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Essays on the Relationship Between Fertility and Child Mortality

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Notice

Chapters 1 to 3 of this thesis are three independent research articles and therefore the terms "paper" and "article" might appear in multiple instances of the chapters. Some information might also, as a consequence, be found in multiple chapters.

The responsibility for opinions expressed in this thesis rests solely with the author and Aix-Marseille University neither approves nor disapproves these opinions.

Avertissement

Les chapitres 1 à 3 de cette thèse sont issus d'articles de recherche rédigés en anglais et dont la structure est autonome et explique la présence des termes "paper" et "article" dans les chapitres. Ainsi, cetaines informations sont suceptibles d'être répétées dans ces chapitres.

Les opinions exprimées dans cette thèse n'engagent que l'auteur et l'Université d'Aix-Marseille n'entend ni approuver, ni désapprouver ces opinions.

Abstract

This dissertation attempts to contribute to the understanding of current demographic trends in sub-Saharan Africa by examining the role of child mortality in shaping fertility behavior.

In the first chapter of this dissertation, I examine the relationship between child mortality and fertility at the micro level. Count data models are employed to investigate the determinants of completed fertility of women from a Senegalese rural community. The global effect of child mortality on total and net fertility is found to be positive. I further identify an inverted-U shaped relationship between child mortality and net fertility. The implication is that health policies aiming at reducing child mortality have indirect effects on desired fertility; yet only a steep decrease in child mortality would be likely to trigger substantial fertility declines.

In the second chapter of this dissertation, I analyze the effects of child mortality changes on fertility behaviors in an endogenous fertility model where child survival is stochastic. I adopt a functional form for the cost of children that allows for four different scenarios, each of which is representative of a particular socio-economic setting. Demographic transition theories usually predict a positive relationship between child mortality and total fertility. I prove analytically that this relationship can be reversed depending on the shape of the total child cost curve considered. My model can predict both positive and negative fertility responses to child mortality depending on whether children are "time-intensive" or "time-supplying", respectively. I argue that this model can provide one explanation for the stalled demographic transition in today's sub-Saharan Africa.

Finally, the third chapter analyzes the effects of childhood mortality and morbidity on the fertility decision-making process among rural Senegalese women. I estimate nonlinear dynamic panel data models of fertility behavior. I am able to identify the causal effect of individual child mortality, and also that of community child mortality and morbidity, on subsequent fertility choices. The results provide consistent support for both the child-replacement hypothesis and the precautionary demand for children. I find that community child mortality and morbidity attributable to malaria, which capture exogenous changes in the epidemiological context, exert a joint influence on fertility behaviors. Community-level malaria incidence among children has a positive effect on subsequent fertility choices, and this positive effect is stronger the more the disease is fatal to children who are infected. I argue that the persistently high childhood disease incidence is not responsible *per se* for sub-Saharan Africa's sluggish demographic transition. Rather, childhood morbidity contributes to the slow transition to the extent that uncertainty about child survival remains a major concern in the region.

Keywords: Fertility · Child mortality · Child morbidity · Malaria · Sub-Saharan Africa · Replacement hypothesis · Insurance hypothesis · Endogenous fertility · Cost of children · Uncertainty.

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Naturellement, je reste le seul responsable des imperfections et des erreurs qui peuvent demeurer.

Notre route est droite, mais la pente est forte.

Contents

General introduction

Sub-Saharan Africa is experiencing a slow demographic transition. Although child mortality shows a downward trend from the 1950s, net fertility rates remain particularly high (Bongaarts and Casterline, 2013). Everything happens as if most countries in sub-Saharan Africa were still at the early stage of the transition. The average fertility gap between sub-Saharan African countries and the rest of the world is unambiguously large and constant (United Nations, 2013). Reducing the growing demographic pressure appears to be a major challenge for the socio-economic development of these countries. A conventional view of the demographic transition is that fertility is likely to follow the decline in childhood mortality. This view apparently fails to explain contemporary demographic patterns in sub-Saharan Africa. In view of the aforementioned statistical regularities, it appears that additional theoretical and empirical analyses of the child mortality-fertility relationship are needed. This dissertation attempts to analyze the roles that child mortality and the uncertainty about child survival play in explaining sub-Saharan Africa's sluggish demographic transition.

It is important to examine sub-Saharan Africa's demographic trends in light of the fertility transition theories. The literature on the fertility transition can be broadly divided into two parts, following a classification dating back at least to Carlsson (1966).[1] On the one hand, the *innovation approach* postulates that reductions in fertility are only due to improvements in family planning technologies, stressing also the importance of the spread of information about birth control. High fertility levels would then be the consequence of an unmet need for contraception. On the other hand, the *adjustment* approach considers fertility as a volitional variable rather than an implicitly-imposed constraint. Households are expected to alter their fertility behavior according to the surrounding socio-economic environment and the epidemiological context, which can change incentives for childbearing. In the latter approach, access to contraception is a necessary but not sufficient condition for fertility reduction. I argue throughout this disseration that the *adjustment* approach prevails in explaining current fertility trends in sub-Saharan Africa. The availability of family planning services is crucial for the fertility transition to occur. Increased

[1]The reader is also referred to Bleakley and Lange (2009) for a more detailed description of both categories with an emphasis on recent literature.

use of modern contraception is obviously strongly associated with fertility decline. In developing countries, knowledge about the availability of methods to delay or avoid a pregnancy is now quite widespread. However, better levels of knowledge are far from being associated with higher use. Cleland et al. (2011) report that attitudinal resistance remains a severe barrier in sub-Saharan Africa, so that progress towards the adoption of contraception has been dramatically slow. Studies reveal that even though some components of fertility arise from an unmet need for family planning, its largest share is actually desired by households (Bongaarts, 2011). Acknowledging the statistical regularities previously reported, I will consider fertility as a choice variable, in the tradition of economic theory. This dissertation thus falls within the scope of the *adjustment approach*.

This dissertation attempts to contribute to the understanding of current demographic trends in sub-Saharan Africa by examining the role of child mortality in shaping fertility decisions. From a theoretical viewpoint, the effect of child mortality on fertility choices can be twofold. On the one hand, the *replacement effect* is at work when households have additional children to replace the ones that they lose, involving a sequential process. This strategy implies that households have a target number of surviving children. Thus, they react to a child's death by replacing that child, and as a result, mortality directly affects the total fertility rate. Replacement behavior alone is therefore sufficient to generate a positive relationship between child mortality and total fertility. Yet, net fertility (*i.e.* the number of survivors) would not be affected by such a strategy. On the other hand, the *insurance effect* (also know as the hoarding motive, the precautionary motive, or the child survival hypothesis) prevails when households anticipate that not all children will survive to adulthood. In an adverse epidemiological environment, households would have additional children in order to insure themselves against potential future losses and ensure a sufficiently large family size. Thus, net fertility would be likely to be positively affected by the adoption of such a strategy. From an economic theory standpoint, the replacement motive can be linked to the concept of adaptive expectations, while the insurance behavior can be linked to the concept of rational expectations. Indeed, in the former only part of the information is taken into account by households, while in the latter all available information is processed. In view of the growing demographic pressure in today's sub-Saharan Africa, addressing the extent to which households adjust their behavior in a context of low child survival probability appears to be a decisive research question. All three chapters of this dissertation examine the relationship between child mortality and fertility, although from different perspectives and with different approaches.

In Chapter 1, I empirically examine the relationship between child mortality and fertility at the micro level. More specifically, I consider the actual realization of a fertility strategy at the end of a woman's reproductive life. I use individual data collected quarterly within the Demographic Surveillance System of the rural community of Niakhar (Fatick, Senegal), which is Africa's oldest and still operational statistical

observatory. An in-depth analysis of the child mortality-fertility relationship is conducted based on the birth histories of 2,884 women born between 1932 and 1961. Here the focus is on completed fertility, that is, the number of births to a woman by the end of her childbearing period. Count data models are employed to investigate the determinants of completed fertility. The global effect of child mortality on total and net fertility is found to be positive, providing consistent support for the *insurance hypothesis*. I further identify an inverted-U shaped relationship between child mortality and net fertility. I show that mothers with the largest number of survivors are those for whom the burden of child mortality has not been particularly high. Therefore, the insurance mechanism mainly prevails for non-zero but relatively low values of child mortality. In addition, the positive mortality effect is found to decrease with the mother's age at first birth. Policies aiming at postponing the onset of childbearing are thus likely to have a direct negative impact on fertility, as well as an indirect negative one through the child mortality channel. I also find that the effect of child mortality is the highest for women belonging to older cohorts, offering hope for the diffusion of the fertility transition in the region. In view of the results of this chapter, the main implication is that health policies aiming at reducing child mortality have indirect beneficial effects on households' wanted fertility. However, only a steep decrease in child mortality would be likely to trigger substantial fertility declines. This does not mean that traditional family planning policies should be abandoned. Such policies have direct effects on households' unwanted fertility, and thus are a necessary condition for the fertility transition to be completed.

In Chapter 2, I analyze the effects of child mortality changes on fertility behaviors in an endogenous fertility model where child survival is stochastic. Endogenous fertility models analyzing the child mortality-fertility relationship systematically assume that the birth of each child entails a fixed cost to parents. In this chapter, I question whether a fixed cost function for children is appropriate in modeling the relationship between child mortality and fertility in an endogenous fertility framework. The motivation for building such a model is to contribute to the understanding of contemporary demographic trends in sub-Saharan Africa. Allowing the shape of the child cost function to vary across socio-economic settings can potentially explain the region's sluggish demographic transition relative to more rapid contemporary or historical transitions. I adopt a functional form for the cost of children that allows for four different scenarios, each of which is representative of a particular socio-economic setting. The empirical foundations of these four scenarios are supported by studies estimating the cost of children in high- and low-income settings. Demographic transition theories usually predict a positive relationship between child mortality and total fertility. I prove analytically that this relationship can be reversed depending on the shape of the total child cost curve considered. By allowing for a more flexible cost function that includes the cases of increasing and decreasing marginal cost of children, as well as total cost that is decreasing in the number of children, I show that the fertility response to child mortality may be either positive, in the former,

or negative, in the latter. Therefore, my model can predict both positive and negative fertility responses to child mortality depending on whether children are "time-intensive" or "time-supplying", respectively. More specifically, I show that when the cost of children is low, which is the case in low-income settings, total fertility increases with improvements in child survival. This would therefore result in a growing demographic pressure. This chapter contributes to recent theoretical discussion of whether existing fertility models attribute past and current demographic transitions to the appropriate mechanisms. In my model, fertility behaviors are jointly determined by health concerns and perceptions of economic opportunities. I argue that this model can provide one explanation for the stalled demographic transition in today's sub-Saharan Africa.

Finally, Chapter 3 investigates the effects of childhood mortality and morbidity on the fertility decision-making process. This analysis is conducted at the empirical level using longitudinal micro data from the rural community of Niakhar (Fatick, Senegal), for the period 1984-2011. I use a dynamic panel data framework in order to focus on the timing of fertility. The main purposes of this chapter are as follows. I attempt to estimate the magnitude of the child-replacement effect, if any, and to provide novel evidence shedding light on the behavioral component of replacement.[2] I also attempt to identify the causal effect of community child mortality and morbidity attributable to malaria on subsequent fertility choices. Malaria is both a disease of poverty and a cause of poverty. This disease is a leading cause of death among children under five in sub-Saharan Africa.[3] Beyond the adverse direct impacts on health, malaria has also important indirect effects and results in large social costs.[4] In this chapter, I examine whether improvements in malaria control are likely to have desirable indirect effects on fertility rates through the reduction of child mortality and morbidity. I estimate nonlinear dynamic panel data models of fertility behavior, allowing for state dependence and unobserved heterogeneity. I am able to identify the causal effect of individual child mortality, and also that of community child mortality and morbidity, on subsequent fertility choices. The results provide consistent support for both the child-replacement hypothesis and the precautionary demand for children. I find that community child mortality and morbidity attributable to

[2]A major issue of the empirical literature on the effect of child mortality on fertility has been to disentangle the behavioral and biological components of the replacement effect. Indeed, an infant death abruptly terminates breastfeeding and truncates the period of postpartum amenorrhea, which in turn can shorten the interval to the next birth. From a research and policy perspective, it is important to determine the relative contribution of behavioral and biological factors to the replacement effect.

[3]See Black et al. (2010) for an analysis of the global, regional, and national causes of child mortality.

[4]For instance, Thuilliez (2010) shows that the link between *falciparum* malaria endemicity and primary repetition rates is strong and positive. This suggests that malaria contributes to impede children's human capital accumulation, and, in turn, economic growth. See also Sachs and Malaney (2002) for a review of the (potential) indirect effects of malaria.

malaria, which capture exogenous changes in the epidemiological context, exert a joint influence on fertility behaviors. Community-level malaria incidence among children has a positive effect on subsequent fertility choices, and this positive effect is stronger the more the disease is fatal to children who are infected. I argue that the persistently high childhood disease incidence is not responsible *per se* for sub-Saharan Africa's sluggish demographic transition. Rather, childhood morbidity contributes to the slow transition to the extent that uncertainty about child survival remains a major concern in the region.

The main policy implication from all three chapters is that, along with economic development and tradition family planing programs, health policies aiming at reducing child mortality and morbidity have additional indirect effects on fertility decisions. To the extent, however, that uncertainty about child survival is greatly reduced, these health policies would be likely to trigger substantial fertility declines and, therefore, would contribute to reduce the demographic pressure in today's sub-Saharan Africa.

Chapter 1

The effect of child mortality on fertility behaviors is non-linear: New evidence from Senegal*

1.1 Introduction

Sub-Saharan Africa (SSA) has long been trapped in a crisis characterized by high mortality and fertility rates. However, mortality rates are continuously decreasing in a significant number of SSA countries from the 1950s, and life expectancy is projected to increase (United Nations, 2012). Concurrently, fertility rates remain markedly high: 39 out of the 55 African countries exhibit high current fertility, namely 5 children or more per woman (United Nations, 2011). This results in a growing demographic pressure. Demographic transition theories usually postulate that fertility is likely to follow the decrease in child mortality after some lag. Concerns are raised about the validity of these theories for the sub-Saharan Africa context, as they apparently fail to explain contemporary demographic patterns. Everything happens as if most countries in SSA were still at the early stage of the transition (Bongaarts and Casterline, 2013). The availability of family planning services is crucial for the fertility transition to occur. In developing countries, knowledge of contraception is now quite widespread. However, Cleland et al. (2011) report that attitudinal resistance remains a severe barrier in SSA, so that progress towards adoption of contraception has been dramatically slow. Studies reveal that even though some components of fertility arise from an unmet need for family planning, its largest share is actually desired by households (Bongaarts, 2011). Acknowledging the statistical regularities previously reported, I will consider fertility as a choice variable. Households are expected to alter their fertility behavior according to the surrounding socio-economic

*The work presented in this chapter has been published in the Review of Economics of the Household (see Bousmah, 2014).

and epidemiological context, which can change incentives for childbearing.

Addressing the extent to which households adjust their behavior in a context of low child survival probability is a decisive research question. Aside from direct health outcomes, health policies aiming at improving child survival could have indirect demographic effects via changes in household fertility behavior.[1] There is as yet no clear consensus in the economic and demographic literature as regards the relationship between child mortality and fertility in developing countries. A distinction is made between total fertility (the number of births per woman), and net fertility (the number of surviving children). The *replacement hypothesis* posits that households have additional children to replace the ones that they lose, involving a targeted number of children chosen by parents. Replacement behavior would then generate a positive relationship between child mortality and total fertility, leaving net fertility unchanged. However, it is worth noticing that among micro studies which attempt to empirically test the *replacement hypothesis*, estimates of direct replacement rates are systematically smaller than 0.5, as pointed out in Kalemli-Ozcan (2003). This means that a replacement strategy cannot be fully realized, as one-to-one replacement is never achieved, due for instance to physiological limits such as birth spacing, breastfeeding period or limited fecundity. In practice, replacement behavior is thus likely to generate a positive (negative) relationship between child mortality and total (net) fertility. The *child survival hypothesis* (also known as the *hoarding motive*) postulates a positive effect of child mortality on the number of surviving children. When uncertainty about child survival is high, households would generate a precautionary demand for children in order to ensure a sufficiently large family size. This theory predicts that improvements in child survival will lead to decreasing total *and* net fertility rates.

Clearly, replacement behavior alone is inconsistent with the observation of past and present patterns of demographic transition, during which both total and net fertility follow the decrease in child mortality. Within the endogenous fertility framework, several theoretical models have been developed to reveal the underlying factors behind the demographic transition. Research on the topic dates back to the seminal works of Becker *et al.* (1973; 1976; 1988), in which parents are assumed to have a dynastic utility function depending on their own consumption as well as on the number and consumption of descendants in all generations. In this framework, an increase in income, by affecting the cost of surviving children, should lead to a trade-off between child quantity and child quality. A permanent decline in the level of child mortality would, however, not directly affect the demand for surviving children (Becker and Barro, 1988).[2] An increase in the survival probability has nonetheless the effect of

[1] See for instance Sachs and Malaney (2002) for a review of the (potential) indirect demographic effects of malaria control policies.

[2] The idea that mortality decline alone is not sufficient to trigger net fertility decline can also been found in Doepke (2005), Boucekkine et al. (2009) or in the *unified growth theory* (*e.g.,* Galor,

lengthening the period during which returns to investment in children's human capital (quality) are realized. Parents may thus substitute child quality for child quantity following an exogenous mortality decline, which will lead to a decrease in net fertility, as pointed out in O'Hara (1975). Another factor that has been shown to play a central role in the demographic transition is the uncertainty about child survival. Sah (1991) predicts a positive impact of child mortality on fertility in the first attempt to incorporate uncertainty in a model of endogenous fertility choice. Also allowing for uncertainty about the number of surviving children, Kalemli-Ozcan (2003) builds a quantity-quality model of children and provides support for the *hoarding motive*, so that child mortality has a positive effect on both total and net fertility. There is a lack of micro-level studies that quantitatively test the actual realization of a fertility strategy at the end of the reproductive life span, partly due to the lack of data availability. The demographic and epidemiological monitoring of the population of Niakhar (Fatick, Senegal) allows me to empirically investigate this research question.

This paper conducts an in-depth quantitative analysis of the determinants of completed fertility in order to provide a better understanding of the relationship between child mortality and fertility in a rural SSA context. Empirical analysis of both gross and net effects of child mortality on fertility is carried out. I use a large longitudinal data set to analyze the birth histories of 2,884 women born between 1932 and 1961. The determinants of completed fertility are investigated using a standard Poisson Regression Model. Different model specifications are estimated to emphasize the potential heterogeneous effects of child mortality. The global impact of child mortality on total and net fertility is found to be positive. To my knowledge, this is the first paper providing evidence for the *child survival hypothesis* at the micro level. However, the effect on net fertility, even though positive and significant, is fairly small. I further show that the relationship is of non-linear nature, as the amplitude and the direction of the effect of child mortality vary with the number of child losses. More specifically, an inverted-U shaped relationship between child mortality and net fertility is exhibited. Finally, I find that the effect of child mortality is the highest for women belonging to older cohorts and for women who start childbearing earlier.

Although infant and child mortality rates are declining continuously since the 1950's, progress have been slower in the past two decades (United Nations, 2012).[3] In line with this empirical observation, I show that the highest fertility rates are essentially associated with relatively low values of child mortality. The implication is that, along with family planning policies which only affect unwanted fertility, health policies aiming at reducing child mortality have indirect effects on desired fertility. However, the decrease in child mortality should be steeper than the one experienced in SSA so far in order to trigger fertility declines.

2005).

[3]Contrastingly, the decline of infant and child mortality that have preceded the demographic transition in Europe and the rest of the industrialized world has been faster and sharper on average (Corsini and Viazzo, 1997).

1.2 Background and existing studies

The determinants of completed fertility are clearly identified in the demographic and economic literature. We thus understand completed fertility to be primarily influenced by economic conditions, educational and occupational status, religion, access to family planning services, child mortality, marital status, ethnicity, and social norms.[4] Accumulating evidence has shown that child mortality and fertility are inextricably linked (Montgomery and Cohen, 1998). A large number of studies document a positive association between gross fertility and child mortality (Taylor et al., 1976; Balakrishnan, 1978; Olsen, 1980; Olsen and Wolpin, 1983; Rosenzweig and Schultz, 1983; Eckstein et al., 1999; Handa, 2000; Hossain et al., 2007). Empirical studies providing evidence of a net effect of child mortality on fertility are much scarcer. To my knowledge, only three studies document both effects of child mortality on total and net fertility (Schultz, 1997; Angeles, 2010; McCord et al., 2010), and solely at the macro level. Using calorie availability per capita as an instrument for child mortality, Schultz (1997) finds that improvements in child health are associated with slower population growth during the period 1972-1988 in a panel of 80 developing countries. Angeles (2010) employs dynamic panel GMM techniques to account for the potential endogeneity of fertility and shows that both gross and net fertility are negatively affected by reductions in child mortality for a panel of 118 countries over the period 1960-2000. McCord et al. (2010) instrument child mortality with an ecologically-based spatial index of the stability of malaria transmission across countries and find that child mortality declines are likely to account for over half of the fertility decline during the demographic transition. The lack of micro data collected over a sufficiently long time-span, in particular in low-income countries, makes it difficult to quantify the net effect of child mortality on fertility at the individual level. Hence, microeconometric estimates of the effect of child mortality on fertility in developing countries usually rely on dynamic models and are based on data covering relatively short time periods.

