence that cities in the larger European monarchies whose trade may have been less restricted by internal tariffs also grew faster (Epstein, 2000; Grafe, 2012).

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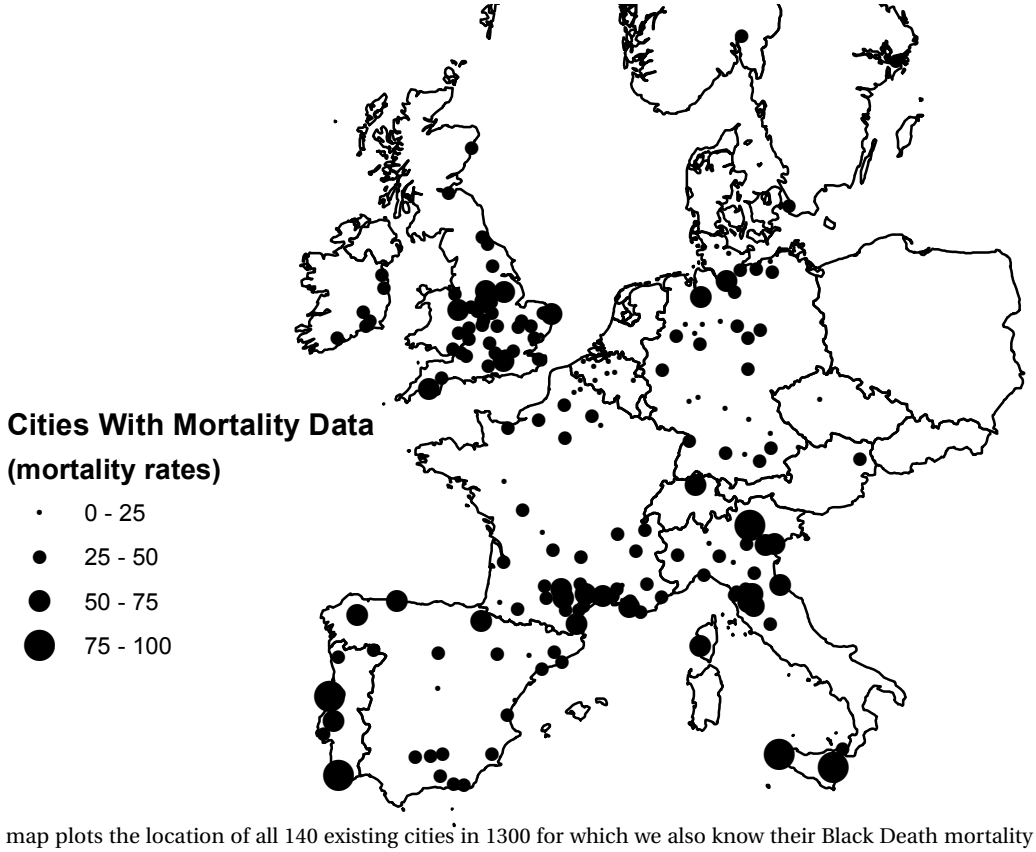
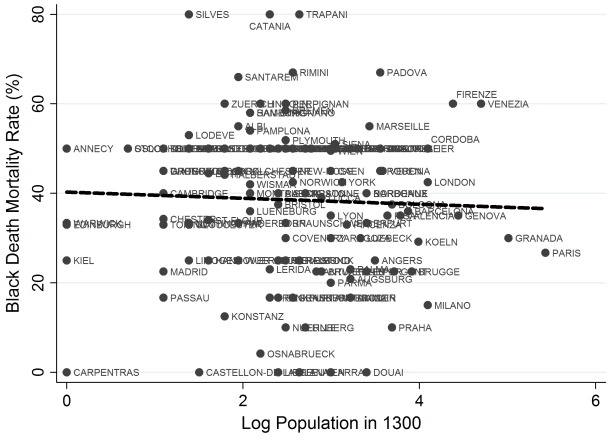
Figure 1: Black Death Mortality Rates (%) in 1347-1352



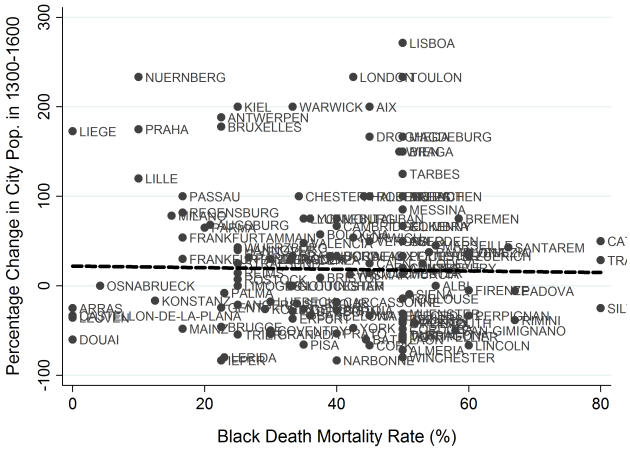
Notes: This map plots the location of all 140 existing cities in 1300 for which we also know their Black Death mortality rate (%) in 1347-1352. The map also shows the modern boundaries of the 17 European countries of our main analysis. The main source for the mortality data is Christakos et al. (2005). See Web Data Appendix for more details on data sources.

