ORIGINAL PAPER

Demographic transitions: analyzing the effects of mortality on fertility

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Received: 18 July 2008 / Accepted: 14 April 2009 / Published online: 16 May 2009 © Springer-Verlag 2009

Abstract The effect of mortality reductions on fertility is one of the main mechanisms stressed by the recent growth literature in order to explain demographic transitions. We analyze the empirical relevance of this mechanism based on the experience of developed and developing countries since 1960. We distinguish between the effects on gross and net fertility, take into account the dynamic nature of the relationship, and control for alternative explanatory factors and for endogeneity. Our results show that mortality plays a large role in fertility reductions, that the change in fertility behavior comes with a lag of about 10 years and that both net and gross fertility are affected. We find comparatively little support for explanations of the demographic transition based on changes in GDP per capita.

Keywords Mortality · Fertility · Demographic transitions

JEL Classification J1 · J13

1 Introduction

The economic mechanisms explaining demographic transitions have attracted an increasing amount of attention from the profession over the last decade.

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Responsible Editor: Junsen Zhang

In this paper, we contribute to this ongoing literature by providing an extensive empirical analysis of one of the most important mechanisms used by researchers to explain these transitions: the effect of mortality reductions on fertility.

Probably the main reason behind the regain of interest in the demographic transition has been the development of what are usually called "unified growth theories." This body of theoretical work has extended the traditional post-war time horizon of previous models of economic growth in order to understand the passage from a near-zero steady state growth regime in preindustrial times to a positive steady state growth regime from the Industrial Revolution on-wards. Most researchers in the area have stressed the role of the demographic transition in this context as both cause and effect of the transition in growth regimes.

In order to explain the demographic transition, most papers in the unified growth literature have made use of the seminal work of Becker (1960) to model fertility decisions and have featured its most paradigmatic result: the quantity–quality tradeoff for children. As is well-known, Becker's result derives from the assumption that parental utility is a function of both the number of children they have ("child quantity") and of the education or human capital of each child ("child quality"). Since both rearing and educating children are costly, a tradeoff between these two activities arises.¹

In this framework, fertility and child education are two endogenous variables jointly determined by socioeconomic factors that make child quantity and child quality more or less attractive. That is where mortality may come into play.

High mortality makes investments in children's human capital less attractive by reducing the expected time horizon over which such capital can be used. In other words, parents would not want to waste resources in educating children who are unlikely to benefit from that education because of premature death. According to this mechanism, falling mortality rates would thus induce a substitution of child quantity for child quality and could explain a demographic transition. Among the contributors to the unified growth literature that have used some version of the above argument, we can count Galor and Weil (1999), Kalemli-Ozcan (2002, 2003), Lagerlöf (2003), Weisdorf (2004), Soares (2005), Azarnet (2006), Tamura (2006), and Falcao and Soares (2008).²

There are, however, many other factors besides mortality rates that could explain a shift towards more child quality and less child quantity. Perhaps the most important ones are the two closely related factors of economic development and technological progress. Indeed, a richer and more technologically

¹Further developments made parental utility dependent not on the children's human capital but on the children's future utility. The results of the model remain similar, however, once we consider that the children's future utility is a function of their human capital (Becker and Barro 1988).

 $^{^{2}}$ Cervellati and Sunde (2005) also rely on mortality changes to generate a demographic transition, but their mechanism works through changes in longevity. Hazan and Zoabi (2006) criticize this part of the literature on theoretical grounds.

advanced economy may remunerate human capital better, and this would induce parents to increase their children's education at the expense of their number. The seminal paper of Galor and Weil (2000) uses a version of this argument while Jones (2001), Kogel and Prskawetz (2001), and Hansen and Prescott (2002) also put the accent on technological change or economic development, although without featuring a quantity–quality tradeoff.³ Moreover, the argument has been extended in a number of directions: economic development and technological change may make child labor relatively less effective, reducing the interest of having many children (Hazan and Berdugo 2002), or it may make female labor relatively more effective, shifting women's choices towards more market employment and less child rearing (Galor and Weil 1996).

Other potential factors of the shift in the quantity–quality tradeoff that have been discussed in the literature include Darwinian evolution (Galor and Moav 2002) and the effects of trade specialization (Galor and Mountford 2006). It is thus apparent that the unified growth literature is in no short supply of potential causes for the fall of fertility rates during the demographic transition. What is more, to the above mentioned candidates, we ought to add several other factors that have, for a long time, been the focus of attention in demography.

Demographers were, of course, the first ones to define and study demographic transitions. In what became known as "classical transition theory," demographers stated that a series of changes that could globally be described as "modernization" would necessarily imply a change in fertility behavior.⁴ Among the most frequently cited elements of "modernization," we can include falling mortality rates, economic development, education, and urbanization.

Demographers have given mortality rates considerable attention as an explanatory factor of fertility. Three mechanisms that have been used to link mortality and fertility rates are the physiological effect, the replacement effect, and the hoarding effect.

