




SPECIAL ISSUE ARTICLE

Immigration, Poverty, and Infant and Child Mortality in the City of Madrid, 1916–1926

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Abstract

In this paper, we study differential infant and child mortality according to the origin of the mothers, natives of Madrid or immigrants, between 1916 and 1926. From 1880 to 1939, Madrid experienced spectacular demographic growth, with a massive influx of immigrants, mainly from the Castilian Plateau. Using the city's records of births and deaths, which we linked for the study period, we demonstrate an important spatial heterogeneity in infant and child mortality across the city. Although the development of the town was planned in the 1860s, the infrastructure and the real estate market were overwhelmed by the continuous arrival of new inhabitants. Moreover, major investments in public health increased the gap between the wealthy districts and peripheral areas. These improvements deepened inequality. During years marked by the waves of the influenza pandemic, we isolate the impact of poverty, which threatened the survival of newborns through poor nutrition, deficient hygienic infrastructures and deplorable housing conditions. Such features explain the impressive association between summer and the risk of dying from enteritis, diarrhea and other diseases of the same type among weaned children. However, the mortality differentials between the offspring of native and migrant mothers were surprisingly small, which we explained in terms of behavioral adaptation to the large city and its mass society.

Introduction

In this paper, we study the differential infant and child mortality in Madrid between 1916 and 1926, according to the birthplace of the mothers. Historical demographers have studied the integration of newcomers into growing urban populations through the lenses of intermarriage (Dribe et al. 2018; Weiss and Stecklov 2020; Pagnini and Morgan 1990) and fertility (Kulu et al. 2019; Moreels and Vandezande 2012; Schumacher et al. 2013). The immigrant mortality paradox is also well known, describing the undermortality of international immigrants but also of rural-urban migrants because of their selection among healthier people (Kesztenbaum and

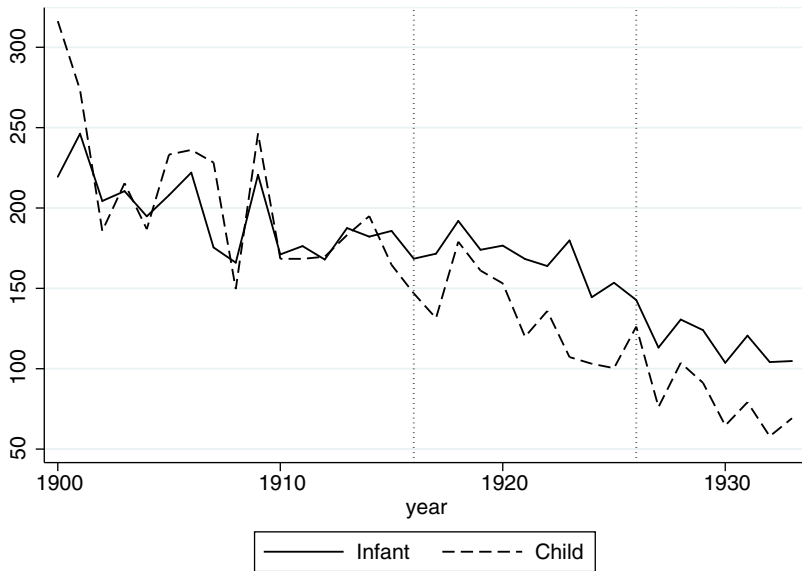


Figure 1. Infant and child mortality in Madrid, 1900–1960.

Source: Adapted from Diego (2008).

Rosental 2011; Oris and Alter 2001; Puschmann *et al.* 2016). However, the mortality of the children of migrants has rarely been considered, and then mainly in the North American context (Dribe *et al.* 2020; Bakhtiari 2018; Olson and Thornton 2011; Preston *et al.* 1994).

To contribute to a cumulative science and question differentials in infant and child mortality by origin of the mother, early 20th-century Madrid is a relevant case. Migration was the main motor of the transformation of a modest town that counted 279,370 inhabitants in 1860 into a dominant metropolis that approached one million in 1930 (Ramiro-Fariñas *et al.* 2021). The capital of Spain attracted 21 to 23 percent of all permanent Spanish internal migrants (Silvestre 2005). Most of the newcomers were unskilled workers coming from the large surrounding Castilian Plateau, but a population of civil servants recruited all across the country also came to work in the national capital (Ramiro-Fariñas *et al.* 2021).

Moreover, at the dawn of the 20th century, Madrid was named “the city of death” (Revinga 1901) and reputed to be a dangerous place. Its demographic expansion exceeded its infrastructure, causing a deterioration in its hygiene and housing conditions (Porrás-Gallo 2002). Between 1916 and 1926, Madrid was characterized by high infant and child mortality, with levels higher than those in Glasgow, Edinburgh, and towns in Prussia, Belgium, Sweden and the United States during the same period (Cage and Foster 2003; Vögele and Woelk 2002; Debuissou 2001; Helgertz and Önnersfors 2019; Eriksson *et al.* 2018). Madrid’s vulnerability was quite evident in the early 20th century, when the brutal waves of Spanish influenza from April 1918 to April 1919 and November 1919 to February 1920 (Cilek *et al.* 2018) did not interrupt but rather slowed down the mortality transition (Figure 1). In this transition, Spain was a late-comer at the European level, but within the country, the

decrease was faster in cities such as Madrid (Reher 2001). This has been attributed to more effective progress in water sanitation and health policies in urban environments than in rural areas (Casado-Ruiz and Ramiro-Fariñas 2018; Haines 2001; Oris and Ramiro-Fariñas 2016).

Indeed, between 1880 and 1930, the Madrid city center gained the attributes and appearance of a modern capital after important investments were made in public hygiene, the construction of new buildings, the development of national administrative entities, educational facilities, and business and shopping districts (Cardesín-Díaz and Mirás-Araujo 2017). Conversely, living conditions were extremely poor in peripheral areas. Progress was associated with growing differences between neighborhoods where inhabitants accumulated advantages and the areas the contemporaries called “barrios negros” or “barrios bajos” (meaning low neighborhoods), which accumulated disadvantages (Díaz-Símon 2016; Vicente-Albarrán 2015). Social stratification and spatial segregation characterized Madrid’s urban environment and heavily affected the inhabitants’ relationships with life and death (Ramiro-Fariñas et al. 2021).

In this paper, building on the literature and considering the Madrid-specific context, we test two hypotheses. The first builds on the “fundamental cause” theory, which considers that differences in access to both preventive and curative resources are related to socioeconomic status (Link and Phelan 1995; 2010). Migrant families tended to be concentrated in disfavored neighborhoods, to face the worst lodging conditions, to have less access to healthy food for both mothers and babies (Moch 2003; Walter and Pinol 2003; Parella et al. 2023), and consequently to be more responsive to environmental conditions (Ramiro Fariñas et al. 2021; Thiede and Brooks 2018; Preston and Haines, 1991). In brief, they were more at risk of poverty than natives, and their limited access to critical resources was the ultimate cause of the overmortality of their children (Dribe et al. 2020: 59; Bakhtiari 2018).