Figure 2: Mortality Rates, Initial City Size and City Population Growth

(a) Mortality and Initial City Size, 1300

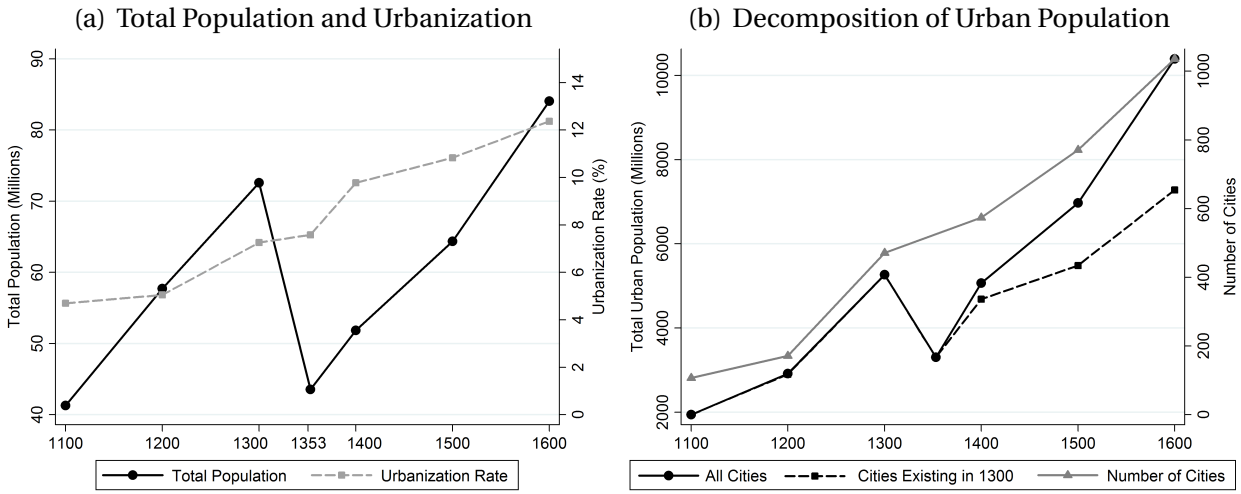


(b) Mortality and City Pop. Growth, 1300-1600



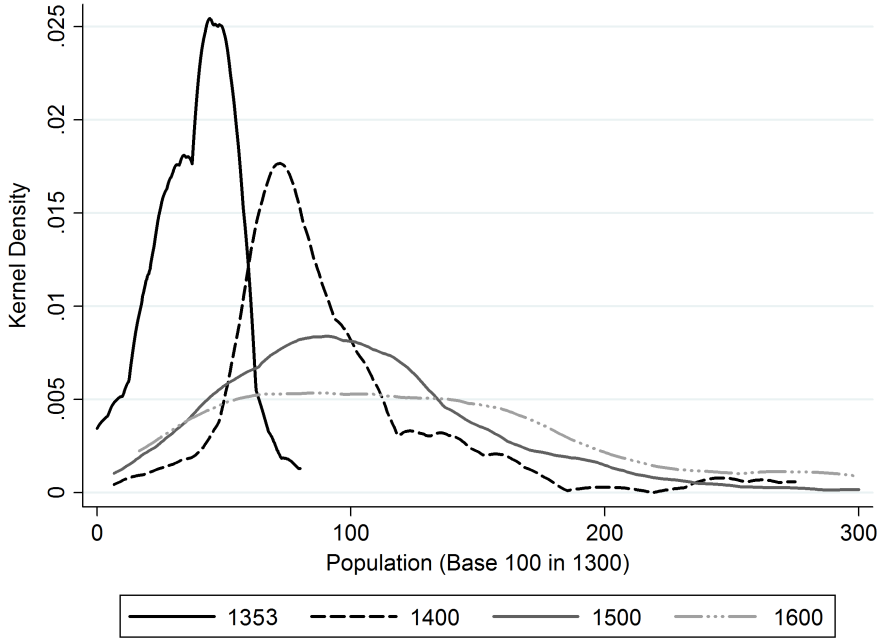
Notes: Subfigure A shows the relationship between the mortality rates (%) for 140 existing cities in 1300 for which we have mortality data and the log of the population of the city in 1300. Subfigure B shows the relationship between the percentage change in city population for the 140 cities for which we have mortality data between 1300-1600 and the mortality rate (%) in 1347-1352. To improve visibility we excluded the top and bottom 5% of observations. See Web Data Appendix for more details on data sources.

Figure 3: Evolution of Europe's Total Population and Urbanization Rate, 1100-1600



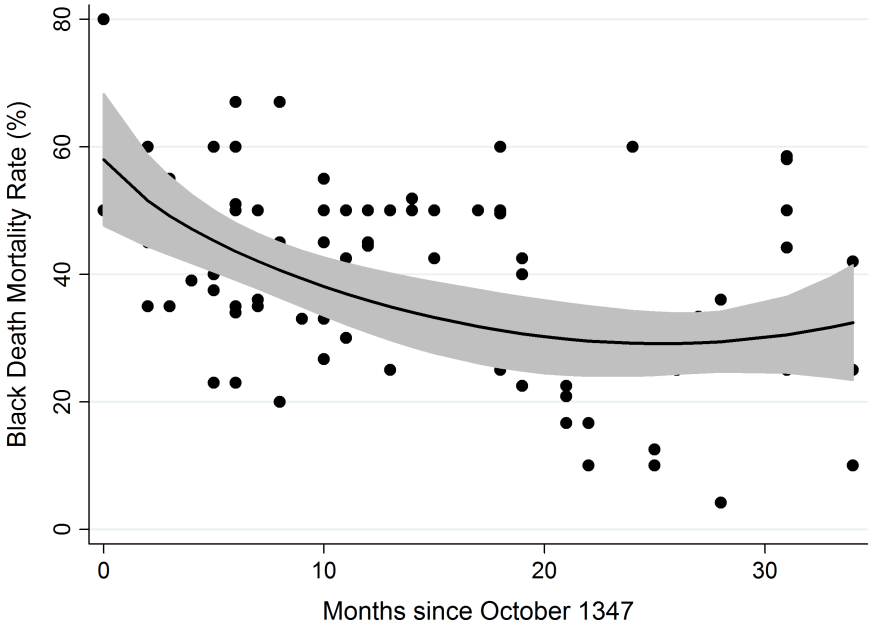
Notes: Subfigure A shows the respective evolutions of the total population (millions) and urbanization rate (%) of the 17 European countries of our main analysis. Subfigure B presents the evolution of the total urban population (millions). The modern countries in our sample are Austria, Belgium, Czech Republic, Denmark , France, Germany, Italy, Luxembourg, Norway, Poland, Portugal, Spain, Sweden, Switzerland, the Netherlands, and the United Kingdom. The main sources for total population are Malanima (2009) and Malanima (2010). The urbanization rate is defined as the share of all the localities above 1,000 in the total population. Total (urban) population in 1353 is proxied by the total (urban) population in 1300 times the average (urban) mortality rate in 1347-1352, which we estimate as 40% (37.5%). City population data is from Chandler (1974, 1987) and Bairoch (1988).