Other attempts have been made to estimate the determinants of fertility using count data models of fertility, which are suitable for the analysis of birth histories.[5] However, these studies are mainly based on household composition data, and not on completed fertility. The effect of child mortality on fertility is investigated in some of these empirical papers. Nguyen-Dinh (1997) documents a small and positive effect of community-level child mortality on fertility in Vietnam.[6] In his analyses of fertility in four Arab countries, Al-Qudsi (1998a,b) finds that infant mortality exerts

[4] See Bulatao and Lee (1983) for an extensive review of the determinants of fertility in developing countries, and Bongaarts et al. (1984) for a focus on SSA countries.

[5] Winkelmann (2008) provides a review of the applications of count data models in the analysis of individual fertility with an emphasis on methodological issues.

[6] Concerns may be raised about the measurement of child mortality in Nguyen-Dinh (1997) as it is assumed that all children ever born were still alive at the time of the survey. Child mortality may thus be underreported.

a positive impact on fertility for replacement purposes. Finally, Atella and Rosati (2000) stress the importance of the uncertainty about child survival in determining fertility choices. Using data from India, the authors show that the negative fertility response to a decrease in child mortality may be dampened if it is also associated with a reduction of its variance. Such studies are typically designed to test the direct replacement effect of child mortality on fertility (birth spacing and timing) during the observation period. They provide a better understanding of the short-term aspect of the child mortality-fertility relationship, yet they do not bring any information about the actual realization of a strategy at the end of a woman's reproductive life.

The determinants of completed fertility appear to have been investigated in only a few empirical papers. Since these studies are based on data from developed countries, where child mortality is low, none of them examines the effect of child mortality on fertility. The present paper aims at filling this gap by determining the quantitative contribution of child mortality on completed fertility in a fairly representative rural West African setting.

1.3 Data: Niakhar Demographic Surveillance System

The rural community of Niakhar is located in the Fatick region of Senegal, 135 km east from Dakar. A Demographic Surveillance System (DSS) has been set up in 8 villages since 1962, and has been extended to 22 more villages in 1983.[7] Since 1983, the geographical boundaries of the study zone comprehends a total area of 203 km^2 and encompasses 30 villages. Niakhar is Africa's oldest and still operational statistical observatory, and world's second-oldest (after Matlab, Bangladesh). This is a fairly representative rural West African setting, and the study population is relatively homogeneous in terms of socio-economic characteristics. The Serer ethnic group comprises 96.5% of the population. Islam and Christianity are the main religions, representing respectively 76% and 21% of the population. The major cause of migration is seasonal migration to Dakar, the closest major urban center, for economic motives. Agriculture is the main source of livelihood, and formal education is very low. Villages are subdivided into hamlets, which are themselves subdivided into compounds. Compounds are constituted of one or more "kitchens" (households) which bring together members of the extended patrilineal family. The average household size is approximately 13 persons. Further information on the study area can be found in Delaunay et al. (2002). There are large variations between households regarding both fertility and child mortality rates. Hence, it is a convenient setting to

[7]Niakhar DSS has originally been set up and is still maintained by the *Institut de recherche pour le développement* (IRD).

analyze the relationship between these two variables.[8]

At the onset of the DSS, major life events such as birth histories were collected retrospectively among the individual residents of the area to serve as a baseline for the follow-up. The DSS consists in conducting quarterly exhaustive surveys within the study area. Thoroughly reliable data on all demographic events are systematically recorded. These events include pregnancies, deaths, marriages, migrations (inside or outside the study area), as well as changes in social characteristics. Such events are also being retrospectively and then systematically collected among the immigrants as they enter the study area. Accounting of pregnancies is practically comprehensive as a result of the quarterly follow-up. Although likely limited, the possibility of under-reporting of pregnancies is still possible, especially those ending in induced abortion. Data on mortality events are reported via verbal autopsy, and causes of death follow World Health Organization's ICD-9 classification. Altogether, this results in an exhaustive and systematic monitoring of the study population.

Alongside the systematic collection of data, several cross-sectional surveys were conducted for specific purposes. This study uses a cross-sectional survey conducted in 2003 to derive economic characteristics of households which remained supposedly path-dependent until the mid 2000s. In this survey, non-monetary data on living and economic conditions were collected in an exhaustive manner, which allows me to estimate measures of multidimensional poverty. More specifically, I estimate an index of deprivation in living standards. The dimensions taken into consideration are: access to electricity, type of sanitation facilities, source of drinking water, type of cooking fuel, possession of certain assets, and flooring material of housing. This index has been calculated following the methodology outlined in Alkire and Santos (2010), which is one of the international standards for the measurement of multidimensional poverty with non-monetary data.

The long time period of data collection (from 1962 to 2010 given the last data extraction) allows me to conduct an individual analysis on completed fertility. I analyze the complete birth histories of 2,884 women born between 1932 and 1961. The sample is left censored because of data availability, and is right censored as the fecund period is usually assumed to last from 15 to 49 years old (following United Nations standards). The analysis does not include women who died before the end of their reproductive years. Furthermore, the study is zero-truncated, as it excludes *nulligravidas* (women who have never been pregnant), but not necessarily *nulliparas* (women who never gave birth). The drivers of fertility are obviously not the same as the factors determining infertility. Moreover, infertility and voluntary childlessness are very rare in the study area (Ronsmans et al., 2001). The long time-span of the data also allows me to distinguish between six different birth cohorts of five-year

[8]The present study would have greatly benefited from the inclusion of a variable capturing the *risk* of dying before the age of five (*i.e.*, the uncertainty about child survival). A variable which is commonly used to capture this *risk* is the incidence of childhood diseases at the community level. However, such data were not available for the time period considered in this study.

intervals (1932-1936 for the earliest cohort, and 1957-1961 for the latest).

For the sake of simplicity, I will denominate total fertility (TF) the number of times a woman has been pregnant (her obstetrical history, that is, *gravidity*) and net fertility (NF) the number of her children who survived to age 5. TF and NF will be the two dependent variables used in the different model specifications. The occupation variable has been defined as the occupation held by a woman for the longest period of time during her reproductive life. Descriptive statistics of the variables used in the analyses are given in Table 1.1. The methodology employed in the study to explore the link between child mortality and fertility is presented in the next section.

Table 1.1 – Descriptive Statistics

	Mean	**Standard deviation**
Complete Fertility (Total)	5.59	2.80
Complete Fertility (Net)	4.90	2.43
Child Mortality Level (0-5 years) (per woman)	.70	1.06
Child Mortality Rate (0-5 years) (per 1,000 live birth)	112	155
Mother's age at first birth	22.85	5.44
	Percentage	
Religion		
Islam	76.01	
Christianity	20.98	
Atheism and traditional religions	3.02	
Marital status in the household		
Wife of the household head	56.73	
Other link	43.27	
Living Standards		
Extremely deprived	3.09	
Less deprived	96.91	
Occupation		
Housewife	88.18	
Maid	0.97	
Active	10.85	
Household head education		
None	86.93	
Primary and religious school	11.34	
Higher	1.73	
Birth cohort		
1932-1936	10.51	
1937-1941	12.52	
1942-1946	13.52	
1947-1951	15.08	
1952-1956	20.15	
1957-1961	28.22	
N	2884	

1.4 Model specifications

The use of an inappropriate model to estimate fertility outcomes can lead to serious drawbacks (Caudill and Mixon, 1995; Winkelmann, 2008). However, suitable models appear to be used only in a few empirical applications. We need a model that allows to capture the discrete and non-negative nature of the two dependent variables (TF and NF). Obstetric and birth histories are typical count data, which call for the use of count data models. Winkelmann and Zimmermann (1994) indicate that the mean-variance equality of the standard Poisson Regression Model is often violated due to the recurrent presence of underdispersion in fertility data. Yet, this is most likely to occur when infertility is taken into consideration, which is not the case for my study. The outcome of zero counts in fertility data is mostly driven by physiological issues rather than by a choice process. For that reason, Santos Silva and Covas (2000) argue that the underdispersion problem that characterizes completed fertility data cannot be solved by replacing the Poisson distribution by a model with a flexible mean to variance ratio. Instead, the authors build a modified hurdle model which takes into account the fact that zeros and positive counts are generated by different mechanisms. Such a model has some desirable features for fertility studies, yet its application is heavily constrained by data availability. For example, in their application to Portuguese data, Santos Silva and Covas (2000) cannot interpret the results of the model for zero counts, as only 37 out of 1093 observations correspond to childless couples. I thus estimate a standard Poisson Regression Model for the determinants of completed fertility. Other count data models have also been employed, namely Generalized Poisson Regression Models and Hurdle Models. However, predicted probabilities for the two dependent variables were less accurately estimated compared with the standard Poisson Regression Model.

Let the count outcome y_i - alternatively the TF and NF of a woman i, be a Poisson random variable. Hence :

$$Pr(y_i|\mu_i) = \frac{e^{-\mu_i}\mu_i^{y_i}}{y_i!} \quad \text{for } y = 0, 1, 2... ,$$

$$\text{where } \mu_i = E(y_i|X_i) = e^{X_i\beta} \text{ and } Var(y_i) = \mu_i \,.$$

X_i is the vector of covariates of woman i. TF is taken to be the dependent variable in a first set of model specifications, and NF in a second set. Recall that $y = 0$ is only possible for NF. The explanatory variables include all individual-level determinants of fertility identified in the demographic and economic literature. Yet the lack of data on knowledge and use of contraception makes it impossible to decompose fertility into desired and undesired components. I include measures of educational attainment and women's employment in the same regressions as I expect both variables to have a specific impact on fertility rates.

Different model specifications are estimated to account for the potential hetero-

geneous effects of child mortality on both TF and NF.[9] Model 1 aims to capture the *global effect* of child mortality on the two dependent variables. For that purpose, child mortality is included as a binary variable for having experienced a child death. The *level effect* of child mortality is analyzed in Model 2 and Model 3. In Model 2, the child mortality variable stratifies the sample into four mutually exclusive groups according to the total number of child deaths per woman during her reproductive life (namely, 0, 1, 2 and 3 or more). Model 3 attempts to improve the estimation of the effect of child mortality by using fractional polynomial transformations of the variable in levels. Several models are fitted and compared using a set of fractional polynomial terms. The model with the lowest deviance, defined as twice the negative log likelihood, is finally chosen (see Royston and Sauerbrei, 2008, for further methodological information). This allows me to capture the potential non-linear nature of the relationship between fertility and child mortality. More specifically, the marginal effects on fertility of an increase in child mortality levels will be allowed to vary across the distribution of child mortality. As already mentioned in the second section of this paper, there are only few studies examining the effect of child mortality on fertility that use count data models. Moreover, fertility is generally specified as a linear function of child mortality among these studies. Such linear restriction is likely to lead to a misspecification of the underlying relationship, especially when there is *a priori* no reason to expect a linear relationship. Loken et al. (2012) provide methodological support for the necessity of relaxing the linearity assumption when one wants to thoroughly investigate a certain relationship. Finally, Model 4 and Model 5 investigate the *rate effect* of child mortality, calculated at the individual level (namely, the number of child losses divided by the dependent variable). When child mortality is expressed in levels, an offset is likely to occur due to the mechanical link between the number of child losses and the number of pregnancies. In turn, estimates of regression coefficients may be artificially inflated. This potential issue is avoided here by using the rate of child mortality. Such a formulation allows us to properly establish the behavioral link between child mortality and fertility, and

[9] Although the causal link from child mortality to fertility is more tenuous, part of the correlation may be due to increased mortality due to higher fertility. Greater family size may adversely affect household constraints such as nutrition and childcare. In their systematic review of the literature on the effect of the number of births on women's mortality, Hurt et al. (2006) do not find any negative effect of parity on mortality. And this is also true within our study area, as documented in Ronsmans et al. (2001). I argue that the direct effect of fertility on child mortality is likely to be negligible compared to the opposite effect. Endogeneity might still be a concern due to potential omitted variable bias. In addition to the inclusion of all individual-level determinants of fertility, I also include time effects in the model in order to circumvent the problem of omitted variables. Time effects are meant to capture unobserved factors common to all individuals in the sample - in particular the contextual (exogenous) development and spread of medical technology - thereby reducing the main potential source of omitted variable bias. With the impossibility to find legitimate instruments for child mortality, I nonetheless acknowledge the possibility of endogeneity bias due to omitted variables and should still remain cautious in pointing to the existence of a causal link from child mortality to fertility.

thus to capture the net effect of child mortality. The variable is categorical in Model 4, with four categories: women with a child mortality rate equals to 0, between 0 and 0.2, 0.2 and 0.3, and higher than 0.3. Fractional polynomial transformations of child mortality rate are used in Model 5. All other explanatory variables remain unmodified across all specifications.

1.5 Results

Regression results are presented in Table 1.2. Coefficients are to be interpreted as incidence rate ratios (IRR). Figure 1.1 shows that both TF and NF are well predicted by the model, as the difference between observed and predicted probabilities lies in a small interval (-0.5 ; 0.5). The goodness-of-fit of the model is also assessed in view of the deviance statistics at the bottom of Table 1.2. Figure 1.2 focuses on the relationship between child mortality and fertility and shows that average predicted counts given all values of child mortality are accurately estimated.

Covariates other than child mortality are included in the regressions in order to control for their potential influence on completed fertility; yet their analysis is not the main focus of this paper. Their magnitude and significance hardly change across specifications.[10] I now turn to the analysis of the results regarding the effect of child mortality. Model 1 reports a positive global impact of child mortality on both total and net fertility. More precisely, I find that women who have experienced at least one child death have a total fertility that is 1.39 times higher than women who have not. Such an increase is proved to be larger than the one needed to replace lost lives, as net fertility is also 1.06 times higher. Furthermore, Model 2 seems to indicate that the relationship between fertility and the level of child mortality may be of a non-linear nature. Indeed, women that have experienced three or more child deaths have a TF 1.61 times higher than women who have never lost a child, but the two

[10]The estimated coefficients are consistent with theoretical considerations and other related empirical studies. I find that Muslim and Christian women exhibit higher fertility rates than women having a traditional religion or being atheist. Women married to the household head have a fertility rate that is roughly 1.2 times higher than that of women who are not. Women belonging to less deprived households have lower fertility rates in comparison with extremely deprived ones (that is, deprived in each and every dimension of the calculated index). Women who were maids ended their reproductive life with significantly lower fertility rates than housewives and other active women. The fact that there is no significant difference in fertility rates between housewives and other active women is certainly due to the nature of Niakhar's economy. Indeed, women reported as housewives dedicate their time to household's agricultural production activities. The educational achievement of the household head appears to be positively associated with fertility rates (see Diamond et al., 1999 for a review of the complex education-fertility relationship). Concerns may be raised about the use of husbands' educational attainment - used because of data availability - to tackle the eduction-fertility relationship. However, studies which use data collected from each spouse show that their education level (Cleland and Rodriguez, 1988) or fertility desire (McIntosh, 1999) have equal influence on fertility decision-making. Finally, mother's age at first birth has a negative impact on fertility rates.

Table 1.2 – Exponentiated coefficients from Poisson Regression Models of Total and Net Fertility

	Total Fertility					Net Fertility				
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 1	Model 2	Model 3	Model 4	Model 5
main										
Child Mortality										
Global effect										
Child Mortality dummy	1.394***					1.064***				
	(20.83)					(3.57)				
Level effect										
Child Mortality (level) categories (ref. = 0)										
1		1.296***					1.091***			
		(14.56)					(4.55)			
2		1.470***					1.075**			
		(19.46)					(3.13)			
>= 3		1.609***					0.954			
		(22.32)					(-1.63)			
Continuous: Fractional polynomial transformations of Child Mortality (level)[1]										
CML1			0.586***					0.633***		
			(-17.65)					(-6.35)		
CML2			1.001**					0.985***		
			(3.07)					(-6.46)		
Rate effect										
Child Mortality (rate) categories (ref. = 0)										
$\in]0; 0.2]$				1.438***					1.228***	
				(21.26)					(11.71)	
$\in]0.2; 0.3]$				1.434***					1.058**	
				(17.10)					(2.69)	
> 0.3				1.262***					0.714***	
				(9.45)					(-12.00)	
Continuous: Fractional polynomial transformations of Child Mortality (rate)[1]										
CMR1					0.000***					0.286***
					(-9.78)					(-8.85)
CMR2					0.000***					0.526***
					(-9.84)					(-9.27)
CMR3					0.104***					0.925***
					(-9.93)					(-9.52)
CMR4					0.000***					0.012***
					(-9.67)					(-16.01)

Table 1.2 (continued)

Religion (ref. = atheism and traditional religions)										
Islam	1.046	1.064^{+}	1.059	1.040	1.041	1.070	1.064	1.067	1.037	1.041
	(1.20)	(1.66)	(1.54)	(1.04)	(1.09)	(1.54)	(1.44)	(1.50)	(0.89)	(1.02)
Christianity	1.049	1.069^{+}	1.067	1.038	1.048	1.083^{+}	1.076	1.076	1.038	1.051
	(1.22)	(1.70)	(1.64)	(0.97)	(1.21)	(1.74)	(1.61)	(1.63)	(0.86)	(1.19)
Marital status in the household										
Wife of the household head	1.190***	1.182***	1.183***	1.187***	1.181***	1.206***	1.208***	1.208***	1.200***	1.187***
	(11.07)	(10.74)	(10.81)	(11.01)	(10.88)	(10.63)	(10.79)	(10.78)	(11.09)	(10.82)
Living Standards										
Less deprived	0.935^{+}	0.948	0.949	0.930^{+}	0.933^{+}	0.957	0.952	0.949	0.933^{+}	0.941
	(-1.77)	(-1.48)	(-1.45)	(-1.94)	(-1.82)	(-1.05)	(-1.16)	(-1.23)	(-1.79)	(-1.57)
Occupation (ref. = maid)										
Housewife	1.376***	1.323**	1.327**	1.373***	1.330***	1.355**	1.371**	1.375**	1.350**	1.311**
	(3.46)	(3.10)	(3.12)	(3.52)	(3.33)	(2.97)	(3.08)	(3.11)	(3.29)	(3.07)
Active	1.362**	1.316**	1.319**	1.355***	1.314**	1.351**	1.364**	1.368**	1.336**	1.300**
	(3.27)	(2.96)	(2.98)	(3.29)	(3.10)	(2.88)	(2.96)	(2.99)	(3.09)	(2.89)
Household head education (ref. = none)										
Primary and religious school	1.078***	1.085***	1.086***	1.073**	1.065**	1.101***	1.097***	1.096***	1.079***	1.066**
	(3.46)	(3.79)	(3.84)	(3.24)	(2.95)	(4.06)	(3.89)	(3.85)	(3.30)	(2.85)
Higher	1.160**	1.190***	1.185**	1.152**	1.097^{+}	1.215***	1.206***	1.208***	1.173**	1.106^{+}
	(2.86)	(3.29)	(3.21)	(2.76)	(1.82)	(3.43)	(3.34)	(3.38)	(2.99)	(1.93)
Maternal age										
Mother's age at first birth	0.959***	0.959***	0.959***	0.960***	0.961***	0.955***	0.955***	0.955***	0.959***	0.960***
	(-27.45)	(-27.68)	(-27.70)	(-26.38)	(-25.91)	(-27.35)	(-27.45)	(-27.54)	(-25.67)	(-25.29)

Table 1.2 (continued)

Birth cohort (ref. = 1932-1936)										
1937-1941	1.023	1.029	1.029	1.024	1.030	1.018	1.015	1.014	1.021	1.029
	(0.59)	(0.74)	(0.74)	(0.62)	(0.76)	(0.44)	(0.37)	(0.36)	(0.52)	(0.72)
1942-1946	1.162***	1.171***	1.172***	1.162***	1.168***	1.159***	1.155***	1.153***	1.161***	1.167***
	(4.16)	(4.40)	(4.43)	(4.15)	(4.31)	(4.02)	(3.90)	(3.86)	(4.12)	(4.32)
1947-1951	1.190***	1.194***	1.195***	1.195***	1.207***	1.182***	1.179***	1.176***	1.199***	1.220***
	(4.90)	(5.04)	(5.10)	(5.01)	(5.33)	(4.67)	(4.58)	(4.51)	(5.11)	(5.67)
1952-1956	1.226***	1.222***	1.221***	1.232***	1.238***	1.211***	1.211***	1.210***	1.228***	1.238***
	(5.76)	(5.69)	(5.72)	(5.86)	(6.02)	(5.33)	(5.34)	(5.32)	(5.77)	(6.05)
1957-1961	1.255***	1.241***	1.240***	1.266***	1.260***	1.231***	1.237***	1.237***	1.267***	1.263***
	(6.56)	(6.26)	(6.29)	(6.77)	(6.66)	(5.98)	(6.11)	(6.08)	(6.87)	(6.81)
Constant	6.873***	6.951***	11.813***	6.754***	0.000***	7.279***	7.279***	11.665***	6.963***	71.347***
	(16.81)	(17.29)	(21.39)	(16.96)	(9.96)	(15.66)	(15.62)	(17.01)	(16.81)	(14.90)
statistics										
Chi2 (df_m)	2828.285 (15)	3182.372 (17)	3132.896 (16)	2948.532 (17)	2745.481 (16)	1602.622 (15)	1640.950 (17)	1656.162 (16)	2256.772 (17)	3946.645 (18)
Log likelihood	-6125.413	-6093.124	-6092.078	-6113.201	-6080.204	-6023.605	-6015.558	-6011.547	-5892.032	-5809.407
AIC	12282.826	12222.247	12218.156	12262.402	12194.408	12079.211	12067.117	12057.094	11820.063	11656.813
BIC	12378.297	12329.652	12319.594	12369.807	12295.845	12174.682	12174.522	12158.532	11927.468	11770.185
N	2884	2884	2884	2884	2884	2884	2884	2884	2884	2884
deviance										
Residual df	2868	2866	2867	2866	2865	2868	2866	2867	2866	2865
Deviance goodness-of-fit	2344.164	2279.586	2277.494	2319.740	2253.746	2501.362	2485.268	2477.245	2238.214	2072.964
(1/df) Deviance	.8174	.7954	.7944	.8094	.7861	.8722	.8672	.8641	.7810	.7235
Pearson goodness-of-fit	2257.723	2199.527	2196.618	2240.500	2173.868	2384.174	2368.175	2362.062	2149.637	1998.052
(1/df) Pearson	.7872	.7675	.7662	.7818	.7582	.8313	.8263	.8234	.7500	.6974

$^{+}$ $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. t statistics in brackets.