The second hypothesis stresses economic, social and cultural capital. We assume that children born in Madrid of mothers also born in Madrid benefited more from the ongoing progress and were less vulnerable to environmental factors. Indeed, people rooted in a city had extended knowledge of their urban space acquired throughout their life course. They were aware of both avoidable risks and available resources, for example, clean water or the existence of a dispensary. They also had a better understanding of the housing market (Dribe et al. 2020; Bakhtiari 2018; Oris and Perroux 2007; Preston et al. 1994). All this knowledge could make a difference at any time but must have been particularly important during the early stages of the epidemiological transition, when Pasteurian principles were spreading (Dribe et al. 2020: 60), as was the case in early 20th century Madrid. Additionally, natives tended to have a more powerful social network than most migrants, although the concentration of newcomers in religious and/or ethnic neighborhoods could create sociocultural enclaves where among others, child care practices from the region of origin could be maintained (Olson and Thornton 2011; Preston et al. 1994). In short, compared to those who grew up in the city, immigrants had to acquire human and social capital specific to their place of destination. This challenge was even bigger when they had to find their way in a large city (Alter and Oris 2005).

Those two hypotheses seem compatible. The first one insists on the migrants’ penalties, the second one on locals’ advantages, and both assume an excess mortality

among migrants' children. However, the promoters of the “fundamental cause” as the ultimate explanation of health and mortality differences recognized that the healthy migrant paradox (poor socioeconomic conditions but better health) contradicts their theory (Link and Phelan 2010: 15; Bakhtiari 2018: 140). What about their children?

To answer this question, the heterogeneity of the under-five mortality must be considered because the main risk factors evolved between birth and the fifth anniversary. During the first 28 days of life, neonatal mortality was mainly due to endogenous causes such as congenital malformation (Mosley and Chen 1984; Oris *et al.* 2004). Those causes were explained by biological factors, which could, however, be associated with poverty and malnutrition, which are keys for maternal depletion syndrome (Manfredini *et al.* 2020; Scalone, 2014).

For babies who survived the first trials of life, breastfeeding offered direct protection through the maternal antibodies present in the mother's milk and indirect protection as well, since the newborns have low immunity against pathogens that may be present in artificial food. Weaning was consequently a dangerous transition that opened a new period of life that saw young children facing new threats (Preston *et al.* 1994; Reid 2002; Thornton and Olson 2011). Particularly, in an urban context under strong demographic pressure, water of poor quality and spoilt animal milk could result in deadly digestive diseases (Olson and Thornton 2011; Preston and Haines, 1991; Vögele and Woelk 2002). This was especially the case when hot temperatures increased viral proliferation (Reher and Sanz-Gimeno 2006; Van Poppel *et al.* 2018). Moreover, weaned children were also susceptible to other infectious illnesses. In large and growing cities such as Madrid, population density, mobility and contacts, and crowding favored the spread of infectious diseases, especially airborne diffused diseases (Dribe *et al.* 2020; Preston and Haines, 1991), during the winter season (Breschi and Livi-Bacci 1986). Weaned children were also responsive to fluctuations in food prices, parental poverty and, ultimately, malnutrition (Oris *et al.* 2004; Dribe *et al.* 2020).

In the following sections of this paper, exploiting a new database, we test origin and sociospatial segregation as competing or complementary explanations of the differentials in infant and child mortality. Origin is a proxy of knowledge about Madrid's dangers and opportunities, while localization in a segregated urban environment is a proxy of living conditions.

Individual nominal data in Madrid

Data

To test our two hypotheses, we used the Madrid birth and death certificates for the period 1916–1926. Specifically, birth information was acquired from the Civil Register of the city for the period 1916–1925, as well as the death information from 1916 to 1926 for a total amount of 184,739 birth events and 182,133 death events, including 55,745 deaths of children under five years of age. In the hospital “*la Inclusa*” (founding hospital), many unmarried mothers went to give birth, abandoning most of their children shortly after birth, resulting in very high levels of infant mortality (Revuelta-Eugercios 2013). We excluded from our analyses

children born in the Inclusa institution to avoid confusion between de facto and de jure populations (Revuelta-Eugercios and Ramiro-Fariñas 2016).

Madrid's birth certificates are very informative. For each individual, the following information is recorded: date of birth, name (or names), two surnames for each parent, sex, place of birth within the city (district, street and number), province of birth of parents and a note that includes information on the nature of birth, such as the case of twins. A major limitation, however, is the absence of an indication of the occupation of the parents. The death certificate, on the one hand, is limited to information about the deceased individual, including date of death, name (or names), names of the parents, age at death (expressed in years, months and days) and the place of death in Madrid, the latter with a structure compatible with that of the birth certificate (district, street and number). On the other hand, a very special feature of death certificates is the systematic mention of the cause of death for each deceased individual in the city.

Several checks were carried out on the raw data, such as the calculation of the sex ratio, which remained consistent across the years of observation, as well as in the different districts of Madrid. Sex ratios were also calculated for the births from native mothers and for the births from mothers of various origins and were similarly consistent.¹ We also adjusted the Bourgeois-Pichat (1951a, 1951b) model for each group of origin. The linearity hypothesis was verified, and no significant difference was observed between the studied subpopulations. Furthermore, a crosscheck with the official statistics produced by the Madrid City Council (*Ayuntamiento de Madrid Estadística Demográfica*) confirmed, albeit with some very slight differences, the consistency of the collected data.

Linking birth and death certificates in the context of a large capital city in historical times can be a challenging task. Such a large number of events, combined with the great heterogeneity of the local population, required the use of advanced techniques and a long, meticulous data preparation to match both birth and death information in a single record. This work consisted of the removal of typing errors, upper- and lower-case letters, accents, unnecessary spaces and the standardization of the information.² During data preparation, phonetic algorithms for names and surnames were implemented to code the information and facilitate linkage.³

After the data preparation phase, the two sources (births and deaths) were linked through a probabilistic technique based on the well-known Fellegi-Sunter model (1969). Stata 17 and a *Datalink* module were used for the actual implementation of the linkage (Kranker 2018). Subsequently, a deduplication process, similarly based on a probabilistic approach, was used to identify the births from the same mother and father. The final product consists of a file containing, for each born in Madrid in the studied period, the date of birth, the date of death and the cause of death – if present – and all the information mentioned above. The final link rate was

¹The period value is approximately 106, which is fully within the compatible values in human populations, with 105–107 male births for every 100 female births (Hesketh and Xing 2006).

²By standardization we refer, for example, to the different ways in which a same name can be written in the birth or death certificate. The name “Maria”, for example, can be written in full or abbreviated forms such as “Maria”, “M.” or “M^a”.

³The use of phonetic algorithms significantly improved the efficiency of record linkage between the two sources. In particular, we use both the “SOUNDEX” and the “NYSIIS” algorithms (Vykhovanets et al. 2020).

satisfactory, reaching 94 percent for the first five years of life and rising to 95 percent when considering only the first year of life. This suggests very low levels of family mobility when children were young and is moreover consistent with the results of an analysis of the 1905 population census where duration of residence in Madrid was mentioned. This analysis showed that while single people were highly mobile, married couples durably settled. When they moved, it was mainly inside the capital (Ramiro-Fariñas *et al.* 2021).