Figure 4: Distribution of City Sizes for the Existing Cities in 1300, 1300-1600



Notes: This figure shows the Kernel distribution of city sizes (base 100 in 1300) for the 140 existing cities in 1300 for which we also know their Black Death mortality rate, for the years 1353, 1400, 1500 and 1600. These cities belong to the 17 European countries of our main analysis (see the notes below Figure 3 for a list). Their population in 1353 is proxied by their population in 1300 times their Black Death mortality rate in 1347-1352. The main sources for the city population data are Chandler (1974, 1987) and Bairoch (1988). For ease of readability we exclude 21 outlying observations whose population is more than 3 times their population in 1300.

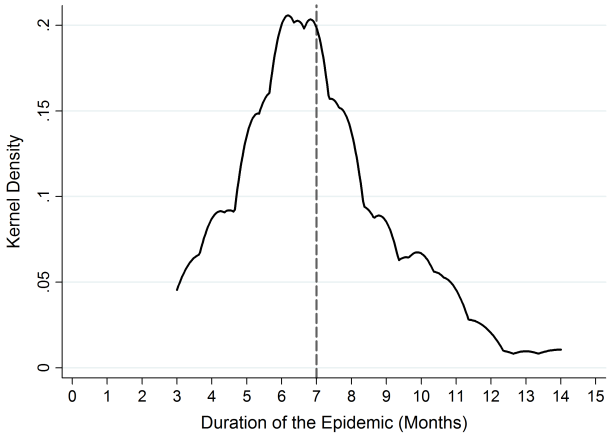
Figure 5: Timing of the Onset of the Black Death and Black Death Mortality: IV(1)



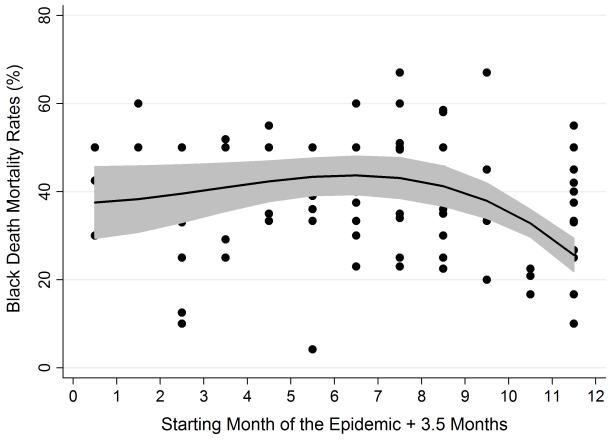
Notes: This figure depicts the relationship between Black Death mortality rates (%) in 1347-1352 and the timing of the onset of the Black Death, here the number of months since October 1347, the month Messina – the port of entry of the Black Death in Europe – was first infected by it. Cities which were affected by the Black Death earlier had higher mortality rates. The main source for the mortality data is Christakos et al. (2005). See Web Data Appendix for more details on data sources.

Figure 6: Duration and Month of Onset of the Black Death and Mortality: IV(2)

