DEMOGRAPHIC MODELS OF HEALTH AND MORTALITY AT BOTH EXTREMES OF THE LIFESPAN

Julio E. Romero Prieto

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Supervisor of Dissertation

_____

Hans-Peter Kohler
Frederick J. Warren Professor of Demography and Professor of Sociology

Graduate Group Chairperson

_____

Hans-Peter Kohler
Frederick J. Warren Professor of Demography and Professor of Sociology

Dissertation Committee

Michel Guillot, Professor of Sociology
Douglas Ewbank, Research Professor Emeritus of Sociology

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To María Alejandra and Enrique Samuel

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ABSTRACT

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Julio E. Romero Prieto
Hans-Peter Kohler

This dissertation consists of three essays. The first chapter proposes a model life table to investigate the human mortality at early ages. The model was estimated from the vital records, observing the experiences of 24 countries, which in some cases are at end of the nineteenth century and much of the twentieth century. Using few input values, the model predicts a mortality schedule for the first days, weeks, months, and years of life. Furthermore, the model is flexible to represent age patterns in conditions of either low or high mortality. Thus, the main application is as a method for indirect estimation, in contexts where vital records are incomplete, imperfect, or non-existent. In this direction, the second chapter takes advantage of the model to investigate the mortality patterns and the quality issues of the mortality estimates from self-reported data. To this end, a total of 252 *Demographic and Health Surveys* were analyzed in light of the predictions of the model, in order to identify particular characteristics of these populations. These comparisons lead to the conclusion that populations with high levels of mortality are more likely to show late patterns of under-five mortality. The model was also used to examine data quality issues regarding misreported ages at death. Particularly, this chapter proposes a simple solution to the problem of heaping at the age of 12 months and the underestimation of the infant mortality. The third chapter investigates the relationship between health status and survival expectations on a sample of mature adults aged 45+, who participated in the *Malawi Longitudinal Study of Families and Health* between 2006 and 2012. In particular, structural equation models were estimated assuming intertemporal relationships between physical health, mental health, and the formation of survival expectations. These models identify different pathways that have been discussed from theoretical and empirical approaches showing evidence of the concomitancy of physical and mental health issues, and the relevance of the expectations about life. This paper quantifies a significant impact of mental health on the prospective physical health and provides evidence on the differentiated adaptation pathways for men and women.

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Under-five mortality: a model life table approach

Abstract

The lack of reliable records is one of the main problems in the study of mortality at early ages in less developed countries. However, indirect methods are used to estimate basic demographic statistics. This paper proposes a model life table for under-five mortality, which is estimated from the vital records of 24 countries. Records included in the model are dated from the end of the nineteenth century and much of the twentieth century. The pertinence of this time period is that it encloses the pronounced decline in infant mortality, where prosperity, medical developments, and social transformations played a decisive role. The model incorporated a diversity of countries, enriching the estimation while adding different socioeconomic backgrounds and climate conditions, which largely affected mortality patterns. Using a few input values, the model allows estimating a complete mortality schedule: days in the first week of life, weeks to complete the first month, months at postneonatal ages, trimesters in the second year, and single years of age to complete childhood. Existing models are general and do not examine variations in the mortality pattern occurring at an early age. By using different mortality indexes and multiple quantitative methods, the model was estimated by identifying a group of input parameters that amplify the predictive power of the model. Finally, the paper presents a model open to the estimation of mortality schedules by using a set of entry values, predefined by the researcher. By formulating indirect estimations based on incomplete information, this model constitutes an essential tool in the demographic analysis.

Key words: Infant and child mortality; under-five mortality; demographic estimation; indirect methods; models of mortality; historical sources of mortality data.

1.1. Introduction

Model life tables constitute an essential tool for the demographic analysis. They are stylized representations of the force of mortality as a function of age (Coale & Demeny, 1966), and models predict the change on the mortality pattern resulting of a change in the level of mortality. According to the approach involved, model life tables could be mathematical, relational, or demographic (Ewbank, et al., 1983). Demographic models are empirically constructed from a collection of life tables; tables that were calculated directly from the vital records whose quality is reliable. In the practice, models are used for validation purposes, as well as for indirect estimation (Brass, 1971; Coale & Trussell, 1996). In the first case, models provide a standard to examine the consistency of the data. In the second case, models reproduce an entire mortality schedule from incomplete information (United Nations, 1982). The simplicity of its implementation, the demographic consistency, and the statistical adjustment are three aspects that should be guaranteed by a model life table (Murray, et al., 2003). Therefore, the purpose of a demographic model is to estimate the force of mortality given a reduced number of parameters that best represent the shape and the level of mortality.

Model life tables do not fit in the strict definition of a model, since they imply neither theoretical constraints nor causal arguments (Murray, et al., 2003). However, models rely on the strong correlation of the mortality at different ages (Wilmoth, et al., 2012). This correlation emerges from the influence of the environment and the socioeconomic background on the risk of dying of all individuals at a particular time, regardless of their age and sex (Preston, 1972). When several populations at different times in history are put together, correlations become more evident. Taking advantage of this regularity, models estimate the mortality of a wide range of ages as a function of a single index, inasmuch as it predicts intrinsic characteristics such as the shape and level of mortality. Since models produce the hypothetical mortality schedule that would be inferred from a set of real populations (Murray, et al., 2003), empirical models are as consistent as the life tables used to estimate them.