The physiological and replacement effects are similar in that both of them point towards an increased likelihood of pregnancy following the death of a child. With the physiological effect, this happens by necessity through the "sudden termination of breastfeeding, which, in turn, triggers resumption of menses and ovulation and thus increases the period of exposure to a new conception" (Palloni and Rafalimanana 1999). The replacement effect, on the other hand, refers to the deliberate actions taken by a couple to have an additional birth in order to "compensate" for the death of an offspring; maybe because of the existence of a target family size.⁵

³In Galor and Weil (2000), it is actually the rate of change of technological progress (and not its level) that shifts parental choices in fertility.

⁴See Kirk (1996) for a useful description and commentary of the theory.

⁵See Ben-Porath (1976) for empirical evidence of the replacement effect.

The hoarding effect has been the object of more rigorous economic modelling. We talk of a hoarding effect when a family decides to have more births than their optimal number of children in order to protect themselves against the possibility of future high mortality in the family. Hoarding is an ex-ante precautionary measure that arises once the randomness of mortality events is taken into account and induces families to "insure" themselves against high mortality scenarios by having more births. Sah (1991) and Kalemli-Ozcan (2002, 2003) present models of fertility choice in which this effect is at play and where mortality has a positive effect on both gross and net fertility.⁶

Besides mortality, demographers have also claimed that the joint effects of economic development, education, and a change from rural to urban living would produce a shift in attitudes and preferences that imply lower fertility choices. An important point to note here is the very different conception that economists and demographers have of the link between education and fertility.

Economists, as we have seen, link fertility decisions with the educational levels *of the children*. Fertility and children's education are two endogenous variables jointly determined by a set of exogenous variables. Demographers, on the other hand, have stressed that fertility decisions may be a function of the educational levels *of parents*. In other words, the education of parents would be an exogenous explanatory factor of fertility. This view emphasizes the idea that more educated parents would embrace "modern" (or "western") values where limiting the size of one's family is both desirable and morally acceptable. Less controversially, education also plays an important role in spreading information about contraceptive methods and encouraging their use.⁷

In the same vein, economic development and urbanization are thought to alter the perceptions towards fertility control and emancipate women from a traditional paternal society in which they were expected to stay at home and raise children. In the empirical section of this paper, we will control for several of the aforementioned factors when analyzing the effects of mortality on fertility.

Alongside this theoretical work, what does the empirical record tell us about the factors causing changes in fertility? As it turns out, economists' efforts on the empirical side of the question have been rather modest. Although our understanding of the potential mechanisms affecting fertility is quite advanced, few attempts have been made to distinguish quantitatively

⁶Before relying too much on the hoarding effect as an explanation for the mortality–fertility link, we must note that its magnitude depends rather heavily on the particular modelling assumptions being made. Several realistic generalizations would eliminate or at least attenuate this effect: parents would be less likely to hoard children if each birth is costly (as in Cigno 1998) or if they can take the decision of replacing a child *after* the occurrence of mortality (as in Doepke 2005).

⁷For examples of this view in demography, see, inter alia, Bogue (1969), Stolnitz (1964), and Cox (1970). The idea has also been mentioned by economists such as Schultz (1973) and Easterlin (1974). A review and criticism can be found in Graf (1979), while Caldwell (1980) takes it into consideration within a general view of the role of education on fertility.

important factors from secondary ones. Galor (2005a, b) compares alternative explanations for the demographic transition but limits himself to a graphical analysis and does not consider the evidence from developing countries. Most other papers only provide some "stylized facts" based on the experience of one or a few developed countries.

Very few papers in economics have provided careful econometric analyses of the determinants of fertility levels across countries. A notable exception is Schultz (1994), who analyzes the role of factors such as schooling and urbanization in this context.⁸ Schultz (1994) does not examine the role of mortality changes, which will be the focus of this paper, and does not control for economic development as we do here. We also improve on Schultz's work in terms of country and time coverage and in the use of more advanced econometric techniques.

Two recent empirical papers that are also related to the present work are Acemoglu and Johnson (2007) and Lorentzen et al. (2008). These two papers are mainly concerned with the effect of mortality on economic growth, but some of their results are relevant for us. Lorentzen et al. (2008) argue that mortality may affect economic growth through several channels, one of them being its effects on fertility. To investigate whether this channel is indeed at work, the authors regress a measure of fertility against the adult mortality rate and the infant mortality rate, which are included simultaneously. Both measures are found to exercise a positive, large, and statistically significant effect on fertility. Acemoglu and Johnson (2007) do not estimate the effect of mortality on fertility but they do analyze its effect on two related variables: total population and total number of births. They find that life expectancy (a negative function of mortality rates) is positively related with both variables but that the effect on births is stronger. If we consider that fertility rates are nothing more than ratios of total births to total population, their results suggest that mortality has a negative effect on fertility, contrary to the findings of Lorentzen et al. (2008). As we discuss in the next section, the contrasting results obtained by these two papers may be explained by the fact that none of them consider the dynamics of the relationship between mortality and fertility.

Besides the above mentioned papers, demographers have also produced a very large body of empirical work on this subject over the past few decades. Their analyses, however, have usually suffered from the failure to adopt econometric techniques that are now common in the economics literature and control for relevant empirical biases.⁹

⁸Earlier contributions by this same researcher are Schultz (1969, 1978).

⁹For instance, a large body of work in demography argues against the importance of socioeconomic factors such as mortality or GDP per capita in demographic transitions (see Coale and Watkins 1986 for an overview). These analyses do not use panel data techniques such as the inclusion of fixed effects to control for unobserved country-specific characteristics. Recent research has shown that controlling for these aspects restores the role of socioeconomic factors (Brown and Guinnane 2007).

