Journal of Economic Growth (2021) 26:1–30
https://doi.org/10.1007/s10887-020-09185-4

Disease and demographic development: the legacy of the plague

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Accepted: 12 December 2020 / Published online: 25 January 2021

Abstract

This paper provides an empirical investigation of the hypothesis that population shocks such as the repeated outbreaks of the plague affected the timing of the demographic transition. The empirical analysis uses disaggregate data from Germany and exploits geographic variation in the exposure to medieval plague shocks. The findings document that areas with greater exposure to plague outbreaks exhibited an earlier onset of the demographic transition. The results are consistent with the predictions of the unified growth literature and provide novel insights into the largely unexplored empirical determinants of the timing of the transition from stagnation to growth.

Keywords Plague · Demographic transition · Fertility · Outbreak · Epidemics

JEL-classification O10 · J10

The authors thank the Oded Galor, three reviewers as well as Lars Boerner, Stephen Broadberry, Davide Cantoni, Matteo Cervellati, David de la Croix, Lukas Rosenberger, Eric Schneider, Battista Severgnini, Andreas Steinmayr, Fabian Wahl, Jacob Weisdorf, Rudolf Winter-Ebmer, and audiences at the ifo Institute, LMU Munich, the University of Bayreuth, the Spring Meeting for Young Economists 2017, ESPE 2017, ifo Conference Economic Uncertainty and the Family, and the 5th CEPR Economic History Symposium 2017, London, for helpful suggestions and comments.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s10887-020-09185-4.

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1 Introduction

The reason for development differences across countries and regions remains one of the long-standing questions in economics. In view of the non-monotonic dynamics of long-run development and the crucial role of the demographic transition for the take-off in economic development, the answer to this question is closely related to the timing of the demographic transition, as maintained by unified growth theories. In particular, it was the deliberate reduction in fertility that allowed for intensified child rearing, increased human capital investment, and ultimately a sustained increase in incomes per capita in the context of continuing productivity improvements (Galor and Weil 2000; Galor 2011). Consequently, the onset of the fertility transition represents the key turning point, not only for population dynamics and education, but also for the transition to sustained growth. While there is widespread agreement about the role of the fertility transition for the economic take-off as the central building block of the mechanisms underlying unified growth theory and ample evidence regarding the mechanics of these transitions, there is relatively little empirical work in economics that has investigated the determinants of the timing of the fertility transition.

This paper explores the empirical relevance of the hypothesis that the exposure to repeated population shocks related to epidemics influenced the population equilibrium and ultimately the timing of the fertility transition. This hypothesis is motivated by earlier research that conjectured that population shocks related to the Black Death triggered adjustment mechanisms that led to shifts in the Malthusian equilibrium with the consequence of higher population density (Voigtländer and Voth 2013a, b), which provided the ground for the transition from Malthusian stagnation to a modern growth regime (Galor and Weil 2000). Evidence on this conjecture is scant, however.

The empirical analysis investigates whether the timing of the onset of the fertility transition in cities or regions in Germany was influenced by the exposure to repeated outbreaks of the plague. The implementation of an empirical strategy to test the hypothesis requires a plausible measure of exposure to repeated plague outbreaks. Since the effect of interest is that of an accumulated exposure, and since conceptually this exposure can be measured in various ways, the empirical strategy is based on a proxy variable approach that makes use of various, mutually non-exclusive proxies for plague exposure. The first measure of plague exposure is the number of plague outbreaks. Empirical results based on this proxy indeed provide evidence that the experience of more frequent plague-related population shocks was also associated with an earlier fertility transition.

In addition to plague outbreaks, the empirical strategy also makes use of a second set of proxy variable that accounts for variation in the spread of repeated outbreaks of plagues in Europe during the Middle Ages by measuring the travel distance to the ports from which the plague spread after new outbreaks. The respective measures make use of the fact that cities that were closer to these harbors were affected more by outbreaks of the plague. Outbreaks spread inland along medieval travel routes with an intensity that decreased in travel distance to these entry ports, regardless of whether outbreaks of the plague after the 1348 outbreak of the Black Death occurred spontaneously from reservoirs in Europe or from reintroductions from Asia. A second set of empirical results reveals that greater exposure to plague outbreaks as proxied by greater proximity to the nearest entry port is associated with a significantly earlier onset of the demographic transition.

Both plague outbreaks and geographic proximity to entry ports of plague waves are imperfect proxies that contain distinct but mutually non-exclusive variation that is

relevant for the hypothesis to be tested. To rule out spurious results, the analysis controls for an extensive set of additional variables that potentially affect the timing of the fertility transition. The extensive specification of the empirical model makes it unlikely that the finding is driven by third factors, but at the same time it is difficult if not impossible to fully disentangle the role of medieval trade from exposure to plague outbreaks. To explore this issue, the empirical analysis applies various robustness checks that verify that plague-related population shocks affect the timing of the demographic transition. The results are robust to controlling for other characteristics, including access to medieval and 19th century trade routes that have been conjectured to be relevant predictors of the demographic transition, as well as accounting for additional historical and geographic information. In particular, by accounting for access to maritime trade routes, access to the hanseatic trade network, and trade networks during the 19th century, the analysis disentangles the role of population shocks related to medieval plague exposure from effects that are exclusively related to 19th century trade or other mechanisms, such as the demand for human capital, that affect the timing of the transition but are not related to population shocks. An instrumental variables approach that uses travel time to the nearest entry port as instrument for the number of plague outbreaks also confirms the finding that cities that were more exposed to plague outbreaks showed an earlier onset of the demographic transition. Additional analyses reveal that similar patterns are also found for France, providing additional support for the external validity of the results. Taken together, the empirical results support the hypothesis that the fertility decline in the context of the demographic transition occurred earlier in cities and regions that were more exposed to the plague and correspondingly experienced more frequent plague-related population shocks.

The paper contributes to the literature in several ways. The results provide empirical support for some of the central predictions of unified growth theory, according to which the demographic transition, which was the prerequisite for long-run development, was fostered by reduced Malthusian population pressure and an increase in the demand for skills (Galor and Weil 2000; Galor 2011). Despite the important negative short-run consequences of disease shocks (see, e.g., Chakraborty et al. 2010; Bhattacharya and Chakraborty 2016) and the set-backs in long-run development caused by repeated epidemic shocks (Lagerlöf 2003), the evidence shown here suggests that frequent exposure to diseases might indeed have induced transitions to Malthusian steady states with higher population density as response to major population shocks and as consequence of behavioral responses that foster development in the long-run. The findings thereby provide empirical support for the implications of the mechanisms proposed by Voigtländer and Voth (2013a), who suggest that exogenous disease shocks like the outbreak of the Black Death might have triggered a transition to a new Malthusian equilibrium with higher wages and population density, with important consequences for long-run development. For instance, plague-related population shocks might have ultimately triggered fertility reduction by fostering female employment and delaying marriage and childbirth (Voigtländer and Voth 2013b, see also Clark 2008), although this pattern was more prevalent in Northern Europe than in Southern regions like Italy (de Moor and van Zanden 2010). The empirical validity of this channel is also a matter of ongoing debate in light of findings that delayed marriage did not affect total fertility Ortmayr (1995) and that the plague exhibited a similar age pattern in mortality for men and women, while there are no significant gender-differences in mortality (De Witte

2010; Curtis and Roosen 2017; Alfani and Murphy 2017; Alfani and Bonetti 2019), implying that the potential comparative advantage underlying this mechanism might have been weaker than previously thought. Alternatively, plague shocks might have led to changes in household composition that favored investments in child quality. If smaller households were more affected by plague shocks than larger households, as suggested by recent evidence by Alfani and Bonetti (2019), and had a greater propensity toward child quality and more resources to spend on each child, a quantity-quality argument would imply that this led to a shift in the Malthusian equilibrium and ultimately led to an earlier transition from a Malthusian or post-Malthusian equilibrium to a modern growth regime.[1]

While the findings presented in this paper are not suited for disentangling the empirical relevance of the different mechanisms that have been proposed in the literature, they suggest that greater plague exposure was associated with an earlier fertility decline. In this sense, the results also complement recent evidence for England by Crafts and Mills (2017) that is overall consistent with the view that the plague shifted the pre-industrial Malthusian equilibrium and eventually gave rise to a demographic transition that marked the onset of modern growth. Likewise, Dittmar and Meisenzahl (2020) find evidence that plague outbreaks led to the adoption of policies and institutions that were favored by the protestant reformation and that fostered human capital acquisition and greater population growth until the 19th century. While their argument rests on the randomness of the timing outbreaks during a short period, our analysis is based on the overall exposure to plague-related shocks. Our results add to their findings by providing new information about the heterogeneity of the timing of the fertility transition about one century later.

Using spatial variation in the plague-related mortality at the city level, Jebwab et al. (2019) explore the impact of the outbreak of the Black Death 1347–1352 on city growth. They present new evidence for the duration until the populations recovered as well as its determinants and document heterogeneity regarding geographic endowments of cities in terms of land suitability and access to trade networks. On the other hand, recent work by Alfani and Percoco (2019) on Italian cities suggests that the plague epidemic of 1629-30 represented a productivity shock that caused a long-run decline in city growth and urbanization rates. The empirical analysis in this paper provides evidence that complements these findings by documenting that repeated plague outbreaks might in fact have led to an earlier fertility transition once controlling for heterogeneity in other factors.[2] At the same time, the approach focuses on a confined area of comparable geography, demography, and institutional environment in Northern Europe, thereby to a certain extent implicitly accounting for the heterogeneity of the impact of the plague that has been documented by Pamuk (2007) and more recently by Alfani (2013) in the context of Europe. The findings thereby also contribute an explanation for the heterogeneity in fertility dynamics across regions that eventually converged in the context of changes in transportation and migration, as recently documented by Daudin et al. (2019).