Back in 1955, the Population Branch of the United Nations Department of Social Affairs proposed a method to estimate the life expectancy and mortality rates for countries with unreliable or nonexistent vital statistics. The method was called *Model Life Tables for Under-developed Countries*, and the main objective was to infer a mortality schedule for all ages using the infant mortality rate as a single entry parameter. The infant mortality was used as the index, inasmuch as it exhibited a wide dispersion. Besides the statistical argument, two additional characteristics were considered (United Nations, 1955). On the one hand, infant mortality has been much more sensitive to changes in the socioeconomic background. On the other hand, infant mortality has been approached through surveys in countries lacking vital records, thus it would be a convenient entry value for indirect

estimation (United Nations, 1955; Brass, 1996). The UN model was estimated applying Ordinary Least Squares to a set of 158 empirical life tables. The model assumes a quadratic relation between the probability of dying at a particular age interval and that of the previous interval. Life tables were chosen in favor of the geographic and time dispersion, although excluding years of wars and epidemics. Criticism and improvements did not wait so long. One of the issues of the UN model is the lack of accuracy in estimating other values of a life table (those that were not used for the estimation), since the model overestimated the life expectancy at birth by more than two years of life (Gabriel & Ronen, 1958). Inasmuch as some life tables were not reliable, the main concern with the UN model was the prediction of probabilities of dying using already predicted values, something that might introduce systematic bias (Coale & Trussell, 1996). As a second approach, Ledermann & Breas (1959) estimated a model life table using principal components and virtually the same database of the UN model. The three components of the model proposed by Ledermann & Breas explained the 93% of the variation in mortality rates at all ages, but an additional fourth component would had helped to explain the variation in infant mortality rates (Hogan, 1976). However, the fourth component was not included in the main model.

Although the estimation based on factor analysis and principal components offers quite an interesting approach, the original idea of one single predictor and a quadratic specification has prevailed in the demographic estimation. In this regard, Coale & Demeny (1966) estimated the first version of their famous *Regional Model Life Tables and Stable Populations*, using as the index the life expectancy at the age of 10 years. One of the most important improvements introduced by Coale & Demeny (1966) was a collection of 326 empirical life tables estimated for countries with censuses and vital statistics. The clearest innovation was the analysis of predefined mortality families: *North*, *South*, *West*, and *East* models, representing different epidemiological patterns and leading causes of death. The family or region, and the level of mortality are the two entry parameters in this approach. Each family reproduces a particular composition of the mortality at early ages and the mortality at adult ages. The combination of region and life expectancy at age 10 allowed the model to represent a variety of mortality patterns that might illuminate the assessment of the mortality schedules of countries with nonexistent or unreliable data. This model was reformulated in 1989 in order to improve the fitting of lower mortality schedules and to provide a revisited estimation of mortality at older ages (Coale & Guo, 1989). Following the approach of modeling the level and the shape of mortality, Wilmoth et al. (2012) propose an empirical model of two entry parameters fitted to 719 life tables covering a broad set of mortality patterns with more geographical disperssion and a broad number of years. The first entry parameter is the probability of dying within the first five years of life (henceforth referred to as the under-five mortality), while the second is an adjustable value shaping the excess of mortality at adult

ages. The clear innovation of Wilmoth's model is regarding the indirect estimation, since the shape of the mortality is tailored to the particular characteristics of the population to be studied.

Despite the great strengths of previous models to describe the mortality for a complete lifespan, there are three limitations in the study of the age patterns of infant and child mortality using these models. The first limitation is regarding the composition of ages. In conventional models, age patterns of infant and child mortality are typically represented using two groups: the *infant mortality* which is occurring within the first year of life, and the *child mortality* ranging from 1 to 5 years. Since these are broad intervals, conventional models investigate mortality patterns neither at infant ages nor during the childhood. It is well known that the number of deaths of a birth cohort descends rapidly as the age of the infant increases. Nevertheless, the fall in the mortality force does not always occur at the same rate and intensity. The second limitation is the broad or general adjustment to all ages. Although infant and child mortality play a leading character in predicting the mortality at adult ages, less attention has received the problem to make predictions at early ages using limited information. Thus, country and time variations within the first months and years of life were not investigated as an independent problem. Perhaps, one exception was the *Age Patterns of Infant Mortality* estimated by Hogan (1976) using the method of principal components. However, the first 12 months of life are not wide enough time to examine all changes in the risk of death occurring at early ages; hence one of the problems of Hogan's approach is the exclusion of the child mortality. The third limitation is the futility to apply the analytical or mathematical approach. Even though mortality is typically high at both extremes of the life span, the force of mortality during the first weeks and months of life does not fit a law of mortality that can be deduced from a single equation. Although the Gompertz Law of Mortality applies with some reliability to the ages 30+ (Coale & Demeny, 1966), the mathematical approximation proposed by Bourgeois-Pichat only describes porbabilities of dying at postneonatal ages and is more suitable for historical populations (Galley & Woods, 1999; London, A, 1993).

This paper estimates a model life table for under-five mortality using vital records from historical and contemporaneous populations. The model is able to reproduce mortality schedules of an under-five population by detailed subintervals of age: days in the first week of life, weeks to complete the first month, months at postneonatal ages, trimesters in the second year, and single years of age to complete childhood. A database was constructed from the empirical life tables of 24 countries; some of them dated at the end of the nineteenth century and most of the twentieth century. This period is of major relevance since it covers the great decline in infant mortality: an epoch of economic progress, medical advances, and social transformations. In the same sense, the

heterogeneity of countries included constitutes a valuable aspect of the model, since socioeconomic background and environmental conditioning may produce different mortality patterns even if the overall mortality level would be the same. The model was estimated using different mortality indexes and a variety of quantitative methods. This comparative approach allows identifying a group of input parameters that maximize the predictive power of the model. However, the model is open to estimate a mortality schedule using a set of entry values (up to four parameters) predefined by the researcher. This makes the model an indispensable tool in the demographic analysis since it allows indirect estimations based on limited information.