It is thus apparent that the significant theoretical advances in the study of demographic transitions by unified growth theorists have not been matched on the empirical side. We believe that research effort in this area needs to be reallocated towards more empirical work and act in consequence. This paper focuses on the role of mortality as a driver for fertility change, but we will also discuss the effects of other factors that will be included as control variables in our empirical analysis. Our results show that changes in mortality have a large and statistically significant effect on fertility over time. Mortality changes go a long way towards explaining fertility transitions, and they may be the single largest explanatory factor behind these events.

The rest of this paper is organized as follows. The next section discusses the empirical methodology to be used and presents the data. Results are discussed in Section 3, and Section 4 summarizes and offers some concluding remarks.

2 Methodology and data

2.1 Methodology

Our aim is to study how mortality rates affect fertility controlling for other potential determinants of fertility that have been discussed in the literature. There are, however, at least three points that ought to be given serious consideration in an empirical analysis of this nature.

The first point refers to the dynamics of the relationship between mortality and fertility. This dimension is absent from theoretical models, where it is always assumed that parents know all relevant mortality rates at the moment of making their fertility decisions. This may be a natural assumption within theoretical models but it needs to be revised for empirical work. Since the early days of the classical transition theory, it has been a natural assumption among demographers that a lag of several years exists between mortality declines and the corresponding changes in fertility. Falling mortality rates are not readily observable for households and it is only after one or two decades, when cumulated changes become obvious to everyone, that families might feel confident enough to take them into account in their fertility plans. Our empirical study allows for this dynamic pattern by using several lags of mortality rates as potential determinants of current fertility.

Incidentally, the absence of this dynamic dimension may help us understand why Acemoglu and Johnson (2007) find results in opposition to Lorentzen et al. (2008). The analysis of birth rates in Acemoglu and Johnson (2007) covers the period from 1940 until 1980 or 1990. Lorentzen et al. (2008), on the other hand, run regressions over the period 1960–2000. If we consider, as Acemoglu and Johnson (2007) long document, that mortality rates began falling in developing countries only from the 1940s onwards, the lagged response of fertility would imply that we will not see any effect on this variable until, say, the 1960s. According to this interpretation, the study of Lorentzen et al. (2008) would be carried entirely over the period when fertility rates were responding to lower mortality, whereas Acemoglu and Johnson (2007) would be considering a time period whose first half saw no reaction at all.

The second point we make refers to the importance of country-specific factors that may have a large impact on fertility, such as culture, religion, and climate. We address this point by including country-specific fixed effects in all our regressions. This specification is superior to the alternative of including dummy variables for, say, Muslim countries or African countries, and that for at least two reasons. First, there are several country-specific characteristics that we cannot properly measure, such as culture. Second, the fixed effects specification is less constraining since it allows all Muslim or African countries to be different from each other.

A third point, finally, is that, even after controlling for all time-invariant, country-specific characteristics by the inclusion of fixed effects, a regression of fertility on mortality may be biased by the existence of reverse causality. Indeed, since the presence of many children implies fewer resources available per child, we can conjecture that causality may also run from high fertility to high mortality. We propose to deal with this endogeneity problem by using, next to traditional regression methods, the GMM methodology of Arellano and Bond (1991). Note, however, that while an endogeneity problem might affect the coefficient on contemporaneous mortality, this should not be a problem for lagged values of mortality. In other words, we may assume safely that today's fertility does not have an effect on past values of mortality.

With all of the above in mind, our baseline econometric specification will be as follows:

$$f_{i,t} = \alpha_i + \sum_{s \in S} \beta_s m_{i,t-s} + \sum_j \gamma_j x_{i,t,j} + \varepsilon_{i,t}, \tag{1}$$

where $f_{i,t}$ is a measure of fertility for country *i* at time *t*, $m_{i,t-s}$ is a corresponding measure of mortality with a lag of *s* years, $x_{i,t,j}$ are a set of control variables that also affect fertility, and α_i are country-specific fixed effects.

As stated, Eq. 1 will incorporate several lags of the mortality measure in order to account for the effects of this variable over time. We will use three versions of Eq. 1 throughout our empirical work: one in which mortality affects fertility only contemporaneously ($S = \{0\}$) and two in which the effect extends over 10 and 20 years, respectively ($S = \{0, 10\}$ and $S = \{0, 10, 20\}$).¹⁰ In each case, we will be interested not only in the individual coefficients β_s but also in the sum of coefficients $\sum_s \beta_s$, which can be interpreted as the overall long run effect of a change in mortality rates.