(a) Duration of the Black Death in Each City

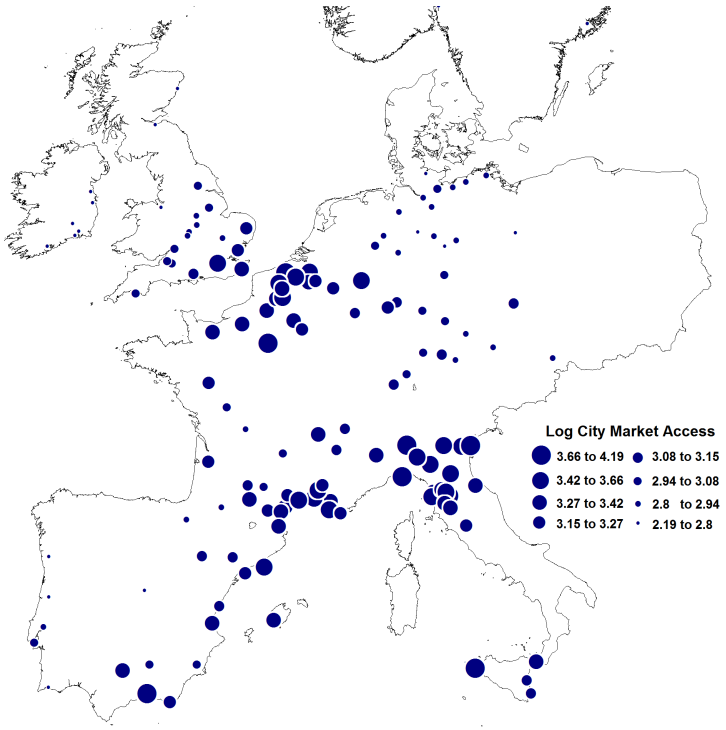


(b) Month of Black Death Onset and Mortality



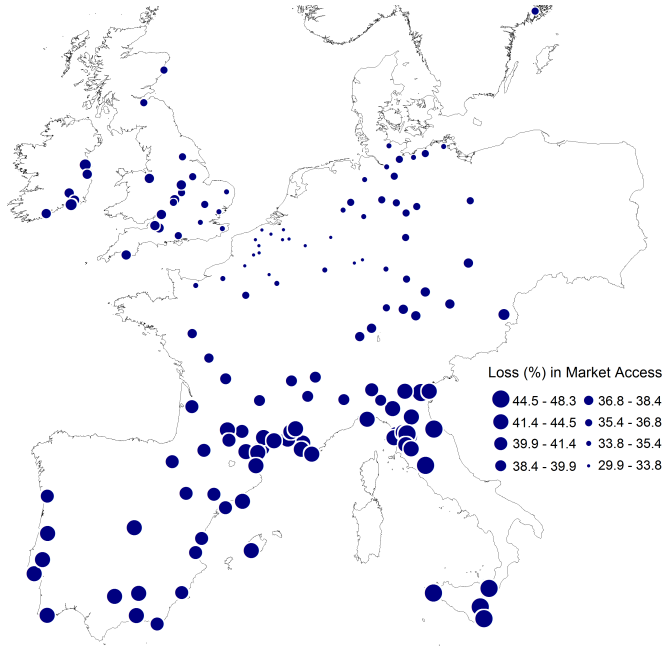
Notes: Subfigure A shows the Kernel distribution of the duration of the Black Death in each city, i.e. the time difference between the month of the first infection in the city and the month of the last infection in the city. The average duration of the Black Death was 7 months. According to Christakos et al. (2005), mortality on average peaked 3.5 months after the first infection, i.e. about halfway. Subfigure B shows the relationship between Black Death mortality and the month of onset + 3.5 months. The Black Death was more virulent when peak mortality in the city was reached during the summer months (6 = June; 7 = July; 8 = August) and less virulent when peaked mortality was reached in the months leading to winter (10 = October; 11 = November; 12 = December). The main source for the mortality data is Christakos et al. (2005). See Web Data Appendix for more details on data sources.

Figure 7: City Overall Market Access in 1300



Notes: This figure plots log city market access for the 140 cities in our data set for the year 1300. We report the measure of market access to the 473 existing cities in 1300 used in rows 1-2 of Table 3 (Incl. Own Pop. / (5 Km x Trade Cost of Porters)$^{\sigma}$, σ = 1, Speeds: Porters (speed normalized to 1), roads (0.50), rivers (0.24) and seas (0.18)). See Web Data Appendix for more details on data sources.

Figure 8: Loss of Market Access (%) after the Black Death Shock, 1300-1353



Notes: This figure plots the percentage loss in market access between 1300 and 1353 to the 473 existing cities in 1300 for the 140 cities in our main sample using the extrapolated mortality rates (based on 263 localities). For the years 1300 and 1353, we use the same definition of market access as in rows 1-2 of Table 3 and Figure 7 except we exclude the population of the same city (we thus use the same definition of market access as in row 4 of Table 3). The population of each city in 1353 is reconstructed as its population in 1300 times (100 - the Black Death mortality rate (%) in 1347-1352) / 100. See Web Data Appendix for more details on 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	1400-1500	1500-1600	1300-1600	1300-1700	1300-1750	1100-1200	1200-1300
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
β	-0.84***	0.46**	0.39	-0.13	0.09	0.44	-0.15	0.05
	[0.31]	[0.23]	[0.29]	[0.51]	[0.85]	[1.03]	[0.37]	[0.62]
	[-1.5–0.2]	[0.1-1.0]	[-0.1–1.0]	[-1.1–0.9]	[-1.6–1.8]	[-1.6–2.5]	[-0.9–0.6]	[-1.2–1.3]
Obs.	140	139	139	139	139	139	59	87
R^2	0.11	0.00	0.00	0.02	0.01	0.00	0.00	0.00

Notes: This table shows the effect β_t of the Black Death mortality rate (%) in 1347-1352 on the percentage change in city population (%) for each period t. The main sample consists of 140 cities (i.e. loc. $\geq$ 1,000 inh.) that already existed in 1300 and for which the mortality rate is available. We use the population of each city 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 Data 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)	-0.77	[1.26]					-0.74	[1.69]
Elevation (m)	-0.01	[0.01]					-0.02	[0.01]
Cereal Suitability Index	1.51	[1.87]					2.83	[2.14]
Potato Suitability Index	0.35	[2.14]					-0.69	[2.34]
Pastoral Suitability Index	1.58	[4.70]					-0.12	[5.27]
Coast 10 Km Dummy	2.62	[3.58]					0.71	[4.65]
Rivers 10 Km Dummy	-3.89	[3.12]					-3.00	[3.40]
Longitude (d)	-0.10	[0.23]					0.14	[0.37]
Latitude (d)	-1.33*	[0.75]					-1.42	[0.90]
Increasing Returns:								
Log City Population in 1300			0.08	[1.59]			-1.99	[2.20]
Maj.Roman Rd (MRR) 10 Km Dummy			-1.93	[7.96]			-1.04	[6.40]
MRR Intersection 10 Km Dummy			4.63	[4.32]			0.92	[4.55]
Any Roman Rd (ARR) 10 Km Dummy			4.08	[8.66]			1.08	[7.51]
ARR Intersection 10 Km Dummy			-0.44	[5.02]			2.90	[5.00]
Medieval Route (MR) 10 Km Dummy			-0.69	[3.04]			0.20	[3.16]
MR Intersection 10 Km Dummy			-3.01	[5.27]			-0.54	[5.71]
Market and Fair Dummy			-5.70	[3.89]			-5.52	[4.48]
Hanseatic League Dummy			1.68	[5.11]			6.98	[6.43]
Aqueduct 10 Km Dummy			0.49	[4.05]			0.25	[4.01]
University Dummy			3.89	[4.71]			1.81	[4.87]
Institutions:								
Monarchy in 1300 Dummy					3.78	[4.67]	3.05	[5.62]
State Capital in 1300 Dummy					0.80	[4.19]	3.44	[5.00]
Autonomous City in 1300 Dummy					-3.46	[3.98]	-0.08	[4.26]
Parliamentary Activity in 1300-1400					1.80	[3.89]	2.15	[4.59]
Battle w/i 100 Km in 1300-1350 Dummy					-4.70	[3.08]	-2.26	[3.28]
Obs.; R^2	140; 0.15		140; 0.06		140; 0.07		140; 0.22	