[1] Transformations are specified in the Appendix;

Figure 1.1 – Observed and Predicted Probabilities from Model 5 for each value of the two dependent variables

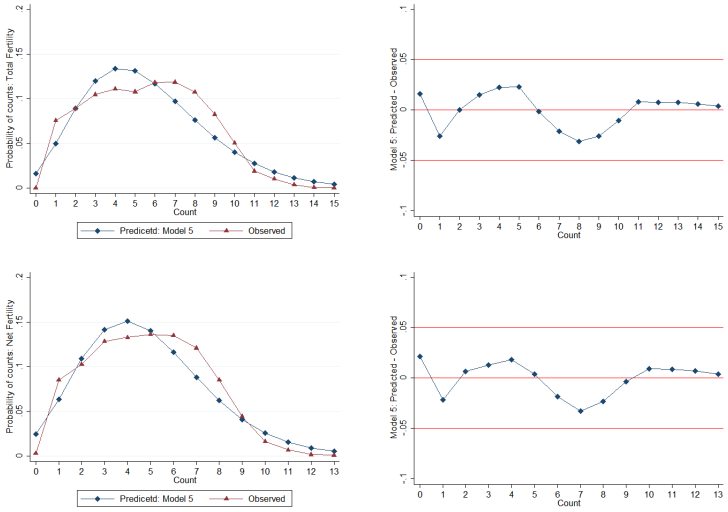
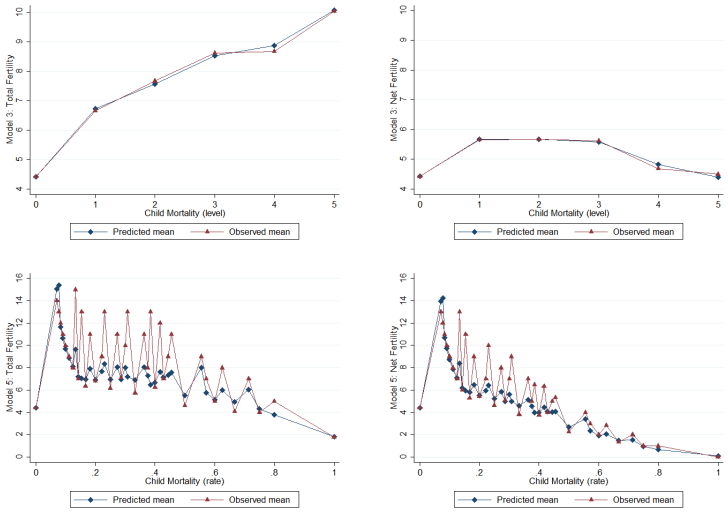


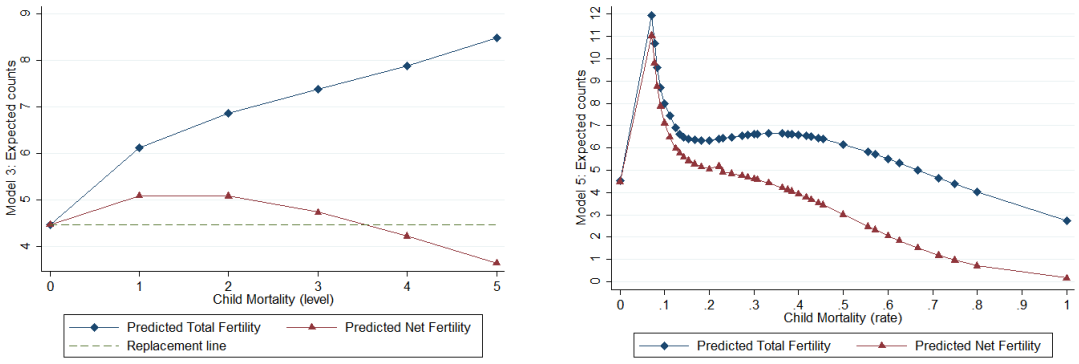
Figure 1.2 – Average predicted values vs Average observations given all values of Child Mortality from Model 3 and Model 5



groups are not significantly different in terms of NF. In order to confirm this result, I analyze the estimates of Model 3, in which fractional polynomial transformations of child mortality level are considered. A graphical analysis is required as it is not possible to interpret the coefficients directly from the regression table. Figure 1.3a gives the predicted values of TF and NF from Model 3 given all levels of child mortality. Although TF is continuously increasing with the level of child mortality, we clearly see that such an increase is insufficient to compensate for lost lives when child mortality levels are very high. Women exhibiting the highest NF are those who have experienced relatively low levels of child mortality (that is, one child loss). Model 4 and Model 5 are similar to Model 2 and Model 3 in all respects except that child mortality is expressed in rates (calculated at the individual level). The results remain unchanged, as shown in Figure 1.3b.

Figure 1.3 – Predicted values of Total and Net Fertility given Child Mortality

(a) Model 3 **(b)** Model 5

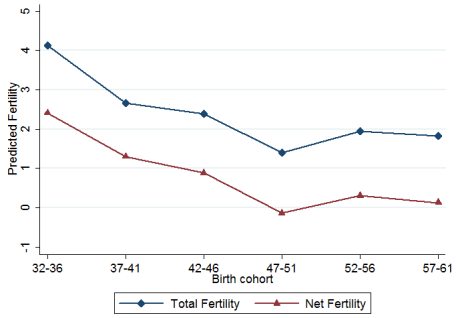


To gain more understanding about the child mortality-fertility relationship, I also investigate whether the mortality effect changes over time. Time effect is likely to capture the contextual (exogenous) development and spread of medical technology. The interaction of child mortality with the mother's birth cohort is thus included in Model 1.[11] Figure 1.4 displays the average marginal effects of child mortality on total and net fertility for the different cohorts. A downward inter-cohort trend is observed, that is, the positive effect of child mortality on total fertility is decreasing for recent cohorts. The mortality effect on net fertility is even slightly negative for the 1947-1951 cohort. More specifically, mothers from the 1932-1936 cohort who experienced at least one child death have 4.13 more pregnancies (and 2.41 more survivors) than women from the same cohort who have not lost a child. For the 1957-1961 cohort, these figures fall to 1.83 more pregnancies and 0.13 more survivors. Mortality is hence likely to have a smaller impact on the fertility of more recent cohorts. With the exception of two distinct mortality peaks that occurred in 1985 and 1998-1999

[11]The table is not presented here but is available from the author upon request.

due to cholera and meningitis outbreaks, infant and child mortality rates have been continuously declining in Niakhar since the 1960s (Delaunay et al., 2001). This could explain the slight rebound in the mortality effect for the last two cohorts, as only women from these cohorts were still in their childbearing years at the time of the mortality outbreaks.

Figure 1.4 – Average marginal effect of Child mortality by Birth cohort



Finally, I also investigate interaction of child mortality with maternal age, as the effect of child mortality on total and net fertility might change depending on the mother's entry into reproductive life. For that purpose, interaction of child mortality with the mother's age at first birth is included in Model 1.[12] Predicted values and marginal effects are shown in Figure 1.5. We see that the positive effect of child mortality on total fertility is likely to decrease with the mother's age at first birth. Women who had their first delivery at the age of 15 years (49 years) and who experienced at least one child death have 1.90 (1.31) more pregnancies than women who have not lost a child. The positive mortality effect on net fertility - although quite small - also decreases with maternal age. Mothers who have lost a child and started childbearing at the age of 15 years (49 years) have 0.32 (0.18) more survivors than women who have not lost a child.

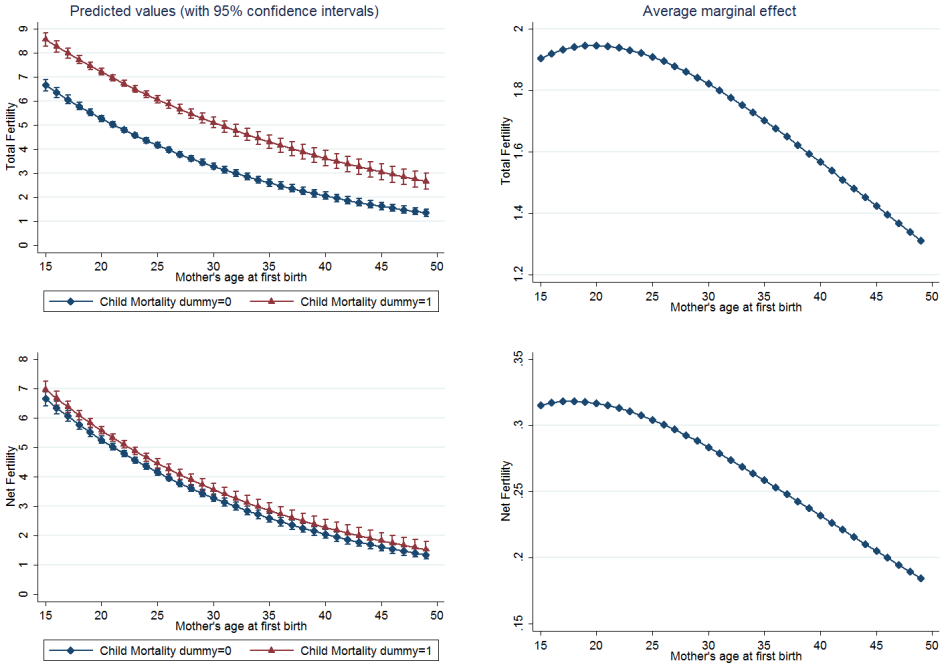
1.6 Discussion

Fundamentally, this study highlights the role that under five mortality plays in determining changes in households fertility behavior. Coefficient estimates for child mortality are overwhelmingly significant and robust across specifications. The positive association between total fertility and child mortality is exhibited, in line with other empirical studies in the economic and demographic literature.[13] Looking

[12]The table is not presented here but is available from the author upon request.

[13]Although I attempt to address the possibility of omitted variables bias, endogeneity might still be present. I thus remain cautious in arguing for the identification of a causal link from child mortality to fertility.

Figure 1.5 – Effect of Child Mortality on Total and Net Fertility given Mother's age at first birth



at the *global effect* of child mortality, this positive association holds for both total and net fertility, as women who have experienced at least one child death have a total and net fertility being, respectively, 1.39 and 1.06 times higher than women who have not. In turn, a precautionary demand for children arises, giving support for the *child survival hypothesis* rather than for the *replacement hypothesis*. Such a statement is also supported by the fact that, as mentioned earlier, one-to-one replacement has never been exhibited in dynamic models of fertility. To my knowledge, this is the first paper providing evidence for the *child survival hypothesis* at the micro level.

Further understanding is gained by examining more thoroughly the child mortality-fertility link. I show that the relationship is of non-linear nature, as the amplitude and the direction of the effect of child mortality vary with the number of child losses. These results are robust to changes in econometric specifications, and hold true when both level and rate effects of child mortality are accounted for. More specifically, an inverted-U shaped relationship between child mortality and net fertility is exhibited. Mothers with the largest number of survivors are those for whom the burden of child mortality has not been particularly high. An insurance mechanism is thus at work for non-zero but relatively low values of child mortality. As mothers who have lost only one child represent the largest share of mothers who have experienced child mortality, the *global effect* of child mortality on net fertility has been found to be positive. When child mortality further increases and reaches dramatic values, ferti-

lity do not respond anymore to compensate for child losses, so that net fertility falls under the level where any child dies at all. The explanation for this slowing may seem obvious when child mortality is expressed in levels, as there are biological limitations on the number of pregnancies a woman can experience. In order to circumvent the mechanical link between the number of child losses and the number of pregnancies, I also consider the rate of child mortality (calculated at the individual level). Then the behavioral link between child mortality and fertility is properly established. A negative child mortality-fertility relationship is documented for high child mortality rates, holding for both total and net fertility. There is a gap of two pregnancies between women who have never lost a child and those who have lost all of them, and their mean total fertility is around 4.5 and 2.5 respectively. This implies that women who have lost a large proportion of their children were less engaged in childbearing than other women.

In addition, my results provide evidence of a downward trend in the positive effect of child mortality on total *and* net fertility with time. This offers hope for the diffusion of the fertility transition in the region. The mortality effect also interacts with the timing of the onset of childbearing. I find that the positive effect of child mortality on total *and* net fertility is likely to decrease with the mother's age at first birth. The postponement of the onset of childbearing is thus likely to have a direct negative impact on fertility, as well as an indirect negative one through the child mortality channel. This gives support to family planning policies that aim to delay the onset of childbearing in developing countries.

This study helps to explain why fertility rates in SSA are not decreasing as traditional demographic transition theories would have expected. Indeed, the decrease in child mortality rates has not been steeper enough to really influence fertility behaviors. Therefore, I argue that any policy that aims to bring about the diffusion of the fertility transition in SSA should include, along with traditional family planning programs, measures to thoroughly reduce child mortality. And it is worth noticing that rapid progress in child survival is possible, for instance through targeted interventions (Bhutta et al., 2010).

Appendix 1.A Transformations used in Model 3 and Model 5

	Total Fertility		**Net Fertility**	
	Model 3	Model 5	Model 3	Model 5
CML1	$1/X$		$\sqrt{X}$	
CML2	X^3		X	
CMR1		$1/\sqrt{Y}$		$1/Y$
CMR2		$(1/\sqrt{Y})ln(Y)$		$(1/Y)ln(Y)$
CMR3		$(1/\sqrt{Y})ln(Y)^2$		$(1/Y)ln(Y)^2$
CMR4		$ln(Y)$		Y^2

where: X = (CML+1) and Y = (CMR+.0054945051670074)

Chapter 2

The effect of child mortality on fertility: Does the cost-of-children function matter?

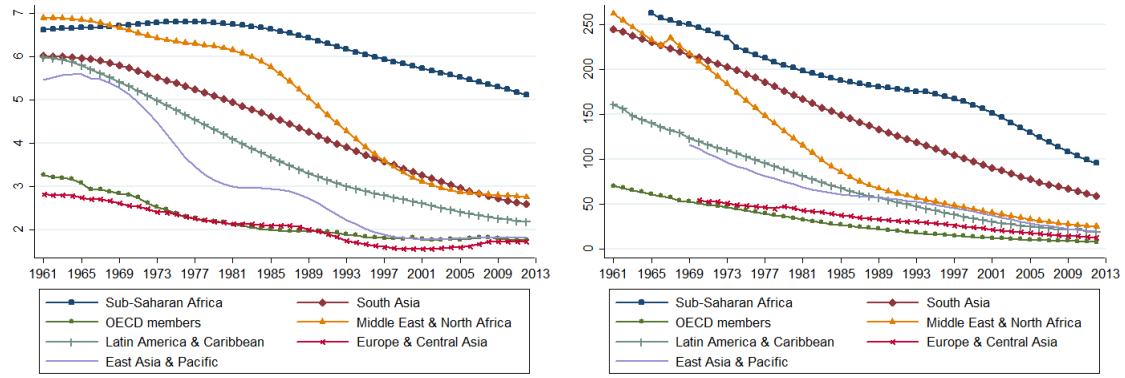
2.1 Introduction

Sub-Saharan Africa has long been trapped in a crisis characterized by high mortality and fertility rates. However, mortality rates have fallen steadily in a significant number of sub-Saharan African countries from the 1950s, and life expectancy is projected to increase. Concurrently, fertility rates are remaining markedly high, and the average fertility gap with the rest of the world remains unambiguously large and constant (United Nations, 2011). These trends are clearly illustrated in Figure 2.1. Altogether, sub-Saharan Africa is experiencing a growing demographic pressure. Nonetheless, studies reveal that even though some components of fertility arise from an unmet need for family planning, its largest share is actually desired by households (Bongaarts, 2011). The availability of family planning services is crucial for the fertility transition to occur. Increased use of modern contraception is obviously strongly associated with fertility decline. In developing countries, knowledge about the availability of methods to delay or avoid a pregnancy is now quite widespread. However, better levels of knowledge are far from being associated with higher use. Cleland et al. (2011) report that attitudinal resistance remains a severe barrier, so that progress towards adoption of contraception has been dramatically slow.

Demographic transition theories usually postulate that fertility is likely to follow the decrease in child mortality after some lag. Concerns are raised about the validity of these theories for the sub-Saharan Africa context, as they apparently fail to explain contemporary demographic patterns. Within the economic literature, there is as yet no clear consensus as regards the relationship between child mortality and fertility in developing countries. A distinction is made between total fertility (the number of births per woman), and net fertility (the number of surviving children). The *replace-*

Figure 2.1 – Demographic transitions since 1960

(a) Total fertility rate **(b)** Child mortality rate



Source: World Bank's World Development Indicators

ment hypothesis posits that households have additional children to replace the ones that they lose, involving a target number of children chosen by parents. Replacement behavior will then generate a positive relationship between child mortality and total fertility.[1] The *child survival hypothesis* (also known as the *hoarding motive*) supposes an effect of child mortality on net fertility rates. Households would generate a precautionary demand for children in order to ensure a sufficiently large family size. This theory predicts that improvements in child survival will lead to decreasing total and net fertility rates. The theoretical findings of the endogenous fertility literature are mixed. Some papers exhibit an effect of child mortality on total fertility only (Doepke, 2005; Boucekkine et al., 2009), while others find that both gross and net fertility are affected by child mortality (Kalemli-Ozcan, 2003).

This paper analyzes the relationship between child mortality and fertility behaviors in a model of endogenous fertility choice. Theoretical research on the topic dates back to the seminal works of Becker and Barro (1988) and Barro and Becker (1989), in which a permanent decline in the level of child mortality should lead to a quantity-quality trade-off. The authors assume that altruistic parents have a dynastic utility function depending on their own consumption as well as on the number and consumption of descendants in all generations. The decrease in child mortality lowers the cost of rearing surviving children in the initial generation, such that the demand for survivors increases. Hence, birth rates may rise in the first place. As the rate of decline in child mortality slows once it approaches zero, fertility rates fall in later generations and parents decide to invest more in (the education of) each child.

Several endogenous fertility models have been developed so far in order to better

[1] One can argue that replacement strategy could not be perfect because of physiological limits such as birth spacing, breastfeeding period or limited fecundity.

understand the child mortality-fertility relationship. Cigno (1998) builds a model where child survival is endogenous, such that fertility and child mortality may move in the same direction when parents realize that they can improve the survival chances of their children by devoting more resources to each child. Otherwise, when child survival probability is independent of parental action, fertility and child mortality move in opposite directions. The author then argue that the observed positive correlation between child mortality and fertility during the demographic transition in industrialized countries is evidence for the endogeneity of infant survival. Sah (1991) predicts a positive impact of child mortality on fertility in a model where child survival is stochastic, but where only surviving children entail a cost to parents. Kalemli-Ozcan (2003) also allows for uncertainty about child survival and provides support for the *hoarding motive*, so that child mortality has a positive effect on both total and net fertility. Doepke (2005) adds more realism in allowing for the sequential nature of fertility decisions and in differentiating between (fixed) costs per birth and per surviving child. He finds that child mortality has a positive effect on gross fertility solely, so that other factors must be responsible for the decrease in net fertility rates observed in developed countries during the demographic transition. Stressing the importance of the timing of child mortality relative to education, Azarnert (2006) shows that if child mortality is realized before education starts, an exogenous decline in child mortality leads to an increase in fertility. Aksan and Chakraborty (2013) emphasize the role of uncertainty about both child mortality and morbidity in a model of fertility choice. Fertility rates are predicted to follow mortality and infectious disease morbidity, but the fertility response may be dampened or non-monotonic as morbidity and mortality can move in opposite directions. Consequently, their model includes both positive and negative fertility responses to the disease burden.