Equation 1 will be estimated under three alternative econometric procedures. The first one will be a standard panel estimation with fixed effects. The

¹⁰Other lag structures give similar results. Including lags at 5-year intervals results in multicollinearity.

second one will be similar to the first one but will also incorporate time dummies. Time dummies may be important to take into account particular periods with unusually high or low fertility such as the post-war "baby-boom." The third procedure, finally, will be the GMM estimation suggested by Arellano and Bond (1991). In this procedure, we difference Eq. 1 and instrument the endogenous regressors in differences with the adequate lags of the regressors in levels. This estimation strategy relies on the assumption that lagged values of the regressors are uncorrelated with changes in the error term, which we will maintain throughout this work.¹¹

2.2 Data

Two concepts of fertility are usually used in the literature: gross and net fertility. Gross fertility relates to the number of births per person in the population, whereas net fertility relates to the number of surviving children per person. In this context, surviving children are usually understood as those reaching sexual maturity (and, thus, able to reproduce themselves, in turn). In this paper, we will explore the consequences of mortality changes on both gross and net fertility.¹²

The measures of gross and net fertility that we use are, respectively:

- The total fertility rate (TFR), which is defined as the number of *children* that would be born per woman if she faced the age-specific fertility rates prevailing in a given country at a given year during each of her childbearing years.
- The net reproduction rate (NRR), defined as the number of *daughters* that would be born per woman if she faced the age-specific fertility rates

¹¹A second estimation strategy that has also gained popularity in the literature is the system-GMM methodology of Arellano and Bover (1995) and Blundell and Bond (1998). We do not apply this methodology here since the assumption that it requires in our context, namely, that changes in the regressors are uncorrelated with the country-specific fixed effects, is unrealistic. High fixed effects correspond to the less-developed countries, and it is to be expected that these countries will also present the largest changes in variables such as mortality or GDP per capita. As discussed by Roodman (2007), system-GMM requires that "throughout the study period, individuals sampled are in a kind of steady-state"; which is clearly not our case since we are analyzing transitions from high to low fertility.

¹²Notice that, if mortality has a positive effect on fertility, the effect on net fertility will necessarily be smaller than the effect on gross fertility. Indeed, if higher mortality leads parents to increase their number of births, their number of surviving children will increase less than proportionally because fewer children survive under higher mortality.

A simple formalization of the argument can be provided in a two-period model in which gross fertility is a positive function of mortality rates, GF(m) with GF' > 0, and mortality takes place only in the first period. Net fertility would be defined as the number of children surviving to the second period: NF = (1 - m)GF.

It follows that the elasticity of net fertility with respect to mortality would be $\varepsilon_{\text{NF}} = \varepsilon_{\text{GF}} - \frac{m}{1-m}$, where $\varepsilon_{\text{GF}} > 0$ is the elasticity of gross fertility with respect to mortality. In other words, ε_{NF} is necessarily smaller than ε_{GF} and potentially of negative sign.

prevailing in a given country at a given year during each of their childbearing years and the age-specific mortality rates from her birth until her child-bearing years.¹³

Turning our attention to mortality, we have three alternative measures at our disposition:

- The child mortality rate, the number of deaths between ages 0 and 5 per 1,000 live births.
- The adult mortality rate, the number of deaths between ages 15 and 60 per 1,000 persons reaching the age of 15.
- Life expectancy at birth, the average number of years that a person would live if faced with all age-specific mortality rates prevalent in a given country at a given year. Unlike the two previous measures, life expectancy is a negative function of mortality rates.

All the theoretical channels linking mortality to fertility that we have discussed in the first section of this paper predict a positive relationship between these two variables. Indeed, high mortality should lead to high fertility either because the death of a child increases the probability that parents will have a new birth (physiological and replacement effect), or because an environment of high mortality leads parents to insure themselves by having more children (hoarding effect), or because high mortality will lead parents to chose child quantity over child quality (quantity–quality tradeoff). In terms of Eq. 1, we would thus expect the overall effect of mortality, $\sum_{s} \beta_{s}$, to be positive. Some of the individual coefficients β_{s} may not be positive though, due to the potential lagged reaction of fertility.

Regarding our three measures of mortality, life expectancy will be our preferred measure since it is the only one incorporating mortality rates at all stages in life. A reason for this preference is that, while the physiological and replacement effects are caused mainly by mortality during the first years of life, the hoarding effect and the quantity–quality tradeoff are affected by mortality at all ages. It may, thus, be the case that only life expectancy will be able to account for the full effects of mortality changes on fertility.

The control variables that we use, finally, are chosen to take into account the most relevant factors used in the economic and demographic literatures to explain fertility transitions.¹⁴ These are:

• The level of education in the adult population, as measured by the average number of years of schooling for the population aged 15 or over

¹³Since the NRR counts only daughters and the TFR counts sons and daughters, the ratio of NRRs to TFRs is about 1/2 in developed countries (where mortality rates during childhood are very low) and considerably less than 1/2 in developing countries.

¹⁴There are other potential explanatory factors that have not been included due to lack of appropriate data. Of particular importance may be the presence or absence of public policies such as transfer payments and tax deductions for children. Such policies, however, are less relevant for developing countries, where most of the variation in the data takes place.

Table 1	Descriptive statistics	Variable	Mean	Standard deviation	Min.	Max.
		Total fertility rate	4.59	2.08	0.94	8.70
		Net reproduction rate	1.74	0.64	0.45	3.31
		Child mortality	105.50	90.76	3.0	500.0
		Adult mortality	271.28	150.48	56.73	889.04
		Life expectancy	58.32	12.50	23.60	80.53
		Average years of schooling	4.87	2.91	0.09	12.05
		GDP per capita (in logs)	7.90	1.10	5.35	10.38
		Urban ratio	44.08	24.36	1.40	100.0

- The level of economic development, as measured by the country's GDP per capita (measured in logs)
- The level of urbanization, as measured by the urban ratio

An important point is that our education variable measures average schooling in the *adult population*. It does not, therefore, correspond to the educational concept in the quantity–quality framework but to the educational concept in the demographers' classical transition theory. This is only natural since, as we have argued, in the quantity–quality framework, fertility and children's education are both endogenous variables; it would be wrong to estimate the effect of one on the other. In classical transition theory, on the other hand, parental education is an exogenous determinant of fertility, and its inclusion as an exogenous regressor is thus correct.