Our findings also complement evidence that fertility reductions in Germany and France were linked to increased education (Becker et al. 2010, 2013, Murphy 2015, de la Croix and Perrin 2018) consistent with the unified growth perspective of a close link between

[1] This conjecture is in line with recent evidence reported by Galor and Klemp (2019).

[2] Higher disease exposure also exerts greater evolutionary pressure, with important implications for long-run development, see, e.g., Galor and Moav (2002). However, the lack of immune resistance to plague and the short period since the medieval outbreaks makes the evolutionary channel appear less relevant in the present context.

the fertility transition, education and economic development. This paper adds the exposure to population shocks during the middle ages as a long-run determinant of the relative timing of the transition in different regions. The empirical findings also complement evidence of higher education attainment in predominantly Protestant areas (Becker and Woessmann 2008, 2009, 2010), while Protestantism was mainly adopted in regions where the return to education was comparably high, related to, e.g., access to major trade routes of the time, which affected the demographic dynamics above and beyond the distance to entry ports of reintroduced plague outbreaks (Cervellati and Sunde 2016). The results are also consistent with a role of greater life expectancy for long-run development (Cervellati and Sunde 2013, 2015), because plague outbreaks represent infrequent epidemics that unfold their consequences through population dynamics at the macro level rather than through individual incentives for education attainment. Finally, the use of disaggregate data complements recent evidence for the role of policies, such as the introduction of public health systems, for longevity and development (Strittmatter and Sunde 2013).

The remainder of the paper is structured as follows. Section 2 describes the background of the resurgent outbreaks of the plague in Europe and the resulting hypothesis. Section 3 describes the data and the empirical strategy. Section 4 presents the main results. Section 5 provides a discussion of the findings.

2 Background and main hypothesis

2.1 The plague in medieval Europe: some background

The repeated outbreaks of the plague in medieval Europe after the Black Death of 1347, which marks one of the largest pandemics in human history, have influenced the social and cultural thinking in ways present even in today's consciousness regarding public health (see, e.g., Cantor 2002; Slack 2012). In light of the contagiousness and fast spread of the epidemic, there has been a debate as to whether the plague had always been caused by the *yersinia pestis* bacterium or potentially by some other pathogen, possibly a virus.[3] Recent DNA evidence from grave samples confirmed an infection by *yersinia pestis* throughout Europe (see, e.g., the discussion in chapter 4.06 of Campbell 2016). Nevertheless, the transmission of plague is not well understood (see, e.g., Alfani and Murphy 2017 for a survey of the state of the literature). In terms of intensity, recent research points towards substantially higher plague-related mortality than earlier estimates, indicating that the impact of the plague might have even been underestimated previously (Benedictow 2004; Alfani 2013; Alfani and Murphy 2017).

The prevalent view regarding repeated outbreaks is that the bacterium had stayed in Europe after the introduction of *Yersinia pestis* in 1347 and reproduced in rodent reservoirs

[3] Caused by the bacterium *Yersinia pestis*, the (bubonic and pneumonic) plague primarily affects mammals. Without treatment, *yersinia pestis* leads to an infection in the entire body (sepsis) and subsequent death. Transmission of the disease can occur through direct contact or ingestion, but transmission is mostly through fleas (in particular the oriental rat flea *Xenopsylla cheopis*), which acquire the pathogen from mammals through blood meals.

in wildlife or urban environments. From these reservoirs, repeated spontaneous outbreaks were thought to have led to waves of plague in Europe, until the disappearance of the plague during the 19th Century (see, e.g., Davis 1986; Keeling and Gilligan 2000). The origins and dynamics of these outbreaks have been an issue of some debate in the literature (see, e.g, Cohn 2008 for a survey). Outbreaks have been related to the relative abundance of host populations and vector populations (Reijniers et al. 2012).[4] Schmid et al. (2015) conjectured that instead of persisting in hidden reservoirs in Europe, *Yersinia pestis* was repeatedly reintroduced from Asia following particular climatic conditions that favored the outbreak and spread of the pathogen, with the respective entry ports all located at trade points connecting Europe with trading routes to Asia. In contrast, newer work based on ancient DNA (aDNA) analysis has been able to show that later European outbreaks of plague were caused by strains of *Y. pestis* that are related to the strains found in burial cites of victims of the 14th century Black Death (e.g., Bos et al. 2016 report evidence for burial sites of victims of the outbreak of 1722 in Marseille, France, whereas Seifert et al. 2016, report genetic similarities in aDNA of *Y. pestis* strains across different burial sites in Germany that span 300 years, and Spyrou et al. 2016, report similarity of *Y. pestis* strains among plague victims of the Black Death in Barcelona and two subsequent historical outbreaks in Russia and Germany). This evidence supports the view of reoccurrence of the plague in Europe from local reservoirs, although the location of these reservoirs is still debated. While it seems accepted by now that the bacterium can survive and remain active in soil for prolonged periods (e.g., Ayyadurai et al. 2008), recent evidence points at plague foci close to the sea as consequence of the salt tolerance of *Y. pestis* (Malek et al. 2017). This is consistent with the finding that plague outbreaks in Europe can be traced back to outbreaks in the vicinity of ports or to maritime imports from other cities. In fact, outbreaks at the beginning of the chain of maritime transmissions can be isolated as outbreaks for which there was no earlier plague outbreak (within a time span of two years) within a 500km radius on land, or 1000km radius for harbors. Figure 1 provides a map of the location of the main entry ports of outbreaks of new waves of the plague.

2.2 Empirical hypothesis

Regardless of the underlying reasons for renewed outbreaks of the plague during the middle ages, new waves of the plague repeatedly spread across Europe, initiating from ports and spreading through overland trade routes and waterways. The spread of the plague was related to human interaction, and the speed of this spread has been estimated to have been several kilometers per day (e.g., Benedictow 2004). Hence, geographic location to a large extent determined the exposure to plague outbreaks, with cities and regions closer to the ports where the new waves originated facing a higher risk of being hit by a new outbreak. Due to this opaque and irregular pattern, outbreaks of the plague were taken as random events, possibly caused by metaphysical or other forces (Cantor 2002). As consequence, there was no systematic migration related to the infrequent outbreaks of the plague that would indicate that individuals avoided particular ports and the related trade routes. Moreover, recent work by Skog and Hauska (2013) suggests that the spread of the Black Death in Sweden in 1350 is well approximated by travel distances on the medieval road network,

[4] The outbreaks and transmission dynamics have also been shown to heavily depend on climatic conditions, which might have favored a synchronization of host and vector populations, and thereby an increased risk of an outbreak, as documented by evidence from Asia (Stenseth et al. 2006; Kausrud et al. 2007; Cohn 2008; Samia et al. 2011).

and evidence by Dittmar and Meisenzahl (2020) and Yue et al. (2016) indicates that locations close to ports, rivers and trade routes were particularly affected by the diffusion of the plague.[5] Taken together, this suggests that, ceteris paribus, the mortality shocks caused by outbreaks of the plague were more frequent and intense in locations closer to the ports where the new plague waves originated.

With medieval Europe being governed by a Malthusian population regime, repeated outbreaks of the plague were followed by higher birth rates that compensated the population loss in the aftermath of the outbreak (see, e.g., Keyser 1941). The loss of lives caused by an epidemic outbreak also led to a temporary scarcity of labor and increased land-labor ratios, which favored more land-intensive production in terms of animal husbandry, and in the medium run led to increased urbanization, birth and death rates, and ultimately to a transition from one Malthusian regime to a another Malthusian regime with higher population density (Voigtländer and Voth 2013a, b). In a longer perspective, greater population density fostered the demand for skills, while lower fertility, in turn, implied lower opportunity costs for undergoing the demographic transition, from quantity to quality investments in children. This development was accompanied by institutional changes, for instance in inheritance rules, that were triggered by repeated plague outbreaks (see, e.g., Alfani and Di Tullio 2019) and ultimately constituted the basis for the economic take-off (Galor 2011). Hence, regardless of the (potentially multiple) mechanisms that were triggered by the population shocks due to epidemics, the consequences and the resulting change in fertility behavior were presumably more prevalent and powerful in the locations hit harder by the plague.

The core hypothesis that follows from this discussion is that greater exposure to the plague might have accelerated the demographic development and ultimately led to an earlier fertility transition. By spreading from city to city, the outbreak of the plague might have had a major impact on many cities. Importantly, however, this impact was likely to be heterogeneous, depending on the location of the city which determined the exposure to the occasional outbreaks of the plague. Hence, cities and regions that were more exposed to these outbreaks faced more frequent and pronounced population shocks and, ceteris paribus, a faster demographic development along the lines outlined before. In particular, the greater exposure to plague outbreaks is expected to be reflected by an earlier onset of the fertility transition. This paper provides a reduced form analysis of the effect of variation in the exposure to repeated plague outbreaks on the timing of the fertility transition across regions in Germany and France.[6]

3 Empirical methods

3.1 Empirical approach

The empirical analysis tests the hypothesis that cities with greater exposure to plague epidemics experienced an earlier fertility transition during the 19th Century. The analysis is based on a simple linear regression model

[5] Conversely, the spread of epidemics like the plague has been used as proxy for relative trade intensities, which is consistent with the approach taken here, see, e.g., Boerner and Severgnini (2014).