1.2. Background

The relevance of the mortality pattern at early ages

The nutritional status, the immune status, and the exposure to infections are proximate factors of children's health. These factors interact with each other during the first years of life producing changes in the probabilities of dying (Garenne, 1982). Passive immunity is inherited from the mother and is often improved by breastfeeding. When breast milk ceases its contribution to the immune status of infants, they depend on their autonomous immunity. However, the weaning by itself increases the exposition to contaminated food. Hence, the transition to a solid diet is a period of nutritional stress that is not observed in other ages (Jelliffe & Jellife, 1979). However, breastfeeding also enhances birth intervals and increases the chances of survival in consequence (Huffman & Lamphere, 1984). In poor economic environments, prolonged birth intervals reduce the probability of dying as infants receive exclusive care and feed from their mothers (Palloni & Millman, 1986); and exclusive breastfeeding in the first semester of life reduces the risk of morbidity and mortality due to diarrheal diseases (Lamberti, et al., 2011). In historic populations, breastfeeding reduced the mortality in the first semester of life, although infants who were artificially fed had a lower mortality in the following six months of life (Knodel & Kintner, 1977). In modern or high-income populations vaccination and nutritive formulas are available, thus the benefits of breastfeeding are lower. However, the long-term effects of breastfeeding are associated with the intelligence performance and the prevention of infections and diseases related to inadequate diets such as diabetes and overweight (Victora, et al., 2016). In light of the foregoing, the probability of survival depends on the nutritional and immune status to resist infections.

Nutritional stress can also occur during gestation. Low birth weight is associated with neonatal mortality (McCormick, 1985; The Lancet, 1988; Victora, et al., 1988), and the probabilities of survival increase as newborns gain weight (Bourgeois-Pichat, 1950). Regardless of height and body mass index of the mother, low birth weight is associated

with low protein intake at the end of pregnancy and high carbohydrate intake at the beginning (Godfrey, et al., 1996). In seasons of food scarcity and through situations of extreme poverty, food supplements for pregnant mothers increase birth weight and reduce early neonatal mortality (Prentice , et al., 1987; Ceesay, et al., 1997). Furthermore, nutritional stress during gestation and the first months of life is also associated with a lower probability of survival after puberty (Moore, et al., 1997; Moore, et al., 1999), and a higher prevalence of degenerative diseases in adult ages (Barker, 1990; Roseboom, et al., 2001). From this perspective, nutrition during the perinatal period has an impact on the health status at all ages.

The significance of the mortality decline at early ages

In the second half of the nineteenth century, an unprecedented decline in mortality at early ages is observed in most developed countries. Economic wellbeing increased and population had access to more nutritious diets (Beaver, 1973; McKeown & Record, 1962; McKeown, 1983). However, social transformations and technological innovations were the leading forces of change in reducing the exposure to the risk of dying. In France, the first regulations that sought to protect the lives of infants were promulgated in the 1870s and the practice of baby farming was abolished because of the excess of mortality (Rollet, 1997).

The nineteenth century culminated with significant advances in the field of bacteriology, such as the discovery of the bacillus of tuberculosis and the cholera microorganism in the decade of 1880s, and antitoxins to treat diphtheria in the decade of 1890s (Preston & Haines, 1991). Pasteurized milk and other nutritional substitutes could have some effect in reducing the burden of infant deaths (Beaver, 1973). In England, the first national conference on infant mortality took place in 1906 and discussed the fact that many of the causes of death were preventable (Dyhouse, 1978). In the United States, the beginning of the twentieth century was the onset of the formal medical knowledge of obstetrics and pediatrics (Preston & Haines, 1991). Medical developments such as vaccines, introduced in the late nineteenth century, and antibiotics, since the mid-twentieth century, would be the result of a better understanding of the causes and treatment of some diseases (Easterlin, 2004; Preston & Haines, 1991).

In addition to innovations in the practice of medicine, public health transformed the cities. Clean water, better sewage disposal, paved streets, and other sanitary measures were effective actions to prevent the spread of some diseases (Easterlin, 2004). Moreover, filtration and chlorination of water would be a leading innovation behind the decline of the infant mortality rates in the early twentieth century for cities in the United States (Cutler & Miller, 2005). Before these transformations occurred, the most effective ways to protect the lives of the infants and children were to be very vigilant of their

feeding, and to send them to the rural areas during the warmest months of the year in order to prevent infections.

1.3. Data

Primary sources

A total of 1,319 life tables were calculated from the vital records of 24 countries. These were mostly European countries: Austria, Belgium, Denmark, England and Wales, Finland, France, Germany, Hungary, Ireland, Italy, Netherlands, Norway, Poland, Portugal, Spain, Sweden, and Switzerland; in addition to: Australia, Canada, Chile, Israel, Japan, New Zealand, and the United States. This is the group of countries of the Human Mortality Database (HMD), nevertheless excluding Iceland and Luxemburg because of the small population, and states from the former Soviet Union.

Life tables were calculated after the consolidation of two sources of information: *(1)* Census enumerations, estimated population for intercensal years, and deaths (by single years of age) were taken from the HMD. *(2)* The distribution of deaths within the first year of life was extracted from the United Nations repository of vital statistics (henceforth referred to as the UN database). With a few exceptions, the standard format of the UN database enumerates deaths by days during the first week of age, weeks during the first month (28 days of life), and then months at postneonatal ages. These two sources alone allow calculating a total 732 life tables. However, the UN database includes information after 1970 and given the selection of countries, it describes a context of low mortality. Therefore, a total 587 additional tables were calculated directly from the demographic yearbooks of selected countries before 1970 (henceforth referred to as the historical database). Country-years included in the analysis are described in Table 1.

The same sources of the HMD were investigated, yet were only considered those country-years reporting deaths by sex and subintervals of age with similar characteristics. Considering that the historical sources report some figures collected at the end of the nineteenth century, the addition of historical data enriches the model so it is able to reproduce the mortality pattern of high infant and child mortality. Additional life tables have two advantages. In the first place, they cover the historical period of the infant mortality decline. In the second place, historical data provide more detailed information of the distribution of deaths at early ages. In some cases (Belgium, Netherlands, Norway, and Sweden), historical data also include the number of deaths within the second year of life by trimesters of age. These additional life tables were calculated using the same methods applied to the UN life tables.