Economic development, as we have discussed, has figured prominently both in the economists' and in the demographers' explanations of fertility decline, while urbanization has been considered only by demographers as an important factor. According to the theoretical mechanisms previously reviewed, we should expect a negative coefficient for all our control variables. Indeed, demographers would argue that higher levels of education, economic development, and urbanization contribute to the change in attitudes that bring about fertility decline. Economists would argue that economic development makes investments in human capital more attractive and tips parental choices towards less child quantity and more child quality.¹⁵

The source for our measures of gross and net fertility, child and adult mortality, life expectancy, and the urban ratio is the United Nations' Common Database. This source provides us with 11 quinquennial observations per country from 1955 to 2005.¹⁶ The average number of years of schooling is taken

¹⁵One might note, however, that economists have also proposed other mechanisms linking economic development with fertility that do not work through the quantity–quality tradeoff. For example, children can be regarded as "normal goods," and higher incomes would lead parents to increase fertility.

¹⁶For life expectancy, there are no observations for 2005. We have used linear interpolation to fill in the gaps in the data for child mortality, adult mortality, and life expectancy.

Table 2 Correlation	n matrix							
	Total fertility	Net reproduction	Child	Adult	Life	Average years	GDP per capita	Urban
	rate	rate	mortality	mortality	expectancy	of schooling	(in logs)	ratio
Total fertility rate	1							
Net reproduction	0.9241	1						
rate								
Child mortality	0.8228	0.6027	1					
Adult mortality	0.7138	0.5050	0.8095	1				
Life expectancy	-0.8457	-0.5910	-0.9525	-0.9153	1			
Average years of	-0.8495	-0.7409	-0.8208	-0.7163	0.8600	1		
schooling								
GDP per capita	-0.7258	-0.5636	-0.7871	-0.7592	0.8336	0.8178	1	
(in logs)								
Urban ratio	-0.6722	-0.4935	-0.7487	-0.7131	0.8022	0.7647	0.8328	1

from Barro and Lee (2000) and also consists of quinquennial observations, but these cover the period 1960–2000. Quinquennial observations of GDP per capita from 1955 to 2005 are taken from Maddison (2006). Our regressions cover up to 118 countries over the period 1960–2000.¹⁷ Most developing countries experienced a demographic transition, or the initial stages of one, during this period.

Table 1 presents descriptive statistics for all the variables, and Table 2 is a matrix of correlations among variables. A fertility transition is usually understood as the passage from a TFR of around six children per woman to two or fewer children per woman. NRRs are typically at or below replacement level (one surviving daughter per woman) following a demographic transition; down from levels of two or more. All our regressors are clearly correlated with gross and net fertility, although mortality and education present stronger correlations than GDP per capita and the urban ratio, and the correlations are stronger with gross than with net fertility.

3 Empirical results

We start by analyzing the effects of child mortality on TFRs (Table 3) and on NRRs (Table 4). These and each of the following tables report the results of nine regressions since we estimate each relationship using three econometric procedures and, for each procedure, three alternative lag structures for mortality.

The first result that emerges from Table 3 is that the time dimension is indeed important in the relationship between mortality and fertility. Columns 1, 4, and 7 would lead us to think than there is a contemporaneous effect of child mortality on fertility, but this coefficient is statistically significant only when the lagged values of mortality are not present. Once we include the 10-year or 20-year lags of child mortality, we obtain an intuitive dynamic pattern with very mild effects contemporaneously and strong effects arriving with a 10-year lag. We also observe that the effect "dies out" after 10-years, with a small residual effect being reported for the 20-year lag.

The inclusion of time dummies alters results only marginally, as a comparison of columns 1–3 with columns 4–6 shows. In both cases, the total effect of child mortality on gross fertility after 20 years, the sum of coefficients $\sum_{s} \beta_{s}$, is close to 0.0125. This number implies that a rise in child mortality of one standard deviation would produce an increase in TFRs of 1.13 children per woman, a large and meaningful effect. The size of the effect is reduced in the GMM estimates, though it remains clearly statistically significant.

¹⁷Most variables are available for as many as 152 countries, but the data on education severely reduce this number. It is still the case, though, that all major developing and developed countries are included in our regressions.