[6] Since the focus of this paper is on the long-run development implications of population shocks, and for reasons of data availability, this study focuses on variation in the timing of the fertility transition across regions in Germany and France and does not investigate the short and medium-run implications of the plague for development in urban versus rural areas, as done, e.g., by Alfani (2013).

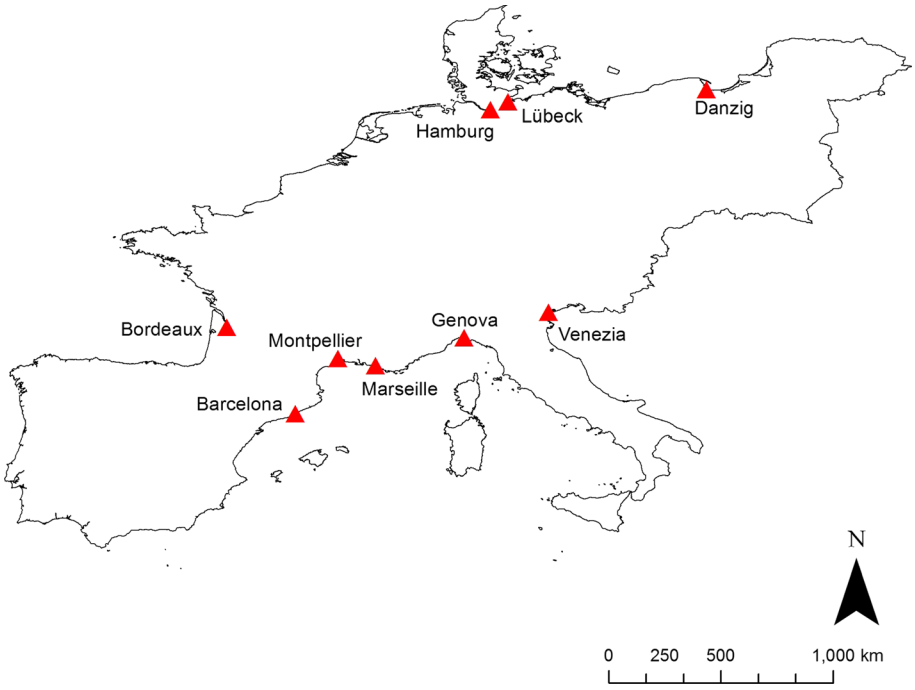


Fig. 1 Plague reintroductions in Europe. Triangles denote main plague entry ports in terms of maritime harbors exhibiting plague outbreaks that are not related to nearby land-based or maritime outbreaks. In particular, the entry ports represent the cities that exhibited plague outbreaks that had not been preceded by a plague outbreak on land within a 500 km radius and on harbors within a 1000 km radius for two years prior to the outbreak. Source: Biraben (1975, 1976) and Keyser (1974)

$$Transition\ Year_i = \beta_0 + \beta_1 Plague\ Exposure_i + \gamma X_i + \varepsilon_i \tag{1}$$

where i indicates city, $Transition\ Year_i$ is the year of the onset of the demographic transition, which is measured in terms of the onset of the fertility decline, and $Plague\ Exposure_i$ is the exposure of city i to the plague. X is a vector of control variables, which include other relevant determinants of the timing of the fertility transition. The empirical analysis accounts for cities located within the same administrative region by clustering the standard errors correspondingly.[7]

The identification of the coefficient of interest, β_1, requires a reliable measure of plague exposure that is exogenous, conditional on variation captured by the control variables included in the vector X_i. The focus on city-level data in Germany has the advantage of comparing variation across environments that are otherwise rather comparable, other than when comparing across countries or even world regions like Europe and Asia. The obvious limitation of this approach is that the analysis does not provide insights as to why the demographic transition happened earlier in Europe than in other parts of the world.

[7] The data by Knodel (1974) is based on 56 regions.

The main problem in this context is that existing measures of plague exposure, such as counts of outbreaks are fairly coarse measures while more detailed measures like plague-related casualties are fraught with measurement error and potential problems of third factors. Moreover, by construction, the timing of the fertility transition, which took place in the late 19th and early 20th century, was related to the long-run exposure to plague epidemics centuries earlier through potentially various, mutually non-exclusive channels whose consequences unfolded over time.

To address these issues, the empirical strategy is based on the use of proxy measures for the exposure to repeated plague outbreaks in history. In particular, the main measure is the number of outbreaks affecting cities during different historical phases, which is coarse but measured fairly reliably. To account for geography as a potential driver of outbreaks, we also apply geographic proxies of the exposure to plague outbreaks. In particular, we use exposure in terms of the travel distance to the nearest entry port for new plague waves. The use of this geographic proxy enables various approaches to identify the effect of plague exposure on the timing of the fertility transition, either in a reduced form approach in addition to information about outbreaks, or in combination with outbreaks in terms of an instrumental variables approach.

According to the empirical hypothesis, β_1 is expected to be negative, in the sense that greater exposure to the plague led to an earlier fertility transition. The identification of the effect rests on the assumption that exposure to repeated outbreaks of the plague (in terms of number of outbreaks and location relative to entry ports) is conditionally exogenous to the timing of the fertility transition of a city. The key issue for identification is therefore to account for confounding factors, such as access to trade that would affect the timing of the demographic transition during the 19th century, e.g., by affecting the demand for human capital, or other historical or geographical features that might be picked up by the measure of exposure to repeated outbreaks of the plague. We thus apply specifications with various sets of controls, including geographic controls such as population controls, religion controls, institutional controls, controls for agricultural yields, and exposure to wars.

While these extensive controls help isolating the role of plague exposure, it is hard if not impossible to disentangle the role of different mechanisms through which plague exposure influenced the timing of the fertility transition as consequence of the singular nature of the fertility transition and the resulting restriction to the availability of cross-sectional variation in the timing of the fertility transition. For instance, an important determinant of plague exposure is related to closer access to the main trade network of medieval Europe, since plague contagion is related to human interaction. While it should be clear that the empirical analysis in this paper is confined to a reduced form approach, it is nevertheless possible to disentangle the role of contemporaneous trade access during the 19th century, which might have had a direct effect on the timing of the demographic transition, from the variation in long-run effects of plague exposure that is related to access to medieval trade by exploiting variation in the importance of trade ports over time. Below, we present results from an extensive number of robustness checks.

3.2 Data

Fertility transition data The baseline analysis is conducted for Germany. The demographic information central to our analysis is the timing of the fertility transition. The main

data source is Knodel (1974), who provides detailed data on the on fertility and age distribution of the population in Germany on a regional level within the boundaries of 1900 (district boundaries from 1901).

Marital fertility rates, which provide the most reliable source of fertility data, are used to calculate the onset of the fertility transition for 237 cities in 56 German regions based on data covering the time from 1871 to 1939. Among a variety of definitions of fertility rates, Knodel (1974) puts most emphasis on the marital fertility instead of total fertility, which also includes illegitimate births since these are more likely to be misreported as result of social pressure. The marital fertility rates take into account different age distributions in different German regions, and thus provide a comparable measure of fertility in terms of the actual number of births during a year relative to the potential fertility.[8] The onset of the fertility transition is defined as the year in which marital fertility reached a threshold.[9] There is some arbitrariness associated with this definition, since it does not measure the onset of the decline in fertility, but the time of reaching a threshold. However, at the same time this definition is transparent and avoids confusion of the onset of the fertility transition with a temporary decline or fluctuations in fertility, e.g., due to a war or German unification. Figure 2 provides a map that illustrates the timing of the fertility transition. To demonstrate the robustness and external validity of the main finding, below we also consider the timing of the fertility transition in France using data from the Princeton European Fertility Project (Coale and Coats-Watkins 1986).[10]

Plague outbreaks We constructed the total number of plague outbreaks in a given city, which serves as proxy for plague exposure, by using information for plague outbreaks in cities by Biraben (1975, 1976). In particular, for each outbreak in Biraben (1975, 1976), we construct a bandwidth in terms of time (1 year for exact match, 3-year window for outbreaks ±1 year, 5-year window for outbreaks ±2 years).

We verified and complemented these data using information about plague outbreaks and their timing in records from city archives compiled by (Keyser 1974) and with other sources of information about plague outbreaks in cities taken from Büntgen et al. (2012). While the precise timing of outbreaks is likely fraught with measurement error, the cumulative counts of outbreaks deliver relevant information of plague exposure for the purpose of this study.[11]

Travel distance from plague entry ports The second proxy for the exposure to plague outbreaks is the geographic travel distance from the initial entry ports depicted in Fig. 1. These ports are: Danzig (Gdansk), Hamburg and Lübeck, Venice, Genova, Marseille, Montpellier, Bordeaux, and Barcelona. The final dataset is constructed on the basis of about 5.7 million road/river segments with elevation data at both the start and end of each

[8] The marital fertility is defined as $I_g = B_L/(\sum_i m_i F_i)$ where B_L is the number of legitimate births, m_i is the number of women in the (five-year) age interval i, and F_i is the age-specific natural fertility, proxied by the fertility of a married Hutterite woman in 1921-1930, see also Table A.3 in the Supplementary Appendix and Henry (1961).

[9] Consistent with the interpretation by Knodel (1974), this threshold is taken to be 0.5 in the baseline analysis. In robustness checks, we consider an alternative threshold of 0.6.

[10] In fact, the data collected by Knodel (1974) constitute the basis for the data on Germany contained in the Princeton European Fertility Project.

[11] Even greater concerns apply to data of plague casualties before the outbreak of the 30-year war, e.g., by Biraben (1975, 1976), or Büntgen et al. (2012), or mortality data (in terms of the percentage of the population killed by the Black Death after 1348) that has been constructed for selected cities by Olea and Christakos (2005), which is why we refrain from considering data on casualties.