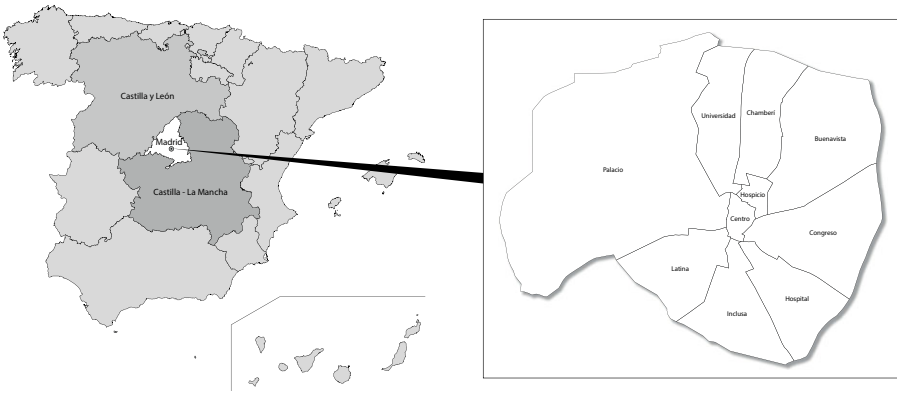
As Bailey *et al.* (2020) note, such automated methods can introduce false matches (Type I errors) and missed matches (Type II errors). However, in Spain, each person has two surnames, the first inherited from the father and the second from the mother, and this characteristic, as well as the use of the “middle name,” increases both the efficiency and the reliability of the linkage process (Bailey *et al.* 2020). Additionally, we tested the quality of the procedure through direct source control and internal coherence checks, for example, by testing the consistency of birth intervals.

The causes of death were coded using the method proposed by Bernabeu-Mestre *et al.* (2003), which aims to classify historical diagnostic expressions. It is an adaptation of the procedure proposed by McKeown (1976) that is specific to Spanish expressions and medical knowledge in Spain during the studied period. Causes of death are first divided into two large clusters: infectious diseases and noninfectious diseases. Within the first cluster, water- and food-borne infectious diseases were distinguished from airborne infectious diseases for the purposes of this paper.

Methods

Individual life histories from the first day of life until the fifth birthday were analyzed through the event history technique.⁴ Starting from a simple structure, where a person corresponds to one record, we progressively created spells following the subject’s individual history. This is particularly useful for accommodating time-dependent variables such as temperature and current season. Subsequently, individual life histories were analyzed using parametric (piecewise constant hazard) models. We preferred this approach to the Cox model (Cox 1972) because in the Piecewise constant hazard model, the baseline hazard function is specified and is exponential. In particular, within each segment, the hazard is constant, but between segments, the hazard may be different with discrete changes between intervals. One of the advantages of using a parametric regression is that the calculation of a shared frailty model is much more convenient than in a semi-parametric model, as in the case of Cox regression, which must simultaneously estimate the baseline and the parameters of the regression (Bouaziz and Nuel 2017). Therefore, we used piecewise constant hazard models with shared frailty (Gutierrez 2002) at the mother level, that is, episodes of individuals born to the same mother are identified by the same ID number. The results were reported in the form of hazard ratios (HR).

⁴Individual life histories are followed until the completion of the fifth birthday or, in the case of death, until the date of the event or the date of the end of the study (censoring), which is December 31, 1926.



Map 1. Regional subdivision of Spain and division into districts of Madrid.

For the analysis of causes of death as competing risks, we used a semi-parametric approach based on the Fine-Gray model (Fine and Gray 1999). This model provides a better estimation for the risk of the main outcome of interest when one or more competing risks exist and, unlike Cox regression, does not treat the competing event as a simple right censor. The results of the competing models were reported in the form of subdistribution hazard ratios (SHRs). All the methods of analysis mentioned are based on the proportional hazard assumption.

For the various multivariate models, to test our two hypotheses, we considered several variables that can be summarized in three clusters: environmental (hypothesis 1), mothers' origin (hypothesis 2) and control variables. The first group includes the variable "District," which refers to the place of residence in one of the ten districts into which the capital was divided at the time of this study (see Map 1). This variable is indicative of the spatial and social segregation and the diversity of housing and hygienic conditions across the city. In the same cluster, we also included two time-varying covariates: "Current season," which identifies the season of the year, and another categorical covariate, called "Temperature," which identifies the particularly warm (>95 percentile) and cold (<5 percentile) months (Institute of Statistics of the Community of Madrid 2022). The environmental group is completed by a dummy variable called "Influenza" that identifies months corresponding to the pandemic waves in Madrid (Cilek et al. 2018). The second cluster consists of a single variable divided into four categories (regions) derived directly from the province of birth of the mother. The actual regional breakdown of Spain was used as the subdivision: Native mother (mothers born in the province of Madrid, dominated by the capital city), Castilla-La Mancha, Castilla y León and other regions. Finally, we used a set of fixed control variables, which are presented and discussed below. Appendix 1 provides the table of frequency of the variables.

We ran separate models according to the age of the children, first for those aged 1 to 28 days, to isolate neonatal mortality. Starting at day one could seem strange, but under Spanish law, until 1975, those who died at birth or during the first 24 hours of life were considered "abortive creatures" (art. 745). The Spanish Civil Code literally

said: “for civil purposes, a fetus shall only be considered as born if it has a human figure and lives for twenty-four hours completely detached from its mother’s womb” (Gómez-Redondo 1985: 100). Therefore, stillbirths and babies who died during the first day of life are absent from our sources. The next age groups cover post-neonatal mortality, and to identify as much as possible the weaning timing, we separated the 29–90 days of life, 91–179, and 180–365. Finally, infant mortality (below 1 year) was dissociated from childhood mortality (between the first and the fifth birthdays).

The impact of mothers’ origins: death and survival of children from native and immigrant mothers

In the multivariate models, we controlled for several important determinants of infant and child mortality (Tables 1a–d). The excess mortality of twins, orphans or illegitimates, boys compared to girls, and children of young mothers is consistent with the results of other studies (Oris *et al.* 2004; Reid 2002) and attests to the quality of the data.

The impact of the mother’s origin (birthplace) is illustrated by Kaplan–Meier survival curves (Figure 2). Children born to Madrid natives, as well as those whose mothers came from the surrounding Castilian Plateau, had similar chances of survival during the first five years of life: between 76.0 and 77.6 percent. However, when the mother was born in another part of Spain, an advantage already appeared during the first year of life, increasing during childhood, resulting in 80.4 percent of the babies still being alive at the age of five. The multivariate model shows no difference in the first days of life. However, this model shows that compared to the children of native mothers, those from Castilla-La Mancha had a limited but significant survival advantage from 29 days until 5 years of life (11–19 percent better chances). From 90 days, those from other regions of Spain were slightly favored (6–14 percent), while those from Castilla y León were slightly more at risk (4–7 percent).

This situation in early 20th century Madrid can be compared with what was observed at the same time in the regions of origin (Figure 3). Data on infant mortality indicate that for the period 1916–1926, the levels in the Madrid province and Castilla y León were the same (approximately 170‰). Risks of dying before the first birthday were lower in the rest of Spain, and we found an indication of this advantage among the children of migrants from this origin. The infant mortality rate was, however, somewhat higher in Castilla-La Mancha (181‰), which makes the survival chances of children to mothers from this region even more intriguing.

After this descriptive analysis, we turn to the piecewise constant hazard models with shared frailty (at the mother level). To approach the impact of urban environmental conditions on the various groups of children, the district of residence of the parents emerged as one of the most discriminating factors of death below age five. It was expected, as Madrid’s expansion was associated with growing social inequalities and segregation. Several districts, especially those covering the city’s expansion to the south, showed worse living conditions than the rich and modern northern part of the capital.

The models stratified by maternal origin confirm that children from all groups saw their risks of death influenced by their place of residence in Madrid (Tables 1a–d).