Notes: This table shows the effects of various characteristics proxying for locational fundamentals, increasing returns and institutions on the mortality rates (%) in 1347-1352. See the text for a description of the variables. We use the main sample of 140 cities. Columns (1)-(4) represent four different regressions. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. See Web Data Appendix for data sources.

Table 3: CITY MARKET ACCESS AND BLACK DEATH MORTALITY RATES

Dependent Variable: Black Death Mortality Rate (%, 1347-1352)

Effect of Log City Market Access in 1300:	Coeff.	SE	Obs.
1. Incl. Own Pop. / (5 Km x Trade Cost of Porters)$^{\sigma}$, σ = 1, Speeds 1	-5.93	[5.55]	140
2. Incl. Own Pop. / (5 Km x Trade Cost of Porters)$^{\sigma}$, σ = 1, Speeds 1, Controls	-10.65	[9.04]	140
3. Incl. Own Pop. / (5 Km x Trade Cost of Roads)$^{\sigma}$, σ = 1, Speeds 1, Controls	-11.32	[9.16]	140
4. Excl. Own Pop., σ = 1, Speeds 1, Controls	-9.79	[8.85]	140
5. Incl. Own Pop. / (5 Km x Trade Cost of Porters)$^{\sigma}$, σ = 3.8, Speeds 1, Controls	-0.53	[1.53]	140
6. Incl. Own Pop. / (5 Km x Trade Cost of Porters)$^{\sigma}$, σ = 2, Speeds 1, Controls	-2.32	[3.19]	140
7. Incl. Own Pop. / (5 Km x Trade Cost of Porters)$^{\sigma}$, σ = 1, Speeds 2, Controls	9.64	[8.81	140
8. Incl. Own Pop. / (5 Km x Trade Cost of Porters)$^{\sigma}$, σ = 1, Costs 1, Controls	10.10	[9.01]	140

Notes: This table shows the effect of log market access in 1300 on mortality (%) in 1347-1352. To calculate market access, we use the population of the 473 cities in 1300 and the travel costs between each pair of cities. Rows 1-2: Market access of city i is the weighted sum of pop. in all cities j (incl. city i), weighted by the travel cost between each pair of cities (i, j) to the power of $\sigma = 1$. The travel cost is the travel time of the least cost path across cells of 5x5 km between city i and city j using 4 transport modes depending on their availability in each cell: porters (speed normalized to 1), roads (0.50), rivers (0.24) and seas (0.18). We divide the pop. of the same city by 5 km and use the speed of porters to account for city congestion. Row 3: For the same city, we use the speed of roads. Row 4: For the same city, we exclude its own pop. Rows 5-6: We employ $\sigma = 3.8$ and $\sigma = 2$. Rows 7-8: For "speeds 2" and "costs 1", the trade costs are {1; 0.5; 0.5; 0.13} and {1; 0.81; 0.21; 0.08} respectively. Robust SE's: † p<0.15, * p<0.10, ** p<0.05, *** p<0.01. See Web Data Appendix for data sources.

Table 4: 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 (4) of Table 1)	-0.84***	[0.31]	-0.13	[0.51]
2. Dropping Top and Bottom 10% in Mortality (Obs. = 109)	-1.06***	[0.39]	0.73	[1.04]
3. Dropping More Hygienic Cities (Obs. = 136)	-0.88***	[0.34]	0.03	[0.57]
4. Controls: Locational Fundamentals	-0.73***	[0.27]	0.32	[0.54]
5. Controls: Increasing Returns	-0.73***	[0.27]	-0.29	[0.52]
6. Controls: Institutions	-0.89***	[0.28]	-0.18	[0.51]
7. Controls: All (Rows 4-6)	-0.66***	[0.22]	-0.41	[0.64]
8. Row 7 + Log Market Access in 1300 (see Row 1 of Table 3)	-0.67***	[0.21]	-0.43	[0.64]
9. Row 7 + Dummies for Each Coastline	-0.70***	[0.21]	-0.34	[0.56]
10. Row 7 + Dummies if Plague Reccurrence in 1350-1400 (-1600)	-0.65***	[0.22]	-0.45	[0.63]
11. Row 7 + Dummies if Battle w/i 100 Km in 1350-1400 (-1600)	-0.68***	[0.21]	-0.50	[0.65]
12. Row 7 + Dummies if Burned or Sacked in 1300-1400 (-1600)	-0.66***	[0.22]	-0.51	[0.64]
13. Row 7 + Dummies if Jewish Presence and Persecution in 1347-52	-0.62***	[0.24]	-0.33	[0.70]
14. Fixed Effects for 13 Countries in 2015	-0.59**	[0.28]	-0.35	[0.53]
15. Fixed Effects for 57 States in 1300	-0.71†	[0.43]	-0.59	[1.20]
16. Fixed Effects for 57 States in 1300 (for States with > 3 Cities)	-1.09**	[0.43]	-0.37	[0.70]
17. Unconditional IV 1: # Months of Infection since Oct 1347 (F: 18.4)	-1.63**	[0.72]	-1.09	[1.23]
18. Conditional IV 1: # Months of Infection since Oct 1347 (F: 21.3)	-0.85**	[0.42]	0.78	[1.59]
19. Unconditional IV 2: Month of First Infection Dummies (F: 9.2)	-1.96***	[0.56]	-1.27	[0.93]
20. Conditional IV 2: Month of First Infection Dummies (F: 4.7)	-0.81***	[0.31]	-0.34	[0.78]
21. Unconditional IV 3: Log Travel Cost to Messina (F: 30.0)	-1.11***	[0.45]	-0.94	[1.14]
22. Conditional IV 3: Log Travel Cost to Messina (F: 12.0)	-1.75**	[0.74]	-4.07*	[2.10]