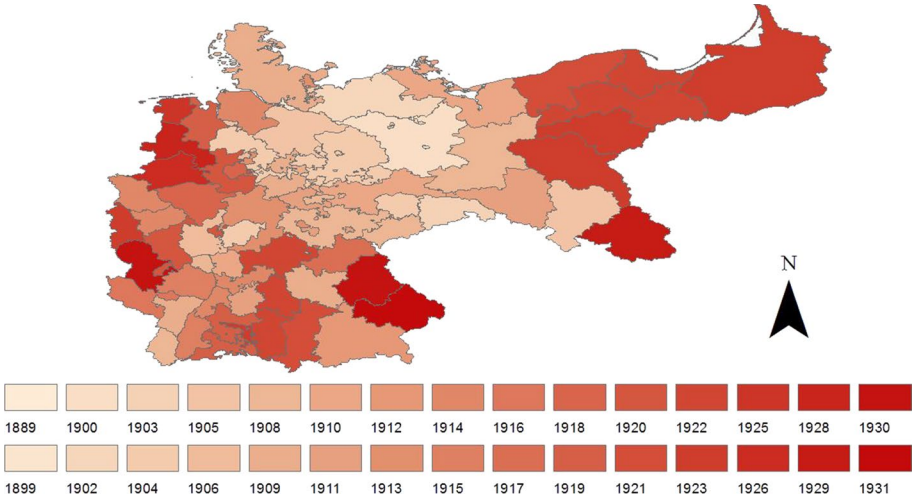


Fig. 2 The timing of the fertility transition in Germany. Districts colored by the year of the fertility transition (threshold 0.5) according to Knodel (1974). Map projection: Europe Equidistant Conic (Color figure online)

of these line segments. The data covers continental Europe West of, and including, Poland and the Czech Republic. In order to measure the travel times from the harbors to the different cities, we combine data from two sources. The data for the road and river network of Europe is taken from Openstreetmap.org via MapCruzin.com. These data comprise of about 8 million line segments, representing roads and about 2 million line segments representing waterways in all over Europe and parts of western Asia. The dataset includes countries ranging from Portugal to parts of western Russia and Turkey.

The additional data for the elevation is taken from DIVA-GIS. This data is available for each country and provides precise elevation data for a fine raster. The elevation data for the individual countries was merged to create an elevation profile for Western and central continental Europe.[12]

The travel distance is constructed from a road map that is based on contemporaneous road network, adjusted for historic travel times. In order to ensure the validity of this measure, the basic dataset is adjusted as follows. Historically, the existing roads in Europe were continuously developed up to the road network observed today. This has been done mainly by expanding existing roads. The most prominent examples for this are Roman roads like the "Via Appia" that are still used today. Obvious deviations are, e.g., the system of motor ways (Autobahn) which was built for a completely different purpose and without historic predecessors. Hence, motor ways and other constructions that were obviously not in place in medieval and early modern times, such as tunnels and canals, were excluded from the dataset. This implies a rather realistic dataset for measuring the distances, especially in areas with

[12] In order to check the accuracy of the elevation data, the DIVA-GIS elevation data was compared to the elevation data provided by Bosker et al. (2013). The reported elevation difference was in the range of up to four meters. The difference could be a result of a different raster size of the elevation data. Furthermore, the maximal elevation difference of four meters lies well in the range one can expect to be within a certain city.

mountains such as the Alps.[13] For some robustness checks, we also distinguish between roads within the borders of the Roman Empire at maximum extent, and roads in areas that were never under the control of the Roman empire, taking into account evidence that road density in Europe is still heavily affected by Roman influence (see, e.g., Dalgaard et al. 2018).

A comparison between maps of the historical road network in Germany during the 19th century and the network obtained by this methodology confirms its validity. To illustrate this, Fig. 3 provides a direct comparison for the region around Leipzig, Halberstadt and Wittenberg. Panel (a) shows the map of this region with medieval trade routes as depicted in the atlas of hanseatic routes by Bruns and Weczerka (1962). Panel (b) shows the digitized data for roads. All streets that have been used for determining travel distance are shown in grey, the most important hanseatic routes are marked with red (including the modern street labels and numbers). These are the basis for the computation of travel distance in terms of time as discussed below.

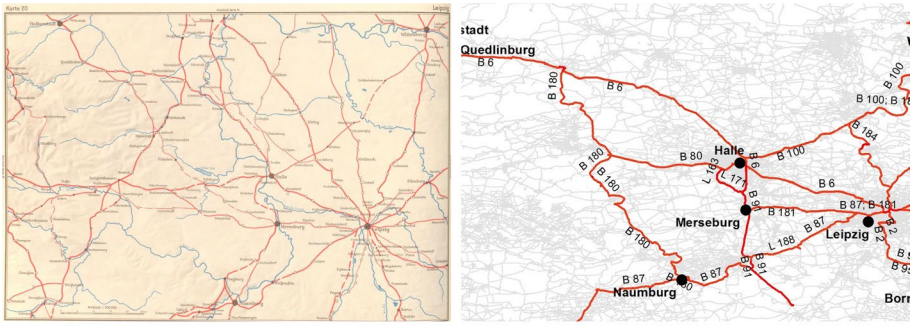
Figure 4 shows the projected road and river map of Europe.[14] In addition to the road and river network shown in Fig. 4, the data use about 5.7 million line segments including detailed elevation data. Using elevation data at both ends of these line segments, we computed the absolute difference in elevation over the length of the line segment and calculated the corresponding slope of the line segments (in degrees) as the arctangens of the elevation difference relative to the length of the segment.

Based on the information from the road and river network map and the slopes of the street segments, we calculated the travel time from the individual ports to each city. The travel times depend crucially on the assumptions about travel speeds for the different means of transportation. Transport via ships on rivers used to be substantially faster than traveling by foot. Transport of people and goods over land was mostly performed by horse coaches, which were just little faster than walking. The average speed of travel was around five to seven kilometers per hour (Ritter 1966, p. 28). This corresponds to alternative sources according to which goods transport was possible at a speed of about 30 kilometers per day (in flat areas up to 40 kilometers), which corresponds to about six to eight hours at a speed of five kilometers per hour (Bruns and Weczerka 1962, 1967). Similarly, historical accounts of mail deliveries over long distances managed travel speeds of approximately 5.5 to 6.5 kilometers per hour (Hitzer 1971). Since for horse coaches it was virtually impossible to travel on very steep roads, the travel time decreases with the slope and roads with a slope of more than 45^o constituted a natural barrier. Hence, following this literature, we assume specific travel speeds by surface type and slope of line segment.[15]

[13] Furthermore, areas that are not relevant for the empirical analysis, such as Turkey and Russia, were excluded from the data.

[14] The map projection is only relevant for illustrative purposes and does not affect the empirical results. Travel times are calculated using the segment lengths included in the road network data throughout the empirical analysis.

[15] See Table A.4 in the Supplementary Appendix for details. For slopes steeper than 45^o, the transport was mostly done by physical man labor for purposes other than travel. Even today the transport of food and other necessary equipment to remote cottages in the hills is done by carrying. Line segments with a slope larger than 45^o are assigned a speed of zero and are thus assumed to be bypassed on other roads. Obviously, the measured time depends on the assumed travel speed. The precise assumptions about the travel speed itself are irrelevant for the empirical analysis (and only affect the size of the coefficient). The important feature is the relative decline in the travel speed for the different slope brackets. Since assigning the travel speed contains an arbitrary element, this constitutes the most serious threat to validity. The main problem is that there are not many sources that provide reliable travel speeds at the medieval times, other than that traveling was exhausting and took a long time. In order to check for robustness, the regression is performed with alternative speed schedules, with similar results as shown below.



(a) Historical Road Map **(b)** Digitized Road Map

Fig. 3 Comparing historical and contemporaneous road networks. Panel (**a**) depicts a map of historical hanseatic trade routes reproduced from Bruns and Weczerka (1962). Panel (**b**) depicts the digitized road map that is used to determine the distances and travel times for the empirical analysis. Map projection: Europe Equidistant Conic (Panel **b**)

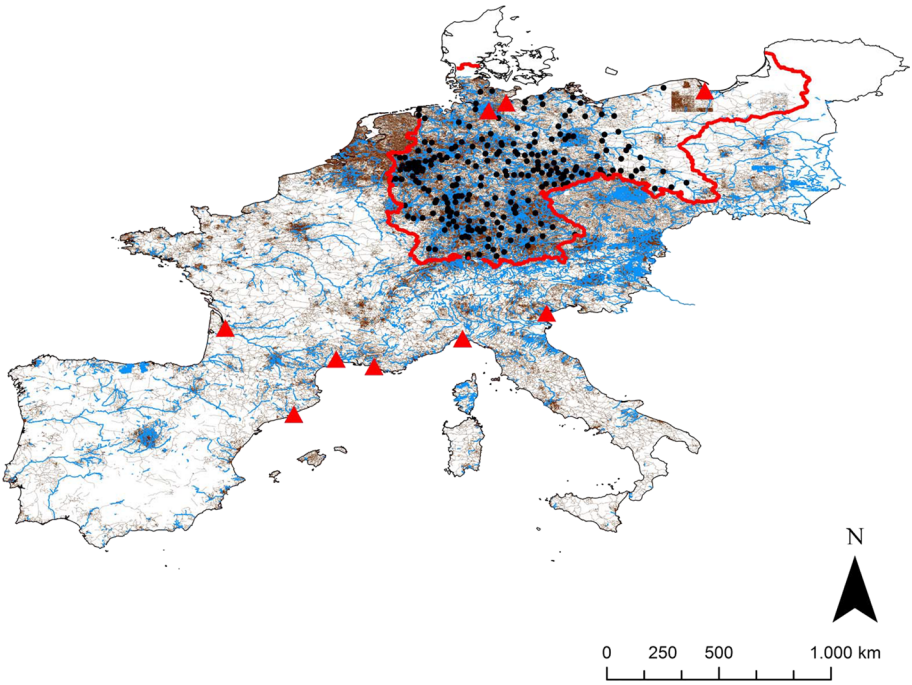


Fig. 4 Travel distances from entry ports. Map of roads (brown) and waterways (blue) used to compute travel distances from entry ports (red triangles). Cities are depicted as black dots. Red line represents German border as of 1900. Map projection: Europe Equidistant Conic (Color figure online)