	Dependent v	/ariable: TFRs							
	Panel with fi	xed effects		Panel with f time dummi	ixed effects an es	p	Difference-	GMM	
Child mortality	0.0049	-0.0002	-0.0020	0.0053	0.0012	-0.0014	0.001	-0.0161	-0.0079
3	0	0.887	0.255	0	0.475	0.387	0.801	0.011	0.107
Child mortality, lagged 10 years		0.0086	0.0126		0.0075	0.0136		0.0149	0.0145
		0	0		0	0		0.001	0
Child mortality, lagged 20 years			0.0017			0.0005			0.0008
			0.454			0.818			0.822
Sum of coefficients on	0.0049	0.0084	0.0123	0.0053	0.0087	0.0127	0.0007	-0.0012	0.0074
child mortality	0	0	0	0	0	0	0.801	0.762	0.051
Average years of education	-0.395	-0.301	-0.231	-0.323	-0.308	-0.249	-0.637	-0.38	-0.364
	0	0	0	0	0	0	0	0.036	0.029
GDP per capita (in logs)	-0.304	-0.212	0.027	-0.392	-0.196	0.063	0.153	0.436	0.371
	0.03	0.042	0.781	0	0.072	0.544	0.591	0.163	0.124
Urban ratio	-0.019	-0.027	-0.026	-0.017	-0.027	-0.027	-0.024	-0.031	-0.048
	0.005	0	0.001	0.011	0	0	0.336	0.157	0.016
Observations	898	711	514	898	711	514	779	593	399
Countries	117	117	115	117	117	115	105	105	103
Instruments							122	113	88
Estimated coefficients are in bold	and <i>p</i> -values a	are given belov	<i>x</i> them. <i>p</i> -valu	tes are calculat	ed using robu	st standard err	ors		

Table 3The effects of child mortality on gross fertility

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Table

	Dependent va	ariable: NRRs							
	Panel with fix	ed effects		Panel with fix time dummies	ed effects and		Difference-G	MM	
Child mortality	-0.0017	-0.0036	-0.0041	-0.0017	-0.0029	-0.0038	-0.0053	-0.0114	-0.0074
	0	0	0	0	0	0	0	0	0.002
Child mortality, lagged 10 years		0.0033	0.0040		0.0024	0.0044		0.0060	0.0048
		0	0		0.007	0		0.005	0.004
Child mortality, lagged 20 years			0.0015			0.0005			0.0007
			0.167			0.64			0.667
Sum of coefficients	-0.0017	-0.0003	0.0014	-0.0017	-0.005	0.0011	-0.0053	-0.0054	-0.0019
on child mortality	0.0000	0.558	0.1405	0.0000	0.4329	0.3036	0.0000	0.0016	0.3118
Average years of education	-0.184	-0.153	-0.124	-0.141	-0.141	-0.117	-0.267	-0.167	-0.169
	0	0	0	0	0	0	0.001	0.064	0.038
GDP per capita (in logs)	-0.161	-0.106	0.012	-0.201	-0.092	0.036	-0.068	-0.198	0.2
	0	0.03	0.804	0	0.069	0.48	0.625	0.205	0.116
Urban ratio	-0.006	-0.010	-0.012	-0.004	-0.009	-0.011	-0.006	-0.012	-0.023
	0.37	0.001	0	0.108	0.003	0.001	0.569	0.264	0.02
Observations	898	711	514	868	711	514	779	593	399
Countries	117	117	115	117	117	115	105	105	103
Instruments							122	113	88
Estimated coefficients are in hald	io sulta a pue p	moled aeries	them n volu	as are coloniate	andor a distribution	t standard arr	540		

Estimated coefficients are in bold and *p*-values are given below them. *p*-values are calculated using robust standard errors

The magnitude of the effect that we find is in the same range of the estimates in Lorentzen et al. (2008). These authors find that one standard deviation in infant mortality would produce a change in TFRs of 0.73.¹⁸ The difference between our estimate and theirs may be explained by two factors: first, Lorentzen et al. (2008) include adult mortality and infant mortality simultaneously, whereas we have only child mortality in our regressions. Second, Lorentzen et al. (2008) only use contemporaneous mortality, whereas we include lagged values. As Table 3 makes clear, studying the effect of mortality on fertility using only contemporaneous mortality rates would give us a much reduced effect.

Turning to our control variables, education and the urban ratio appear to have a sizeable effect on fertility while GDP per capita turns out to be a much weaker predictor of fertility. Our measure of education is statistically significant in all regressions, and its value is maintained when we include time dummies or when we use the GMM methodology. With a coefficient of around -0.300, a one-standard-deviation increase in education is associated with a decline in TFRs of 0.87. The urban ratio is also significant in the first six columns but loses significance in some GMM regressions. The size of its coefficient implies an effect on fertility of roughly similar size to that of education.

The coefficient of GDP per capita, which has the expected negative sign in columns 1 and 3, is considerably reduced when the 10-year lag of mortality is included and ends up becoming positive and not significant when the 20-year lag of mortality is added. In the GMM regressions, GDP per capita is never significant, and its coefficient is always positive. These results are a strong indication of a weak role of GDP per capita when compared with mortality or education.

In Table 4, we turn our attention to the effects of child mortality on net fertility. The results are consistent with those presented in Table 3. Table 3 revealed that, once we include lagged values of child mortality, the contemporaneous effect of child mortality on gross fertility is small and not statistically significant, in accordance with the hypothesis that families need some time to "internalize" changes in the socioeconomic environment. It follows that a rise in child mortality should decrease net fertility contemporaneously. The reason is simply that higher mortality rates will decrease the number of surviving children immediately; there is no lag for this effect. This is indeed the case in our results, as the negative and strongly significant coefficients on contemporaneous mortality in all columns of Table 4 clearly show.