Notes: This table shows the effect β_t of the mortality rate (%) on the percentage change in city population (%) for period t (sample of 140 cities). Rows 2: Dropping the top and bottom 10% mortality rates. Row 3: Dropping cities with a better hygiene system. Row 8: Row 7 + log market access in 1300 (row 1 of Table 3). Row 9: Row 7 + dummies if within 10 km from the Mediterranean Sea, the Atlantic Ocean or the North-Baltic Sea. Row 10: Row 7 + dummy if plague reoccurrence in 1350-1400 ((1)) or 1350-1600 ((2)). Row 11: Row 7 + dummies for whether a battle occurred within 100 km from the city in 1350-1400 ((1)) or 1350-1600 ((2)). Row 12: Row 7 + dummies for whether the city was burned or sacked in 1300-1350 and 1350-1400 ((1)) or 1350-1600 ((2)). Row 13: Row 7 + dummies if Jews were present in 1347 and were persecuted in 1347-52. Row 14: Adding 13 country FE. Rows 15-16: Adding 57 state in 1300 FE (row 16: excl. states with $\leq$ 3 obs.). Rows 17-18: Instrumenting by the number of months between the city-specific date of first infection and Oct 1347. Rows 19-20: Instrumenting by twelve dummies for the month at the peak of the infection, while adding dummies for the year of infection. Rows 21-22: Instrumenting by the log of the travel cost to Messina, while controlling for log market access in 1300. In rows 18, 20 and 22, we add the controls and quartics of latitude and longitude. Robust SE's: † p=0.17, * p<0.10, ** p<0.05, *** p<0.01. See Web Data Appendix for data sources.

Table 5: MORTALITY RATES AND CITY GROWTH, MAIN 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 (4) of Table 1; Obs. = 140)	-0.84***	[0.31]	-0.13	[0.51]
2. City Population Data: Bairoch Only (Obs. = 140)	-0.71*	[0.43]	0.04	[0.53]
3. Dummies for Source of Mortality Data (Obs. = 140)	-0.91***	[0.33]	-0.24	[0.55]
4. Excluding Description-Based Mortality Data (Obs. = 115)	-0.81**	[0.36]	-0.31	[0.58]
5. Excluding Desertion-Based Mortality Data (Obs. = 119)	-0.96***	[0.34]	-0.21	[0.57]
6. Excluding Clergy-Based Mortality Data (Obs. = 135)	-0.84***	[0.31]	-0.11	[0.51]
7. Raw Mortality Data (Obs. = 89)	-0.96**	[0.41]	-0.38	[0.67]
8. Extrapolated Rates Based on 263 Mortality Rates (Obs. = 467)	-0.69***	[0.22]	0.34	[0.46]
9. Extrapolated Rates Based on 140 Mortality Rates (Obs. = 467)	-0.74***	[0.23]	0.29	[0.50]
10. Conley-Type SEs: 100 km (Obs. = 140)	-0.84***	[0.20]	-0.13	[0.60]
11. Adding % Change in Pop. in 1200-1300 (Obs. = 87)	-1.10***	[0.38]	-0.14	[0.61]
12. Regressing Log Change in Pop. on Log Mortality (Obs. = 134)	-0.34***	[0.11]	-0.12	[0.16]
13. Regressing Pop. on Number of Dead and Initial Pop. (Obs. = 140)	-0.87*	[0.46]	0.00	[0.64]
14. Panel Model with City FE and Year FE in 1200-1600 (Obs. = 564)	-1.07*	[0.56]	-0.05	[0.52]
15. Excluding Cities in France (N = 33; Obs. = 107)	-0.75**	[0.38]	-0.21	[0.63]
16. Excluding Cities in Germany (N = 29; Obs. = 111)	-1.04***	[0.36]	-0.28	[0.57]
17. Excluding Cities in Italy (N = 22; Obs. = 118)	-1.09***	[0.41]	0.14	[0.77]
18. Excluding Cities in the United Kingdom (N = 19; Obs. = 121)	-0.80**	[0.311]	-0.15	[0.510]
19. Excluding Cities in Spain (N = 14; Obs. = 126)	-0.92***	[0.32]	-0.07	[0.50]

Notes: This table shows the effect β_t of the mortality rate (%) on the percentage change in city population (%) for period t. (sample of 140 cities). Row 2: Employing the raw Bairoch pop. data. Row 3: Including dummies for the source of mortality data: raw number (N = 89), literary description (N=25), desertion rates (N=21) and clergy mortality rates (N=5). Rows 4-7: Excluding the description-based, desertion-based or clergy-based mortality rates, or all of them altogether. Rows 8-9: Using spatially extrapolated mortality rates based on the mortality rates of 263 localities and the 140 cities of the main sample respectively. Row 10: Using Conley-type SEs (100 Km). Row 11: Controlling the percentage change in city pop. in 1200-1300. Rows 12: Regressing the log difference in city pop. on log mortality. Row 13: Regressing city pop. on the number of dead (estimated as pop. in 1300 x the mortality rate) and the initial city pop. Row 14: Running a panel model with city FE and year FE in 1200-1600. We show the relative effects in 1300 and 1600. Rows 15-19: Excluding the cities of the most represented countries in the sample. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. See Web Data Appendix for data sources.