Table 1: Country-years included in the analysis

Country	Years	Historical	UN database
Australia	1970-71, 1973-2009	-	39
Austria	1970-2010	-	40
Belgium[1]	**1841-60**, **1861-70*(average)***, **1878-84**, 1970, 1972-83, 1986-87, 1992, 2007-09	28	19
Canada	1970-75, 1977-86, 1988-90, 1992, 1995-97, 1999-2006	-	31
Chile	1992-2005	-	14
Denmark[2]	**1890-94**, **1896-69**, 1970-93, 1997, 2000-10	79	36
England and Wales[3]	**1905-45**, 1970-1985	41	16
Finland[4]	**1881-85*(a)***, **1886-90*(a)***, **1891-95*(a)***, **1896-1900*(a)***, **1901-27**, **1928-29*(a)***, **1930-36**, **1937-39*(a)***, **1948**, 1970-90, 1994, 1996-98, 2000-06, 2008-09	41	35
France[5]	**1885-88**, **1893-19**, **1926-29**, **1931**, **1933**, **1935-36**, **1942**, **1947**, 1970-72, 1974-92, 1994-99, 2001-09	41	37
Germany	1991-94, 1996-97, 2001-07, 2010	-	14
Federal Republic[6]	**1956-65**, 1970-90	10	21
Democratic Republic[7]	**1956-89**	34	-
Hungary	1970-94, 1996-09	-	39
Ireland	1970-88, 1990-99, 2001-06, 2008	-	36
Israel	1983-98, 2000-09	-	26
Italy	1970-72, 1974-85, 1988-94, 2001-08	-	30
Japan[8]	**1947-50**, **1954-56**, **1958-60**, **1962-64**, 1970-94, 1996-2000, 2002-09	13	38
Netherlands[9]	**1850-64**, 1970-94, 1996, 1998, 2000-01, 2004-08	15	34
Norway[10]	**1876-1900**, **1901-05*(a)***, **1906-26**, **1927-30*(a)***, **1931-75**, 1976-1992, 1995-2001, 2003-09	93	31
New Zealand	1970-75, 1977-2008	-	38
Poland	1970-99, 2001-09	-	39
Portugal[11]	**1940**, **1942-59**, **1962**, 1970-93, 1996-97, 2001-09	20	35
Spain	1976-83, 1987-91, 1995-98, 2000-09	-	26
Sweden[12]	**1891-2001**, 2002, 2004-2010	111	8
Switzerland	1970-82, 1984-94, 1996, 1998-2010	-	38
United States[13]	**1933-1993**, 1995-2003, 2007-09	61	12
Total	-	587	732

[1]Ministre de l'Intérieur, *Statistique Générale de la Belgiqu*e - *Exposé de la Situation du Royaume* 1841, 1851, 1861, Bruxelles: TH. Lesigne. *Annuaire Statistique de la Belgique* 1879-90, Bruxelles: Imprimerie Félix Callewaert Père – Imprimerie Veuve Monnon – Imprimerie & Lithographie Ad. Mertens.
[2] Danmarks Statistik, *Statistisk Tabelværk - Vielser, Fødte og Døde* 1890, 1895, 1901, 1906, 1911, 1916, 1921, 1926, 1931, 1941, 1956, København (Copenhague): Statens Statistiske Bureau.
[3] The Registrar General, *Annual Report, Births, Deaths and Marriages* 1905-1919, *Annual Report* 1920, *Statistical Review (Text)* 1921-38, *Statistical Review (Medical)* 1930-37, 1940, London: His Majesty's Stationery Office.

The sample of country-years included in the analysis is not random in the sense that were selected those cases reporting deaths by sex and subintervals of age within the first year of life. In addition, there were selected the cases with consistent values comparing the cumulative number of deaths, by years of life, reported on the UN database with the effective number of deaths informed on the HMD for each single age. In other words, data collected from the UN repository were validated using information from the HMD as a standard of completeness. Since both datasets, the historical and UN database, were reported by calendar years, life tables are considered period data. Thus, mortality rates and empirical life tables were calculated using the assumption of synthetic cohort. Moreover, it is assumed that the empirical life tables used in this paper are of good quality as they permit the direct estimation of the age-specific mortality rates within neonatal and postneonatal ages. Therefore, mortality patterns calculated from reliable

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[4] Statistiska Centralbyrån, *Statistisk Årsbok för Finland* 1904-08, 1911-14, 1921, 1923, 1925, Helsingfors (Helsinki): Kejserliga Senatens Tryckeri – Statsrådets Tryckeri. Tilastollisen Päätoimiston, *Suomen Tilastollinen Vuosikirja* 1922, 1924, 1926-29, 1932-37, 1941-44, 1950, Helsinki: Valtioneuvoston Kirjapaino – Statistiska Centralbyrån.

[5] Service de la Statistique Générale de France, *Annuaire Statistique de la France* 1888-89, Nancy: Imprimerie Berger-Levrault et Cie. 1890-91, Paris: Imprimerie Nationale. Bureau de la Statistique Générale, *Annuaire Statistique de la France* 1898, *Annuaire Statistique* 1900-04. Statistique Générale de la France, *Annuaire Statistique* 1905-07, 1911-12, 1928-35. Direction de la Statistique Générale et de la Documentation, *Annuaire Statistique* 1936-39. Institut National de la Statistique et des Études Économiques, *Annuaire Statistique* 1940-45, *Statistique du Mouvement de la Population* 1946-47, Paris: Imprimerie Nationale.

[6] Statistisches Bundesamt (Wiesbaden), *Statistisches Jahrbuch für die Bundesrepublik Deutschland* 1952-88, Stuttgart – Köln – Mainz: W. Kohlhammer GmbH.

[7] Staatlichen Zentralverwaltung für Statistik, *Statistisches Jahrbuch der Deutschen Demokratischen Republik* 1962, 1967-89, Berlin: Van Deutscher Zentralverlag – Staatsverlag der Deutschen Demokratischen Republik.

[8] Statistics Bureau of the Prime Minister's Office, *Japan Statistical Yearbook* 1950-52, 1957-58, 1961, 1965, 1967, Tokyo: Nihon Statistical Association.

[9] Departement van Binnenlandsche Zaken, *Statistisch jaarboekje voor het koningrijk der Nederlanden* 1851-55, 1857-60, 1863, 1865, 1867-68, ś-Gravenhage (The Hague): Algemeene Landsdrukkerij – Van Weelden en Mingelen.