Table 1a. Piecewise constant hazards models with shared frailty (mother level) on time to death. Mothers born in the Madrid province

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Buenavista ref.	1.000	1.000	1.000	1.000	1.000	1.000
Centro	0.929	0.912	1.237	1.042	1.027	1.160*
Chamberí	1.135	1.394***	1.303**	1.198**	1.243***	1.374***
Congreso	1.151	1.187	1.380***	1.122	1.197***	1.137*
Hospicio	1.012	1.017	1.037	1.295***	1.122*	1.236***
Hospital	1.346***	1.370***	1.397***	1.393***	1.386***	1.582***
Inclusa	1.521***	1.697***	1.694***	1.504***	1.586***	1.855***
Latina	1.215*	1.590***	1.674***	1.464***	1.480***	1.576***
Palacio	1.170	1.060	1.351**	1.103	1.151**	1.297***
Universidad	1.186	1.368***	1.605***	1.285***	1.343***	1.546***
Influenza	1.206***	1.284***	1.174**	1.274***	1.243***	1.607***
Winter ref.	1.000	1.000	1.000	1.000	1.000	1.000
Spring	0.630***	0.555***	0.664***	0.944	0.727***	0.994
Summer	0.733***	0.745***	1.052	1.384***	1.038	0.841***
Autumn	0.652***	0.562***	0.424***	0.526***	0.539***	0.597***
Normal temperature ref.	1.000	1.000	1.000	1.000	1.000	1.000
Very low	1.246**	1.329***	0.897	0.991	1.102**	1.016
Very high	1.028	1.033	1.295***	1.185***	1.176***	1.176***

(Continued)

Table 1a. (Continued)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Age of mother						
15–19	1.417***	1.343***	1.271**	0.945	1.185***	0.892
20–24	1.129*	1.117*	1.210***	0.918*	1.056*	0.935*
25–29 ref.	1.000	1.000	1.000	1.000	1.000	1.000
30–34	1.138**	1.004	1.213***	0.964	1.053*	1.073**
35–39	1.114	1.239***	1.336***	1.160***	1.201***	1.106**
40+	1.481***	1.264**	1.439***	1.223**	1.329***	1.135*
Age of child						
1–9 days ref.	1.000				1.000	
10–28	0.883***				0.857***	
29–90		(omitted)			0.445***	
91–179			(omitted)		0.359***	
180–365				(omitted)	0.335***	
1y – 2y ref.						1.000
3y – 4y						0.296***
Female	0.840***	0.790***	0.822***	0.901***	0.847***	0.966
Twin	6.645***	4.249***	2.944***	2.735***	4.195***	1.750***
No father	1.949***	2.006***	1.358***	0.986	1.485***	0.921
Constant	0.343***	0.154***	0.102***	0.095***	0.312***	0.051***

(Continued)

Table 1a. (Continued)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
/lntheta	1.625***	1.046	0.799	0.367***	0.457***	0.292***
Number of subjects	73,296	71,365	69,430	67,289	73,296	63,428
Number of deaths	1931	1935	2141	3861	9868	6326
Time at risk	5738	11,938	16,649	33,110	67,435	184,008
Log likelihood	–10,602	–10,732	–11,534	–18,441	–41,245	–29,770
LR test of $\theta = 0$	62***	28***	26***	21***	171***	32***

Note: * $p < .1$. ** $p < .05$. *** $p < .01$.

Table 1b. Piecewise constant hazards models with shared frailty (mother level) on time to death. Mothers born in Castilla-la Mancha

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Buenavista ref.	1.000	1.000	1.000	1.000	1.000	1.000
Centro	1.480	0.724	1.413	1.259	1.211	1.043
Chamberí	1.394*	1.131	1.698***	1.496***	1.428***	1.163
Congreso	1.531**	1.194	1.498**	1.012	1.247***	0.991
Hospicio	1.076	0.907	0.955	1.11	1.029	0.905
Hospital	1.414*	1.104	1.668***	1.394***	1.378***	1.278***
Inclusa	1.868***	1.391*	1.877***	1.419***	1.585***	1.340***
Latina	1.185	1.167	1.874***	1.540***	1.446***	1.250**
Palacio	0.857	1.082	1.346	1.045	1.072	1.046
Universidad	0.852	0.856	1.692***	1.250*	1.155	1.268**
Influenza	1.007	1.232*	1.138	1.420***	1.238***	1.405***
Winter ref.	1.000	1.000	1.000	1.000	1.000	1.000
Spring	0.711***	0.520***	0.612***	0.987	0.750***	1.017
Summer	0.787*	0.782**	0.974	1.202**	0.981	0.859**
Autumn	0.712***	0.467***	0.526***	0.545***	0.554***	0.616***
Normal temperature ref.	1.000	1.000	1.000	1.000	1.000	1.000
Very low	1.550***	1.296*	1.105	0.998	1.195**	1.145
Very high	1.447**	0.898	1.190	1.131	1.159**	1.269**

(Continued)

Table 1b. (Continued)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Age of mother						
15–19	1.478	2.125***	1.637*	0.94	1.433***	0.868
20–24	1.068	1.198	1.077	0.921	1.034	0.819***
25–29 ref.	1.000	1.000	1.000	1.000	1.000	1.000
30–34	0.921	1.047	1.182	1.089	1.066	1.085
35–39	1.013	1.343**	1.252*	1.173**	1.182***	1.089
40+	1.232	1.480**	1.412**	1.268**	1.324***	1.078
Age of child						
1–9 days ref.	1.000				1.000	
10–28	0.811***				0.778***	
29–90					0.394***	
91–179		(omitted)			0.291***	
180–365			(omitted)		0.313***	
1y – 2y ref.				(omitted)		1.000
3y – 4y						0.297***
Female	0.818**	0.802***	0.878	0.886**	0.851***	1.034
Twin	7.070***	5.136***	4.175***	3.516***	4.839***	2.009***
No father	2.027***	2.340***	1.383*	1.07	1.615***	0.761**
Constant	0.334***	0.166***	0.077***	0.089***	0.318***	0.058***

(Continued)

Table 1b. (Continued)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
/lntheta	1.771**	1.066	1.131	0.372**	0.329***	0.308***
Number of subjects	26,022	25,336	24,691	24,033	26,022	22,664
Number of deaths	686	645	658	1369	3358	2156
Time at risk	2036	4242	5941	11,834	24,053	65,668
Log likelihood	–3771	–3591	–3551	–6505	–14,155	–10,264
LR test of $\theta = 0$	28***	9***	12***	8***	31***	10***

Note: * $p < .1$. ** $p < .05$. *** $p < .01$.