This paper's model investigates to what extent the way the cost of children is introduced affects the results found in the recent literature on the topic. Endogenous fertility models analyzing the child mortality-fertility relationship systematically assume that the birth of each child entails a fixed cost to parents, so that the total cost is proportional to the number of children. I adopt a more general and empirically appealing child cost function that allows for four different scenarios, each of which is representative of a particular socio-economic setting. I prove analytically that the positive relationship between child mortality and total fertility can be mitigated - and even reversed - depending on the shape of the total cost curve considered. My model can predict both positive and negative fertility responses to child mortality depending on the relative cost of children. Fertility and child mortality move in the same direction when the cost of children is high, which is the case in countries where a certain level of economic development has been reached. However, when the cost of children is low, as observed in low-income countries, fertility and child mortality move in opposite directions. I argue that this paper's model can provide one explanation for the stalled demographic transition in today's sub-Saharan Africa.

2.2 Background: the cost of children

Since the influential works of Becker *et al.* (1973; 1976; 1988), endogenous fertility models usually assume a fixed cost of child rearing which is proportional to the number of children - the cost of *quantity* - and a variable cost of formal education - the cost of *quality*. Fundamentally, the cost of quality introduces a non-linearity in the budget constraint of altruistic parents, which will be ultimately translated into a trade-off between quality and quantity of children.

Recent papers focusing on the relationship between child mortality and fertility only rely on the cost of quantity to derive their core results, although human capital/educational investment decisions are eventually incorporated in further model extensions. Such a framework is plausible, as schooling decisions are to be taken after the uncertainty about infant and child survival has occurred. Hence, although it is part of the maximization problem in Kalemli-Ozcan (2003), solving the model without educational investment yields exactly the same results regarding the child mortality-fertility relationship, which also coincides with the analytical findings in Kalemli-Ozcan (2002): both papers attribute the cause of fertility reduction to declining mortality only.

Theoretical models on the child mortality-fertility relationship always assume that the cost of quantity is proportional to the number of children, a modeling assumption that is not consistent with theoretical considerations or empirical estimations of the cost of children. More generally, very few models with endogenous fertility consider another functional form for the cost of children than the usual fixed cost which implies a linear total cost function.[2] Azariadis and Drazen (1993), Chakrabarti (1999), Tertilt (2005), Oshio and Yasuoka (2009) assume that the time cost of raising children is convex and non-decreasing in the number of children. Such a functional form can be viewed as additional children taxing a woman's health by increasing amounts (Tertilt, 2005). Palivos (1995) and Yip and Zhang (1997) build models of endogenous growth and fertility where the marginal cost of raising children can be increasing, constant, or decreasing, the latter corresponding to a situation where there are economies of scale in child-rearing.

A lot of consideration in the empirical literature has long been given to the definition and estimation of the costs of children.[3] As Apps and Rees (2002) point out, the costs of children has too often been viewed solely as market consumption costs. It is now widely recognized that parental time inputs constitute another essential element of child costs. In taking care of a child, the amount of time devoted to other activities, such that leisure, market work or household production, has to be shortened. And it is worth noticing that the price of child quantity relative to the price of quality increases with wages (Moav, 2005). In turn, the relative shares of monetary and time costs used in child rearing vary with the socio-economic setting

[2]However, none of these papers focus on the child mortality-fertility relationship.

[3]See Lewbel (1997) for a summary of the various estimation methods.

taken into consideration.

Studies estimating the cost of children in terms of opportunity cost of work within developed countries often conclude that parents, and especially mothers, incur increasing marginal costs of having children (see for instance Connelly, 1992). However, large scale economies may be reached in the production of children, due for instance to joint consumption. Using US data, Lazear and Michael (1980) find evidence of increasing returns to scale in the number of children. Estimates of the monetary cost of children in France show that parents dedicate an important share of total expenditure to children, but economies of scale in multi-person households are likely to be large (Bargain and Donni, 2012). Gustafsson and Kjulin (1994) document large economies of scale in time used for child care among Swedish families. Considering respectively monetary and time costs, Percival and Harding (2007) and Craig and Bittman (2008) provide evidence of substantial entry costs to parenthood for Australian households, but considerable economies of scale in cost per child thereafter. Child costs to the household may also be further reduced thanks to macro-level factors. The institutional environment can produce incentives for fertility in socializing the costs through subsidies for housing, education, reduced taxes or direct transfers related to the presence of children (DiPrete et al., 2003). Pro-natalist policies in countries such as France or Sweden tend to lower child costs, especially for additional children. The costs of children may also vary across individuals' life courses, for instance due to changes in labor market conditions (Adsera, 2011), and adjustments of the desired family size can be made in a sequential manner (Heiland et al., 2008).

Due to the lack of data availability, empirical estimates of the cost of children are much more scarce in low-income settings. The opportunity cost of time is low in poor countries (Moav, 2005), and the consequences of rearing children on adult leisure are presumably quite limited (Deaton and Muellbauer, 1986). Moreover, formal schooling is often deficient and a large amount of total time spent to rear children is shared jointly with household production activities. Lindert (1983) argues that, in the course of economic development, a child switches from being "time-supplying" to being "time-intensive". Within an overlapping-generations model with different social norms of filial obligations, López-Calva and Miyamoto (2004) provide a theoretical explanation for the empirical regularity according to which poorer (richer) countries exhibit high (low) prevalence of child labor activities and time-intensive (money-intensive) care of the elderly. For low-income countries, it therefore seems much more realistic to consider a definition of the cost of children in terms of net value, as children also generate (gross) economic benefits related to child labor or household production activities (Schultz, 1973; Lanjouw and Ravallion, 1995). Time and money inputs are required, as well as generated, by children. Taking this phenomenon into consideration, one can consider the total cost of children to be a decreasing function of the number of existing children. However, even in developing countries, evidence suggests that a child seemingly entails a net economic cost (Caldwell, 1978, 1982; Lindert, 1983; Lee and Kramer, 2002). Consequently, the total

(net) cost of children is likely to remain always positive. Having additional children may also lower the financial and perceived costs of using contraception (Grubb, 1987; Casterline and Sinding, 2000). In some low-income settings, contraception is viewed as more dangerous than childbirth (Campbell and Potts, 2006). Subjective cost-benefit considerations of parents at the time of the fertility decision have to be taken into consideration. These subjective considerations include the perceived costs of additional children, for instance in terms of expected expenses, benefits, or maternal health. Using a very rich Ghanaian dataset, Kodzi et al. (2012) prove that both subjective elements and normative considerations of costs are incorporated into fertility decisions.

The various possible aspects of the cost of children are not captured by the linear total cost function usually adopted in endogenous fertility models. I argue that this may have a strong influence on the results regarding the child mortality-fertility relationship, as the shape of the total cost curve is likely to change across socio-economic settings. In light of the theoretical literature and empirical evidences, I adopt in this model a functional form for the cost of children that allows for four different possible scenarios: (1) increasing marginal cost, (2) fixed cost, (3) decreasing marginal cost and (4) decreasing total cost (while remaining always positive).

2.3 The model

The methodology draws on the overlapping generations model proposed by Kalemli-Ozcan (2003). I do not incorporate human capital investment decisions in the model.[4] Let n be the number of children, and N the number of survivors. Members of generation t maximize their utility on their own consumption, c_t, and are altruistic toward children, in the sense that the future income of survivors, $N_t w_{t+1}$, is also part of their utility:

$$U_t = \gamma ln[c_t] + (1-\gamma) E_t \left\{ ln[N_t w_{t+1}] \right\} . \tag{2.1}$$

The budget constraint is the following:

$$w_t(1 - v(n_t)) = c_t \, , \tag{2.2}$$

with the total cost of children taking the following functional form:[5]

[4] As previously mentioned, the result regarding the child mortality-fertility relationship holds both with and without human capital investment decisions.

[5] The cost function could also depend only on N_t, the number of survivors. However, two important aspects of child costs would be omitted when choosing such a specification: the cost per birth and the perceived cost of children, as discussed in the second section of this paper. Let us recall that the individual's problem is modeled here as a one-time fertility decision. Finally, N_t is defined as the total number of children who will survive to adulthood, so even children who do not survive to adulthood can consume and/or produce resources.

$$v(n_t) = \theta_1(\theta_2 + n_t)^{\epsilon}\,. \tag{2.3}$$

Each child involves a positive cost which implies a reduction of income. As pointed out in Doepke (2005), child costs can be interpreted here either in terms of good or time costs since the full income of a member of generation t, w_t, is taken as given. The total cost function satisfies $v(0) = 0$ when $\theta_2 = 0$.[6] ϵ can be viewed as the elasticity of the cost of raising children with respect to the number of children. Under the restrictions $\epsilon = 1$ and $\theta_2 = 0$, my model is a special case of Kalemli-Ozcan (2003), where each child entails a fixed cost θ_1, implying a linear total cost function. For $\epsilon > 0$, the total cost of children is such that: $v\prime > 0$, $v\prime\prime > 0$ when $\epsilon > 1$ and $v\prime\prime < 0$ when $0 < \epsilon < 1$. Hence, when $\epsilon > 1$ the cost of children is convex and non-decreasing in the number of children, while when $0 < \epsilon < 1$ there are increasing returns to scale in child production. Finally, for $\epsilon < 0$, we have that $v\prime < 0$ and $v\prime\prime > 0$, so the total cost of children, while remaining always positive, is decreasing in the number of children. I discussed in the previous section to what extent are each of these scenarios representative of a particular socio-economic setting. In a first step, the model is solved with certainty about child survival. Uncertainty is added in a second step.[7]

2.3.1 Certainty case

The survival probability of each child, $q \in (0, 1)$, is known by parents without uncertainty, which implies that $N_t = E_t(N_t) = n_t q$. Hence, the choice problem is as follows :

$$\begin{aligned} &n_t = argmax\left\{\gamma ln[w_t(1 - \theta_1(\theta_2 + n_t)^{\epsilon})] + (1-\gamma)ln[n_t q w_{t+1}]\right\}, \\ &s.t.\;\; n_t \geq 0\,. \end{aligned} \tag{2.4}$$

The first order condition with respect to n_t gives:

$$(1-\gamma)\theta_1(\theta_2 + n_t)^{\epsilon} + \gamma\epsilon\theta_1(\theta_2 + n_t)^{\epsilon-1}n_t - (1-\gamma) = 0\,. \tag{2.5}$$

A proof of concavity of the maximization problem is contained in Appendix 2.A. Note that the maximization problem is always concave in the definition domain of n, although the cost function can be either concave or convex. Considering the special

[6] $\theta_2 > 0$ would correspond to a case where not having children is costly (for instance due to birth control costs or perceived costs), as discussed in the second section of this paper. Since θ_2 is only a scale parameter, considering different values for θ_2 would only change the level of fertility but not the shape of the child mortality-fertility relationship, which is ultimately our main interest.

[7] Uncertainty about child survival is undoubtedly a prominent feature of low-income countries. Kalemli-Ozcan (2003) argues that individuals will have a precautionary demand for children once this uncertainty is accounted for in their optimization problem.

case where $\theta_2 = 0$,[8] the fertility choice is:

$$n_t = \left(\frac{(1-\gamma)}{(1-\gamma+\gamma\epsilon)\theta_1} \right)^{\frac{1}{\epsilon}} . \tag{2.6}$$

Proposition 1. *When the child survival probability is known with certainty, exogenous variations in child survival have no effect on fertility.*

We clearly see that, since there is no uncertainty about child mortality, the number of children only depends on exogenous parameters. Note, however, that ϵ shapes the fertility choice in the following way:

Proposition 2. *For $\theta_2 = 0$ and $\theta_1 = 1$ the number of children increases less and less rapidly as the cost of children increases.*

$$\frac{dn_t}{d\epsilon} > 0, \quad \forall\epsilon \tag{2.7a}$$

and

$$\frac{d^2 n_t}{d^2\epsilon} < 0, \quad \forall\epsilon\,. \tag{2.7b}$$

2.3.2 Uncertainty case

Now the survival probability of each child is stochastic. As in Sah (1991), Kalemli-Ozcan (2003), or Doepke (2005), consider the child survival probability to be the following binomial density:[9]

$$f(N_t; n_t, q) = \binom{n_t}{N_t} q^{N_t}(1-q)^{n_t - N_t}\,, \qquad N_t = 0, 1, ..., n_t, \forall t\,. \tag{2.8}$$

The expected utility of a member of generation t can thus be rewritten as follows:

$$E_t(U_t) = \sum_{N_t=0}^{n_t} \{\gamma ln[c_t] + (1-\gamma) ln[N_t w_{t+1}]\}\, f(N_t; n_t, q)\,. \tag{2.9}$$

Using the Delta Method to approximate the utility through a third-order Taylor series expansion about the mean of the binomial distribution,[10] the expected utility maximization problem becomes:

[8] The result regarding the child mortality-fertility relationship remains unmodified when considering $\theta_2 \neq 0$, but a closed-form analytical solution would not be obtainable.

[9] As argued in Sah (1991), such a discrete representation has both realistic and tractable properties for the modeling of fertility outcomes.

[10] See Kalemli-Ozcan (2003).

$$n_t = argmax \left\{ \gamma ln[w_t(1-\theta_1(\theta_2+n_t)^{\epsilon})] + (1-\gamma) ln[n_t q w_{t+1}] - \frac{(1-\gamma)(1-q)}{2n_t q} \right\}, \quad s.t. \;\; n_t \geq 0\,. \tag{2.10}$$

The first order condition with respect to n_t gives:

$$\frac{(1-\gamma)}{n_t} + \frac{(1-\gamma)(1-q)}{2n_t^2 q} - \frac{\gamma\epsilon\theta_1(\theta_2+n_t)^{\epsilon-1}}{1-\theta_1(\theta_2+n_t)^{\epsilon}} = 0\,. \tag{2.11}$$

As shown in Appendix 2.A, the second-order conditions are satisfied. When uncertainty about child survival is added to the model, the child mortality-fertility nexus becomes apparent. The core results are contained in the following proposition:

Proposition 3. *When mortality is stochastic, fertility decreases with improvements in child survival when there are increasing, as well as diminishing, marginal costs of children. This relationship is reversed when the total cost of children is decreasing in the number of children.*

$$\frac{dn_t}{dq} < 0, \quad \forall \epsilon > 0, q \tag{2.12a}$$

and

$$\frac{dn_t}{dq} > 0, \quad \forall \epsilon < 0, q\,. \tag{2.12b}$$

Proof. See Appendix 2.B.

Even though mortality is driving fertility change, the sign of the relationship ultimately depends on the relative cost of children. Equation (2.12a) corresponds to the cases where there are increasing returns to scale in child production and where the cost of children is convex and non-decreasing in the number of children. Both cases are representative of a socio-economic setting where children are "time-intensive", typically in countries where a certain level of economic development has been reached. For these cases, total fertility and child mortality move in the same direction. However, a reversal of the child-mortality-fertility relationship occurs when the cost of children is low. Equation (2.12b) refers to the case where the total cost of children is decreasing in the number of children. This corresponds to a socio-economic setting where children, while always entailing net economic costs to parents, also generate (gross) economic benefits, and are thus "time-supplying". I argue that this analytical finding can provide one explanation for the current stalled demographic transition in most of the least developed sub-Saharan African countries. Note that

when $\epsilon = 1$ and $\theta_2 = 0$, solving for n_t gives $\frac{dn_t}{dq} < 0 \ \forall q$, which matches the special case where child costs are fixed.[11]

2.4 Concluding remarks

This paper analyzes the effects of child mortality changes on fertility behaviors in an endogenous fertility model where child survival is stochastic. Endogenous fertility models analyzing the child mortality-fertility relationship systematically assume that the birth of each child entails a fixed cost to parents, so that the total cost is proportional to the number of children. The importance is stressed of considering a more refined functional form for the cost of children which is more general and empirically appealing, as the shape of the total cost curve is likely to change across socio-economic settings. my model can predict both positive and negative fertility responses to child mortality depending on the relative cost of children. The positive relationship between child mortality and total fertility, exhibited in Kalemli-Ozcan (2003) for fixed child costs, is generalized in my model to increasing, as well as diminishing, marginal costs of children. This result is usually predicted by demographic transition theories, but only holds in my model for socio-economic settings where children are "time-intensive", typically in developed countries. It is shown that this positive relationship may be reversed, even when uncertainty is added in the utility maximization problem. The latter result is derived from a functional form for the cost of children which takes into account the net value of children as regards cost-benefit considerations. Hence, even when they perceive future benefits in producing children (when children are "time-supplying"), parents tend to lower their total fertility when child mortality rises. These analytical findings can be linked to those of Cigno (1998), where fertility and child mortality are positively correlated only when infant survival is endogenous. The author argues that this corresponds to countries having already experienced a certain level of development. The idea that mortality decline alone is not sufficient to trigger fertility decline can also been found in Doepke (2005), Boucekkine et al. (2009) or in the *unified growth theory* (*e.g.,* Galor, 2005). As shown empirically by Kodzi et al. (2012), perceptions of economic opportunities and health concerns are joint determinants of fertility behaviors. When the cost of children is low, I find that total fertility increases with improvements in child survival. I claim that this situation can be particularly relevant for some low-income settings, and more specifically in sub-Saharan Africa where infant and child mortality rates are declining continuously since the 1950's and where fertility rates are remaining markedly high, thus resulting in a high demographic pressure.

[11] The proof is contained in Appendix 2.B.

Appendix 2.A Proof of concavity of the maximization problem

The bordered Hessian determinant, which is the last principal minor, equals :
- for the certainty case :

$$\det \bar{H} = \frac{(1-\gamma)}{n_t^2} + \lambda w_t \theta_1 \epsilon(\epsilon-1)(\theta_2+n_t)^{\epsilon-2} + \Big(w_t\theta_1\epsilon(\theta_2+n_t)^{\epsilon-1}\Big)^2 \left(\frac{\gamma}{c_t^2}\right) \quad (2.13)$$

- for the uncertainty case :

$$\det \bar{H} = \frac{(1-\gamma)}{n_t^2} + \frac{(1-\gamma)(1-q)}{q n_t^3} + \lambda w_t \theta_1 \epsilon(\epsilon-1)(\theta_2+n_t)^{\epsilon-2} + \Big(w_t\theta_1\epsilon(\theta_2+n_t)^{\epsilon-1}\Big)^2 \left(\frac{\gamma}{c_t^2}\right) \quad (2.14)$$

As for both cases $\det \bar{H} > 0$, it follows from the second-order conditions of the maximization problem that the solution is a local maximum.

Appendix 2.B Proof of Proposition 3

Equation (2.11) can be written as:

$$F(n_t, q) = -n_t \left(\frac{\gamma\epsilon\theta_1(\theta_2+n_t)^{\epsilon-1} n_t}{1-\theta_1(\theta_2+n_t)^{\epsilon}} - (1-\gamma) \right) + \frac{(1-\gamma)(1-q)}{2q} = 0\,. \quad (2.15)$$

Suppressing t subscript and rewriting Equation (2.15) in the following way:

$$\frac{(1-\gamma)(1-q)}{2q} = n \left(\frac{\gamma\epsilon\theta_1(\theta_2+n)^{\epsilon-1} n}{1-\theta_1(\theta_2+n)^{\epsilon}} - (1-\gamma) \right), \quad (2.16)$$

we see that the left-hand side only depends on q and the right-hand side only depends on n.

Using the implicit functions theorem:

$$\frac{dn}{dq} = -\frac{F_q}{F_n}\,, \quad (2.17)$$

with:

$$\begin{aligned} &LHS_q(q) = F_q, \\ &RHS_n(n) = -F_n\,. \end{aligned} \quad (2.18)$$

Since $0 < q \leq 1$, $LHS(q)$ is always negative, as in Kalemli-Ozcan (2003):

$$\begin{aligned} LHS_q(q) &= -\frac{(1-\gamma)}{2q^2} < 0 \\ LHS_{qq}(q) &= -\frac{(1-\gamma)}{q^3} > 0 \\ \lim_{q\to 0} LHS(q) &= +\infty \\ \lim_{q\to 1} LHS(q) &= 0\,. \end{aligned} \tag{2.19}$$

Hence:

$$F_q < 0, \quad \forall q\,. \tag{2.20}$$

Let's now analyze $RHS(n)$. We have that:

$$RHS_n(n) = \frac{\gamma\epsilon\theta_1(\theta_2+n)^{\epsilon-1}n}{1-\theta_1(\theta_2+n)^{\epsilon}} + \frac{\gamma\epsilon\theta_1(\theta_2+n)^{\epsilon-2}n(n\epsilon+\theta_2-\theta_1\theta_2(n+\theta_2)^{\epsilon})}{(-1+\theta_1(\theta_2+n)^{\epsilon})^2} - (1-\gamma)\,. \tag{2.21}$$

We now need to distinguish between the case where $\epsilon > 0$ and the case where $\epsilon < 0$. Notice that the definition domain of n is determined by both ϵ and the budget constraint in Equation (2.2).

