Cohort and period effects in the long run decline of mortality, 
Paris 1817-2000

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This version: September 2015

Abstract
A growing body of evidence suggests there is a long lasting impact of early life conditions. This suggests an important role for cohort effects in mortality decline. Here we investigate mortality in the very long run so as to estimate the role of cohort effects in the mortality transition. We focus solely on Paris so as to control as best as possible for sanitary improvements. We do three things. First, we compute yearly mortality rates over more than 150 years to measure both cohort and period effects. Second we document where sanitary improvements took place in the city at different times. Third we mobilize individual data on mortality that allow us to distinguish those born in Paris from the whole population so as to control for the huge migration flow.

Keywords: mortality, cohort effects, early-life effects, long run mortality decline
Introduction

The mortality decline over the past two centuries is one of the most striking demographic phenomena. Humans born today in high income economies can expect to live at least twice as long as their forebears of two centuries ago. This gain results from various improvement, either through the rise in income (and its consequences, such as better food or housing) or through large-scale public infrastructure that improve health (such as better access to water or better sanitation. In any cases, these improvements can have both direct effects (the immediate reduction in mortality following a given improvement) and long-lasting effects (the reduction in mortality occurring later in life for those who benefited from the improvement when they were young). The latter has been the subject of growing attention in recent works that focused on so-called early-life conditions (among many others see Elo and Preston, 1992; Almond and Mazumder, 2005; Van den Berg et al., 2006).

This paper is set within that literature focuses on the evolution of mortality in Paris, one of the largest cities in the world (at least at the beginning of our period) over the full span of the mortality transition. Our objectives are to disentangle cohort and period effect, and to evaluate the role of on water improvements (be it water connection or better sanitation).

Indeed, in a seminal paper, Samuel Preston and Etienne Van de Walle (1978) find very strong cohort effect on mortality using historical data from urban areas in France. They suggest these effects are related to better access to water but they cannot directly link the two phenomena.¹ In this paper we start where they stopped using a much larger dataset. We narrow the focus to variation within Paris so as to control as best as possible for sanitary improvements and extend our analysis on the large period that run from the time where there has been no water improvement at all up to the very recent time. To do so, we take advantage of annual reports on the death by age, sex, and marital status in the city of Paris as a whole, starting in 1817 and going up until quite recently.

We do three things. First, we compute yearly mortality rates over more than 150 years to measure both cohort and period effects. Second we precisely document the diffusion of access to clean water and direct connections to sewer across the city and overtime. Third we mobilize individual data on mortality that allow us to distinguish those born in Paris from the whole population so as to control for the huge migration flow.

¹ To be sure they suggest that these cohort effects are related with access to better water but they do not investigate this possibility empirically, they only recall that this improved access occurred in the right time span.
Context

The decline of mortality between the early 19th century and the end of the 20th century was widespread in large cities across the North Atlantic economies. This sharp reduction of mortality occurred in the U. S. (Cutler and Miller 2005, Troesken 1999), in Germany (Brown 1989), and in the U. K. (Szreter 1988) among other countries. This decline initially allowed urban mortality to reach parity with that of rural areas, which had long enjoyed a health advantage. Urban mortality then continued to fall, finally giving cities the life expectancy advantage over rural areas they currently enjoy. Time and again scholars have pointed to income growth (Birchenall 2007, McKeown 1976) and to the diffusion of water infrastructure—piped, filtered, and chlorinated water on one side and sewers on the other (Cutler and Miller 2005, Ferrie and Troesken 2008)–to explain why mortality fell so sharply and quickly throughout the North Atlantic region.

Scholars have tried to evaluate the role of income or wealth by charting the differential adoption of new water infrastructure across cities (Brown 1988). To evaluate the value of clean water they have looked at specific improvements that diffused quickly (e.g. chlorination) after the infrastructure had been put in place (Cutler and Miller 2005). In each case scholars looked at the impact of these measures city-wide—in effect treating it as a public good. These are informative approaches but they also have some limitations, the first being the elision of the huge variations that occurred within cities.