[10] Det statistiske Centralbureau, *Folkemængdens Bevægelse* 1876-1975, Kristiania (Oslo): Norges Officielle Statistik.

[11] Instituto Nacional de Estadistica, *Annuario Demografico* 1940-62, Lisboa: Instituto Nacional de Estadistica.

[12] Sveriges Officiella Statistik, *a) Befolkningsstatistik* 1891-1910, *Befolkningsrörelsen* 1911-60, *Folkmängdens Förändringar* 1961-66, *Befolknings Förändringar* 1967-90, *Befolkningsstatistik*, 1991-2001, Stockholm: Statistiska Centralbyråns.

[13] US Department of Commerce – Bureau of Census, *Birth, Stillbirth, and Infant Mortality Statistics* 1931-36, *Vital Statistics of the United States (Natality and Mortality Data)* 1937-44, Federal Security Agency – United States Public Health Service, *Vital Statistics of the United States (Natality and Mortality Data)* 1945-49. US Department of Health, Education, and Welfare – Public Health Service, *Vital Statistics of the United States (Marriage, Divorce, Natality, Fetal Mortality, and Infant Mortality Data)* 1950-59, *(Mortality)* 1960-75. UD Department of Health and Human Services – Public Health Service, *Vital Statistics of the United States(Mortality)* 1976-93, Washington, DC – Rockville and Hyattsville, MD: United States Government Printing Office.

sources are not affected by errors introduced by procedures used to collect and to report mortality data. In absence of data problems, changes in the mortality schedule would respond to subjacent epidemiological patterns.

Age intervals

The analysis of data from historical sources is desirable, but has one clear limitation: tabulations of deaths at premature ages were reported using heterogeneous formats. The UN data is consistently reported by days and weeks within the first month of age, and months at postneonatal ages. However, some specifics deserve attention. Swedish yearbooks reported neonatal deaths by days of age, postneonatal deaths by months of age, and second-year deaths by trimesters of age. However, second-year deaths were no longer detailed since 1968. Norwegian yearbooks have a similar format, but neonatal deaths were reported by days for the firsts two weeks of life, and then grouped to complete the first month of life (1876-1935). Since 1936, deaths occurring during the third and fourth week of life were reported individually. This is the same format of Finnish yearbooks, although second-year deaths were not tabulated in detail.

Yearbooks of England and Wales reported neonatal deaths by weeks of age, and postneonatal deaths by months of age. Since 1906, deaths occurring the first day of life were tabulated individually. More details were introduced in 1931, when deaths occurring in the first week were reported by days of age and the report also included the number of deaths occurring in the first hour of life. However, postnatal deaths were grouped in trimesters of age since 1926. Demographic yearbooks of the United States have a similar format. Neonatal deaths were tabulated by days of age only for the first week, and then by weeks of age to complete the first month. However, postneonatal deaths were consistently reported by months of age. The number of deaths occurring in the first hour of life was reported since 1952.

In some cases, fewer details were reported regarding the neonatal deaths and the postneonatal deaths were not tabulated by months of age. Statistical yearbooks of the Federal Republic of Germany reported neonatal deaths by days of age for the first two weeks of life, and then by weeks of age. Postneonatal deaths were reported in months of life (1956-1965). However, postneonatal deaths were no longer detailed since 1966 and most of the details at neonatal ages discontinued in 1970. Thus, the database was truncated in 1965 and the period 1970-1990 relies on data from the UN repository. Statistical yearbooks of the Democratic Republic of Germany reported neonatal deaths at 0-3, 4-10, and 11 or more days of age, while postneonatal deaths were tabulated by months of age (1956-1967). Although postneonatal deaths continued to be reported in months of age, no more details were provided for neonatal deaths (1968-1989).

Statistical yearbooks of the Netherlands reported infant deaths by months of age, and second-year deaths by trimesters of age (1850-1864). Thus, no details were provided for the distribution of neonatal deaths. This is the same format of the statistical yearbooks of Belgium (1841-1870). However, more details were provided when neonatal deaths were reported by groups of five days and by individual days for the first five (1878-1884).

The only detail of Danish yearbooks at neonatal ages is in the number of deaths in the first day of life, with the exception of the period 1896-1900 when no details were provided. Postneonatal deaths were reported by months of age for the second and third month of life, and then by trimesters of age. However, more details were later introduced. Postneonatal deaths were reported by months of age since 1910, and neonatal deaths included tabulations for the first day and the first week of life since 1921. Similarly, statistical yearbooks of Portugal reported deaths occurring in the first trimester of life by months of age, and then by trimesters of age to complete the first year of life (1940-1954). Since 1955, neonatal deaths were reported by days of age for the first week, and weeks to complete the first month of life; while postneonatal deaths were tabulated by months of age.

French yearbooks reported neonatal deaths at 0-7, 8-14, and 15 or more days of age. Postneonatal deaths were reported grouping the second and the third month of life, and tabulating the deaths occurring the second trimester and the second semester of life (1885-1887). This format changed from 1888, when neonatal deaths were reported at 0-4, 5-9, 10-14 and 15 or more days of life; and the deaths occurring during the second and third month of life were tabulated independently. Likewise, postneonatal deaths were tabulated by months of age since 1947. Statistical yearbooks of Japan have a similar format. Neonatal deaths were reported at 0-5, 6-10, 11-15, and 16 or more days. Postneonatal deaths were tabulated for the second and the third months of life, as well as the total number of deaths occurring in the second trimester, and the second semester of life (1947-1950). This format changed and neonatal deaths were reported by weeks of age (1954-1959). However, in 1960, 1962, and 1964 no further details were given on the distribution of neonatal deaths.