The travel time is consequently defined as the minimum time necessary to travel the distance of the line segment given the speed restriction of the slope. The travel times to entry ports are then calculated in two steps. First the time to cover the particular line segment is assigned to the individual line segment given surface type and slope as described above. In the second step the path with the shortest sum of travel times is selected among

all paths, and the total travel time from each entry port to each city is computed using the Dijkstra-algorithm (Dijkstra 1959). The resulting variable *Travel Time* represents the travel time in hours from the closest port to the respective city. This variable serves as proxy for the relative risk of being exposed to outbreaks of the plague, which are expected to have occurred more often the closer the nearest entry port. The use of the travel times, rather than the simple distance, is essential to the analysis, since the spread of the plague requires human contact to infected hosts and vectors. The simple horizontal distance is therefore an inadequate proxy for the relative risk of being exposed to plague epidemics, since remote places were less likely to be affected by an outbreak.

Other variables To account for relevant heterogeneity across cities and regions, we use additional information from various sources. The analysis controls for access to the trade network of the Hanseatic League, as well as distance to trade ports that became important after the discovery of the Americas and to the main trade ports of the 19th century. City-level controls for religion, the associated cultural differences, as well as for specific institutions, are taken from data constructed by Cantoni (2012). This data set includes 237 cities in Germany and Austria, with information about population at various points in time and other background information. In particular, the data include binary indicators that denote whether a city was considered protestant after the 15th and 16th century, respectively, whether a city belonged to the Hanseatic League, whether a city was considered a free imperial city, whether a city had a printing press by the year 1517 or whether a city had a university or was located on a navigable river.[16]

To account for agricultural potential, we use information about the soil suitability for agriculture (in terms of caloric yield of the most important crop) based on data constructed by Galor and Özak (2016).[17] Together with the measure for ruggedness, these variables provide valuable insight in the agricultural potential of a region. Additional indicator variables include information whether a city was affected by the 30-year war 1618-1648 or the 7-year war 1756-1763, in terms of plundering or other warfare events.[18]

Descriptive statistics Table 1 provides descriptive statistics for the core variables of the analysis, the onset of the demographic transition, plague exposure in terms of the number of plague outbreaks in different time periods and in terms of travel distance (in hours) to the closest entry port for new plague outbreaks, and other control variables.

4 Results

4.1 Plague outbreaks and the timing of the demographic transition

As a first step, we regress the timing of the demographic transition in Germany, measured as the year in which a city experienced the fertility decline in terms of a marital

[16] Additional variables indicate the number of monasteries within a 10 km radius of the respective city for all monasteries and monasteries of the Order of Saint Augustine.

[17] This variable measures the average potential crop yield in terms of calories (millions of kilo calories per hectare and year) for the most productive crop available for cultivation before 1500CE. In robustness tests, we also consider an index of land suitability for agriculture designed by Ramankutty et al. (2002) that uses the daily sum of temperature over a base temperature of 5 degree Celsius, the pH-level of the soil, the soil carbon density and a moisture index, calculated by the actual evapotranspiration over the potential evapotranspiration, in order to calculate a single number that indicate the suitability for agriculture.

[18] The information for these variables is taken from records of city archives (Keyser 1974).

fertility below 0.5 as dependent variable, on exposure to plague shocks as measured by the total recorded number of plague outbreaks before 1900 in a city. The results, which are presented in Table 2, document a consistently negative effect of the number of plague outbreaks on the year of the fertility transition. This is an indication that cities that experienced more frequent plagues also experienced an earlier fertility transition. In order to account for systematic heterogeneity that might affect this finding, we replicate the analysis by including various sets of control variables. In particular, we add controls for geography (location on a navigable river, maritime port, latitude and longitude), demographic controls (for population density in 1400, as well as for population growth between 1400 and 1600), religion controls (whether a city had adopted Protestantism by 1600, the number of monasteries per capita, and the number of Augustinian monasteries), controls for institutions (the existence of a university, membership in the Hanseatic league, the status of a free imperial city, or the presence of a printing press by the time of the protestant reformation), suitability for agriculture (in terms of caloric yield of the most important crop), and exposure of the city to wars (30-year war and 7-year war). Regardless of the specification, the coefficient estimates for plague outbreaks are significantly negative, indicating an earlier transition in line with the hypothesis. The similarity of the coefficients of interest across the different specifications suggests that the omission of relevant variables or problems due to the inclusion of endogenous ("bad") controls are unlikely to drive the result.

The coefficient estimates for these control variables also reveal a coherent pattern.[19] Consistent with historical accounts that the demographic transition occurred first in France and North-West Europe, but also with the hypothesis that greater exposure to plague outbreaks implied an earlier onset of the fertility transition, we find evidence for an earlier onset being associated with greater latitude and longitude. The results for the religion controls confirm the intuition of an earlier onset of the fertility transition in cities that adopted Protestantism. Adding controls for institutions reveals that the existence of a university or a printing press implied an earlier fertility transition, although the coefficient estimates are not significant. Greater agricultural potential, as proxied by caloric yield of the most important crop, is also associated with a significantly earlier fertility transition, supporting arguments that are based on the interplay between environmental and cultural factors (see, e.g., Galor and Özak 2016). Finally, in line with previous arguments on the role of wars, we find that a greater exposure to the 30-year war was associated with an earlier onset of the demographic transition. Together, these findings are consistent with the joint role of population shocks, access to trade, and war for the demographic development as suggested by Voigtländer and Voth (2013b). Importantly, however, accounting for these control variables leaves the main results regarding the role of the exposure to the plague for the timing of the fertility transition essentially unaffected.[20] Moreover, as suggested by the relatively low variance inflation factors, the estimation results also seem not to be affected by multicollinearity.

This finding provides a first piece of evidence that population shocks, proxied by the exposure to plague outbreaks, indeed had an effect on the timing of the demographic transition. However, while the stability of the coefficient estimates with respect to the addition of different sets of control variables indicates that plague outbreaks exert a largely

[19] See Table A.5 in the Appendix for the respective results.

[20] Estimates following the approach of Oster (2019) suggest that the degree of selection on unobservables relative to observables that would be necessary to explain away the result exceeds the selection on observables ($\delta = 3.9$ for $R_{max} = 0.6$ which corresponds to the suggested benchmark, and 1.4 for for $R_{max} = 1$).

Table 1 Descriptive statistics

	Mean	SD	Min	Max
Onset of the demographic transition (Year)	1912.4	7.6	1889	1931
Number of outbreaks (0–1900)	2.1	4.8	0	25
Number of outbreaks (0–1555)	1.0	2.8	0	16
Number of outbreaks (0–1618)	1.6	3.9	0	20
Number of outbreaks (1360–1618)	1.4	3.6	0	19
Travel time	46.7	18.5	0.19	79
Travel time (Hanseatic city)	20.6	17.7	0	66
Travel time (Cologne)	47.8	25.8	0	118
Travel time (19th century ports)	61.8	21.1	26	121
Population in 1400(log)	0.5	0.9	0	3.7
Population growth 1400–1600(log)	0.5	0.9	−2.2	3
Protestant	0.8	0.4	0	1
Monasteries (p.c.)	1.3	1.8	0	15
Augustian Monasteries (p.c.)	0.1	0.3	0	1
University	0.1	0.2	0	1
Hanseatic city	0.1	0.3	0	1
City with maritime port	0.0	0.2	0	1
Reichsstadt	0.2	0.4	0	1
Printing press	0.1	0.3	0	1
Navigable river	0.4	0.5	0	1
Caloric yield	9105.3	455.6	7614	10,109
30y war	0.5	0.5	0	1
7y war	0.2	0.4	0	1
Latitude	51.0	1.5	48	54
Longitude	10.5	2.7	6.1	18

The statistics refer to 237 cities in Germany

independent effect that is unlikely driven by third factors, the use of historical data on plague outbreaks might be problematic as these data are based on various sources of different quality and reliability. In particular, the data on outbreaks exhibit little variation and are measured very coarsely. This circumvents several shortcomings of historical data about plague-related casualties, including the lack of heterogeneity and reliability, emphasized by recent contributions (see, e.g., Alfani 2013), or comparability as consequence of different data sources and coverage (see, e.g., Roosen and Curtis 2018). Nevertheless, the results of a significant and robust relation between plague outbreaks and the timing of the fertility transition can only be seen as preliminary evidence in support of the hypothesis of the influence of plague exposure. In particular, in light of the lack of variation at the intensive margin and of the likely presence of measurement error, the size of the estimates is not very informative.