Case $\epsilon > 0$

The definition domain of n is:

$$-\theta_2 \leq n \leq \left(\frac{1}{\theta_1}\right)^{\frac{1}{\epsilon}} - \theta_2\,. \tag{2.22}$$

Thus, the sign of $RHS_n(n)$ can be determined by evaluating the following:

$$\begin{aligned}
&\lim_{n\to-\theta_2} RHS_n(n) = -(1-\gamma) < 0\\
&\lim_{n\to(1/\theta_1)^{\frac{1}{\epsilon}}-\theta_2} RHS_n(n) = +\infty\\
&RHS_{nn}(n) = \frac{\gamma\epsilon\theta_1(\theta_2+n)^{\epsilon-3}}{(-1+\theta_1(\theta_2+n)^{\epsilon})^3}[4n\epsilon\theta_2(-1+\theta_1(\theta_2+n)^{\epsilon}) - 2\theta_2^2(-1+\theta_1(\theta_2+n)^{\epsilon})^2\\
&- n^2\epsilon(1+\epsilon+(\epsilon-1)\theta_1(\theta_2+n)^{\epsilon}] \geq 0, \quad \forall\epsilon>0, n\\
&\lim_{n\to-\theta_2} RHS(n) = 0\\
&\lim_{n\to(1/\theta_1)^{\frac{1}{\epsilon}}-\theta_2} RHS(n) = +\infty\,.
\end{aligned} \tag{2.23}$$

Hence:

$$-F_n > 0, \quad \forall n\,. \tag{2.24}$$

Finally, Equation (2.17) with Equations (2.20) and (2.24) implies:

$$\frac{dn}{dq} < 0, \quad \forall\epsilon>0, q\,. \tag{2.25}$$

Case $\epsilon < 0$

The definition domain of n is now:

$$\left(\frac{1}{\theta_1}\right)^{\frac{1}{\epsilon}} - \theta_2 \leq n \leq \left(\frac{(1+\epsilon)}{(1-\epsilon)\theta_1}\right)^{\frac{1}{\epsilon}} - \theta_2\,. \tag{2.26}$$

Thus, the sign of $RHS_n(n)$ can be determined by evaluating the following:

$$\lim_{n\to(1/\theta_1)^{\frac{1}{\epsilon}}-\theta_2} RHS_n(n) = +\infty$$
$$\lim_{n\to((1+\epsilon)/((1-\epsilon)\theta_1))^{\frac{1}{\epsilon}}-\theta_2} RHS_n(n) = -(1-\gamma) < 0$$
$$RHS_{nn}(n) = \frac{\gamma\epsilon\theta_1(\theta_2+n)^{\epsilon-3}}{(-1+\theta_1(\theta_2+n)^{\epsilon})^3}[4n\epsilon\theta_2(-1+\theta_1(\theta_2+n)^{\epsilon}) - 2\theta_2^2(-1+\theta_1(\theta_2+n)^{\epsilon})^2$$
$$- n^2\epsilon(1+\epsilon+(\epsilon-1)\theta_1(\theta_2+n)^{\epsilon}] \leq 0, \quad \forall\epsilon < 0, n$$
$$\lim_{n(1/\theta_1)^{\frac{1}{\epsilon}}-\theta_2\to} RHS(n) = -\infty$$
$$\lim_{n\to((1+\epsilon)/((1-\epsilon)\theta_1))^{\frac{1}{\epsilon}}-\theta_2} RHS(n) = 0\,. \tag{2.27}$$

Hence:

$$-F_n < 0, \quad \forall n\,. \tag{2.28}$$

Finally, Equation (2.17) with Equations (2.20) and (2.28) implies:

$$\frac{dn}{dq} > 0, \quad \forall\epsilon < 0, q\,. \tag{2.29}$$

Chapter 3

Childhood mortality, childhood morbidity, and subsequent fertility decisions

3.1 Introduction

The relationship between child mortality and fertility lies at the heart of demographic transition theories. A conventional view of the demographic transition is that fertility is likely to follow the decline in childhood mortality. In sub-Saharan Africa, child mortality shows a downward trend from the 1950s, yet net fertility rates remain particularly high (Bongaarts and Casterline, 2013). These patterns might be explained by two noticeable phenomena. First, the decrease in child mortality has so far not been substantial enough to trigger fertility declines. In comparison, the child mortality decline that has preceded the demographic transition in Europe and the rest of the industrialized world has been faster and sharper on average (Corsini and Viazzo, 1997). Second, despite declining childhood mortality rates, the incidence of childhood diseases remains very high. The mortality transition in sub-Saharan Africa is specific in the sense that morbidity has not declined along with mortality. Incidence of most diseases is much higher than their fatality rate (Aksan, 2014). In two recent theoretical contributions, Aksan and Chakraborty (2013, 2014) identify infectious disease morbidity as the source of sub-Saharan Africa's sluggish demographic transition.

Additional empirical analyses of the child mortality-fertility relationship are required in order to better understand contemporary demographic patterns in sub-Saharan Africa. From a theoretical viewpoint, the effect of child mortality on fertility can be twofold. The insurance effect (also know as the hoarding motive, the precautionary motive, or the child survival hypothesis) prevails when households anticipate that not all children will survive to adulthood. In an adverse epidemiological environment, households would have additional children in order to insure themselves against

potential future losses and ensure a sufficiently large family size. The replacement effect is at work when households have additional children to replace the ones that they lose, involving a sequential process.[1] From an economic theory standpoint, the replacement motive can be linked to the concept of adaptive expectations, while the insurance behavior can be linked to the concept of rational expectations. Indeed, in the former only part of the information is taken into account by households, while in the latter all available information is processed. Given the theoretical considerations underlying these two distinct effects, I argue that the replacement effect is related to individual child mortality experiences, whereas the insurance effect is related to mortality and morbidity at the community level.

The present paper uses longitudinal micro data from a Senegalese rural community and for the period 1984-2011 to analyze the effects of childhood mortality and morbidity on the fertility decision-making process. More precisely, I attempt to identify the causal effect of individual child mortality, and also that of community child mortality and morbidity, on subsequent fertility choices. For that purpose, I estimate a series of nonlinear dynamic panel data models of fertility behavior, allowing for state dependence and unobserved heterogeneity, and including the determinants of fertility as identified in the demographic and economic literature. My model is able to identify causal effects, to capture both replacement and insurance effects of child mortality, and to assess the relative importance of unobserved heterogeneity and state dependence in explaining persistence in fertility decisions. The influence on fertility of other covariates than child mortality, for instance that of seasonal migrations, is also discussed.

3.2 Previous literature

A large body of evidence suggests that fertility and child mortality are inextricably linked (Montgomery and Cohen, 1998). The relationship between child mortality and fertility lies at the heart of demographic transition theories, and the positive association between the two variables has been extensively documented.[2] Some of these studies focus on the short-term aspect of the child mortality-fertility relationship, and more specifically on the replacement effect. A common finding is that estimates of direct replacement rates are always strictly smaller than unity, implying that a replacement strategy cannot be fully realized. Among studies providing analyses of the child-replacement hypothesis in low-income settings, only a few are able to properly identify the causal effect from mortality to subsequent fertility. Testing the effect of individual child mortality on subsequent fertility decisions (*i.e.*, the replacement

[1] See for instance Taylor et al. (1976) for a detailed discussion about the insurance and replacement hypotheses. Note that these two mechanisms are not necessarily substitutes.

[2] See for instance the works of Taylor et al. (1976), Balakrishnan (1978), Olsen (1980), Olsen and Wolpin (1983), Rosenzweig and Schultz (1983), Schultz (1997), Al-Qudsi (1998b), Eckstein et al. (1999), Handa (2000), Hossain et al. (2007), Angeles (2010), or McCord et al. (2010).

effect), for which a sequential process is involved, requires the use of duration models or dynamic panel data models of individual fertility.[3] Based on a hazard regression model, Hossain et al. (2007) analyze the sequential relationship between childhood mortality and fertility among rural Bangladeshi mothers observed over the period 1982-1993. The authors find that child mortality reduces the time to subsequent conception, providing consistent support to the child-replacement hypothesis. They further show that the replacement effect interacts with the sex of the dead child, which support the hypothesis of volitional replacement. Child-replacement effects are also shown to increase as the demographic transition progresses. Also using a hazard model, Lindstrom and Kiros (2007) provide evidence for the replacement effect in Ethiopia, and show that this effect mainly occur in the case of the most recent born child, and is limited for higher order children. Finally, Bhalotra and Soest (2008) finds evidence of replacement behavior in India. With dynamic panel data models, the author show that 37 in 100 children who die during the neonatal period are replaced by new births.

To date, very few micro-level fertility studies include both individual and aggregate (community) measures of child mortality. Using Vietnamese data and a static count data model, Nguyen-Dinh (1997) documents a small and positive effect of community-level child mortality on fertility. Analyzing marital fertility trends in the Netherlands over the period 1860-1939 with a discrete-time event history model, Schellekens and van Poppel (2012) show that national-level childhood mortality contributed to fertility decline. The authors argue that this pattern is mainly due to a hoarding effect.

From a theoretical viewpoint, reproductive behaviors are not only affected by individual mortality experiences (*i.e.*, the replacement effect), but also by the anticipation that not all children will survive to adulthood (*i.e.*, the insurance effect). Such expectations are likely to be driven by changes in the epidemiological context, which include the risk of dying from infectious diseases. Surprisingly, however, the empirical investigation of the child morbidity channel in fertility studies is very recent. Employing country-level data from sub-Saharan Africa and disease-specific measures of morbidity, Aksan and Chakraborty (2013) provide evidence for the precautionary motive in a static count data model of fertility. Disaggregating child mortality into incidence and case fatality rates, the authors show that declines in child mortality reduce total fertility less where mortality *and/or* morbidity are high. Aksan (2014) also employs a static count data model of fertility, but uses individual data from the nationally representative Demographic and Health Surveys. He reaches the same result as Aksan and Chakraborty (2013), that is, fertility declines with community mortality only weakly at high levels of mortality *and/or* morbidity. The two afore-

[3]Discrete choice dynamic programming models of fertility can also be used to estimate replacement rates (see for instance Wolpin, 1984 or Mira, 2007). However, existing dynamic programing techniques require a high computational burden as well as strong assumptions on the decision-making process.

mentioned studies conclude that sub-Saharan Africa's slow demographic transition is due to the combination of continued uncertainty about child survival and poor expected health for those surviving. To my knowledge, the present paper is the first attempt to analyze the effect of community childhood morbidity on individual fertility choices within a dynamic framework, which allows to properly identify the causal effect from mortality to subsequent fertility.

3.3 Data: the Niakhar Health and Demographic Surveillance System

The rural community of Niakhar is located in the Fatick region of Senegal, 135 km east from Dakar. A Health and Demographic Surveillance System (HDSS) has been set up in 8 villages since 1962, and has been extended to 22 more villages in 1983.[4] Since 1983, the geographical boundaries of the study zone comprehends a total area of 203 km^2 and encompasses 30 villages. The estimated population was 43,000 in January 2012, and the Serer ethnic group comprises 97% of the population. Villages are subdivided into hamlets, which are themselves subdivided into compounds. Compounds are constituted of one or more "kitchens" (households) which bring together members of the extended patrilineal family. The average household size is approximately 13 persons. Agriculture is the main source of livelihood. Niakhar is Africa's oldest and still operational statistical observatory, and world's second-oldest (after Matlab, Bangladesh). Further information on the study area can be found in Delaunay et al. (2002) and Delaunay et al. (2013). This is a fairly representative rural West African setting, and the study population is relatively homogeneous in terms of socio-economic characteristics. There are large variations between households regarding both fertility and child mortality rates. Hence, it is a convenient setting to analyze the relationship between these two variables.

At the onset of the HDSS, major life events such as birth histories were collected retrospectively among the individual residents of the area to serve as a baseline for the follow-up. The HDSS consists in conducting quarterly exhaustive surveys within the study area. Thoroughly reliable data on all demographic events are systematically recorded. These events include pregnancies, deaths, marriages, migrations (inside or outside the study area), as well as changes in social characteristics. Such events are also being retrospectively and then systematically collected among the immigrants as they enter the study area. Accounting of pregnancies is practically comprehensive as a result of the quarterly follow-up. Although likely limited, the possibility of underreporting of pregnancies is still possible, especially those ending in induced abortion. Data on mortality events are reported via verbal autopsy, and causes of death follow World Health Organization's ICD-9 classification. Altogether, this results in an

[4]Niakhar HDSS has originally been set up and is still maintained by the *Institut de recherche pour le développement* (IRD).

exhaustive and systematic monitoring of the study population.

Alongside the systematic collection of data, several cross-sectional surveys were conducted for specific purposes. This study uses a cross-sectional survey conducted in 1998 and 2003 to derive economic characteristics of households. In this survey, non-monetary data on living and economic conditions were collected in an exhaustive manner, which allows me to estimate measures of multidimensional poverty. More specifically, I estimate an index of deprivation in living standards. The dimensions taken into consideration are: access to electricity, type of sanitation facilities, source of drinking water, type of cooking fuel, possession of certain assets, and flooring material of housing. This index has been calculated following the methodology outlined in Alkire and Santos (2010), which is one of the international standards for the measurement of multidimensional poverty with non-monetary data.

The long time period of data collection allows me to conduct a longitudinal analysis of individual fertility decisions. The final sample comprises 3,435 women born between 1969 and 1986. Each woman is observed over 11 consecutive years, from her 15th to her 25th birthday.[5] Hence, women born in 1969 are observed from 1984 to 1994, and women born in 1986 are observed from 2001 to 2011. The data thus covers the period 1984-2011, for a total of 18 birth-year cohorts, resulting in a sample of 37,785 individual-year observations. This sample selection allows to compare women from different cohorts, and more specifically to test whether the replacement effect of child mortality on fertility changes for women of different generations. In the analysis I distinguish between three different birth cohorts of six-year intervals (1969-1974 for the earliest age group, and 1981-1986 for the latest).

The explanatory variables include the individual-level determinants of fertility in low-income settings as identified in the demographic and economic literature.[6] Descriptive statistics of the variables used in the analyses are given in Table 3.1. The mean numbers of births and child deaths are, respectively, 1.95 and 0.22 per woman.[7] These figures are declining for more recent cohorts, as shown in Figure 3.1. The child mortality rate is 111 per 1,000 live births. Figure 3.2 shows that child mortality rates decreased sharply during the period of observation (1984-2011), with the exception of

[5]The large majority of births in the study area occur to women in early adulthood. In addition, the methodology employed in this study calls for the selection of a balanced panel with a large number of cross-sectional units (Wooldridge, 2005). Hence, a trade-off has to be made between the number of cross-sectional units in the panel and the number of observation periods during which each cross-sectional unit is observed. However, the external validity of the results does not seem to be limited by the number of observation periods (from 15 to 25 years of age) chosen in the analysis. I have used three other panels to reestimate the baseline model (Model 1). These three panels exhibit longer observation periods (namely from 15 to 30, 35 and 42 years of age), but less cross-sectional units (namely 1,823, 882, and 59 women). The results show that the sign, magnitude and statistical significance of the coefficients remain very similar. The table of results is not shown here due to space considerations, but is available from the author upon request.

[6]See Bulatao and Lee (1983) for an extensive review of the determinants of fertility in developing countries, and Bongaarts et al. (1984) for a focus on sub-Saharan African countries.

[7]Recall that each woman is observer from her 15th to her 25th birthday.

Table 3.1 – Descriptive statistics

	Mean	Standard deviation	Min	Max	Time period
Births (per woman)	1.949	1.43	0	7	1984-2011
Child mortality (per woman)	0.217	0.49	0	4	1984-2011
Child mortality (per 1,000 live births)	111	197			1984-2011
Temporary migrations (per woman)	2.740	3.09	0	11	1984-2011
Age at marriage	19.621	2.79	15	25	1984-2011
Malaria incidence rate	222.256	63.61	130.291	300.018	1992-2004
Malaria case fatality rate	0.047	0.02	0.030	0.086	1992-2004
Malaria mortality rate	10.423	3.21	4.309	16.279	1992-2004
Rainfall	466.566	94.36	292.610	631.700	1992-2004

Figure 3.1 – Mean births and child deaths (per woman) by women's birth cohort

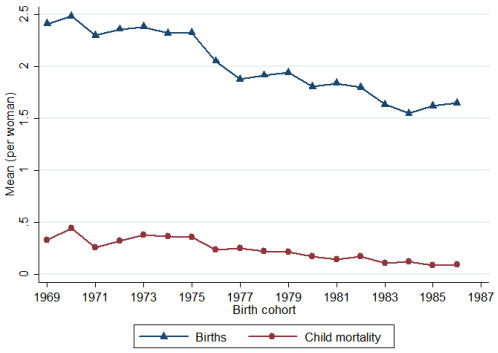
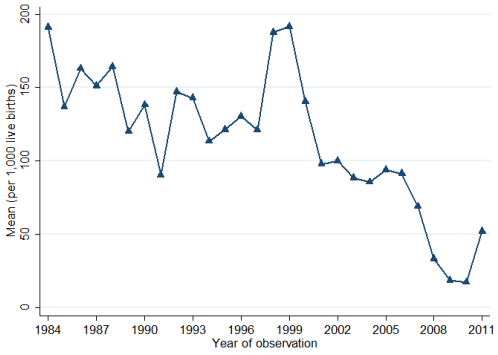


Figure 3.2 – Mean child mortality (per 1,000 live births) over the observation period



a mortality peak that occurred in 1998-1999 due to cholera and meningitis outbreaks (Delaunay et al., 2001). Each woman has temporarily migrated more than twice on average, and only 16% of the sample has never migrated during the period of study. About two-thirds of the seasonal migrations are made for economic reasons, for the most part to Dakar, the closest major urban center. Detailed definitions of the variables used are given in Table 3.2.

The mean age at marriage is 19.6, and 5% of the women were married before age 15. Polygyny is quite common, with approximately 17% of the sample living in a polygynous household. The main purpose of establishing a polygynous household in the area of study is to facilitate a more efficient household production (Mondain et al., 2004). Islam and Christianity are the main religions, representing respectively 77% and 22% of the sample. The occupation variable has been defined as the occupation held the longest by a woman during the observation period. Formal education is very low, with more than three-fourths of the sample having never completed primary school. As the time unit is taken to be a year, the individual longitudinal characteristics are included as dummy variables for each woman-year of the panel.

Table 3.2 – Definitions of the dependent and independent variables

Variable	Type	Definition	Percentage of sample
Fertility status (dependent variable)	Binary	=1 if the woman i had a birth at t; =0 otherwise	
Child mortality			
This year	Binary	=1 if the woman i experienced a child death at t; =0 otherwise	
Last year	Binary	=1 if the woman i experienced a child death at $t-1$; =0 otherwise	
Temporary migration			
This year	Binary	=1 if the woman i migrated temporarily at t; =0 otherwise	
Last year	Binary	=1 if the woman i migrated temporarily at $t-1$; =0 otherwise	
Formal education	Discrete	None (=base category); Primary or religious school; Higher	76.13; 20.64; 3.23
Living standards	Binary	=1 if the woman i lives in an extremely deprived household; =0 otherwise	2.71; 97.29
Religion	Discrete	Islam (=base category); Christianity; Animism	76.86; 22.15; 0.99
Marital status in the household			
Married before 15 years of age	Binary	=1 if the woman married before age 15; =0 otherwise	4.72; 95.28
Polygynous household	Binary	=1 if the woman i lives in a polygynous household; =0 otherwise	16.94; 83.06
Occupation	Discrete	Housewife (=base category); Active; Maid; Student	66.11; 1.37; 23.90; 8.62
Birth cohort	Discrete	1969-1974 (=base category); 1975-1980; 1981-1986	24.19; 35.05; 40.76
Age	Continuous	In years	
State dependence			
Fertility status last year	Binary	=1 if the woman i had a birth at $t-1$; =0 otherwise	
Initial conditions			
Initial fertility status	Binary	=1 if the woman i had a birth at the first observation period; =0 otherwise	
Community child morbidity (last year)			
Malaria incidence rate	Continuous	=Number of malaria cases per 1,000 child population (0-5 years) in year $t-1$	
Malaria case fatality rate	Continuous	=Number of malaria deaths divided by the number of malaria cases in the child population (0-5 years) in year $t-1$	
Malaria mortality rate	Continuous	=Number of malaria deaths per 1,000 child population (0-5 years) in year $t-1$	
Rainfall (last year)	Continuous	Total rainfall (mm) in the study area (recorded at the Niakhar station) in year $t-1$	

These dummy variables take the value of one if an event has occurred in the year considered, and zero otherwise.