Given the characteristics of the sources of data, there were imposed the following age intervals in the model life table: *(1*) days within the first week; *(2*) weeks to complete the first month of life of 28 days longer; *(3)* months of age to complete the first year of life, with a second month unusually longer in order to include deaths occurring after the 28^{th} day of life; *(4)* trimesters of age within the second year of life; and *(5)* years to complete the first five years of life. Yet, some country-years do not fit the age intervals described; the problem of irregular formats is faced under the assumption of a constant force of mortality at each possible age interval. Although this assumption is reasonable when age

intervals are not longer than a year, the assumption is unsuitable at early ages, ever since the most detailed data show a rapid decline of the mortality force at neonatal and postneonatal ages. In this regard, specific observations lacking a direct report by days of life within the first week or by weeks during the first month were excluded from the estimations.

1.4. Methods

Mortality rates and cumulative probabilities of dying

Life tables were calculated directly from death registrations. Usually, this information is tabulated by calendar years; thus, life tables were computed as period estimates. From this perspective, data describe the mortality experience of a particular year (a synthetic cohort), instead of an actual cohort. Compared to cohort life tables, period life tables can be calculated with no concern about migration. Life tables were calculated from age-specific mortality rates; for each sex individually. Mortality rates were estimated from the number of deaths at each age interval, and the population at risk of dying, as shown in the following equation:

$$ {}_{n}m_{x}\ [t, t+1) = \frac{{}_{n}D_{x}\ [t,t+1)}{{}_{n}N_{x}\ [t,t+1)}. \qquad (1) $$

Using a conventional notation in demography, equation (1) shows that a mortality rate at the age interval $[n, n+x)$ is equal to the number of deaths, of those individuals whose age at death was at the age interval; divided by the population at risk of mortality at the same age interval. While the number of deaths was extracted from vital statistics, the population at risk of dying was estimated from the number of people at the same age. Demographic yearbooks tabulate mortality data at early ages by subintervals, such as days, weeks, and months of age, but the population is usually tabulated by years of age. Therefore, the exposure to the risk of mortality was assumed to be proportional to the length of the age interval n, as shown in equation (2), for the population less than 1 year-old.

$$ {}_{n}N_{x}\ [t, t+1) = n \cdot\ {}_{1}N_{0}\ [t, t+1), \qquad 0 < x + n \leq 1. \qquad (2) $$

Under the assumption of proportionality, the exposure to the risk of mortality was calculated for all infants bellow one year of age; and then, it was distributed according to the convenient age intervals by days, weeks, and months. The same approach was used for the second year of life, using population older than one year but younger than two years of age, ${}_{1}N_{1}\ [t, t+1)$. In this case, the risk of mortality was scattered by trimesters of age for those selected countries-years that reported death distributions at the second

year of life. For each country-year in the sample, the exposure was estimated from the population reported by single years of age under the assumption of linear growth, and using the mean of the initial and final population sizes, as shown in equation (3):

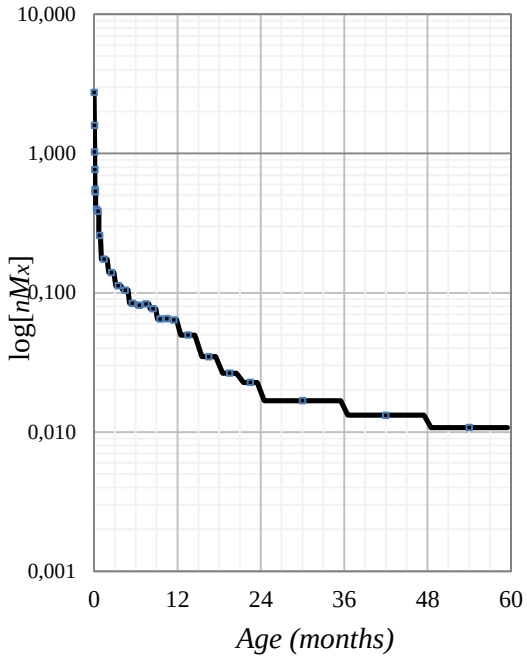
$$ {}_1N_y\ [t, t+1] = \frac{1}{2} \cdot \left[\ {}_1N_y\ (t+1) + \ {}_1N_y\ (t)\right], \qquad y = 0, 1, \ldots, 4. \tag{3} $$

Figure 1 shows the age-specific mortality rates for males, calculated from equation (1), from a selection of country-years. The flat segments represent the assumption of constant mortality rates at each age interval. Once the mortality rates were calculated, the next step was to estimate the cumulative probability of dying using the equation (4):

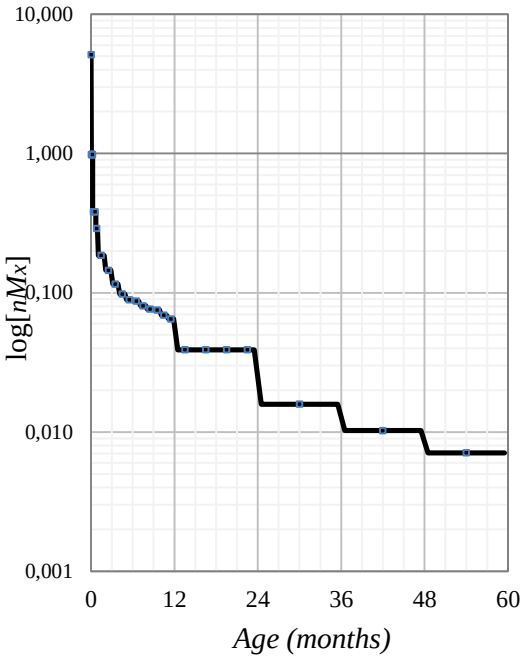
$$ q(x) = 1 - e^{-\int_0^x m(y)dy}. \tag{4} $$

For each life table, a total of 28 values were calculated given the age intervals used in the model. At neonatal ages, 10 cumulative probabilities of dying were calculated for the first seven days of life, and then by weeks to complete the first 28 days of life. At postneonatal ages, 11 values were calculated for each month to complete the first year of life. At the second year of life, 4 values were calculated by trimesters of age, and then 3 additional values to complete the fifth year of live by single years of age. These cumulative probabilities of dying were the input of the model life table. Figure 2 plots the association of the cumulative probabilities of dying at different ages using the sources described in Table 1. In particular, the model exploits the fact that they are strongly correlated at different ages.