Table 6: MORTALITY RATES AND CITY GROWTH: THE EXTENSIVE MARGIN

Regression:	(1) t = 1400		(2) t = 1600		(3) t = 1700		(3) t = 1750	
	Effect of Extrapolated Black Death Mortality Rate (%):							
Panel A: Dummy for City Creation in Year t (Obs. = 1,321):								
1. Unconditional	-0.002***	[0.001]	0.000	[0.001]	-0.001	[0.001]	-0.002*	[0.001]
2. Row 1 + Controls	-0.001	[0.001]	0.002	[0.001]	0.004**	[0.002]	0.004***	[0.001]
3. Row 2 + 18 Country in 2015 FE	0.001	[0.001]	0.005***	[0.002]	0.005**	[0.002]	0.005***	[0.002]
4. Row 2 + 228 State in 1300 FE	-0.001	[0.001]	0.001	[0.002]	0.001	[0.002]	0.001	[0.002]
5. Row 3, $\geq$ Median Dist. City$_{1300}$	0.000	[0.001]	-0.000	[0.002]	0.004*	[0.002]	0.004**	[0.002]
Panel B: Log Population for New Cities in Year t (Obs. = 105; 573; 810; 894):								
1. Unconditional	-0.008	[0.005]	0.007***	[0.002]	0.003*	[0.002]	0.006***	[0.001]
2. Row 1 + Controls	0.007	[0.007]	0.002	[0.003]	-0.001	[0.003]	0.000	[0.002]
3. Row 2 + 18 Country in 2015 FE	0.019	[0.015]	0.010***	[0.004]	0.008**	[0.003]	0.003	[0.003]
4. Row 2 + 228 State in 1300 FE	0.030	[0.023]	0.006	[0.006]	0.010**	[0.005]	0.008**	[0.004]
5. Row 3, $\geq$ Median Dist. City$_{1300}$	0.009	[0.018]	-0.002	[0.005]	0.006	[0.004]	0.006*	[0.003]

Notes: This table shows the effect β_t of the mortality rate (%) on the extensive margin of city growth. The sample consists of 1,321 cities of the Bairoch sample that did not already exist in 1300 but existed at one point (i.e. passed the 1,000 threshold) in 1400-1850. Rows 1-4 of Panel A show the effect of extrapolated mortality rates based on 263 localities on a dummy equal to 1 if a city has greater than 1,000 inh. in year t. Rows 1-4 of Panel B regress log population in year t on mortality for these new cities. In rows 5 of Panels A and B, we show the interacted effect of the mortality rate and a dummy equal to 1 if the new city is farther than the median Euclidean distance to an existing city in 1300 (34.4 km for panel A; 28.7, 34.4, 32.4 and 32.3 km for panel B). See Web Data Appendix for data sources.