4.2 Plague exposure and the timing of the demographic transition

In order to explore the robustness of the results, and to obtain more reliable estimates, we next replicate the analysis using information from a second, geography-based measure of

Table 2 Plague outbreaks and the timing of the demographic transition

Dependent variable	Onset of the demographic transition						
Number of outbreaks (0–1900)	−0.230***	−0.216***	−0.297***	−0.322***	−0.332***	−0.326***	−0.355***
	(0.081)	(0.067)	(0.087)	(0.085)	(0.092)	(0.094)	(0.093)
Controls							
Geography		✓	✓	✓	✓	✓	✓
Population			✓	✓	✓	✓	✓
Religion				✓	✓	✓	✓
Institutions					✓	✓	✓
Agriculture						✓	✓
Wars							✓
Observations	237	237	237	237	237	237	237
R^2	0.021	0.306	0.309	0.366	0.376	0.389	0.396
Adjusted R^2	0.017	0.291	0.288	0.338	0.337	0.347	0.349
Number of cluster	56	56	56	56	56	56	56
Highest VIF	1.00	1.27	2.11	2.18	2.63	2.64	2.65

Results of OLS regressions. Geography controls include latitude, longitude, and location on a navigable river; Population controls include log city population in 1400 and population growth 1400-1600; Religion controls include protestant denomination of city in 1600, the number of monasteries per capita, and the number of Augustinian monasteries; Institutions controls include dummies for university, Hanseatic city, imperial city, and printing press present in 1517; Agriculture controls include average caloric yield; Wars controls include dummies for battle events during 30-year war and 7-year war. Robust standard errors clustered on region level in parentheses, */**/*** correspond to significance levels at the 10%/5%/1%-level, respectively

plague exposure. In particular, in a reduced form exercise we use the distance to the closest entry port for plague outbreaks in terms of travel time, in addition to the measured number of plague outbreaks, as proxy for plague exposure. Otherwise, we apply the same extensive specification of the empirical model in terms of controls as before.

The results are shown in Table 3 and document that greater exposure to plague outbreaks, proxied by greater proximity to the nearest entry port in terms of travel time is associated with a significantly earlier onset of the fertility transition in German regions. This finding is robust to the inclusion of the full set of geographic, population, religion, institution, agriculture and war controls. The effect of geographic exposure to plague outbreaks is also robust to the inclusion of the total number of plague outbreaks as alternative proxy for plague exposure. The effect of plague outbreaks remains significant but is about 25% smaller than in the estimates with a comparable specification in the last column of Table 2. This suggests that part of the effect of plague outbreaks is accounted for by geographic exposure. The results document that the demographic transition is associated with a significantly earlier onset both in cities with more frequent outbreaks and closer travel distance to entry ports of new outbreaks.[21] In addition, focusing on the number of outbreaks during different phases reveals that the association is particularly strong for the

[21] Sensitivity checks following the approach of Oster (2019) suggest that the degree of selection on unobservables relative to observables needed to erode the effect of outbreaks is $\delta = 2.6$ for $R_{max} = 0.6$ (the suggested benchmark), and 0.8 for for $R_{max} = 1$. The corresponding estimates for travel time are 0.5 and 0.2, respectively.

Table 3 Overall exposure to plague outbreaks and the timing of the demographic transition

Dependent variable	Onset of the demographic transition				
Travel time	0.214***	0.197***	0.196***	0.196***	0.196***
	(0.070)	(0.070)	(0.070)	(0.069)	(0.069)
Number of outbreaks (0–1900)		−0.261***			
		(0.088)			
Number of outbreaks (0–1555)			−0.457***		
			(0.149)		
Number of outbreaks (0-1618)				−0.360***	
				(0.109)	
Number of outbreaks (1360–1618)					−0.415***
					(0.117)
Controls					
Geography	✓	✓	✓	✓	✓
Population	✓	✓	✓	✓	✓
Religion	✓	✓	✓	✓	✓
Institutions	✓	✓	✓	✓	✓
Agriculture	✓	✓	✓	✓	✓
Wars	✓	✓	✓	✓	✓
Observations	237	237	237	237	237
R^2	0.436	0.448	0.448	0.450	0.453
Adjusted R^2	0.392	0.402	0.402	0.405	0.408
Number of cluster	56	56	56	56	56
Highest VIF	5.03	5.14	5.11	5.11	5.10

Results of OLS regressions. Travel time measures time to nearest plague port (in hours). Geography controls include latitude, longitude, and location on a navigable river; Population controls include log city population in 1400 and population growth 1400–1600; Religion controls include protestant denomination of city in 1600, the number of monasteries per capita, and the number of Augustinian monasteries; Institutions controls include dummies for university, Hanseatic city, imperial city, and printing press present in 1517; Agriculture controls include average caloric yield; Wars controls include dummies for battle events during 30-year war and 7-year war. Robust standard errors clustered on region level in parentheses, */**/*** correspond to significance levels at the 10%/5%/1%-level, respectively

number of outbreaks during later periods. Robustness checks with respect to the coding of the number of plague outbreaks confirm these findings.[22]

Overall, the findings are consistent with the main mechanism underlying the onset of the demographic transition in the canonical unified growth model, which relates to population dynamics and greater demand for human capital, as the main factors behind the onset. While the greater proximity to entry ports might also proxy for greater demand for human capital as these ports were also major medieval trade hubs, it should be kept in mind, however, that trade patterns and industry structure changed substantially from the middle ages to the 19th century, when the demographic transition took place. The coefficients suggest that geographic proximity to plague ports takes up part of the effect of plague outbreaks, which nevertheless remain a significant factor for the timing of the demographic transition.

[22] See Table A.6 in the Supplementary Appendix for details.

At the same time, the coefficient estimates individually imply that a reduction in the travel distance to the nearest plague port by ten hours, is roughly equivalent to an onset of the fertility transition that occurs two years earlier.

4.3 Robustness: disentangling plague exposure and trade

The results so far suggest that the exposure to population shocks as reflected by the exposure to plague outbreaks was potentially a key factor for the shifts in the Malthusian equilibrium that eventually gave way to the demographic transition, above and beyond a rich set of control variables. The finding that the travel distance to entry ports captures part of the effect of outbreaks is consistent with the observation that new waves of the plague repeatedly spread across Europe, initiating from ports and spreading through overland trade routes and waterways. However, this finding also raises the potential concern that the timing of the fertility transition might have been determined entirely by trade. Of course, it is very hard if not impossible to disentangle the role of plague-related population shocks from the effect of trade on the timing of the demographic transition, since trade might affect the timing directly and indirectly. Besides the direct effects related to the demand for human capital (see, e.g., Galor and Mountford 2008) there are indirect effects related to greater measurement error as consequence of lower quality of historical records in more remote towns. In the following, we present several additional analyses that address this issue.

Accounting for 19th Century Trade When considering the role of trade for the timing of the fertility transition, it is important to recognize that the plague outbreaks mainly occurred during the (late) middle ages while the fertility transition happened during the late 19th century. To disentangle the direct role of trade and plague exposure that is related to medieval trade we replicated the analysis while accounting for the differences in trade access during the middle ages and the 19th century. The analysis makes use of the fact that not all maritime harbors were recognized as entry points of new epidemic outbreaks, but all were access hubs to trade. In addition, trade networks during the 19th century had changed in comparison to the time of the plague outbreaks. This allows us to disentangle the role of the exposure to plague-related population shocks (which spread along trade routes, but much earlier in time) from access to trade during the 19th century as a direct driver of the timing of the fertility transition.

In order to explore the relevance whether a greater proximity to a port or generally a good connection to trade networks are a driver for the fertility transition, we use information about maritime ports that were not related to plague outbreaks as depicted in Fig. 1. To investigate the robustness of the main results with respect to trade access, we estimated more extensive specifications that account for access to trade in the middle ages and during the 19th century. In particular, we control (separately and jointly) for the distance to Cologne as an important trade center in medieval Germany, access to the Hanseatic trade network, accounting for the shortest way along which goods could be shipped or transported over land to the closest member of the Hanse, and controlling for the closest port that had no relevance during medieval times but during the 19th century. In particular, we use additional information about the proximity to ports that were only founded (or gained importance) after the middle ages and that were important trade hubs in the 19th century (such as Rotterdam). Notice that these estimations can also be seen as a falsification test in the sense that if access to trade was the main trigger for the fertility transition, it should be the distance to the 19th century trade ports that affects the timing of the transition, and not

the distance to medieval entry ports, which already had lost importance. The results leave the main findings unaffected, as shown by Table 4. More extensive specifications with all controls added at once show indications of multicollinearity, with the result that travel time to the nearest entry port loses significance, while the number of plague outbreaks retains a significantly negative effect throughout.[23]

Additional unreported falsification tests based on variables for access to placebo ports including or excluding Hamburg using similar variables (regarding the number of maritime ports in a perimeter of 100 hours travel time and the distance to the closest maritime port for maritime ports that were not entry ports for the plague) confirm the main results and document that the findings are not merely driven the distance to maritime harbors. In particular, while the main results for plague exposure are unchanged, these estimates deliver no evidence for an influence of the placebo ports on the timing of the fertility transition.

2SLS Results: Plague Exposure and Plague Outbreaks As a second robustness check regarding the distinct role of plague exposure and medieval trade and to explore whether and how the main results are affected by measurement error, we report the results of an instrumental variables estimation. This analysis is based on the idea that if travel distance to the entry ports represents a proxy for plague exposure that exhibits sufficient (continuous) variation, this variable can be used as an instrument for the number of plague outbreaks in the most extensive specification of control variables. Notice that the validity of an instrumental variables approach requires relevance of the instrument in terms of a first stage relationship between travel distance to entry ports and outbreaks, and validity in terms of an exclusion restriction. The latter appears implausible as far as a direct effect of medieval trade on the timing of the demographic transition is concerned, even when accounting for trade, since this effect might be accumulative. However, in the context of accounting for measurement error in plague outbreaks, the IV approach promises new insights regarding the effect sizes and channels. Since the geographic distance from entry ports is unlikely to be relevant for the reliability of the count of plague outbreaks that is based on archival information, the IV estimates deliver insights regarding the role of plague exposure in terms of outbreaks when restricting to geographic variation only.