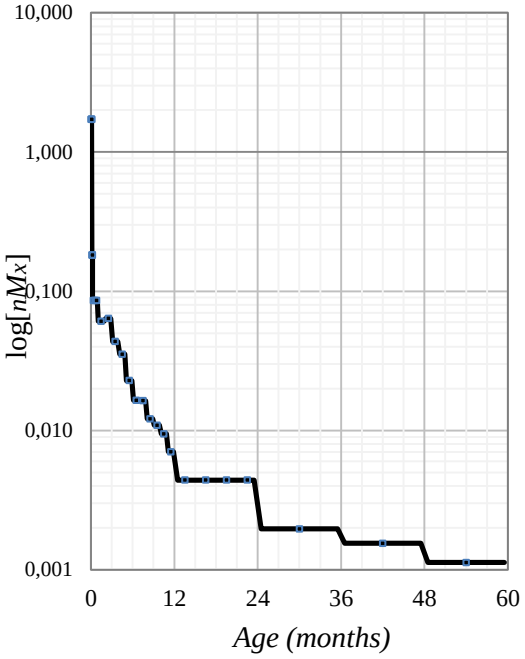
Figure 1: Age-specific mortality rates for males from selected country-years
Male



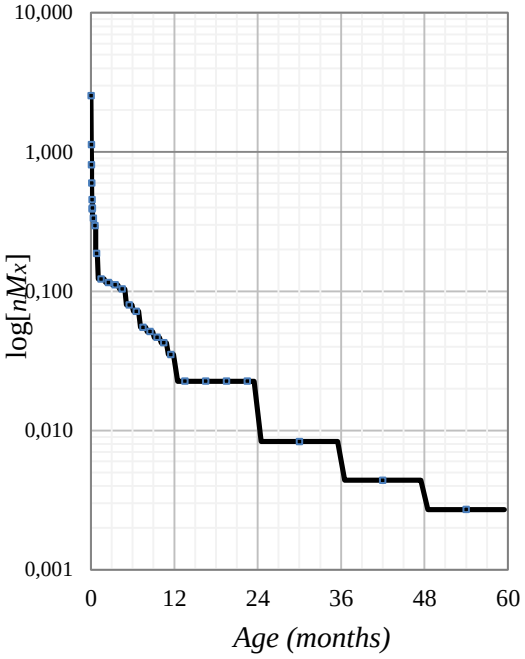
Sweden, 1899



England & Wales, 1907

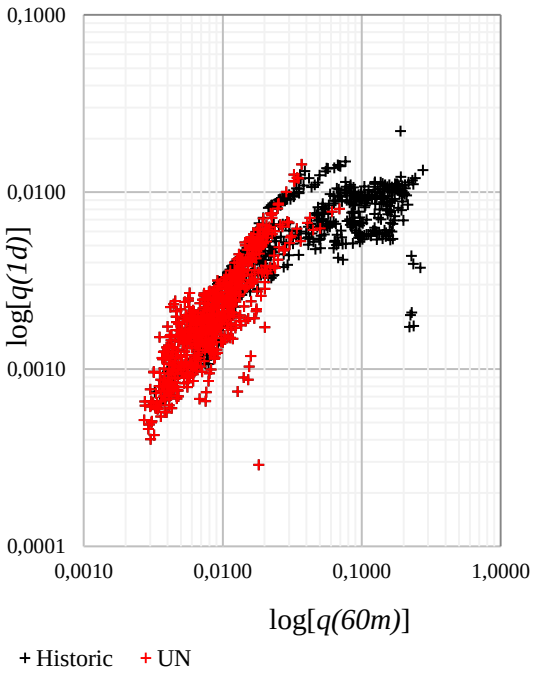


Democratic Republic of Germany, 1957

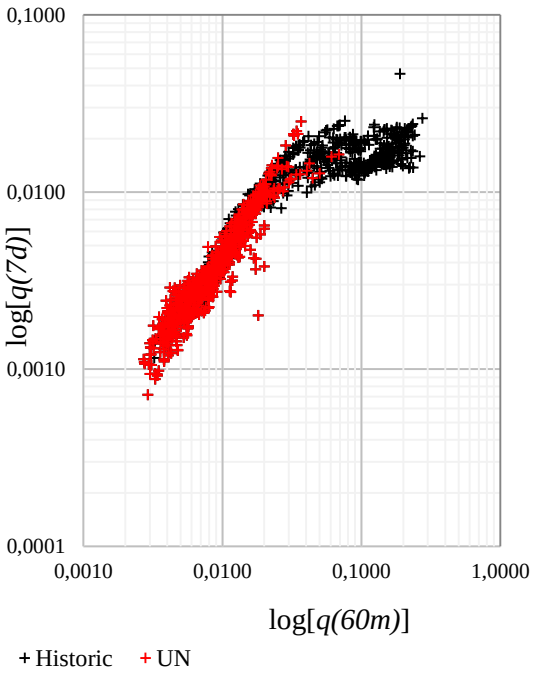


Portugal, 1959

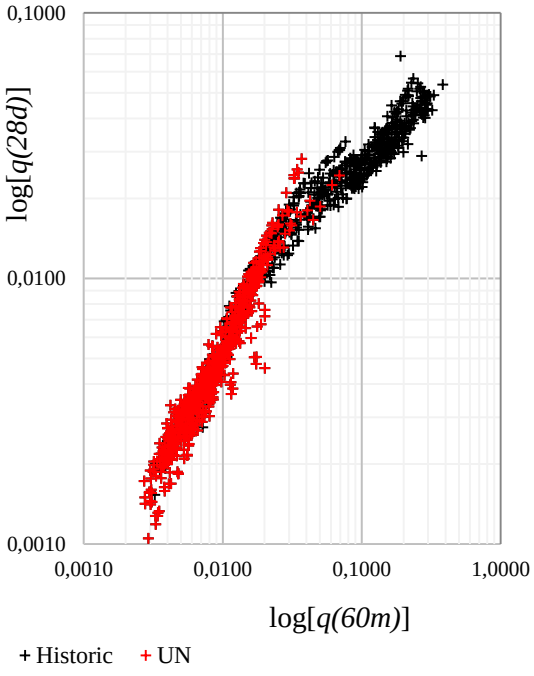
Figure 2: Cumulative probabilities of dying $q(x)$ vs. under-five mortality $q(60m)$
Female