Table 1c. Piecewise constant hazards models with shared frailty (mother level) on time to death. Mothers born in Castilla y León

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Buenavista ref.	1.000	1.000	1.000	1.000	1.000	1.000
Centro	0.882	0.890	0.935	0.817	0.872	1.143
Chamberí	0.935	1.093	1.275*	1.033	1.067	1.347***
Congreso	1.111	0.940	0.910	0.907	0.956	0.974
Hospicio	0.739	0.904	0.928	0.855	0.847*	1.333**
Hospital	1.283	1.024	1.188	1.114	1.144*	1.379***
Inclusa	1.223	1.378*	1.125	1.186	1.208**	1.504***
Latina	1.005	1.403**	1.408**	1.215*	1.242***	1.570***
Palacio	0.835	1.298	1.034	0.914	0.983	1.206*
Universidad	1.107	1.077	1.313*	1.112	1.143*	1.364***
Influenza	1.020	1.224**	1.124	1.454***	1.250***	1.635***
Winter ref.	1.000	1.000	1.000	1.000	1.000	1.000
Spring	0.612***	0.505***	0.686***	0.986	0.737***	1.005
Summer	0.87	0.774**	1.010	1.252***	1.024	0.798***
Autumn	0.728***	0.504***	0.489***	0.526***	0.554***	0.602***
Normal temperature ref.	1.000	1.000	1.000	1.000	1.000	1.000
Very low	1.133	1.138	1.175	1.109	1.130*	0.991
Very high	1.176	0.995	1.172	1.259**	1.190***	1.11

(Continued)

Table 1c. (Continued)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Age of mother						
15–19	1.353	1.566*	1.273	0.955	1.241*	0.84
20–24	1.058	1.306**	1.012	0.967	1.058	1.018
25–29 ref.	1.000	1.000	1.000	1.000	1.000	1.000
30–34	1.037	1.205*	0.994	1.106	1.082*	1.221***
35–39	1.004	1.191	1.088	1.122	1.106**	1.333***
40+	1.327*	1.476***	1.294**	1.315***	1.332***	1.323***
Age of child						
1–9 days ref.	1.000				1.000	
10–28	0.923				0.867**	
29–90		(omitted)			0.408***	
91–179			(omitted)		0.375***	
180–365				(omitted)	0.349***	
1y – 2y ref.						1.000
3y – 4y						0.269***
Female	0.781***	0.752***	0.888*	0.862***	0.831***	1.02
Twin	9.959***	5.020***	2.756***	2.224***	4.630***	1.995***
No father	2.378***	2.775***	1.714***	0.804	1.692***	0.793**
Constant	0.411***	0.164***	0.141***	0.119***	0.381***	0.053***

(Continued)

Table 1c. (Continued)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
/lntheta	2.356***	1.584	0.659	0.230***	0.456***	0.344***
Number of subjects	31,703	30,829	30,035	29,038	31,703	27,258
Number of deaths	874	794	997	1780	4445	2770
Time at risk	2480	5161	7199	14,268	29,108	78,074
Log likelihood	–4720	–4432	–5232	–8324	–18,299	–12,990
LR test of $\theta = 0$	54***	19***	8***	4***	68***	18***

Note: * $p < .1$. ** $p < .05$. *** $p < .01$.

Table 1d. Piecewise constant hazards models with shared frailty (mother level) on time to death. Mothers born in other regions of Spain

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Buenavista ref.	1.000	1.000	1.000	1.000	1.000	1.000
Centro	1.137	1.226	1.364*	1.781***	1.427***	1.537***
Chamberí	1.421**	1.139	1.221	1.598***	1.385***	1.590***
Congreso	1.521***	1.504***	1.210	1.386***	1.415***	1.536***
Hospicio	1.196	0.985	1.415**	1.395**	1.271***	1.390***
Hospital	1.170	1.508***	1.395**	1.900***	1.538***	2.057***
Inclusa	1.769***	1.881***	1.898***	2.406***	2.067***	2.409***
Latina	1.435**	1.652***	1.669***	1.734***	1.646***	2.133***
Palacio	1.173	0.992	0.921	1.570***	1.217**	1.485***
Universidad	1.267	1.555***	1.621***	1.859***	1.607***	1.839***
Influenza	1.515***	1.139	1.267**	1.163**	1.252***	1.656***
Winter ref.	1.000	1.000	1.000	1.000	1.000	1.000
Spring	0.731***	0.690***	0.624***	0.817***	0.731***	0.899**
Summer	0.810**	0.774**	1.207*	1.111	1.000	0.763***
Autumn	0.596***	0.574***	0.529***	0.455***	0.525***	0.560***
Normal temperature ref.	1.000	1.000	1.000	1.000	1.000	1.000
Very low	1.231	1.530***	1.275*	0.989	1.201***	0.946
Very high	1.394**	0.990	0.961	1.297***	1.186***	1.049
Age of mother						
15–19	1.696***	1.562**	1.113	1.086	1.327***	0.962

(Continued)

Table 1d. (Continued)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
20–24	1.239**	1.181*	1.007	1.010	1.094*	0.862**
25–29 ref.	1.000	1.000	1.000	1.000	1.000	1.000
30–34	0.949	0.877	1.186*	1.028	1.006	1.076
35–39	1.179	1.122	1.187	1.260***	1.197***	1.146**
40+	1.334**	1.366**	1.197	1.240**	1.282***	1.403***
Age of child						
1–9 days ref.	1.000				1.000	
10–28	0.681***				0.648***	
29–90		(omitted)			0.337***	
91–179			(omitted)		0.254***	
180–365				(omitted)	0.251***	
1y – 2y ref.						1.000
3y – 4y						0.283***
Female	0.882*	0.787***	0.790***	0.812***	0.812***	0.972
Twin	7.822***	4.712***	5.318***	2.921***	5.123***	1.679***
No father	1.357**	2.027***	1.632***	1.057	1.450***	0.729***
Constant	0.329***	0.124***	0.082***	0.069***	0.319***	0.037***
/lntheta	2.882***	1.915**	1.608*	0.370**	0.593***	0.539***
Number of subjects	38,849	37,859	36,975	36,069	38,849	34,326

(Continued)

Table 1d. (Continued)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Number of deaths	990	884	906	1742	4522	2717
Time at risk	3039	6346	8900	17,842	36,126	100,239
Log likelihood	–5578	–5033	–5010	–8585	–19,770	–13,292
LR test of $\theta = 0$	65***	28***	25***	7***	86***	27***

Note: * $p < .1$. ** $p < .05$. *** $p < .01$.

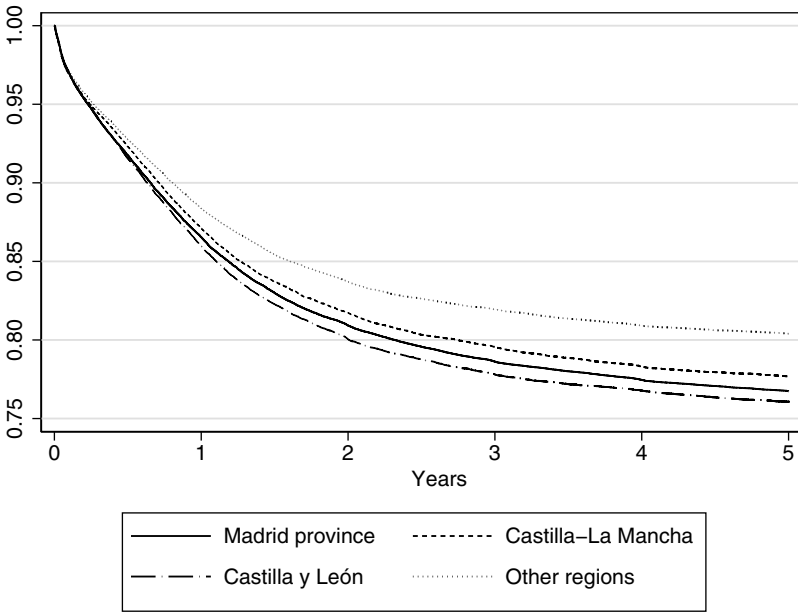


Figure 2. Kaplan-Meier survival estimates according to mother birth place. Madrid, 1916-1926.