Table 5 presents the corresponding estimation results. The first column replicates the OLS results of the last column of Table 2 for all plague outbreaks before 1900. The remaining columns of Table 5 report the 2SLS results for different specifications of the variable of plague outbreaks for the most extensive specification of controls. Throughout all specifications, the first stage results suggest that the instrument is relevant with the proxies of plague exposure being correlated (positively) with the frequency of plague outbreaks in different epochs, although the first stage is not very strong. The center panel reports the respective coefficient estimates of the first stage regressions. Consistent with the hypothesis, a greater travel distance to entry ports of new plague outbreaks is associated with fewer outbreaks.[24] The coefficient estimates for the instrumented number of plague outbreaks is negative and significant throughout all specifications, indicating an anticipation of the onset of the

[23] In particular, even though empirical specification does not include controls for latitude and longitude, the variance inflation factors are higher than in the baseline specification. Results with latitude and longitude controls and all trade controls added jointly are qualitatively similar and reported in Table A.7 in the Supplementary Appendix.

[24] Estimation results suggest that the number of outbreaks is only related to the distance to entry ports for plague outbreak, but is not just a reflection of access to maritime harbors, see Table A.8 in the Supplementary Appendix.

Table 4 Robustness: access to trade

Dependent variable	Onset of the demographic transition			
Panel A: Outbreaks 0–1900				
Travel time	0.178***	0.163***	0.200***	0.119**
	(0.044)	(0.057)	(0.045)	(0.059)
Number of outbreaks (0–1900)	−0.276***	−0.284***	−0.274***	−0.327***
	(0.092)	(0.089)	(0.092)	(0.093)
Additional trade controls				
Cologne	✓			
Hanse		✓		✓
19th century ports			✓	✓
Observations	237	237	237	237
R^2	0.427	0.414	0.427	0.458
Adjusted R^2	0.382	0.369	0.383	0.414
Number of cluster	56	56	56	56
Highest VIF	2.68	2.87	2.68	5.93
Panel B: By timing of outbreaks				
Travel time	0.119**	0.117*	0.118**	0.119**
	(0.059)	(0.059)	(0.058)	(0.058)
Number of outbreaks (0–1900)	−0.327***			
	(0.093)			
Number of outbreaks (0–1555)		−0.566***		
		(0.161)		
Number of outbreaks (0–1618)			−0.438***	
			(0.116)	
Number of outbreaks (1360–1618)				−0.490***
				(0.121)
Additional trade controls				
Cologne				
Hanse	✓	✓	✓	✓
19th century ports	✓	✓	✓	✓
Observations	237	237	237	237
R^2	0.458	0.458	0.461	0.464
Adjusted R^2	0.414	0.413	0.417	0.420
Number of cluster	56	56	56	56
Highest VIF	5.93	5.90	5.91	5.90

Results of OLS regressions. Travel time measures time to nearest plague port (in hours). Geography controls include location on a navigable river; Population controls include log city population in 1400 and population growth 1400–1600; Religion controls include protestant denomination of city in 1600, the number of monasteries per capita, and the number of Augustinian monasteries; Institutions controls include dummies for university, Hanseatic city, imperial city, and printing press present in 1517; Agriculture controls include average caloric yield; Wars controls include dummies for battle events during 30-year war and 7-year war. Additional Trade Controls include travel distance to Cologne, travel distance to the closest Hanseatic city, and travel distance to the closest 19th century trade port. Robust standard errors clustered on region level in parentheses, */**/*** correspond to significance levels at the 10%/5%/1%-level, respectively

Table 5 Plague exposure and the timing of the demographic transition: IV Results

Dependent variable	Onset of the demographic transition				
	OLS	2SLS			
	(1)	(2)	(3)	(4)	(5)
Number of outbreaks (0–1900)	−0.355***	−3.333**			
	(0.093)	(1.317)			
Number of outbreaks (0–1555)			−5.490**		
			(2.154)		
Number of outbreaks (0–1618)				−4.443**	
				(1.785)	
Number of outbreaks (1360–1618)					−4.960**
					(2.046)
First stage					
Travel time		−0.064***	−0.039***	−0.048**	−0.043**
		(0.024)	(0.014)	(0.019)	(0.018)
Controls					
Geography	✓	✓	✓	✓	✓
Population	✓	✓	✓	✓	✓
Religion	✓	✓	✓	✓	✓
Institutions	✓	✓	✓	✓	✓
Agriculture	✓	✓	✓	✓	✓
Wars	✓	✓	✓	✓	✓
Observations	237	237	237	237	237
Number of cluster	56	56	56	56	56
F-Stat in FS		6.90	7.56	6.25	5.71
Kleinbergen-Paap rk LM (p-value)		0.02	0.01	0.02	0.02

Results of OLS and 2SLS regressions. In 2SLS, the instrument is travel time measures time to nearest plague port (in hours). Geography controls include latitude, longitude, and location on a navigable river; Population controls include log city population in 1400 and population growth 1400–1600; Religion controls include protestant denomination of city in 1600, the number of monasteries per capita, and the number of Augustinian monasteries; Institutions controls include dummies for university, Hanseatic city, imperial city, and printing press present in 1517; Agriculture controls include average caloric yield; Wars controls include dummies for battle events during 30-year war and 7-year war. Robust standard errors clustered on region level in parentheses, */**/*** correspond to significance levels at the 10%/5%/1%-level, respectively

fertility transition that is quantitatively substantially larger than the effect obtained with OLS. The estimates are even larger when restricting attention to plague outbreaks before the peace of Augsburg 1555, or before the onset of the 30-year war 1618, as indicated by the results in Columns (3) and (4). Column (5) presents results for the number of outbreaks between 1360 and 1618, i.e., excluding the first wave of the Black Death that began in 1347. The significant negative coefficient estimate of number of outbreaks, which suggests an earlier onset of the fertility transition in cities that experienced more plague shocks, suggests potentially substantial attenuation of the OLS results due to measurement error, or captures an independent effect of port proximity, e.g., through medieval trade.

4.4 Robustness: additional results

Taken together, the results presented so far suggest that trade per se is unlikely to be the sole determinant of the timing of the fertility transition. Instead, plague outbreaks in the centuries before appear to be systematically related to the onset of the fertility transition.

To investigate the robustness of these results, we conducted different robustness checks. We computed the Moran statistic for spatial autocorrelation in the residual in order to rule out that the results are driven by spatial dependence (Kelly 2019). The results reveal no evidence for systematic spatial dependence in the residuals for large perimeters. Not surprisingly, however, the results are sensitive to the choice of perimeter and reveal spatial dependence for small distances, which is natural in light of the fact that the timing of the demographic transition is measured at the region level and that the distance variables to ports exhibit strong spatial patterns. In fact, the inclusion of travel distance substantially reduces the spatial dependence in the residuals.

As further robustness check, we replicated the analysis while restricting attention to outbreaks during various subperiods. This allows exploring the sensitivity of the findings with respect to particular outbreaks or eras. For instance, after the fall of Constantinople in 1453, trading routes changed as consequence of the loss of control over the Bosporus. Similarly, the end of the first wave of the protestant reformation ended with the Peace of Augsburg in 1555, which marked a change in the influence of the Catholic church in Germany. The analysis thus explores whether the main findings are affected when splitting the sample in terms of plague outbreaks before/after these dates. The results document the robustness of the estimates for the coefficient on travel time throughout the different specifications, while the coefficient on the number of outbreaks exhibits some variation. Overall, however, the results indicate a systematic relationship between a larger number of outbreaks and an earlier onset of the demographic transition.[25] Another robustness check refers to measuring travel time distance along roads within the Roman empire separately from roads outside in order to rule out that the results are driven by systematic heterogeneity related to historical patterns and institutional factors that correlate with road quality or travel time, such as Roman influence. Roman influence on roads predates the plague outbreaks but might nevertheless influence the spread of the plague by affecting road density (Dalgaard et al. 2018). The results are unaffected when measuring travel time distance distinguishing between roads in the Roman empire and roads outside to account for the persistent role of Roman institutions and the Roman road network for long-run development (see, e.g., Wahl 2017; Dalgaard et al. 2018).[26] Overall, the results are qualitatively similar and quantitatively even slightly larger when considering Roman roads, but the coefficients are not significantly different from each other and from those in Panel A. The plague exposure proxy alone explains around 25 percent of the variation in the timing of the fertility transition, whereas adding the extensive set of additional control variables does not deliver a drastic increase in explanatory power of the empirical model. The estimation results seem not to be affected greatly by multicollinearity.

To explore the robustness with respect to the particular measure of the timing of the fertility transition, we replicated the analysis using an alternative measure based on a threshold of marital fertility of 0.6 instead of 0.5 relative to maximum (Hutterite) fertility applied

[25] The respective estimation results are reported in Panel A of Table A.9 in the Supplementary Appendix.

[26] The respective estimation results are reported in Panel B of Table A.9 in the Supplementary Appendix.

to same data source (Knodel 1974). The results obtained for the alternative measure of the timing of the fertility transition are qualitatively very similar to the baseline results.[27] The results of unreported estimates also reveal similar results when weighting the distance to the nearest port by the number of plague outbreaks.