Measures of child mortality and morbidity at the community level are included in further analyses.[8] More specifically, I use annual data on malaria mortality and morbidity among the child population of the study area, which are likely to capture changes in the epidemiological context.[9] These analyses are based on a smaller sample of 806 individuals since the epidemiological data is only available for the period 1992-2004.[10] Three different contextual variables are used, expressed in the following forms:

$$\text{Malaria incidence rate} = \frac{\sum \text{Malaria cases}_{t-1}}{\text{Child population (0-5 years)}_{t-1}} \times 1000$$

$$\text{Malaria case fatality rate} = \frac{\sum \text{Malaria deaths}_{t-1}}{\sum \text{Malaria cases}_{t-1}}$$

$$\begin{aligned}\text{Malaria mortality rate} &= \frac{\sum \text{Malaria deaths}_{t-1}}{\text{Child population (0-5 years)}_{t-1}} \times 1000 \\ &= \text{Malaria incidence rate} \times \text{Malaria case fatality rate}\end{aligned}$$

Each of these variables is meant to capture a specific aspect of the epidemiological profile of the population. The incidence rate measures the yearly occurrence of malaria among children, and thus quantifies the risk of developing the disease. The case fatality rate - the proportion of malaria deaths among infected children - is a marker of disease severity.[11] The mortality rate - the number of malaria deaths per 1,000 children per year - is the product of incidence and case fatality and measures

[8] The term *community* refers to the rural community of Niakhar, in which all the individuals of the sample live. These variables nonetheless exhibit both temporal (annual) variations and individual variations, as the first period of observation (the year of the 15$^{\text{th}}$ birthday) is not the same for all individuals in the sample.

[9] Changes in the malaria burden are mostly exogenous and are conditioned by climate and ecology (Sachs and Malaney, 2002; Kiszewski et al., 2004; McCord et al., 2010). Thus, these changes are unlikely to act as a proxy for an effect not directly related to epidemiological conditions. In addition, the analysis also includes temporal effect (in the form of cohort effects). These effects take into account the contextual changes potentially omitted in the model which might influence the fertility of women in the study area (for instance the development and spread of medical technology).

[10] I exploit routine data from the health care facilities of the study area. The quality of the epidemiological data has been assessed in Munier et al. (2009). Also note that, as the period of observation is shortened in this set of analyses, I distinguish between two different birth cohorts of three-years intervals (1975-1977 and 1978-1980), and no longer six-years intervals.

[11] Annual rainfall recorded at the Niakhar rain gauge will also be used as a proxy for case fatality, since periods of exceptionally high rainfall are usually accompanied by malaria outbreaks with high fatality rates (Kiszewski and Teklehaimanot, 2004). Here the coefficient of correlation between rainfall and case fatality is 0.26 ($p < 0.001$).

the yearly risk of dying from the disease. For the period 1992-2004, on average, 222 malaria cases were reported per 1,000 children per year, among which 4.7% died from the disease, resulting in a mortality rate of about 10 per 1,000 children per year. Figure 3.3 shows that malaria incidence and case fatality rates exhibit large variations over the observation period, and more importantly, that they do not always move in the same direction. In fact, the correlation between malaria incidence and case fatality is markedly negative ($\rho = -0.53,\ p < 0.001$). Not surprisingly, incidence and case fatality are both positively correlated with the mortality rate ($\rho = 0.23,\ p < 0.001$, and $\rho = 0.67,\ p < 0.001$, respectively). It is worth noticing that, for the time periods during which incidence and case fatality move in opposite directions, incidence always increases with decreasing case fatality, as shown in Figure 3.3. This statistical regularity is a common feature of sub-Saharan Africa, where, although child mortality has declined substantially, the morbidity burden remains substantial (Aksan, 2014).

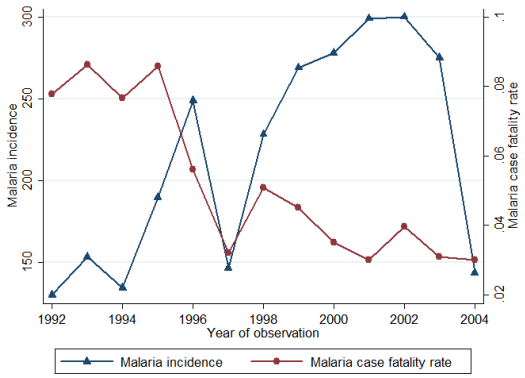


Figure 3.3 – Malaria incidence and case fatality rates over the observation period

3.4 Econometric methodology

One of the main objectives of this paper is to analyze the effects of past and present child mortality and morbidity on fertility behaviors. Two econometric issues have to be carefully considered. The first is the potential presence of strong state dependence between past and present fertility status. Here, state dependence is presumed to be negative, that is, a birth event in year $t-1$ is expected to reduce the birth probability in year t. Secondly, the correlation of the explanatory variables with the unobserved effects yields inconsistent parameter estimates if unobserved heterogeneity is not properly taken into account. The nature of the research question and the two issues needed to be addressed call for the use of a dynamic nonlinear panel data model allowing for state dependence and unobserved heterogeneity. I thus use a dynamic correlated random effects Probit model (Wooldridge, 2005).[12] This methodology allows me to properly identify the causal effect from mortality to subsequent

[12]Heckman's (1981) reduced-form approximation is an alternative method for estimating dynamic nonlinear panel data random-effects models. However, Akay (2012) shows that this method is best suited for short panels (shorter than 5 periods), while Wooldridge's (2005) method works better for panels of moderately long duration (longer than 5-8 periods), in terms of both finite-sample performance and robustness.

fertility.

To formalize the discussion, consider the following econometric model of fertility behavior of woman $i(i = 1, ..., n)$ in year $t(t = 1, ..., T)$:

$$\begin{aligned} P(birth_{it} = 1 | birth_{i,t-1}, ..., birth_{i0}, z_i, c_i) \\ &= \Phi(z_{it}\gamma + \rho birth_{i,t-1} + c_i) \\ &= \Phi(\gamma_1 cm_{it} + \gamma_2 cm_{i,t-1} + \gamma_x x_{it} + \rho_1 birth_{i,t-1} + c_i) \end{aligned} \tag{3.1}$$

where Φ is the standard normal cumulative distribution function. The binary dependent variable, $birth_{it}$, is a birth indicator. A lagged dependent variable ($birth_{i,t-1}$) is also included in the model. Notice that $t = 1$ corresponds to 1985 (2002) and $t = T$ corresponds to 1994 (2011) for women in the first (last) birth-year cohort, that is, women born in 1969 (1986).[13] Past and present child mortality (cm_{it} and $cm_{i,t-1}$) are the main variables of interest in the baseline model. x_{it} is a vector of other explanatory variables (some of these variables are constant over time). Note that $z_i = cm_i + x_i$ is the row vector of all non-redundant explanatory variables in all time periods, so that cm_i is the $1 \times T$ vector of child mortality indicators. c_i is the unobserved time-invariant individual effect. State dependence and unobserved heterogeneity are captured by $birth_{i,t-1}$ and c_i, respectively. The unobserved effect satisfies the following assumption:

$$c_i | birth_{i0}, z_i \sim \text{Normal}(\alpha_0 + \alpha_1 birth_{i0} + z_i\alpha_2, \sigma_a^2) \tag{3.2}$$

so that the distribution of the unobserved effect is conditional on the initial value and any exogenous explanatory variables. σ_a being the conditional standard deviation of c_i, it follows that $Rho = \sigma_a^2/(1 + \sigma_a^2)$ measures the contribution of the unobserved effect in the unexplained variance of the composite error. I control for the initial fertility status ($birth_{i0}$), that is, whether a woman had a birth in the first time period, in order to solve the potential initial condition problem, although this problem is unlikely to be severe in the present analysis. The dynamic binary response model (Equation 3.1) is estimated using conditional maximum likelihood.

I include both age and cohort effects in the model.[14] Period effects (year dummies) are not included due to the age-period-cohort identification problem and the impossibility to disentangle these three effects, as they are mathematically confounded (Bell and Jones, 2013). I argue that cohort effects are more likely to capture unobserved factors common to all individuals in the sample - for instance the contextual (exogenous) development and spread of medical technology - than do period effects.

[13]For each woman, the first of the 11 consecutive years of observation automatically drops out of the estimation, since lagged values of the dependent and some of the independent variables are included in the model. The econometric analysis is thus based on 10 consecutive years per woman.

[14]Since each woman is observer from her 15th to her 25th birthday, the age effect is unlikely to be non-linear, so that the use of a quadratic age polynomial is unnecessary.

An infant death abruptly terminates breastfeeding, thereby truncating the period of postpartum amenorrhea. The mother will therefore become susceptible to pregnancy more rapidly than if the child has survived (Grummer-Strawn et al., 1998). Even if the household does not intend to engage in any replacement strategy, the interval to the next birth may be nonetheless shortened, particularly in societies where access to modern contraception is limited. From a statistical viewpoint, biological and volitional effects of child mortality on fertility are thus difficult to disentangle. Attempting to circumvent this problem, I include in the model both past and current child mortality status. Current child mortality is likely to capture the behavioral component, and, in the case where the child who died was the last born and still being breastfed, the biological component of the effect of a child death. Past child mortality, however, mostly captures the behavioral component, characterized by the household's willingness to replace the child who died in the previous year. Further analyses allow the past child mortality status - the main variable of interest - to take on multiple values. For instance, I distinguish between women who did not experience a child death at $t-1$, women who lost a male child at $t-1$, and women who lost a female child at $t-1$. This allows to gain more understanding about the replacement effect and its behavioral component. A last set of analyses includes community-level measures of child mortality and morbidity. Note that these variables vary across time but not across individuals.

In order to ease the interpretation of parameter estimates, I also calculate average partial effects (APEs), which measure the size of the effect of the independent variables on the probability of birth. A method which allows to identify and estimate APEs even in the presence of unobserved heterogeneity is therefore required. I follow Wooldridge (2005) to compute partial effects on the birth probability, averaged across the population distribution of the unobserved heterogeneity. A consistent estimator of the expected probability of birth with respect to the distribution of c_i is:

$$N^{-1}\sum_{i=1}^{N}\Phi(z_t\hat{\gamma}_a+\hat{\rho}_a birth_{t-1}+\hat{\alpha}_{a0}+\hat{\alpha}_{a1}birth_{i0}+z_i\hat{\alpha}_{a2}) \tag{3.3}$$

Note that the 'a' subscript denotes multiplication by $(1+\hat{\sigma}_a^2)^{-1/2}$, and that $\hat{\gamma}$, $\hat{\rho}$, $\hat{\alpha}_0$, $\hat{\alpha}_1$, $\hat{\alpha}_2$ and $\hat{\sigma}_a^2$ are the maximum likelihood estimates. Finally, I compute differences or derivatives of Expression 3.3 with respect to the elements of $(z_t, birth_{t-1})$ to obtain the APEs.

3.5 Results

3.5.1 Baseline model

Maximum likelihood estimates of the baseline model (Equation 3.1), as well as APEs, are reported in Table 3.3. Not surprisingly, a negative state dependence effect

Table 3.3 – Dynamic correlated random effects Probit model of fertility behavior

	Model 1 (baseline model)	
	Coefficient estimates	Average partial effects
Child mortality		
This year	0.762***	0.1652
	(0.056)	(0.077)
Last year	0.877***	0.1902
	(0.060)	(0.089)
Temporary migration		
This year	-0.414***	-0.0897
	(0.027)	(0.042)
Last year	-0.007	-0.0015
	(0.027)	(0.001)
Formal education (ref. = none)		
Primary or religious school	-0.111***	-0.0240
	(0.032)	(0.011)
Higher	-0.294***	-0.0638
	(0.081)	(0.030)
Living Standards		
Extremely deprived	0.134*	0.0290
	(0.067)	(0.014)
Religion (ref. = Islam)		
Christianity	-0.010	-0.0022
	(0.028)	(0.001)
Animism	-0.044	-0.0095
	(0.115)	(0.004)
Marital status in the household		
Married before 15 years of age	0.365***	0.0791
	(0.053)	(0.037)
Polygynous household	0.140***	0.0304
	(0.030)	(0.014)
Occupation (ref. = housewife)		
Active	-0.329**	-0.0714
	(0.101)	(0.033)
Maid	-0.262***	-0.0569
	(0.027)	(0.027)
Student	-0.679***	-0.1472
	(0.059)	(0.069)
Birth cohort (ref. = 1969-1974)		
1975-1980	-0.007	-0.0015
	(0.030)	(0.001)
1981-1986	0.046	0.0100
	(0.032)	(0.005)
Age	0.149***	0.0323
	(0.003)	(0.015)
State dependence		
Fertility status last year	-1.291***	-0.2799
	(0.031)	(0.131)
Initial conditions		
Initial fertility status	0.132	
	(0.086)	
Constant	-3.817***	
	(0.076)	
$\hat{\sigma}_a$	0.405***	
	(0.015)	
Rho	0.141	
Log likelihood	-14198.938	
Wald test p-value	0.0000	
No. of individuals	3435	
No. of time periods per individual	10	
No. of observations	34350	

NOTES: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Asymptotic standard errors in parentheses. Child mortality indicators (cm_i) are included but not shown in the table. The significance level of the average partial effects corresponds to the one of the coefficient estimates.

of lagged fertility status is found, that is, having experienced a birth in the previous year has a strong and significant negative effect on current fertility. The birth probability given that a woman had a birth in the previous year is 28 percentage points lower than that of a woman who did not gave birth in the previous year. The initial value of fertility status is not statistically significant, suggesting that there is no correlation between the unobserved heterogeneity and the initial condition. The estimate of the conditional standard deviation of the unobserved effect, $\hat{\sigma}_a$, is significant and equals to 0.405. The relative importance of unobserved heterogeneity is assessed by the parameter *Rho*. Here, 14% of the total variance is due to the unobserved effect.

I now turn to the analysis of the main variables of interest, the current and lagged child mortality status. Recall that the econometric methodology allows to identify the causal effects of current and lagged child mortality on fertility behavior.[15] I thus find that both variables exert a substantial positive influence on the probability of birth. The APEs allow me to assess the magnitude of these effects. The likelihood of having a birth among women who have lost a child in the current (previous) year is 16.5 (19) percentage points higher than for women who have not lost a child in the current (previous) year. Such highly significant positive effects are nonetheless insufficient to compensate for lost lives. These results provide consistent support for the replacement hypothesis and are in line with previous findings in the literature.

The other time-varying explanatory variables are past and present migration status, and age. The parameter estimate on current migration status is negative and significant, while that on lagged migration status is not significant. More precisely, women who have migrated temporarily are 9 percentage points less likely to have a birth in the same year compared to women who stayed in the local area. The coefficient estimate on the age variable is positive and significant, with each additional year increasing the probability of birth by 3.2 percentage points.

In order to control for observed heterogeneity and to increase explanatory power, a set of time-constant explanatory variables is included in the model. It is worth noticing that the causal effects on the birth probability of time-invariant regressors cannot be identified without assuming that they are uncorrelated with the unobserved effect. I should therefore interpret these results in terms of association, remaining cautious in pointing to the existence of a causal link from these variables to fertility. Women's formal education is negatively associated with the likelihood of childbirth. Women living in the most deprived households, that is, deprived in each and every dimension of the calculated index, are significantly more likely to have births. Religion is found to have no significant influence on fertility decision-making. Not surprisingly, being married before the age of 15 is strongly associated with a higher likelihood of childbirth. Belonging to a polygynous household is found to be positively associated with the probability of birth. In Niakhar, the main purpose of establishing a polygy-

[15]Note that child mortality indicators (cm_i) are included, but not reported in the tables, to allow for correlation between the unobserved effect (c_i) and child mortality status in all time periods.

nous household is to facilitate a more efficient household production (Mondain et al., 2004). My finding could be interpreted in the same vein: having a large family would increase the value of household production. I also examine the potential influence of women's occupation on fertility behaviors, and find that occupied women have a lower likelihood of childbirth than housewives. Finally, no significant cohort effect is found.

3.5.2 Further analyses of the replacement effect

To gain more understanding about the replacement effect, I also investigate a series of interactions between child mortality and other variables of interest. These interactions are included in the baseline model, and the maximum likelihood estimates are presented in Table 3.4. Note that I still control for child mortality in the current year, which captures most of the biological effect of mortality on fertility due to the interruption of breastfeeding and the truncation of lactational amenorrhea.

In order to assess the short-term nature of the replacement effect, Model 2 adds to the baseline model an indicator of child mortality at year $t-2$. The coefficient on child mortality two years ago is not statistically significant, implying that the replacement effect only prevails in the current or subsequent year following a child's death. This can also be related to biological factors, as a mother becomes susceptible to pregnancy more rapidly after the death of a child who was being breastfed.

In Model 3, the lagged child mortality status - the main variable of interest - is allowed to take on multiple values. I distinguish between women who did not experience a child death at $t-1$ (the reference group), women who lost a male child at $t-1$, and women who lost a female child at $t-1$. The replacement effect is found to be larger in magnitude for the loss of a male rather than a female child in the previous year, and the difference between the two coefficients is statistically significant ($p < 0.10$). Hence, the volitional effect interacts with the sex of the dead child, as also observed among rural Bangladeshi women (Hossain et al., 2007). Such an illustration of the preference for sons through the replacement effect sheds light on the behavioral mechanism underlying the effect of child mortality on fertility decision-making.

Model 4 allows the past child mortality status to take on several values depending on the age of the dead child, if any. In the event of a child's death in the previous year, the replacement effect is significantly greater for the death of a child younger than one year of age than for a child aged 1-2 years ($p < 0.001$). No significant effect is found if the child who died in the previous year was older than 2 years of age, further corroborating the short-term nature of the replacement effect.