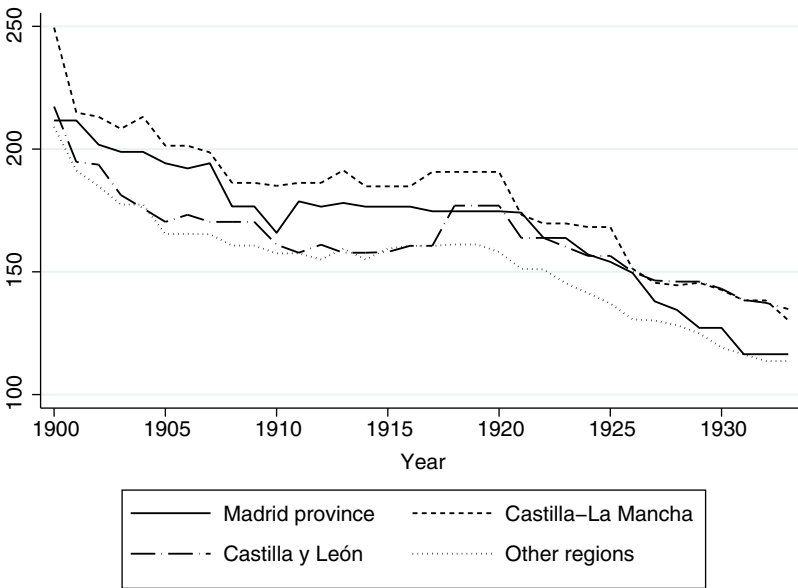


Figure 3. Infant mortality by region. Spain, 1900-1933. Source: Adapted from Diego (2008).

Table 2. Mains causes of death by age groups under five in Madrid (in percent), 1916–1925

Main Causes of death	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–4 y.
Infectious: food	9.4	19.2	27.3	25.8	15.1
Infectious: air	20.8	39.1	38.0	44.4	53.8
Infectious: others	5.4	9.8	14.8	17.5	21.1
Noninfectious	64.4	31.9	19.9	12.3	10.0
Total	100.0	100.0	100.0	100.0	100.0

As suggested by the literature, this environmental condition affected post-neonatal mortality more than neonatal mortality and child mortality more than infant mortality. In addition, suggestive differences in the sensitivity to this factor appear. While they faced the lowest risks of dying, children of mothers born in the rest of Spain were the most responsive to the spatial location within the large city. In this group, childhood mortality in the worst districts (Hospital, Inclusa, Latina) was twice as high as in Buenavista. Children of Madrid natives were also affected by place of residence, although to a lesser extent. They were followed by Castilla y León and then by Castilla-La Mancha.

Another important environmental factor of vulnerability in early 20th-century Madrid was seasonality. During the first five years of life, spring and even autumn were protective, while winter was the most dangerous period. Summer was positive for children younger than six months and between ages one and four. However, when they were aged 180–365 days, summer was more dangerous than winter. This was especially the case for the infants of Madrid natives (H.R. 1.38) and Castilian mothers (H.R. 1.20 for Castilla-La Mancha, H.R. 1.25 for Castilla y León). The perils of summer happened earlier in the life course of children of mothers born in the rest of Spain.

Our results suggest that most of the mothers living in Madrid weaned their children for approximately 6 months, while those from the most distant regions had an even shorter duration of breastfeeding. We ran a separate model for each month of life from birth to the first birthday (Tables not shown) with three patterns emerging. For children of mothers coming from the rest of Spain, summer was dangerous only during the fifth month of life, while for those of mothers born in Castilla-La Mancha, summer was dangerous only during month seven. This suggests a clear transition from breastfeeding to artificial food. Additionally, it was also in the seventh month of life that summer became more harmful for children born to Castilla y León and Madrid native mothers. However, in these two subpopulations, the penalty continued to affect the infants until their first birthday, suggesting a more staggered transition and less secure infant feeding practices in the second half of the first year of life.

Causes of death are noted in Madrid civil certificates. Table 2 unsurprisingly shows that noninfectious diseases were dominant in the first days of life. Endogenous causes of death, for example, congenital malformation, were by far the most frequently cited causes of death during the first month of life. The second one is atresia, which indicates acute malnutrition, followed by “falta de desarrollo,”

which means lack of development. When the surviving children grew, these diseases continued to be major threats to their survival, with stunting, a feature of chronic undernutrition, completing the list of the main killers. This group of noninfectious pathologies, clearly associated with poverty, became decreasingly dominant throughout the transition from infancy to childhood: they accounted for more than two-thirds of the deaths between 1 and 28 days of life but only 10 percent approximately between the first and fifth birthdays. Conversely, the frequency of infectious diseases rose as the children grew older. These diseases caused 90 percent of childhood mortality; the largest contributors were airborne diseases (bronchitis, pneumonia, tuberculosis, etc.). An interesting exception concerned food- and waterborne diseases (mostly gastroenteritis and enteritis), which reached their maximum between 3 months and 1 year of life, around weaning and the following period of adaptation to artificial food.

We estimated a Fine-Gray competing risk model on the risk of dying from three types of causes: food- and waterborne infectious diseases, airborne infectious diseases, and noninfectious diseases. The results are presented in Tables 3a–c and show a striking impact of the summer season on the first group of causes. This association resulted in a multiplication of the risk by 7 when the children were aged 91 to 179 days and by 10 when they were aged between 6 months and 1 year. The hazard ratios were lower but still very high (between 3 and 4) for the younger and older children. This pattern confirms that weaning was especially dangerous in summer and that the impact of hot temperatures on water and food quality was a real threat throughout the first five years of life. Logically, the seasonal pattern was exactly the opposite for airborne diseases, with summer and autumn being the most protective periods while winter was the most aggressive, as well as very low temperatures, especially during the first six months of life. Approximately the same pattern applied to noninfectious pathologies with, however, a more limited impact by seasonality and temperatures.

Tables 3a–c also demonstrate that the deprived areas of the districts Hospital, Inclusa, and Latina in the south of Madrid and Universidad in the north accumulated disadvantages. They suffered from penalties that caused higher infant and childhood mortality risks in these areas, with a particularly high tribute to food- and waterborne diseases and to noninfectious diseases.

Instead, after we controlled for environmental factors, as well as for a few other factors that are not central to this paper, the differences according to the birth region of the mother were relatively limited. Compared to the children of Madrid native mothers, children of immigrants from Castilla-La Mancha were less affected by food- and waterborne diseases during their first year of life and by airborne diseases and noninfectious diseases between 3 and 6 months. Children of mothers born in the regions of Spain more distant from Madrid were most favored, not when newborn but after one or three months of life, whatever the cause of death. Conversely, having a mother born in Castilla y León was a risk factor for childhood mortality (deaths due to infected water and food) and during the second half of their first year of life (airborne infectious diseases). Otherwise, they did not differ from the offspring of Madrid natives.