Instead of exploring the role of the exposure to repeated population shocks as consequence of plague outbreaks using city-level data, we also analyzed the data on the level of regions. Given that the main information source on the timing of the fertility transition is on the regional level (Knodel 1974), this could be viewed as a more natural level of the analysis. On the other hand, however, this means using variation in the data at a higher level of aggregation. Checking the sensitivity of the empirical results with respect to the level of aggregation also provides a sensible robustness check in general. The results confirm the findings on the city level, namely that regions with more entry ports in a reasonably close distance are associated with a significantly earlier fertility transition. Likewise, regions that are less exposed to plague outbreaks as measured by a greater distance in terms of travel time to the nearest plague entry port experience a significantly later onset of the fertility transition.[28] The coefficient estimates are quantitatively very comparable to those obtained for data on the city level. At the same time, the findings regarding an earlier transition for more densely populated regions, for regions that are predominantly of Protestant denomination, and for regions that have better access (in terms of lower distance) to major trade routes are confirmed using regional data.

We also verified the robustness of the results with respect to additional controls, such as population dynamics during the 19th century or a distinct effect of outbreaks of the Black Death of 1347-1352 and later plague outbreaks, which might indicate the prevalence of a permanent reservoir, or controls. The results confirm the main findings.[29]

A further set of robustness checks concerns the relevance of the assumptions about travel speeds and road structures. In general, historical sources on travel speeds both over land and on waterways are both rather scarce and vague. There are several ways of addressing this issue. When performing robustness checks based on a different travel time schedule or when calibrating the Dijkstra-algorithm in order to minimize the travel time avoiding roads whenever possible, and restricting to only roads, the results are qualitatively similar to the baseline results, which suggests that these results are not due to assumptions about travel speeds or transportation mode.[30]

Unreported results using the number of plague entry ports within a travel distance of 100 hours instead of the travel distance to the nearest entry port also delivered similar findings.[31]

4.5 External validity: the demographic transition in France

The hypothesis that greater exposure to population shocks such as repeated plague outbreaks ultimately contributes to an earlier fertility transition is generic and not restricted to the context of Germany. In an attempt to investigate the external validity of the empirical

[27] The respective estimation results are reported in Panel A of Table A.10 in the Supplementary Appendix.

[28] The respective estimation results are reported in Panel B of Table A.10 in the Supplementary Appendix. The travel times are computed as average of the cities located in the region.

[29] The respective estimation results are reported in Panels A and B of Table A.11 in the Supplementary Appendix.

[30] Completely deleting roads would reduce the number of observations dramatically as many cities cannot be reached solely by waterways due to their location away from rivers or within river deltas.

[31] Details are available upon request.

results, we replicated the same empirical approach for France. The case of France is particularly interesting for several reasons. France was the first country to experience the demographic transition. In addition, it has been argued that the fertility transition in Europe was influenced by social and behavioral changes that originated in France (Spolaore and Wacziarg 2019). Moreover, outbreaks of the plague to Europe occurred first in France, and much research on the Black Death has focused on France as consequence of an arguably higher data quality for plague outbreaks in France than in other parts of Europe.[32]

The extension of the main analysis to France requires the use of different data and data sources, which constrains the comparability with the earlier estimation results for Germany, particularly concerning the control variables. At the same time, this is a useful complement to the analysis for data from Germany as it provides insights into the external validity and affords greater statistical power. The timing of the fertility transition is constructed based on data from the Princeton European Fertility Project (Coale and Coats-Watkins 1986) using the same definition as for Germany.[33] Other variables are constructed from different sources. The onset of the fertility transition in France, although occurring earlier than in Germany, also exhibits considerable variation (see Figure A.3 in the Supplementary Appendix).

In terms of the specification of the estimation model, the analysis of France involves several modifications of the baseline model. First, in light of the geography, the empirical specification accounts for additional trade controls. In particular, the empirical model accounts for the distance to Cologne and Paris as the main medieval trade hubs. This also accounts for arguments that social and behavioral changes that affected the timing of the fertility transition spread concentrically from Paris (Spolaore and Wacziarg 2019). In addition, the controls also include the distance to the nearest Hanseatic city or maritime port, and to the nearest 19th century trade port that was not an entry ports for the plague. Preliminary analysis of the data for France reveals that greater exposure to plague outbreaks in terms of travel time is associated with more frequent outbreaks. However, to avoid problems of multicollinearity, the empirical specification does not include controls for latitude and longitude.

Table 6 presents the reduced form results for the proxy measures of plague exposure when including France in the estimation. The findings confirm the earlier result that a greater plague exposure, as proxied by the number of outbreaks and proximity to plague entry ports, is associated with an earlier onset of the fertility transition. The coefficient for plague outbreaks appears more robust and quantitatively larger in comparison to the baseline results, whereas the travel time to the nearest plague port has a positive but insignificant coefficient. The size of the coefficient estimates as well as the findings for outbreaks during different periods are also comparable.[34]

[32] For instance, the coverage of the data by Biraben (1975, 1976) is comparably high for France, while even here full availability and comparability of sources is not ensured, see, e.g., Roosen and Curtis (2018).

[33] As baseline, we code the first year in which marital fertility reached a threshold of 0.5 as the onset, for robustness we also conducted robustness checks for an alternative threshold of 0.6, with similar results.

[34] Additional results also confirm the main findings for an extended specification that includes controls for latitude and longitude, although considerably higher variance inflation factors advise caution when interpreting the results due to potential multicollinearity, see Appendix Table A.12. The qualitative patterns are similar when restricting attention to France only, although for the sample of France the coefficients for travel time appear statistically more robust, or when applying the 2SLS approach, see Tables A.13 and A.14 in the Supplementary Appendix.

Table 6 Plague exposure and the timing of the demographic transition: external validity

Dependent variable	Onset of the demographic transition			
Panel A: outbreaks 0–1900				
Travel time	0.059	0.123	0.017	0.040
	(0.146)	(0.167)	(0.126)	(0.148)
Number of outbreaks (0–1900)	−1.427***	−1.396***	−1.000**	−0.994**
	(0.417)	(0.419)	(0.397)	(0.399)
Additional trade controls				
Cologne/Paris	✓	✓	✓	✓
Hanse/Maritime ports		✓		✓
19th century ports			✓	✓
Observations	518	518	518	518
R^2	0.446	0.451	0.548	0.548
Adjusted R^2	0.434	0.438	0.537	0.537
Number of cluster	136	136	136	136
Highest VIF	1.96	2.11	6.39	6.53
Panel B: By timing of outbreaks				
Travel time	0.040	0.032	0.039	0.039
	(0.148)	(0.149)	(0.148)	(0.148)
Number of outbreaks (0–1900)	−0.994**			
	(0.399)			
Number of outbreaks (0–1555)		−1.202*		
		(0.636)		
Number of outbreaks (0–1618)			−0.988**	
			(0.490)	
Number of outbreaks (1360–1618)				−1.049**
				(0.518)
Additional trade controls				
Cologne/Paris	✓	✓	✓	✓
Hanse	✓	✓	✓	✓
19th century ports	✓	✓	✓	✓
Observations	518	518	518	518
R^2	0.548	0.545	0.546	0.546
Adjusted R^2	0.537	0.533	0.534	0.534
Number of cluster	136	136	136	136
Highest VIF	6.53	6.52	6.55	6.57

Results of OLS regressions. Travel time measures time to nearest plague port (in hours). Geography controls include location on a navigable river; Population controls include log city population in 1400 and population growth 1400-1600; Religion controls include protestant denomination of city in 1600, the number of monasteries per capita, and the number of Augustinian monasteries; Institutions controls include dummies for university, Hanseatic city, imperial city, and printing press present in 1517; Agriculture controls include average caloric yield; Wars controls include dummies for battle events during 30-year war and 7-year war. Additional Trade Controls include travel distance to Cologne, travel distance to the closest Hanseatic city, and travel distance to the closest 19th century trade port. Robust standard errors clustered on region level in parentheses, */**/*** correspond to significance levels at the 10%/5%/1%-level, respectively

5 Conclusion

This paper presented an empirical investigation of the hypothesis that cities or regions that were more (and more often) exposed to major population shocks related to the medieval plague experienced an earlier fertility transition. This hypothesis follows from recent work on the long-run implications of plague outbreaks and complements existing evidence that has not considered the timing of the fertility transition. The findings provide novel evidence in line with the implications of earlier contributions predicting that the population shock associated with the plague led to shifts in the Malthusian equilibrium and ultimately accelerated the mechanisms behind the demographic transition as predicted by the canonical unified growth framework.

Given the importance of the demographic transition for long-run development, the evidence shown here provides new insights into the reasons for regional development differences that might be related to historical coincidences and points to various directions for future research regarding the underlying mechanisms. The literature has described several candidate mechanisms working through fertility which are related to age of first marriage or knowledge and attitudes regarding birth control. However, direct evidence on these mechanisms is scarce and empirical findings indicate no clear correlation between age of first marriage and total fertility. Others have pointed to changes in the population composition as result of plague shocks, which are related to gender ratio, age composition, and household size. Finally, repeated plague outbreaks might have led to the adoption of institutions, e.g., regarding inheritance rules, that might have ultimately led to a reduced demand for children and an earlier demographic transition. More evidence is needed to shed light on the empirical relevance and relative importance of these mechanisms. In addition, the analysis here has focused on the timing of the demographic transition across regions in Europe. More work is needed for a better understanding why the demographic transition occurred earlier in Europe than in other parts of the world such as China that experienced similar plague-related population shocks.

Funding Open Access funding enabled and organized by Projekt DEAL.

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