I then test whether the magnitude of the replacement effect varies with the child's cause of death. Model 5 distinguishes between deaths from infectious diseases (predominantly due to malaria and intestinal infections) and deaths from other diseases (predominantly neonatal and accidental deaths). I find that the replacement

Table 3.4 – Dynamic correlated random effects Probit model of fertility behavior - Extensions of Model 1

	Model 1 (baseline model)	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9
Child mortality									
This year	0.762***	0.733***	0.762***	0.786***	0.762***	0.762***	0.763***	0.762***	0.745***
	(0.056)	(0.057)	(0.056)	(0.056)	(0.056)	(0.056)	(0.056)	(0.056)	(0.056)
Last year	0.877***	0.865***							0.874***
	(0.060)	(0.061)							(0.060)
Two years ago		0.014							
		(0.070)							
Child mortality × Sex of the dead child									
Last year × Male child			0.959***						
			(0.079)						
Last year × Female child			0.780***						
			(0.085)						
Child mortality × Age of the dead child									
Last year × Child aged <1 year				1.613***					
				(0.090)					
Last year × Child aged 1 to <2 years				0.668***					
				(0.111)					
Last year × Child aged 2 years or more				0.056					
				(0.106)					
Child mortality × Cause of death									
Last year × Infectious disease					0.617***				
					(0.085)				
Last year × Other disease					1.098***				
					(0.079)				
Child mortality × Mother's education									
Last year × Mother with no formal education						0.865***			
						(0.064)			
Last year × Mother with formal education						0.956***			
						(0.158)			
Child mortality × Mother's marital status									
Last year × Mother in a polygynous household							0.857***		
							(0.067)		
Last year × Mother in a polygynous household							0.946***		
							(0.120)		
Child mortality × Mother's birth cohort									
Last year × Mother in the 1969-1974 cohort								1.045***	
								(0.096)	
Last year × Mother in the 1975-1980 cohort								0.826***	
								(0.092)	
Last year × Mother in the 1981-1986 cohort								0.700***	
								(0.120)	

Table 3.4 (continued)

	Model 1 (baseline model)	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9
Temporary migration									
This year	-0.414***	-0.414***	-0.414***	-0.417***	-0.414***	-0.414***	-0.414***	-0.414***	
	(0.027)	(0.027)	(0.027)	(0.027)	(0.027)	(0.027)	(0.027)	(0.027)	
Last year	-0.007	-0.018	-0.007	-0.007	-0.007	-0.007	-0.007	-0.007	0.042
	(0.027)	(0.027)	(0.027)	(0.027)	(0.027)	(0.027)	(0.027)	(0.027)	(0.027)
Temporary migration × Migration motive									
This year × Economic reasons									-0.546***
									(0.030)
This year × Family motive									0.156**
									(0.052)
This year × Educational motive									-0.741***
									(0.123)
Formal education (ref. = none)									
Primary or religious school	-0.111***	-0.115***	-0.111***	-0.113***	-0.111***	-0.112***	-0.111***	-0.111***	-0.112***
	(0.032)	(0.033)	(0.032)	(0.033)	(0.032)	(0.032)	(0.032)	(0.032)	(0.032)
Higher	-0.294***	-0.295***	-0.294***	-0.294***	-0.293***	-0.295***	-0.294***	-0.293***	-0.289***
	(0.081)	(0.081)	(0.080)	(0.082)	(0.081)	(0.081)	(0.081)	(0.081)	(0.080)
Living Standards									
Extremely deprived	0.134*	0.126^{+}	0.134*	0.139*	0.135*	0.134*	0.134*	0.135*	0.125^{+}
	(0.067)	(0.068)	(0.067)	(0.068)	(0.067)	(0.067)	(0.067)	(0.067)	(0.066)
Religion (ref. = Islam)									
Christianity	-0.010	-0.011	-0.010	-0.009	-0.011	-0.010	-0.010	-0.011	-0.015
	(0.028)	(0.028)	(0.028)	(0.028)	(0.028)	(0.028)	(0.028)	(0.028)	(0.027)
Animism	-0.044	-0.054	-0.044	-0.045	-0.042	-0.044	-0.044	-0.045	-0.044
	(0.115)	(0.117)	(0.115)	(0.117)	(0.115)	(0.115)	(0.115)	(0.115)	(0.114)
Marital status in the household									
Married before 15 years of age	0.365***	0.306***	0.364***	0.373***	0.368***	0.365***	0.365***	0.364***	0.357***
	(0.053)	(0.055)	(0.053)	(0.054)	(0.053)	(0.053)	(0.053)	(0.053)	(0.053)
Polygynous household	0.140***	0.140***	0.140***	0.139***	0.141***	0.140***	0.138***	0.141***	0.140***
	(0.030)	(0.031)	(0.030)	(0.031)	(0.030)	(0.030)	(0.030)	(0.030)	(0.030)
Occupation (ref. = housewife)									
Active	-0.329**	-0.329**	-0.331**	-0.345***	-0.336***	-0.330**	-0.330**	-0.331**	-0.326**
	(0.101)	(0.102)	(0.101)	(0.102)	(0.101)	(0.101)	(0.101)	(0.101)	(0.100)
Maid	-0.262***	-0.264***	-0.262***	-0.266***	-0.262***	-0.262***	-0.262***	-0.262***	-0.252***
	(0.027)	(0.028)	(0.027)	(0.028)	(0.027)	(0.027)	(0.027)	(0.027)	(0.027)
Student	-0.679***	-0.680***	-0.679***	-0.689***	-0.680***	-0.678***	-0.679***	-0.680***	-0.643***
	(0.059)	(0.059)	(0.059)	(0.060)	(0.059)	(0.059)	(0.059)	(0.059)	(0.059)

Table 3.4 (continued)

	Model 1 (baseline model)	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9
Birth cohort (ref. = 1969-1974)									
1975-1980	-0.007	0.005	-0.007	-0.005	-0.005	-0.007	-0.007	-0.000	-0.018
	(0.030)	(0.031)	(0.030)	(0.031)	(0.030)	(0.030)	(0.030)	(0.030)	(0.030)
1981-1986	0.046	0.067*	0.046	0.050	0.047	0.046	0.046	0.054^{+}	0.051
	(0.032)	(0.033)	(0.032)	(0.033)	(0.032)	(0.032)	(0.032)	(0.033)	(0.032)
Age	0.149***	0.131***	0.149***	0.153***	0.150***	0.149***	0.149***	0.149***	0.147***
	(0.003)	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
State dependence									
Fertility status last year	-1.291***	-1.303***	-1.291***	-1.366***	-1.301***	-1.291***	-1.292***	-1.293***	-1.296***
	(0.031)	(0.032)	(0.031)	(0.033)	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)
Initial conditions									
Initial fertility status	0.132	0.082	0.131	0.145^{+}	0.134	0.132	0.131	0.132	0.130
	(0.086)	(0.087)	(0.086)	(0.087)	(0.086)	(0.086)	(0.086)	(0.086)	(0.085)
Constant	-3.817***	-3.398***	-3.818***	-3.892***	-3.830***	-3.817***	-3.817***	-3.823***	-3.770***
	(0.076)	(0.084)	(0.076)	(0.077)	(0.076)	(0.076)	(0.076)	(0.076)	(0.076)
$\hat{\sigma}_a$	0.405***	0.408***	0.405***	0.420***	0.407***	0.405***	0.405***	0.405***	0.396***
	(0.015)	(0.016)	(0.015)	(0.015)	(0.015)	(0.015)	(0.015)	(0.015)	(0.015)
Rho	0.141	0.143	0.141	0.150	0.142	0.141	0.141	0.141	0.136
Log likelihood	-14198.938	-13601.161	-14197.649	-14129.584	-14189.710	-14198.793	-14198.718	-14196.002	-14120.759
Wald test p-value	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
No. of individuals	3435	3435	3435	3435	3435	3435	3435	3435	3435
No. of time periods per individual	10	9	10	10	10	10	10	10	10
No. of observations	34350	30915	34350	34350	34350	34350	34350	34350	34350

NOTES: $^{+}$ $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Asymptotic standard errors in parentheses. Child mortality indicators (cm_i) are included but not shown in the table. The significance level of the average partial effects corresponds to the one of the coefficient estimates.

effect, while still positive, is significantly lower for deaths from infectious diseases ($p < 0.001$). This result may reflect that the exposure to infectious diseases is more closely related to the hoarding motive than to the replacement motive. The replacement effect consists in a direct fertility response following the sudden death of a child, which is a sequential process. The hoarding motive suggests a different timing of the fertility decision process: in a worsening epidemiological environment, parents would tend to produce more children as an insurance mechanism, but not necessarily subsequent to the actual death of a child.

Models 6 investigates whether maternal education generates a differential in the replacement effect. Here the past child mortality variable distinguishes between women who did not experience a child death at $t-1$ (the reference group), women with no formal schooling who lost a child at $t-1$, and formally educated women who lost a child at $t-1$. In view of the results, it seems that maternal education does not lead to replacement effects of different magnitudes, since the two coefficients are not statistically different from each other ($p = 0.590$). In a similar manner, Model 7 questions whether the replacement effect varies according to the mother's marital status in the household. I find that the replacement behavior of mothers who lost a child in the previous year does not statistically differ depending on whether the mother belongs to a polygynous household ($p = 0.510$).

I now investigate whether the size of the replacement effect changes over time. For that purpose, Model 8 compares the replacement behavior of women from different birth cohorts. The results clearly reveal a downward trend in the size of the replacement effect, which nonetheless remains positive and significant. Mothers from the 1969-1974 cohort who experienced a child death a given year have a higher probability of birth in the next year than mothers from the 1975-1980 cohort ($p < 0.10$), or than mothers from the 1981-1986 cohort ($p < 0.05$). Hence, child mortality is likely to have a smaller impact on the subsequent fertility of women from more recent cohorts. Younger generations may resort to other strategies in a more favourable socio-economic context. Another explanation, linked to the previous one, may be related to the biological component of the replacement effect. The better access of younger generations to contraception may minimize the component of fertility related to the unmet need for family planning. In other terms, not replacing a child can now be done in a more efficient manner. Such a downward inter-cohort trend has also been documented elsewhere for the case of the hoarding motive (Bousmah, 2014).

Finally, Model 9 investigates more thoroughly the causal effect of short-term migrations on fertility decision-making. I show that, in comparison with women who stayed in the local area a given year, women who have migrated temporarily for a family motive are more likely to have a birth in the same year, whereas those who have migrated for economic or educational reasons are less likely to have a birth in the same year. In turn, the impact of current seasonal migration on current fertility is proved to be ultimately related to the motive for migration, and not to the absence

per se.

3.5.3 Insurance effects and the child morbidity channel

The last set of analyses focuses on identifying the potential effects of community-level child mortality and morbidity on subsequent fertility choices. I use annual data on malaria mortality and morbidity among the child population of Niakhar, which capture exogenous changes in the epidemiological profile of the population. Here I test whether contextual child mortality and morbidity affect households' fertility decisions. I thus favor the use of lagged community characteristics, assuming that it takes time for households to process the information about the changing epidemiological context and to adjust their fertility behavior accordingly. This specification seems more realistic and in line with the theoretical considerations underlying the insurance hypothesis, which is related to a learning process. In order to disentangle mortality and morbidity effects, I include in Model 10 lagged annual measures of both malaria incidence and case fatality. The interaction between incidence and case fatality - the malaria mortality rate - is also included to test whether mortality and morbidity jointly influence the fertility decision-making process. The inclusion of these three variables is all the more important since I showed that mortality and morbidity from malaria do not always move in the same direction. Note that I still control for past and current individual child mortality experiences. Maximum likelihood estimates and APEs are reported in Table 3.5.

The effects of previous and current individual child mortality experiences are slightly lower in magnitude than in the baseline model (Model 1). The coefficient estimates on incidence and case fatality are both negative and significant, and the coefficient estimate on their interaction is positive and significant. Given that the interaction term is significant, looking at the main effects of incidence and case fatality is not meaningful. The results reveal that the impact of past malaria incidence on the probability of birth varies depending on the severity of the disease. I provide a graphical analysis in order to ease the interpretation of the results. Figure 3.4a shows estimated the relationship between past malaria incidence among children and the probability of birth for four representative values of case fatality (including its minimum and maximum observed values). Figure 3.4b shows the APE of past malaria incidence on the probability of birth given case fatality rates. When the case fatality rate is very low (*i.e.*, its minimum observed value), the effect of past malaria incidence on fertility decisions is almost zero. The effect eventually becomes positive with higher case fatality rates, and increases with the severity of the disease. For a case fatality rate of 0.047, which is the mean over the observation period, a 5 percentage point increase in the malaria incidence rate leads to a 1.5 percentage point increase in the likelihood of having a birth in the next period. For a case fatality rate of 0.078, the likelihood of having a birth in the next period is increased by 4.5

Table 3.5 – Dynamic correlated random effects Probit model of fertility behavior - Morbidity

	Model 10		Model 11	
	Coefficient estimates	Average partial effects	Coefficient estimates	Average partial effects
Community child mortality and morbidity (last year)[a]				
Malaria incidence rate	-0.003**	0.0002	-0.007***	-0.0016
	(0.001)	(0.000)	(0.002)	(0.001)
Malaria case fatality rate	-26.849***	-0.7173		
	(4.548)	(1.217)		
Malaria mortality rate	0.098***			
	(0.024)			
Rainfall			-0.005***	-0.0001
			(0.001)	(0.000)
Malaria incidence rate × Rainfall			0.000***	
			(0.000)	
Child mortality				
This year	0.748***	0.1581	0.769***	0.1631
	(0.116)	(0.080)	(0.116)	(0.080)
Last year	0.802***	0.1696	0.818***	0.1736
	(0.122)	(0.086)	(0.123)	(0.085)
Temporary migration				
This year	-0.300***	-0.0635	-0.281***	-0.0597
	(0.050)	(0.032)	(0.050)	(0.029)
Last year	0.024	0.0051	-0.005	-0.0010
	(0.052)	(0.00)	(0.052)	(0.000)
Formal education (ref. = none)				
Primary or religious school	-0.047	-0.0099	-0.046	-0.0097
	(0.071)	(0.005)	(0.070)	(0.005)
Higher	-0.334*	-0.0706	-0.333*	-0.0708
	(0.154)	(0.036)	(0.153)	(0.035)
Living Standards				
Extremely deprived	-0.116	-0.0245	-0.110	-0.0234
	(0.158)	(0.012)	(0.157)	(0.012)
Religion (ref. = Islam)				
Christianity	0.118*	0.0249	0.114^{+}	0.0242
	(0.059)	(0.013)	(0.058)	(0.012)
Animism	0.226	0.0478	0.229	0.0486
	(0.291)	(0.024)	(0.289)	(0.024)
Marital status in the household				
Married before 15 years of age	0.372***	0.0787	0.370***	0.0785
	(0.109)	(0.040)	(0.108)	(0.039)
Polygynous household	0.173**	0.0366	0.165**	0.0351
	(0.062)	(0.019)	(0.061)	(0.017)

Table 3.5 (continued)

	Model 10		Model 11	
	Coefficient estimates	Average partial effects	Coefficient estimates	Average partial effects
Occupation (ref. = housewife)				
Active	-0.306	-0.0646	-0.313	-0.0664
	(0.246)	(0.033)	(0.244)	(0.033)
Maid	-0.198***	-0.0418	-0.199***	-0.0422
	(0.051)	(0.021)	(0.051)	(0.021)
Student	-0.821***	-0.1735	-0.820***	-0.1741
	(0.154)	(0.088)	(0.153)	(0.086)
Birth cohort (ref. = 1975-1977)				
1978-1980	-0.025	-0.0053	0.063	0.0133
	(0.059)	(0.003)	(0.057)	(0.007)
Age	0.121***	0.0257	0.147***	0.0312
	(0.013)	(0.013)	(0.010)	(0.015)
State dependence				
Fertility status last year	-1.332***	-0.2814	-1.327***	-0.2817
	(0.067)	(0.143)	(0.067)	(0.138)
Initial conditions				
Initial fertility status	0.291		0.279	
	(0.193)		(0.192)	
Constant	-2.312***		-1.966***	
	(0.342)		(0.528)	
$\hat{\sigma}_a$	0.389***		0.383***	
	(0.032)		(0.032)	
Rho	0.131		0.128	
Log likelihood	-3234.656		-3247.278	
Wald test p-value	0.0000		0.0000	
No. of individuals	806		806	
No. of time periods per individual	10		10	
No. of observations	8060		8060	

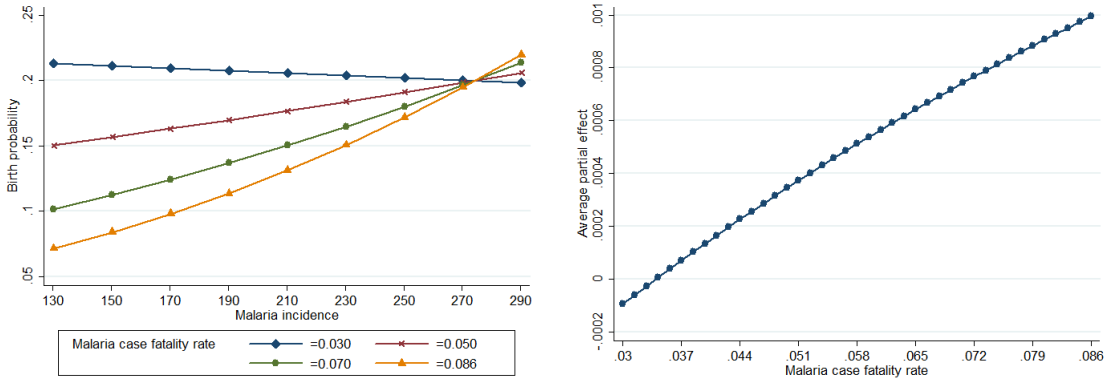
NOTES: $^{+}$ $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Asymptotic standard errors in parentheses. Child mortality indicators (cm_i) are included but not shown in the table. The significance level of the average partial effects corresponds to the one of the coefficient estimates.

[a] The term *community* refers to the rural community of Niakhar, in which all the individuals of the sample live.

percentage points following a 5 percentage points increase in the malaria incidence rate. To sum up, the more malaria is fatal to children who are infected, the more households are likely to produce children in the next period. Finally, the results also suggest that both individual and contextual child mortality positively influence subsequent fertility decisions.

Figure 3.4 – Effect of community morbidity (malaria incidence rate) on fertility given malaria case fatality rate

(a) Predicted probabilities **(b)** Average partial effect



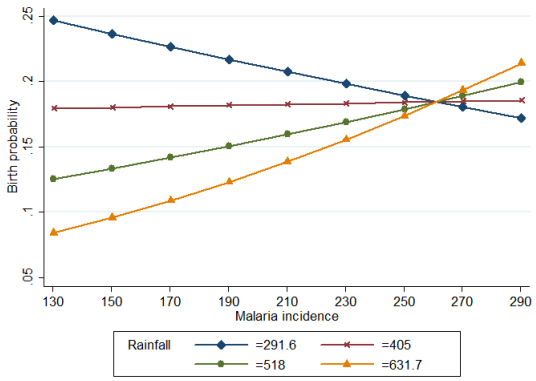
I choose to use contextual mortality and morbidity attributable to malaria since the transmission of this disease has been shown to be mainly driven by exogenous factors, namely climate and ecology (Kiszewski et al., 2004). By doing so, I assume that the potential endogenous component of community child mortality is removed. In order to confirm my results, Model 11 proposes to use annual rainfall as a proxy for malaria case fatality, since periods of exceptionally high rainfall are usually accompanied by malaria outbreaks with high fatality rates (Kiszewski and Teklehaimanot, 2004). Figure 3.5 shows that the qualitative finding of Model 10 is confirmed: the more severe the disease, the higher the effect of malaria incidence on subsequent fertility choices.

3.6 Discussion

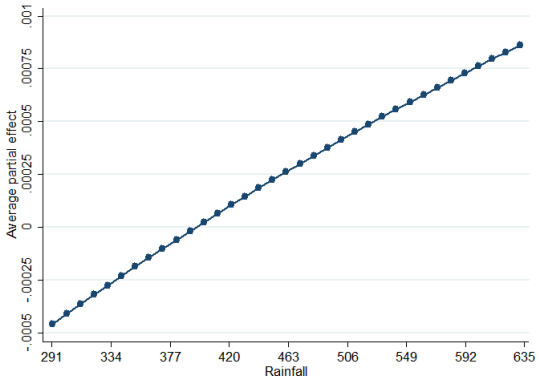
In summary, this paper conducts an analysis of both replacement and insurance effects among women from the rural area of Niakhar, Senegal and for the period 1984-2011. Nonlinear dynamic panel data models of fertility behavior are estimated to analyze the effect of childhood mortality and morbidity on the fertility decision-making process. My methodology allows for state dependence and unobserved heterogeneity, and also explicitly control for observed heterogeneity in including the determinants of fertility as identified in the demographic and economic literature.

Figure 3.5 – Effect of community morbidity (malaria incidence rate) on fertility given rainfall

(a) Predicted probabilities



(b) Average partial effect



I stress the importance of distinguishing between individual and community-level measures of childhood mortality and morbidity. As a result, I am able to identify the causal effect of individual child mortality, and also that of community child mortality and morbidity, on subsequent fertility choices. The results provide consistent support for the replacement hypothesis, as well as for the precautionary demand for children.

The analysis of the child replacement hypothesis yields several findings. First, the impact of contemporaneous and previous period child mortality on fertility are both positive, yet not sufficient enough to compensate for lost lives. The short-term nature of the replacement effect is assessed by showing that child mortality two years ago has no effect on current fertility, and that the effect is greater for the death of a child younger than one year of age. In addition, the replacement effect is found to be larger in magnitude for the loss of a male rather than a female child. This cannot be attributable to biological factors, shedding light on the behavioral mechanism underlying the replacement effect.[16] I also show that child mortality is likely to have a smaller impact on the subsequent fertility of women from more recent cohorts, providing evidence for a downward trend in the size of the replacement effect over time. Part of this downward trend may be due to the increased knowledge and use of modern contraceptive methods, which would dampen the biological replacement effect related to the unmet need for contraception. Finally, I find that the magnitude of the replacement effect varies with the child's cause of death, being lower for deaths from infectious diseases. This result may reflect that the exposure to infectious diseases is more closely related to the insurance effect, which has a different

[16]Recall that the volitional fertility response is likely to act jointly with a purely biological effect which occurs when a child dies during the breastfeeding period. Disentangling the volitional and biological components of the replacement effect has been a major challenge of the empirical literature on the effect of child mortality on fertility.

timing.

The main findings supporting the insurance effect are as follows. My estimates reveal that community-level malaria incidence among children has a positive effect on subsequent fertility choices, and that this positive effect is stronger the more the disease is fatal to children who are infected. Therefore, the effect of the children's risk of developing malaria is not linear and ultimately depends on the relative severity of the disease. To my knowledge, this is the first paper that identifies the causal effects of perceived child mortality and morbidity risks on the fertility decision-making process. My results are only partly in line with those of Aksan and Chakraborty (2013) and Aksan (2014), obtained with static count data models of fertility. Although they also find that both child mortality and morbidity are joint determinants of fertility in today's sub-Saharan Africa, their estimated effect of childhood disease incidence on fertility is different from mine. Their main conclusion is that declines in child mortality reduce fertility less where morbidity persists, whereas my results suggest that childhood disease incidence is likely to reinforce the positive effect of child mortality on fertility. In my analysis, the childhood morbidity effect is somewhat indirect, acting through the relative risk of dying from the disease, and not through the risk of getting infected *per se*. I argue that the decline in childhood mortality, which has so far not been substantial enough to trigger fertility declines, is an explanation for sub-Saharan Africa's sluggish demographic transition. The persistently high childhood disease incidence is not responsible *per se* for the slow transition. Rather, childhood morbidity contributes to the slow transition to the extent that uncertainty about child survival remains a major concern in sub-Saharan Africa.