Table 3a. Fine-Gray model on time to death (fail = death caused by infection from food; compete = death from other causes)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Buenavista ref.	1.000	1.000	1.000	1.000	1.000	1.000
Centro	0.920	0.828	1.440**	1.080	1.095	0.976
Chamberí	1.288	1.211	1.790***	1.387***	1.418***	1.337***
Congreso	1.341	1.078	1.476***	1.136	1.210***	1.328***
Hospicio	0.777	0.765	0.979	0.844	0.850*	0.756*
Hospital	1.355	1.458**	1.577***	1.284***	1.367***	1.404***
Inclusa	1.508*	1.868***	2.008***	1.448***	1.614***	1.800***
Latina	1.319	1.470**	1.885***	1.359***	1.461***	1.583***
Palacio	1.001	1.363*	1.556***	1.173	1.267***	1.244*
Universidad	1.137	1.648***	2.166***	1.515***	1.625***	1.640***
Winter ref.	1.000	1.000	1.000	1.000	1.000	1.000
Spring	0.860	0.853	1.176	1.242*	1.105	0.962
Summer	3.268***	4.111***	7.100***	9.978***	7.002***	3.945***
Autumn	1.489**	1.485***	1.653***	2.311***	1.871***	1.745***
Normal temperature ref.	1.000	1.000	1.000	1.000	1.000	1.000
Very low	0.987	1.227	0.989	1.087	1.064	0.869
Very high	1.725***	1.095	1.408***	1.337***	1.354***	1.408***
Native mother ref.	1.000	1.000	1.000	1.000	1.000	1.000
Castilla-La Mancha	0.984	0.812*	0.874	0.823***	0.851***	1.019

(Continued)

Table 3a. (Continued)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Castilla y León	0.960	0.947	1.075	0.972	0.994	1.107*
Other regions	1.005	0.844*	0.900	0.840***	0.873***	0.812***
Number of subjects	169,870	165,389	161,131	156,429	169,870	147,676
Log likelihood	–4662	–9271	–14,373	–25,187	–53,830	–23,508

Note: We also controlled for age of the mother, gender, twin births and father presence at the time of birth; * $p < .1$. ** $p < .05$. *** $p < .01$.

Table 3b. Fine-Gray model on time to death (fail = death caused by airborne infectious diseases; compete = death from other causes)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Buenavista ref.	1.000	1.000	1.000	1.000	1.000	1.000
Centro	1.258	0.869	1.386**	1.082	1.115	1.136*
Chamberí	1.449**	1.119	1.166	1.182**	1.185***	1.316***
Congreso	1.373**	0.942	1.239*	0.986	1.056	1.068
Hospicio	0.962	0.905	1.036	1.264***	1.102	1.261***
Hospital	1.652***	1.118	1.541***	1.480***	1.413***	1.555***
Inclusa	1.859***	1.330***	1.585***	1.474***	1.477***	1.606***
Latina	1.410**	1.286**	1.588***	1.479***	1.432***	1.534***
Palacio	1.256	0.907	1.289**	1.276***	1.190***	1.229***
Universidad	1.009	1.065	1.509***	1.173**	1.190***	1.326***
Winter ref.	1.000	1.000	1.000	1.000	1.000	1.000
Spring	0.415***	0.444***	0.447***	0.787***	0.587***	0.854***
Summer	0.178***	0.200***	0.247***	0.342***	0.266***	0.400***
Autumn	0.293***	0.248***	0.247***	0.267***	0.263***	0.389***
Normal temperature ref.	1.000	1.000	1.000	1.000	1.000	1.000
Very low	1.604***	1.386***	1.095	1.081	1.206***	1.078
Very high	0.567	0.632*	0.961	1.033	0.936	0.931
Native mother ref.	1.000	1.000	1.000	1.000	1.000	1.000
Castilla-La Mancha	0.904	0.995	0.883*	0.977	0.953	0.907***

(Continued)

Table 3b. (Continued)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Castilla y León	1.008	0.952	1.126*	1.078*	1.054*	0.999
Other regions	0.863	0.910	0.820***	0.871***	0.869***	0.812***
Number of subjects	169,870	165,389	161,131	156,429	169,870	147,676
Log likelihood	–10,263	–19,046	–20,482	–44,570	–94,870	–84,287

Notes: we also controlled for age of the mother, gender, twin births and father presence at the time of birth; * $p < .1$. ** $p < .05$. *** $p < .01$.

Table 3c. Fine-Gray model on time to death (fail = death caused by noninfectious diseases; compete = death from other causes)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Buenavista ref.	1.000	1.000	1.000	1.000	1.000	1.000
Centro	0.939	0.901	1.007	0.995	0.948	1.472**
Chamberí	1.102	1.369**	1.676***	1.568***	1.306***	1.676***
Congreso	1.268***	1.576***	1.198	1.343*	1.333***	1.426**
Hospicio	1.099	1.207	1.338	1.370*	1.200**	1.471**
Hospital	1.314***	1.392**	1.310*	1.780***	1.396***	1.663***
Inclusa	1.496***	1.787***	1.831***	2.344***	1.721***	2.528***
Latina	1.235**	1.822***	1.743***	1.663***	1.487***	2.153***
Palacio	1.012	1.230	0.953	1.011	1.045	1.862***
Universidad	1.197**	1.287*	1.427**	1.479***	1.289***	1.559***
Winter ref.	1.000	1.000	1.000	1.000	1.000	1.000
Spring	0.764***	0.672***	0.969	1.031	0.816***	1.047
Summer	0.846***	0.850*	1.167	1.321***	0.971	0.791***
Autumn	0.773***	0.828**	0.710***	0.653***	0.764***	0.841**
Normal temperature ref.	1.000	1.000	1.000	1.000	1.000	1.000
Very low	1.182**	1.418***	1.238	0.737*	1.165***	1.569***
Very high	1.234**	1.093	1.105	1.253*	1.197***	1.461***
Native mother ref.	1.000	1.000	1.000	1.000	1.000	1.000
Castilla-La Mancha	0.978	0.904	0.723***	0.928	0.912**	0.984

(Continued)

Table 3c. (Continued)

Covariates	1–28 d.	29–90 d.	91–179 d.	180–365 d.	1–365 d.	1–4 y.
Castilla y León	1.038	1.009	1.065	1.097	1.047	0.900
Other regions	0.986	0.894	0.700***	0.832**	0.894***	0.809***
Number of subjects	169,870	165,389	161,131	156,429	169,870	147,676
Log likelihood	–32,107	–15,614	–10,794	–12,351	–71,116	–15,725

Notes: we also controlled for age of the mother, gender, twin births and father presence at the time of birth; * $p < .1$. ** $p < .05$. *** $p < .01$.

Discussion

In this research, we empirically tested two hypotheses. The results clearly validated the first hypothesis, based on the “fundamental cause” theory, using location in a segregated city as a proxy of inequality in living conditions. In Madrid, although urban development was planned in the 1860s, demographic growth increasingly overwhelmed the infrastructure and real estate market. The shantytowns of the suburbs were located in the “least favored districts (corresponding to the districts of Inclusa, Hospital, Latina and Universidad), characterized by the concentration of most of the unhealthy housing and neighborhoods, with most of their streets lacking sewage systems and water, electricity and clean air in the houses” (Casado-Ruiz and Ramiro-Fariñas 2018). Lodgings also suffered from poor aeration and darkness. Coherently, our individual-level data identified a strong excess mortality in these districts, which not only experienced an accumulation of penalties but were also stigmatized for their dirtiness and seen by the local elites as a manifestation of urban degeneration (Manzano-Gómez 2022). Our results illustrate a pattern according to which “the poor lived in the environmentally more dangerous areas and this ‘boosted’ their observed mortality” (Reid 2002: 151), a pattern where the neighborhood is at the right level to reveal poverty (Thornton and Olson 2011).