Table 7: MORTALITY RATES AND CITY GROWTH, HETEROGENEOUS EFFECTS, 1300-1750

Dependent Variable:	Percentage Change in City Pop. (%) in Period t					
	Col.(1)-(3): Existing Rates (Obs. = 139)			*Col.(4)-(6): Extrapolated Rates (Obs. = 462)*		
Period 1300-t:	(1) 1600	(2) 1700	(3) 1750	(4) 1600	(5) 1700	(6) 1750
Separate regression in each row:	Effect of Black Death Mortality Rate (%) x Dummy:					
	Panel A: Non-Linearities in the Shock					
1. ≥Med. Mortality Rate	-0.7 [2.2]	-7.3 [6.7]	-10.8 [7.8]	1.8 [1.3]	1.2 [1.8]	0.5 [1.9]
2. ≥Med. Population 1300	1.4 [1.2]	1.4 [2.3]	1.2 [2.8]	2.5** [1.2]	4.3 [2.9]	5.4* [3.2]
3. ≥Med. Population 1353	2.3 [2.0]	5.5 [4.3]	8.2 [5.1]	4.7*** [1.6]	8.5** [4.0]	11.1** [4.4]
4. ≥Med. ΔMarket Access	-0.3 [1.5]	-3.0 [3.4]	-5.1 [4.1]	0.6 [1.3]	-1.2 [2.6]	-3.3 [3.1]
5. ≥Med. Market Access 1300	0.4 [1.5]	-2.5 [2.1]	-4.4 [2.7]	0.7 [1.1]	-0.6 [1.5]	-0.7 [1.9]
6. ≥Med. Market Access 1353	0.1 [1.3]	-2.3 [1.9]	-3.4 [2.4]	0.6 [1.1]	-0.3 [1.5]	-0.3 [1.8]
	Panel B: Relative Growth of Northwestern Europe					
7. ≥ Med. Av. Temp. 1500-1600	-0.5 [1.3]	-4.8* [2.9]	-7.7** [3.5]	0.9 [1.0]	-1.7 [2.0]	-4.0* [2.4]
8. ≥ Med. Latitude (= 48.4; 47.3)	0.6 [1.3]	3.7 [3.0]	6.4* [3.6]	-0.2 [1.1]	2.6 [2.2]	5.6** [2.7]
9. ≥ Med. Longitude (= 4.0; 7.3)	-1.4 [1.1]	-1.2 [2.1]	-1.5 [2.4]	0.4 [0.9]	1.8 [1.3]	1.4 [1.5]
10. 𝟙 Northwestern (Fr: Lat≥45.0)	2.1 [1.3]	4.2 [2.7]	7.4** [3.3]	1.1 [1.1]	2.7 [2.1]	5.5** [2.5]
11. 𝟙 Northwestern (Fr: Lat≥48.8)	1.9 [1.3]	4.2 [2.8]	7.5** [3.4]	0.7 [1.1]	2.6 [2.2]	5.8** [2.7]
12. 𝟙 Northwestern (Fr: Lat≥48.8 Lon≤2.4)	1.6 [1.3]	4.7 [3.2]	8.7** [3.9]	0.3 [1.1]	2.6 [2.4]	6.2** [3.0]
13. 𝟙 Mediterranean Coast 10 Km	1.6* [0.9]	1.5 [1.4]	0.1 [1.6]	3.1*** [1.1]	3.5*** [1.4]	3.0* [1.6]
14. 𝟙 Maj.RomanRd (MRR) 10 Km	-0.3 [1.2]	-3.6** [1.7]	-5.2** [2.3]	-0.0 [1.0]	-1.6 [1.4]	-2.2 [1.7]
15. 𝟙 Any Roman Rd (ARR) 10 Km	-0.2 [1.6]	-3.8* [2.0]	-6.6** [3.0]	-0.2 [1.1]	-1.5 [1.6]	-2.9 [2.0]
16. 𝟙 Atlantic Coast 10 Km	9.7 [9.1]	20.9* [11.5]	20.1 [18.2]	-2.3 [4.0]	6.1 [8.1]	5.4 [9.3]
17. 𝟙 Baltic Coast 10 Km	5.2 [3.5]	16.0** [6.8]	27.8*** [9.7]	0.1 [4.1]	0.3 [9.9]	8.9 [11.3]
18. 𝟙 Hanseatic League 1300	4.4** [2.1]	7.0 [4.2]	9.9* [5.8]	4.0** [1.8]	6.7* [3.7]	9.2* [5.1]
19. 𝟙 Monarchy 1300	1.0 [1.0]	2.4 [1.6]	2.3 [1.9]	2.3*** [0.8]	2.6** [1.3]	2.7* [1.5]
20. 𝟙 Autonomous City 1300	1.9** [0.9]	0.9 [1.5]	1.1 [1.8]	0.5 [0.8]	0.2 [1.2]	0.3 [1.4]

Notes: This table shows the interacted effects of the mortality rates (%, 1347-1352) and various city characteristic dummies proxying for locational fundamentals, increasing returns and institutions. Each row represents a separate regression for each city characteristic dummy. Columns (1)-(3): We use the mortality rates for the 140 existing cities in 1300 for which we have the mortality data. Columns (4)-(6): We use the extrapolated mortality rates based on 263 localities. In rows 10-12, Northwestern Europe includes Austria, Belgium, Denmark, Germany, Ireland, Luxembourg, the Netherlands, Norway, Sweden, Switzerland and the United Kingdom, as well as Northern France (Row 10: Latitude ≥ 45°; Row 11: Latitude ≥ 48.8° (Paris' latitude); Row 12: Latitude ≥ 48.8° & Longitude ≤ 2.4° (Paris' latitude and longitude)). Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. See Web Appendix for data sources.

Table 8: MORTALITY RATES, MARKET ACCESS AND CITY GROWTH, 1300-1750

Dependent Variable: Percentage Change in City Population (%) in Period 1300-t								
t:	1400	1600	1700	1750	1400	1600	1700	1750
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mortality Rate (%)	-0.42*	-0.13	0.64	0.79	-0.36*	-0.14	0.21	0.07
	[0.24]	[0.72]	[1.53]	[1.85]	[0.21]	[0.68]	[1.41]	[1.64]
ΔMarket Access 1300-1353	-4.73***	-9.25**	-6.39	0.81	-3.03**	-9.61***	-9.61	-3.15
	[1.77]	[4.27]	[9.10]	[11.77]	[1.26]	[3.32]	[6.54]	[7.98]
ΔMarket Access 1353-t	1.09*	0.75**	0.34	0.32	0.74**	0.74***	0.57*	0.51*
	[0.62]	[0.32]	[0.40]	[0.40]	[0.36]	[0.23]	[0.33]	[0.28]
Obs.	140	139	139	139	467	460	462	462
R^2	0.55	0.36	0.30	0.33	0.26	0.26	0.13	0.17

Notes: This table shows the effects of the Black Death mortality rate (%) in 1347-1352 and changes in market access in 1300-1353 and 1353-t on the percentage change in city population (%) for various periods 1300-t. The main sample consists of 140 cities (i.e. localities $\geq$ 1,000 inh.) that already existed in 1300 and for which the mortality rate is available. Market access of city i is the weighted sum of population in all cities $j \neq i$ among the 1792 cities of the Bairoch data set, weighted by the travel cost between each pair of cities (i, j) to the power of $\sigma = 1$. The travel cost is the travel time of the least cost path across cells of 10x10 km between city i and city j using four transportation modes depending on their availability in each cell: porters (speed normalized to 1), roads (0.50), rivers (0.24) and seas (0.18). "ΔMarket Access 1300-1353" and "ΔMarket Access 1353-t" are the changes in market access between 1300 and 1353 and between 1353 and year t respectively (the population of each city in 1353 is reconstructed as its population 1300 times the mortality rate in 1347-1353). We use the population of each city in the initial year as regression weights. Robust SE's: * p<0.10, ** p<0.05, *** p<0.01. The 95% confidence level intervals are shown into brackets. See Web Data Appendix for data sources.