This negative effect of child mortality on NRRs is reversed after 10 years, as is denoted by the positive and statistically significant coefficients on the lagged

¹⁸My own calculations using the results in column 6, Table 9 from Lorentzen et al. (2008).

values of mortality. The overall effect, however, appears to be close to zero. Even the equations that include 10- and 20-year lags of child mortality estimate that the overall effect after 20 years is just about positive but not statistically significant. GMM estimates conserve this same pattern but once again reduce the effect of mortality in most regressions.

Regarding the rest of the variables, results are once again consistent with education being always significant and large, the urban ratio usually significant and with a somewhat smaller effect, and GDP per capita being not significant once mortality lags are included.

When we use adult mortality instead of child mortality in the regressions of Tables 3 and 4, we obtain essentially the same results. The overall effect of a one-standard-deviation increase in adult mortality on TFRs is 0.75, whereas the overall effect on NRRs is, as in the case of child mortality, close to zero and not statistically significant. The coefficients of the control variables suffer only minor changes. We do not report these results for conciseness, but they are available upon request.

To summarize our results up to this point, we have found that the overall effect of mortality rates on gross fertility is, as expected, large and positive but that the overall effect on net fertility is close to zero. The dynamics of the process are in line with demographers' predictions: gross fertility reacts with a lag of about 10 years to mortality changes. This lagged reaction implies that net fertility will initially fall, and, in our estimates so far, its subsequent rise roughly compensates for this initial movement.

The above results have been obtained by considering either child mortality rates or adult mortality rates as the explanatory factor of interest. As we have argued before, however, both the hoarding effect and the quantity–quality tradeoff suggest that fertility choices will depend on mortality rates at all ages, not just on mortality rates early or late in life. It is thus important to consider how these results change when using life expectancy as our measure of mortality rates.

Table 5 reports the effects of life expectancy on gross fertility, while Table 6 contains the corresponding results for net fertility. In these two tables, we have premultiplied life expectancy by the factor (-1) in order to have a positive function of mortality rates and, thus, coefficients of similar sign to those obtained in Tables 3 and 4.

The results in Table 5 have many similarities with those reviewed in Table 3, but also some interesting differences. Changes in life expectancy do not affect TFRs contemporaneously once lagged values are taken into account, as was the case with child mortality rates. On the other hand, the effects of life expectancy do not "die out" after 10 years but have a similar or even larger effect at 20-year lags than at 10-year lags. Correspondingly, the overall effect of mortality rates on gross fertility after 20 years is larger with life expectancy than with child mortality. The sum of β_s coefficients in columns 3, 6, and 9 predict an overall decline of TFRs of between 1.61 and 2.01 children per woman following a one-standard-deviation increase in life expectancy. We interpret this result as evidence that mortality rates at all stages in life matter

Table 5The effects of life expectancy on gross fertility

	Dependent v	ariable: TFRs							
	Panel with fix	xed effects		Panel with fix	ed effects and		Difference-G	MM	
Life expectancy	0.053	-0.016	-0.009	0.058	-0.011	-0.002	0.056	-0.059	-0.016
	0	0.092	0.366	0	0.268	0.838	0.03	0.008	0.332
Life expectancy lagged 10 years		0.132	0.069		0.146	0.073		0.172	0.050
		0	0		0	0		0	0.030
Life expectancy lagged 20 years			0.069			0.090			0.110
			0			0			0.000
Sum of coefficients on	0.053	0.116	0.129	0.058	0.135	0.161	0.056	0.113	0.144
life expectancy	0.000	0	0	0.0000	0	0	0.0300	0	0
Average years of education	-0.365	-0.194	-0.139	-0.286	-0.243	-0.216	-0.413	-0.303	-0.231
	0	0	0	0	0	0	0.01	0.03	0.080
GDP per capita (in logs)	-0.227	-0.281	0.004	-0.305	-0.319	0.025	-0.323	-0.279	0.180
	0.018	0.002	0.966	0.001	0.001	0.794	0.206	0.274	0.403
Urban ratio	-0.017	0.002	-0.013	-0.015	-0.002	-0.017	-0.026	0.027	-0.010
	0.011	0.708	0.045	0.024	0.807	0.006	0.188	0.153	0.547
Observations	911	818	629	911	818	629	791	698	510
Countries	118	118	118	118	118	118	106	106	106
Instruments							136	133	115
Estimated coefficients are in bold	and p -values a	re given below	them. <i>p</i> -valu	ies are calculate	d using robust s	standard err	ors		