General conclusion

This dissertation is related to the strand of literature which analyzes the relationship between child mortality and fertility behaviors. Besides the academic interest, a better understanding of this relationship can be useful from a policy standpoint. In today's sub-Saharan African context, examining the child mortality-fertility relationship is all the more important, as the region is experiencing a slow demographic transition and a growing demographic pressure. Each of the three essays of this dissertation makes a distinct contribution to the literature on the topic.

The first chapter of this dissertation provides empirical support for the precautionary demand for children, that is, a positive effect of child mortality on net fertility. To my knowledge, this is the first evidence of the realization of the insurance strategy at the micro level. I also find that the child mortality-fertility relationship is of non-linear nature, as the amplitude and the direction of the effect of child mortality vary with the number of child losses. Mothers with the largest number of survivors are those for whom the burden of child mortality has not been particularly high. The insurance mechanism is thus revealed to be at work for non-zero but relatively low values of child mortality. In addition, my estimates provide evidence of a downward trend in the positive effect of child mortality on total *and* net fertility with time. The mortality effect is also found to decrease with the mother's age at first birth.

In the second chapter, I develop an endogenous fertility model to analyze the effect of the uncertainty about child survival on fertility behavior. Existing endogenous fertility models analyzing the child mortality-fertility relationship systematically assume that the birth of each child entails a fixed cost to parents. I stress the importance of considering a more refined functional form for the cost of children, that is, more general and empirically appealing. When the total cost of children is allowed to change across socio-economic settings, I show that the fertility response to improvements in child survival may be either positive or negative. The positive relationship between child mortality and total fertility holds for socio-economic settings where children are "time-intensive", typically in settings where a certain level of economic development has been reached. Concurrently, I find that total fertility increases with improvements in child survival in settings where children are "time-supplying", that is, in economically deprived environments. Hence, the results suggest that perceptions of economic opportunities and health concerns are joint determinants of fertility behaviors.

Finally, the third chapter empirically investigates the effects of childhood mortality and morbidity on the timing of the fertility decision-making process. I identify the causal effect of individual child mortality, and also that of community child mortality and morbidity, on subsequent fertility choices. The econometric methodology allows me to identify the causal effect of individual child mortality, and also that of community child mortality and morbidity, on subsequent fertility choices. The results provide consistent support for both the child-replacement hypothesis and the precautionary demand for children. Among other results, I find that community child mortality and morbidity attributable to malaria, which capture exogenous changes in the epidemiological context, exert a joint influence on fertility behaviors. More specifically, the more malaria is fatal to children who are infected, the more households are likely to produce children in the next period. I then argue that the persistently high childhood morbidity contributes to the slow demographic transition in sub-Saharan Africa only to the extent that child mortality remains a major concern in the region.

This dissertation fundamentally highlights the role that child mortality plays in determining changes in households fertility behavior. All three chapters identify child mortality as one of the main cause of the slow demographic transition in today's sub-Saharan Africa. The first chapter concludes that the decrease in child mortality should be steeper than the one experienced in the region so far in order to trigger substantial fertility declines. The second chapter demonstrates that joint improvements in socio-economic conditions and child survival are needed to trigger a fertility transition. Finally, the third chapter suggests that childhood disease incidence is likely to reinforce the positive effect of child mortality on fertility. When choosing to focus on community mortality and morbidity attributable to malaria, I also provide the first empirical support for the desirable indirect effects of malaria control policies on fertility behaviors at the individual level. Therefore, while malaria control is an end in itself, it is also a means to achieving the fertility transition. Altogether, I argue that more ambitious improvements in economic conditions and in child survival and infection rates, along with traditional family planning programs, would be likely to substantially encourage the diffusion of the fertility transition in sub-Saharan Africa. Understanding better the interconnection between economic and health conditions in determining changes in fertility behaviors remains a goal for future work.

Résumé

Cette thèse se donne pour objectif de contribuer à la compréhension des tendances démographiques en Afrique subsaharienne par l'examen de l'influence de la mortalité infantile sur les comportements de fécondité.

Dans le premier chapitre, j'examine la relation entre mortalité infantile et fécondité à l'échelle micro-économique. Des modèles de données de comptage sont utilisés pour analyser les déterminants de la fécondité complète de femmes d'une communauté rurale sénégalaise. Je montre que l'effet global de la mortalité infantile est positif tant sur la fécondité totale que sur la fécondité nette. De plus, j'identifie une relation en U inversé entre mortalité infantile et fécondité nette. La principale implication est que les politiques de santé visant à réduire la mortalité infantile ont des effets indirects sur la fécondité désirée. Néanmoins, seul un fort déclin de la mortalité infantile serait à même d'entraîner une baisse substantielle de la fécondité.

Dans le second chapitre, j'analyse les effets de la mortalité infantile sur les comportements reproductifs dans un modèle de fécondité endogène où la survie infantile est stochastique. J'adopte une forme fonctionnelle de coût des enfants englobant quatre scénarios différents, chacun représentant un contexte socio-économique distinct. Les théories de la transition démographique postulent généralement une relation positive entre mortalité infantile et fécondité totale. Je démontre analytiquement que cette relation peut être inversée en fonction de la forme de la fonction de coût total des enfants considérée. Mon modèle peut prédire des réponses positives et négatives de la fécondité, selon que les enfants sont respectivement "intensifs en temps" ou "pourvoyeurs en temps". Par conséquent, ce modèle peut fournir une explication pour la lenteur de la transition démographique dans l'Afrique subsaharienne contemporaine.

Finalement, le troisième chapitre analyse les effets de la mortalité et de la morbidité infantiles sur le processus de décision de fécondité des femmes rurales sénégalaises. J'estime des modèles dynamiques de données de panel qui permettent d'identifier l'effet causal de la mortalité infantile, ainsi que de la mortalité et de la morbidité infantiles à l'échelle de la communauté, sur les décisions ultérieures de fécondité. Les résultats supportent les hypothèses de remplacement et d'assurance. Je montre que la mortalité et la morbidité palustres à l'échelle de la communauté, capturant les changements exogènes du contexte épidémiologique, ont un effet positif sur les décisions ultérieures de fécondité. Cet effet est d'autant plus fort que la maladie est létale pour les enfants infectés. L'incidence des maladies infantiles, qui reste particulièrement élevée en Afrique subsaharienne, n'est donc pas en soi responsable de la lenteur de la transition démographique. Néanmoins, cette morbidité infantile contribue à la lenteur de la transition dans la mesure où l'incertitude de la survie de l'enfant reste un problème majeur dans la région.

Mots-clés : Fécondité · Mortalité infantile · Morbidité infantile · Paludisme · Afrique subsaharienne · Hypothèse de remplacement · Hypothèse d'assurance · Fécondité endogène · Coût des enfants · Incertitude.

Introduction générale

L'Afrique subsaharienne connaît une transition démographique particulièrement lente. Bien que la mortalité infantile suive une évolution décroissante depuis les années 1950, les taux nets de fécondité restent considérablement élevés (Bongaarts et Casterline, 2013). Tout se passe comme si la plupart des pays d'Afrique subsaharienne se trouvaient toujours au premier stade de la transition. L'écart de fécondité entre ces pays et le reste du monde reste considérable et constant dans le temps (United Nations, 2013). Réduire la pression démographique s'avère être un enjeu majeur du développement socio-économique de ces pays. Un postulat conventionnel de la transition démographique veut que la fécondité suive la baisse de la mortalité infantile. Ce postulat ne parvient apparemment pas à expliquer les tendances démographiques de l'Afrique subsaharienne contemporaine. Au vu des régularités statistiques mentionnées ci-dessus, il semble que des travaux théoriques et empiriques supplémentaires soient nécessaires pour mieux comprendre la relation entre mortalité infantile et fécondité. Cette thèse se donne pour objectif d'analyser les rôles joués par la mortalité infantile et l'incertitude de la survie de l'enfant dans l'explication de la lenteur de la transition démographique en Afrique subsaharienne.

Il est important d'examiner les tendances démographiques en Afrique subsaharienne à la lumière des théories de la transition de la fécondité. La littérature sur la transition de la fécondité peut être scindée en deux parties, suivant la classification établie par Carlsson (1966).[1] D'une part, l'*approche de l'innovation* postule que les baisses de fécondité sont uniquement causées par l'amélioration des technologies en matière de planning familial. Cette approche relève aussi l'importance de la diffusion de l'information sur le contrôle des naissances. Les hauts niveaux de fécondité seraient donc la conséquence d'une demande non satisfaite de contraception. D'autre part, l'*approche de l'ajustement* considère la fécondité comme une variable volitionnelle plutôt que comme une contrainte implicite. Selon cette approche, les ménages seraient amenés à modifier leur comportement de fécondité en fonction des changements de contexte socio-économique et épidémiologique, ces derniers pouvant affecter les incitations à la fécondité. Dans cette dernière approche, l'accès à la contraception est une condition nécessaire mais non suffisante à la baisse de la fécondité. Tout au

[1] Le lecteur est aussi invité à se référer à Bleakley et Lange (2009) pour une description des deux catégories plus détaillée et s'appuyant sur la littérature récente.

long de cette thèse, je soutiens que l'*approche de l'ajustement* prévaut pour expliquer les tendances démographiques actuelles en Afrique subsaharienne. La disponibilité des services de planning familial est cruciale pour la diffusion de la transition de la fécondité. L'utilisation de la contraception moderne est indéniablement associée à un déclin de la fécondité. Dans les pays en développement, la connaissance des méthodes pour reporter ou empêcher une grossesse est désormais très répandue. Néanmoins, ces meilleurs niveaux de connaissance ne sont pas nécessairement associés à une plus grande utilisation. Cleland *et al.* (2011) rapportent que le progrès vers l'adoption de la contraception moderne a été particulièrement faible en Afrique subsaharienne, les résistances attitudinales restant une barrière majeure. Des études révèlent que, bien qu'une part de la fécondité résulte d'une demande non satisfaite de planning familial, sa plus grande part est en réalité désirée par les ménages (Bongaarts, 2011). En m'appuyant sur les régularités statistiques reportées ci-dessus, je considère la fécondité principalement comme une variable de choix, dans la tradition de la théorie économique. Cette thèse entre donc dans le champ de l'*approche de l'ajustement*.

L'objectif de cette thèse est de contribuer à la compréhension des tendances démographiques actuelles en Afrique subsaharienne par l'examen du rôle joué par la mortalité infantile dans les décisions de fécondité. D'un point de vue théorique, l'effet de la mortalité infantile sur les choix de fécondité peut être double. D'une part, l'*effet de remplacement* prévaut quand les ménages ont des enfants additionnels pour remplacer ceux qu'ils perdent, ce qui relève d'un processus séquentiel. Cette stratégie implique qu'un nombre cible d'enfants survivants a été établi par les ménages. Ainsi, ils réagissent au décès d'un enfant en le remplaçant directement, et la mortalité affecte donc le taux brut de fécondité. Cependant, le seul comportement de remplacement n'est pas suffisant pour générer une relation positive entre mortalité infantile et taux net de fécondité (*i.e.*, le nombre de survivants). D'autre part, l'*effet d'assurance* prévaut quand les ménages anticipent le fait qu'un certain nombre de leurs enfants ne survivront pas. Dans un contexte épidémiologique défavorable, les ménages auraient des enfants additionnels afin de s'assurer contre les pertes potentielles futures et garantir une taille familiale suffisamment large. Ainsi, l'adoption d'une telle stratégie affecterait positivement la fécondité nette. Du point de vue de la théorie économique, le motif de remplacement peut être rattaché au concept d'anticipations adaptatives, et le comportement d'assurance à celui d'anticipations rationnelles. En effet, seulement une partie de l'information est prise en compte dans le premier cas, alors que toute l'information disponible est exploitée dans le second. Au vu de la pression démographique grandissante en Afrique subsaharienne, le fait de mieux savoir dans quelle mesure les ménages ajustent leur comportement de fécondité dans un contexte de faible probabilité de survie infantile semble donc être une question de recherche décisive. Les trois chapitres de cette thèse examinent la relation entre mortalité infantile et fécondité, toutefois selon différentes perspectives et avec différentes approches.

Dans le premier chapitre, j'analyse empiriquement la relation entre mortalité in-

fantile et fécondité à l'échelle micro-économique. Plus spécifiquement, je considère la réalisation (ou non) d'une stratégie de fécondité à la fin de la vie reproductive d'une femme. J'utilise des données individuelles collectées trimestriellement dans le cadre du Système de Suivi Démographique de la communauté rurale de Niakhar (Fatick, Sénégal), qui est le plus ancien observatoire statistique africain encore en fonction. Une analyse approfondie de la relation entre mortalité infantile et fécondité est conduite en s'appuyant sur les historiques de naissances de 2884 femmes nées entre 1932 et 1961. L'attention est portée sur la fécondité complète, c'est-à-dire le nombre de naissances au terme de la vie féconde d'une femme. Des modèles de données de comptage sont employés pour estimer les déterminants de la fécondité complète. L'effet global de la mortalité infantile est positif tant sur la fécondité totale que sur la fécondité nette, ce qui apporte un soutien empirique à l'hypothèse d'assurance. De plus, j'identifie une relation en U inversé entre mortalité infantile et fécondité nette. Je montre que les mères ayant eu le plus grand nombre de survivants sont celles pour lesquelles le fardeau de la mortalité infantile n'a pas été particulièrement élevé. Ainsi, le mécanisme d'assurance prévaut principalement pour des valeurs positives mais relativement faibles de mortalité infantile. De plus, je montre que l'effet positif de la mortalité décroit avec l'âge de la mère à la première naissance. Les politiques visant à retarder l'entrée en vie féconde ont donc vraisemblablement un effet négatif direct sur la fécondité, ainsi qu'un effet négatif indirect par le canal de la mortalité infantile. Je trouve aussi que l'effet de la mortalité infantile est plus important pour les femmes des cohortes plus anciennes, ce qui caractérise une diffusion croissante de la transition de la fécondité dans la région. Au vu des résultats, la principale implication de ce chapitre est que les politiques de santé visant à réduire la mortalité infantile ont des effets indirects sur la fécondité désirée des ménages. Néanmoins, seul un fort déclin de la mortalité infantile serait à même d'entraîner une baisse substantielle de fécondité. Cela ne signifie pas que les politiques traditionnelles de planning familial devraient être abandonnées. De telles politiques ont des effets directs sur la fécondité non désirée des ménages, et sont donc une condition nécessaire à la réalisation de la transition de la fécondité.

Dans le deuxième chapitre, j'analyse les effets de la mortalité infantile sur les comportements reproductifs dans un modèle de fécondité endogène dans lequel la survie infantile est stochastique. Les modèles de fécondité endogène analysant la relation entre mortalité infantile et fécondité supposent systématiquement que chaque naissance représente un coût fixe pour les parents. Dans ce chapitre, je remets en question l'utilisation d'une fonction de coût fixe des enfants pour la modélisation de la relation entre mortalité infantile et reproduction dans un cadre de fécondité endogène. La construction d'un tel modèle a pour objectif une meilleure compréhension des tendances démographiques contemporaines en Afrique subsaharienne. La lenteur de la transition démographique dans la région peut potentiellement être expliquée en faisant varier la forme de la fonction de coût des enfants selon les contextes socio-économiques. J'adopte donc une forme fonctionnelle de coût des enfants englobant

quatre différents scénarios, chacun représentant un contexte socio-économique distinct. Les études estimant le coût des enfants dans des contextes de hauts et bas revenus constituent les fondements empiriques de ces quatre scénarios. Les théories de la transition démographique prédisent généralement une relation positive entre mortalité infantile et fécondité totale. Je démontre analytiquement que cette relation peut être inversée en fonction de la forme de la fonction de coût total des enfants considérée. Dans le cas de coûts marginaux croissants ou décroissants, je montre que la réponse de la fécondité à la mortalité infantile est positive. En revanche, la réponse devient négative dans le cas d'un coût total décroissant avec le nombre d'enfants. Ainsi, mon modèle peut prédire des réponses positives et négatives de la fécondité, selon que les enfants sont respectivement "intensifs en temps" ou "pourvoyeurs en temps". Plus spécifiquement, je montre que quand le coût des enfants est faible, ce qui est le cas dans les contextes de bas revenus, la fécondité totale augmente avec les progrès en matière de survie infantile. Cela entraînerait donc une augmentation de la pression démographique. Ce chapitre contribue aux discussions théoriques récentes sur les mécanismes des transitions démographiques passées et présentes. Dans mon modèle, les comportements de fécondité sont déterminés conjointement par les questions de santé et les perceptions d'opportunités économiques. Ce modèle peut fournir une explication pour la lenteur de la transition démographique dans l'Afrique subsaharienne actuelle.

Finalement, le troisième chapitre analyse les effets de la mortalité et de la morbidité infantiles sur le processus de décision de fécondité. Cette analyse est conduite au niveau empirique et s'appuie sur des données microéconomiques longitudinales provenant de la communauté rurale de Niakhar (Fatick, Sénégal), pour la période 1984-2011. J'utilise un modèle dynamique de données de panel afin de centrer l'analyse sur le *tempo* (calendrier) de la fécondité. Les principaux objectifs de ce chapitre sont les suivants. Je cherche à estimer la magnitude de l'effet de remplacement, ainsi qu'à mettre en lumière le caractère comportemental de cet effet.[2] Je cherche aussi à identifier l'effet causal de la mortalité et de la morbidité infantiles attribuées au paludisme, et ce à l'échelle de la communauté, sur les choix ultérieurs de fécondité. Le paludisme, étant à la fois une maladie liée à la pauvreté et une cause de pauvreté, représente une des causes principales de mortalité infantile en Afrique subsaharienne.[3] Au-delà de ses impacts directs sur la santé, le paludisme entraîne

[2]Un problème majeur de la littérature empirique sur la relation entre mortalité infantile et fécondité a été de séparer la composante comportementale de la composante biologique de l'effet de remplacement. En effet, le décès d'un enfant interrompt brusquement l'allaitement, tronquant ainsi la période d'aménorrhée post-partum de la mère. Cela peut avoir pour effet de réduire l'intervalle avant la prochaine grossesse. Que ce soit à des fins de recherche ou de politique publique, il apparaît important de déterminer la contribution relative des facteurs comportementaux et biologiques de l'effet de remplacement.

[3]Voir Black *et al.* (2010) pour une analyse des causes globales, régionales et nationales de mortalité infantile.

aussi d'importants effets indirects et de larges coûts sociaux.[4] Dans ce chapitre, je cherche à déterminer si les progrès dans le contrôle du paludisme ont aussi des effets indirects désirables sur les taux de fécondité, passant par la réduction de la mortalité et de la morbidité infantiles. Des modèles dynamiques non linéaires de données de panel sont utilisés pour estimer les probabilités de naissance. La dépendance d'état et l'hétérogénéité non observée sont prises en compte dans la modélisation. Cette méthodologie rend possible l'identification de l'effet causal de la mortalité infantile, ainsi que ceux de la mortalité et de la morbidité infantiles à l'échelle de la communauté, sur les décisions ultérieures de fécondité. Les résultats supportent l'hypothèse de remplacement, ainsi que celle de la demande d'enfants de précaution. Je montre que la mortalité et la morbidité palustres à l'échelle de la communauté, capturant les changements exogènes du contexte épidémiologique, exercent une influence conjointe sur les comportements de fécondité. L'incidence palustre a un effet positif sur les choix ultérieurs de fécondité, et cet effet positif est d'autant plus fort que la maladie est létale pour les enfants infectés. L'incidence des maladies infantiles, qui reste particulièrement élevée en Afrique subsaharienne, n'est donc pas *en soi* responsable de la lenteur de la transition démographique. Néanmoins, cette morbidité infantile contribue à la lenteur de la transition dans la mesure où l'incertitude de la survie de l'enfant reste un problème majeur dans la région.

La principale implication en matière de politique publique qui apparaît au terme des trois chapitres de cette thèse est la suivante. Parallèlement au développement économique et aux programmes traditionnels de planning familial, les politiques de santé visant à réduire la mortalité et la morbidité infantiles ont des effets additionnels sur les décisions de fécondité. Si ces politiques de santé devaient réduire fortement l'incertitude de la survie infantile, elles pourraient entraîner des baisses conséquentes de fécondité. Cela contribuerait à réduire la pression démographique dans l'Afrique subsaharienne contemporaine.

[4]Par exemple, Thuilliez (2010) prouve l'existence d'un lien fort et positif entre l'endémie de paludisme *falciparum* et les taux de redoublement dans le primaire. Cela suggère que le paludisme entrave l'accumulation de capital humain des enfants, et par extension la croissance économique. Voir aussi Sachs et Malaney (2002) pour une revue des effets indirects potentiels du paludisme.

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