The slow diffusion of health-improving infrastructure such as clean water and sanitation remains an important problem in large parts of the world today (Banerjee and Duflo 2007, Baisa et al. 2010) just as it was a hundred years ago in Europe and North America. In cities of the developing world in particular, the diffusion of these technologies is highly uneven though they improve health and reduce water-related expenses (Galiani, Gertler, and Schargrodsky 2009). In fact, an important part of the debate today (as in the nineteenth century) involves who should pay for expanding infrastructure: landlords, users of water, or the rich more generally (Galiani, Gertler, and Schargrodsky 2005).

But the diffusion and increased use of clean water (for whatever purpose) in cities was not always clear-cut. In Paris it covers a period of over twenty years during which spring water was gradually brought to the city by aqueduct. Clean water should have helped reduce the mortality in the city. At the same time, the effect of improvements in water infrastructure may also be long-lasting, they may help reduce mortality later in life (indeed better living conditions early in life may also help reduce morbidity or improve health conditions in old
age, Costa 2000). It is already clear that better water reduce the incidence of other diseases, the so-called Mills–Reincke phenomenon (Ferrie and Troesken 2008).

By 1885 two-thirds of Parisian buildings were connected to the city’s water supply (Cebron de Lisle 1991: 547) and by then the vast majority of homes received pure (spring) water brought in by aqueduct (Deligny 1883: Annexe n°1, p. 49). After that date differential access to clean water was not an issue (Goubert 1986: 90-92, Bocquet, Chatzis, and Sander 2008).

Figure 1. Life expectancy at age 5, Paris and France, 1817-2000

Data
What is also specific to Paris is the quality of the data that we can use to assess the process of change. We rely on different sources published both at the local and at the state level, that provide aggregate accounts of deaths in the city, by age group, marital status, and sex from early 19th century (the earlier we can get is 1807) up to nowadays. Indeed, it does matter to have a time span as large as possible to investigate cohort effects since, if we are to have the full cohort mortality we need approximately 100 years for each cohort. So even with our 200 years data, we can only explore in details the cohorts born before 1914. ² But this is not a severe constraint as it encompasses the cohorts who lived through the most dramatic changes during the mortality transition.

² To be sure, we can restrict ourselves to, say, mortality before 60 years old, which would allow us to consider all cohorts born up until WWII.
The statistical office of Paris also published a series of detailed abstracts for the city drawn from the national population censuses that occurred every five years. These give us the age distribution of the living for the same neighborhoods. Taking these two databases together allows us to compute both period and cohort mortality rate and life expectancy.

Indeed, a purely descriptive exploration of the data show to what extent Paris sum the mortality transition experience (Figure 2). As can be seen on the figure both the intensity and the age composition of mortality change dramatically over the course of these 200 years: the frequency and strength of epidemics (easily seen on the figure) diminishes greatly in the 19th century, infant and children mortality declined since the turn of the century, and the overall mortality also diminishes quite rapidly in this period (which can be seen more easily on Figure 1).

Figure 2. Mortality by age and total population in Paris, 1817-2010

Note: the colored areas are the total number of deaths (on the right axis) by increasing age group (the one closer to the axis is 0-1 years old, the next one is 1-4, then 5-9, etc. The black line is the total population of the city at each census. There is a structural change in the administrative boundaries of the city in 1860, which explain the sudden increase in both deaths and population that year. There is a severe crisis in 1870 (French-Prussian war, the siege of Paris, and the Commune de Paris) and we omit the figures for those years on the graph for the sake of clarity.