115

Table 6 The effects of life expect.	ancy on net fer	tility							
	Dependent v	ariable: NRR							
	Panel with fi	xed effects		Panel with fix time dummie	ced effects and s		Difference-G	MM	
Life expectancy	-0.012 0.007	-0.035	-0.029 0	-0.010 0.038	-0.032 0	-0.025 0	-0.010 0.393	-0.052 0	-0.031 0.001
Life expectancy lagged 10 years		0.048	0.020		0.050	0.019		0.065	0.012
Life expectancy lagged 20 years		0	0.035		0.043	0000		○ ⊂	0.050
Sum of coefficients on	-0.012	0.013	0.026	-0.010	0.018	0.037	-0.010	0.013	0.031
life expectancy	0.007	0.0028	0	0.0380	0.0003	0	0.3930	0.2195	0.0191
Average years of education	-0.193	-0.128	-0.090	-0.153	-0.139	-0.120	-0.244	-0.195	-0.147
	0	0	0	0	0	0	0.002	0.005	0.018
GDP per capita (in logs)	-0.196	-0.209	-0.081	-0.236	-0.225	-0.068	-0.305	-0.275	-0.077
	0	0	0.057	0	0	0.122	0.024	0.043	0.433
Urban ratio	-0.003	-0.004	-0.002	-0.001	0.003	-0.003	0.003	0.022	0.005
	0.272	0.149	0.466	0.486	0.326	0.18	0.73	0.014	0.484
Observations	911	818	629	911	818	629	791	698	510
Countries	118	118	118	118	118	118	106	106	106
Instruments							136	133	115
Estimated coefficients are in bold	and <i>p</i> -values a	tre given below	them. <i>p</i> -valu	tes are calculat	ed using robus	t standard err	STC		

116

L. Angeles

for fertility decisions and that all the potential mechanisms linking these two variables are likely to be at work.¹⁹

With respect to all other variables, their effects remain very similar: education always has a negative and statistically significant effect, GDP per capita is not robust to the inclusion of all lags of mortality or to the GMM estimation, and the urban ratio is somewhere between these two. In addition, we remark that the use of life expectancy instead of child mortality has tended to produce smaller coefficients for most control variables: the size of the effect of education is up to a third smaller than what it was in Table 3, and for the urban ratio, the reduction can be even larger. We hypothesize that the effect of the omitted post-childhood mortality rates in Table 3 was partially taken up by our control variables.

When we turn our attention to net fertility (Table 6), the results are affected in consequence. We find again the intuitive result that net fertility is negatively related to contemporaneous changes in mortality rates, as denoted by the negative and statistically significant coefficient on the nonlagged level of life expectancy. The changes in parental behavior that follow over the next 20 years, however, are now found to more than compensate this initial effect. In our three estimation procedures, the sum of coefficients becomes positive and statistically significant once we extend the lag length to 20 years. This sum of coefficients takes values between 0.026 and 0.037, implying an overall rise of NRRs of between 0.32 and 0.46 for a one-standard-deviation increase in life expectancy, a large and meaningful effect.

Our results using life expectancy suggest that changes in mortality are a large, and possibly the largest, cause of changes in fertility rates; only parental education has effects of similar magnitude in some regressions. If we take the regressions that include fixed effects, time dummies, and up to 20-year lags as our benchmark and we calculate the effect on TFRs of a one-standard-deviation change in each exogenous variable, we find that the effect of life expectancy, 2.01, is much larger than the effects of parental education (0.63), urbanization (0.41), and GDP per capita (0.03). The same calculation for the NRRs reveals an effect of life expectancy of 0.46 against effects of 0.35 for parental education, 0.07 for the urban ratio, and 0.07 for GDP per capita.

4 Conclusions

This paper contributes to the ongoing research effort improving our understanding of demographic transitions. The unified growth literature has produced many valuable theoretical contributions in this area, but we are lacking

¹⁹Unfortunately, it is beyond the scope of this paper to estimate the relative importance of the different mechanisms linking mortality to fertility. All we can say at this point is that the physiological and replacement effects would not be sufficient to explain our results since life expectancy has an effect larger than child mortality and adult mortality remains significant when included alongside child mortality (this last result is not shown in the tables).

empirical studies to help us differentiate between first- and second-order mechanisms. We advance in that direction by analyzing in detail the role of mortality as a cause of fertility reductions while controlling for other prominent factors used in the economic and demographic literatures.

Our main results can be summarized as follows:

- 1. Mortality changes have a large impact on fertility reductions and can account for a major part of the fertility change characterizing demographic transitions. This result is obtained controlling for alternative explanatory factors of fertility, country-specific fixed effects, time dummies, and using the GMM methodology of Arellano and Bond (1991) to control for endogeneity.
- 2. Both gross and net fertility are affected by mortality. The overall effect on net fertility becomes large and statistically significant once we take into account mortality rates at all ages by using life expectancy.
- 3. Gross fertility reacts to mortality changes with a lag of about 10 years; the effects continue to be felt after 20 years. Net fertility has an initial negative relationship with mortality; the direction of the effect being reversed after 10 years.
- 4. Compared with other factors, the effect of mortality is larger than those of GDP per capita and the urban ratio. Only parental education has an effect of similar magnitude in some regressions.

Turning back to the theoretical literature, our results bring support to the large part of the literature emphasizing the role of mortality changes but, at the same time, sides against the equally large part of the literature whose mechanisms are based on changes in GDP per capita or, more often, technological change. While the link between technological change and GDP per capita might be tenuous in the short run, over a time horizon of several decades like the one considered here a strong link can be reasonably expected. We must be cautious, however, before ruling out economic development as a potential cause of fertility change. While we have found a small or even inexistent direct effect of GDP per capita on fertility, many indirect channels might be in place. In particular, economic development might be a major cause of mortality reductions and would, therefore, affect fertility through this last variable.

Acknowledgements I am greatly indebted to two anonymous referees for comments and suggestions. All remaining errors are, of course, mine.

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