From this perspective, it is important to consider how the urban landscape changed. All across Europe, after the cholera pandemics and later the Pasteurian discoveries, massive investments in public hygiene transformed cities and targeted a reduction in infectious diseases (Baldwin 1999; Harris and Helgertz 2019). Madrid was, however, a late-comer. Casado-Ruiz and Ramiro-Fariñas (2018) noted that “it was not until after the Spanish flu epidemic [1918] that a real sanitation plan for the city was implemented: septic tanks were banned, cleaning services were reorganized, the municipal laboratory was modernized, bathrooms and public toilets were built, and water distribution was extended, among other measures.” More than 200 km of underground water pipes were constructed in a decade, completing an old network of 176 km that was renewed (Casado-Ruiz and Ramiro-Fariñas 2018). However, these same authors showed that those improvements were concentrated in the wealthy districts, thus increasing the gap between the favored and disfavored areas. Early twentieth Madrid illustrates a process that has also been observed elsewhere (Harris and Helgertz 2019; Jaadla and Puur 2016), where progress in public health was associated with growing spatial and social inequalities.

Indeed, what the 1916–1926 Madrid data reveal is the importance of the accumulation of disadvantages among the urban poor and of social inequalities inscribed in the urban space. They appear clearly at the very beginning of life. Among the causes of neonatal death, the most important diseases demonstrated a clear association with poverty. Undernutrition affected poor mothers and drastically increased the mortality risks of their children in their first month of life, as well as later on to a lower extent. This pattern was observed in several historical populations in the eighteenth and nineteenth centuries (Oris *et al.* 2004; Alter *et al.* 2004; Van de Walle 1986). However, seeing it in the capital of a European country in the early 20th century, and more so with such intensity, is striking. This is support for the “fundamental cause” theory (Link and Phelan 1995, 2010).

According to our second hypothesis, we expected Madrid native mothers to be more aware of the city's resources and consequently more able to protect their children than immigrant mothers.⁵ Our results show that the reality was far from being so simple. Madrid native mothers' offspring were surprisingly not advantaged when facing death but were also quite responsive to seasonality, as well as to the place of residence across the urban space, an urban space where their mothers spent all their lives. In comparison, children from mothers coming from Castilla-la-Mancha were slightly favored, and those from mothers born in Castilla y León tended to be disfavored. Differences between Castilians and Madrilenians were small, however, only a few percentage points, and for most of them statistically significant mainly because of the large size of the database.

Migrants coming from the rest of Spain were the only real exception. Children of mothers born in regions distant from Madrid had a lower probability of dying as soon as they survived the first three months of life. This result is not a surprise since this nativity group included a medium- to high-bourgeoisie recruited from all across the country to work in the capital ministries, institutions of high education, or the headquarters of major enterprises (de Miguel-Salanova and Diaz-Simon 2015; Pallol-Trigueros 2017).

This group of migrants was, however, a minority. For the large majority of the mothers (77 percent), hypothesis 2 is not validated: their origin was not an important risk factor, and children born in Madrid from mothers born in Madrid were not advantaged. To explain this unexpected result, we build on the research of Bakhtiari. This author considers that for the children of immigrants in the United States in 1910, the ultimate cause of the differentials in health and death was the context of reception, instead of wealth. This was this context that defined "assimilation pathways available to immigrants" and access to resources (Bakhtiari 2018: 140). In Madrid, migrants' insertion also went through the labor and housing markets.

As far as the first one is concerned, we face the most important limitation of our data source, the absence of indication about occupation for both father and mother. For the second one, once again, location in the city provides important information. If mothers born in the distant regions of Spain were 22.9 percent of the mothers between 1916 and 1926, they accounted for 30 percent in Buenavista and 28 percent in Centro, so in the most favored and healthier districts, while they formed the group that showed the greatest sensitivity to the location within a city they discovered. This spatial distribution contributed to the better survival of their children. However, surprisingly, this concentration was exceptional. Madrid native mothers were 42 percent, a proportion varying between 38 percent in Congreso and 46 percent in La Latina, with a fluctuation only from 40 to 44 percent in the eight other districts. Castilians were also equally distributed. Their insertion pathways in

⁵In this paper, we assumed that in a patriarchal society like the Spanish one in the early 20th century, mothers took care of the baby, why our analyses considered their origin. Additional models tested the influence of the father origin, especially if he was a Madrid native, married with another native or with an immigrant. As expected, this variable does not impact neonatal mortality. Moreover, its adjunction in the models does not affect the other results. Only one additional information emerges. The risks of childhood mortality decreased when the father was not coming from Castilla but from the rest of Spain. This is coherent with the results about mothers' origin.

Madrid's urban space – at least at the district level – did not result in spatial discrimination. Contrary to North American towns in the same period (Preston *et al.* 1994; Olson and Thornton 2011; Bakhtiari 2018; Dribe *et al.* 2020), a spatial concentration of immigrants from the same origin was not observed in early 20th century Madrid.

In fact, the outskirts (called *extraradios*) were a poor refuge for the poorest and newly arrived immigrants in a habitat of slums and barracks (Vicente-Albarrán 2015; Vorms 2017). However, these suburban neighborhoods acted not only as a barrage for new migrants but also as a refuge for those who had escaped the center and its excessive rent prices (Carballo-Barral *et al.* 2008). This explains why, contrary to our expectations, the process of spatial segregation did not affect the Castilian immigrants more than the natives. This is an explanation for the unexpected small differences in infant and child mortality according to mothers' place of birth. Much more than origin, location in the city was the dominant factor of death among children under five years of age.

Another explanation of the absence of large differentials between the nativity groups is more speculative. We know that although the turnover of young single adults was impressive, those who married before moving to Madrid or found a spouse in the city matrimonial market tended to settle durably in the Spanish capital (Ramiro-Fariñas *et al.* 2021). The accumulation of newcomers of fertile age sustained the birth rate. In 1905, 65.8 percent of births were from immigrant mothers. This proportion fell to 58.3 percent in the period 1916–1926 studied here. This decrease can be explained because a fair proportion of the 41.7 percent of mothers born in Madrid were daughters of immigrants from the surrounding regions. As we have just seen, approximately 77 percent of the mothers shared the Castilian culture and lived together in the same districts. This facilitated what the Spanish social historians called the “transition to the mass society” (Otero-Carvajal and Pallol-Trigueros 2010: 564–566). This mass society was essentially defined by Madrid's environmental and living conditions. Socialization in the region of origin faded, overwhelmed by the urban living style. Moreover, we have an element that strengthens this interpretation. In Central Spain, 12–18 months was the normal age for weaning (Ramiro-Fariñas and Sanz-Gimeno 2000). However, in the Spanish capital, weaning occurred around months 6–7. Interestingly, this was the case for Madrid native mothers as well as for those born in Castilla y León and Castilla-La Mancha. They all acted the same way, adapting their behaviors to the context of a large city.

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Appendix

Table A1. Table of frequency. Variables at birth

Variable		%	Variable		%
District	Buenavista	10.4	Mother origin	Madrid province	43.2
	Centro	4.6		Castilla-La Mancha	15.3
	Chamberí	12.2		Castilla y León	18.7
	Congreso	10.5		Other regions	22.9
	Hospicio	5.5	Mother age	15–19	2.9
	Hospital	12.3		20–24	19.5
	Inclusa	11.3		25–29	33.1
	Latina	12.9		30–34	24.9
	Palacio	7.5		35–39	14.4
	Universidad	12.8		40+	5.3
Pandemic	No flu	87.8	Sex of newborn	Male	51.4
	Influenza	12.2		Female	48.6
Current season	Winter	26.9	Type of delivery	Single	98.4
	Spring	25.2		Twin	1.6
	Summer	23.1	Presence of father	With father	95.1
	Autumn	24.8		No father	4.9
Temperature	Normal	90.0	Individuals	169,870	
	Very low	5.3			
	Very high	4.7			

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