To be sure, demographic data, however detailed and precise, would not be enough to explain the mortality transition and so we also rely on different sources to evaluate, and

3 Since the French Revolution, censuses were performed every five years; they have been kept in the archives from 1836 on in most cases. Here we use data on censuses from 1831, 1836 and so on.
control for income: real estate censuses (late 19th century, starting in the 1870’s, we rely on crude estimations before that date); rents from the archives of the treasury; GDP estimate from wealth collected at the individual level. We also have information on access to clean water and how soiled water was dealt with (those data are described in more detail in Kesztenbaum and Rosenthal, 2014).

Cohort effects

In this first section we will compute mortality rate by age and sex for almost 200 years. This will allow us to test for the presence of cohort effects for the generations who experienced the most dramatic changes in their mortality pattern. We see the evolution of mortality for these cohorts over all their life and thus can test whether the mortality decline the same for all age groups in a given year (period effect) or the same for all individuals belonging to the same cohort over time (cohort effect).

We can then compare period and cohort life expectancy and compute the gap between the two so as to measure how much each cohort take advantage of the mortality reduction that occur during her whole life (Goldstein and Wachter, 2006). Moreover, we can decompose the gains (or, for that matter, the losses) in life expectancy at birth between cohorts according to age-specific mortality reduction (Vaupel and Canudas Romo, 2003).

Our aim here is not simply descriptive but it is to test whether the large and long-lasting trend in mortality reduction in Paris depends, to some extent, on cohort effects (as Preston and Van de Walle argue). But the only way to do so properly is to consider the determinants of the mortality decline, whether immediate or later in life. In fact, we might guess that some determinants have long-lasting (e.g. cohort) effects (this what Preston and Van de Walle suggested for water) whereas others (for instance reduction in tuberculosis that affected mostly young adults) have only immediate effects. This is what we do in the next section.

Determinants of cohort effects

In order to explain the cohort effects enlightened in the previous section, we will look at specific cohorts who experienced health shocks at a given moment, whether positive or negative. We will look at positive investments such as changes in the quality and quantity of water supply to the city or the development of a better sanitation system to get rid of that water. We will do all this while controlling for changes in income. We will also look at
negative shocks; mainly epidemics that are still quite common in Paris until the 1870’s. We will question if the cohorts affected by these epidemics (at different ages) experience a different mortality patterns over their life. We will use the fact that there are multiple epidemic episodes, which vary greatly in intensity, to separate period and cohort effects. For instance, we have, on the one hand, two cohorts affected by the same shock at different age and, on the other hand, two cohorts affected by different shocks at the same age. We follow all these cohorts toward the end of their life.

**Migration and selection**

In the last section we will turn our attention to individual level data in order to take into account the additional complexity introduced by the large migration flows both in and out of Paris. Indeed, in the initial Preston-Van de Walle paper, they did take migration into account when computing death rates but they did not investigate the effect of migration on their results: they compare death rates by cohorts but the composition of these cohorts vary over age. In other words, among those belonging to their 1861-74 cohort, many of those observed in the Seine (in Paris in our case) at ages 50-64 were not in Paris at ages 5-19 (and vice-versa: many of those who were there while young may have left before 50 years old) which limits the comparability between the death rates at those ages. In fact, it’s all the more impressive that Preston and Van de Walle were able to demonstrate strong cohort patterns given the huge variability over time of the composition of these cohorts.

Indeed migration is the most obvious complication for studying mortality in Paris: like other large cities, its population is not closed. In fact, at the end of the 19th century, six out of ten people living in Paris had not been born there or in the suburbs (census results). Moreover, migrants to Paris are also not a random sample of the world or of France’s population, in fact migrants choose to move to Paris, and Parisians sort themselves into neighborhoods. Indeed, changes in the mortality of Parisians could be simply attributed to changes in rates of migration or in migrants’ characteristics. Instead of considering simply the limitations it induces for measuring mortality in Paris, we aim to take advantage of these features so as to compare mortality within Paris of migrants compare with those born in the city. To do so we will take advantage of a large individual dataset drawn from municipal death records.
References