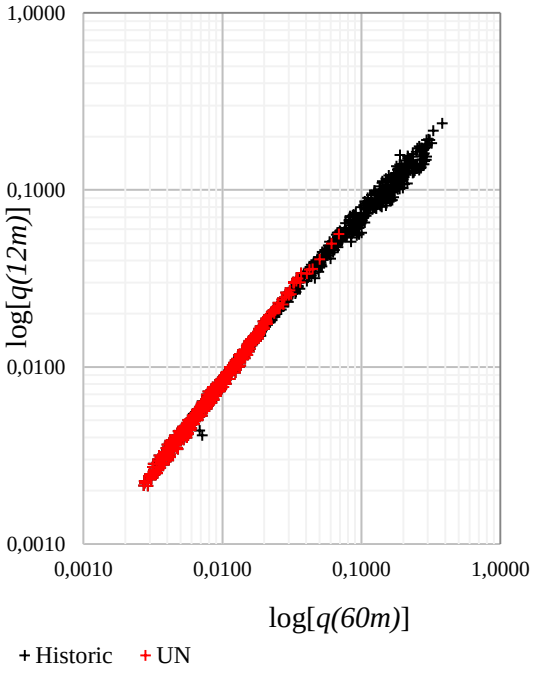
1 day *vs*. 60 months of life



7 days *vs*. 60 months of life



28 days *vs*. 60 months of life



12 months *vs*. 60 months of life

The level of mortality

The model life table was estimated from a set of equations using one equation per age, and exactly the same predictor for all equations. This is, in summary, the main intuition of the empirical model: to use the same predictor at different ages and to exploit the strong correlation of the probabilities of dying at different ages. Cumulative probabilities of dying at all ages were used as the response variable, and the explanatory variable was defined as the probability of dying that best predicts the cumulative probabilities of dying at all other ages. Hence, the model produces a hypothetical mortality schedule given one single entry value. Inasmuch as the explanatory variable is related to the mortality at all ages, it works as an index of the level of mortality.

Following a matrix notation, a column vector y was defined, consisting of cumulative probabilities of dying at different ages within the first five years of life. For convenience, this vector is sorted by age, and then by country and year. Therefore, the probabilities of dying at earlier ages are located at the top of the vector, and the probabilities of dying at older ages are located at the bottom:
$y = [q(1d)', \quad \cdots \quad q(28d)', q(2m)', \quad \cdots \quad q(60m)']'$. Hence, $q(28d)$ is a column vector comprising the probability of dying within the first month of age for each country-year included in the analysis, and for construction all vectors $q(\quad)$ are sorted equally. Since the model was estimated from 1,319 empirical life tables, the length of each $q(\quad)$ vector is 1,319. Similarly, given that the model includes of 28 age intervals, then the length of the response variable is $28 \times 1{,}319$.

Although the dependent variable varies by age, country, and year, independent variables must be the same for all ages. Then, the right hand of the equation can be defined as: $I_r \otimes Z$; where I_r is an identity matrix of dimension $r = 28$, which is the number of ages in the model; Z is a matrix of explanatory variables depending on the level of mortality; and $\otimes$ is the Kroeneker product. Following a conventional approach in model life tables, the dependent variable is assumed to be a log-quadratic function of the level of mortality. Hence, the right hand of the equation is defined as: $Z = [\iota \quad ln(\mathrm{H}) \quad ln(\mathrm{H})^2]$; where ι is a vector of ones that will measure the constant term for each equation; and H is a vector of mortality levels. Thus, each element of the vector H represents the level of a particular country-year in the sample. Given the above description, the model life table model was the result of estimating equation (5).

$$ln(y) = [I_r \otimes Z] \cdot \beta + \epsilon. \qquad (5)$$

Considering that this is a quadratic model with 28 age intervals, the vector of coefficients β has 84 elements to be estimated. For a given level of mortality h, the coefficients of the model predict the cumulative probabilities of dying at different ages. Separate models

were estimated for men and women, using different explanatory variables and various quantitative methods. If equation (5) were to be estimated by Ordinary Least Squares (OLS), the vector of coefficients is consistently estimated using the equation: $\beta_{OLS} = [I_r \otimes (Z'Z)^{-1}] \cdot [I_r \otimes Z'] \cdot ln(y)$. Since the dependent variable is a natural logarithm, the model minimizes the relative error incurred in trying to predict cumulative probabilities of dying.

Although the OLS provide the best linear unbiased estimator, the OLS estimation could be improved in multiple ways. In the second method, weights were assigned to each observation to attenuate the effect of outliers in the model, using the equation: $\beta_w = [(I_r \otimes Z') \cdot w \cdot (I_r \otimes Z)]^{-1} \cdot [I_r \otimes Z'] \cdot w \cdot ln(y)$. Where w is a diagonal matrix that takes continuous values between 0 and 1 that were iteratively calculated by the Bi-weight regression (Beaton & Tukey, 1974), using a scaling constant equal to 6. The third method is the same as the above but taking discrete values of 0 or 1, so that the extreme values were simply excluded from the estimation. For a particular age, as extreme observations were considered those with absolute errors equal or greater than six times the median residual. Once the model was estimated, the vector of coefficients β was conveniently reshaped into a matrix of dimension 28×3. Thus, the model life table q^m would result from multiplying the coefficient matrix $\hat{\beta}_{28\times3}$ by a column vector containing information for the only input parameter h, using the equation (6):

$$ln(q^m) = \hat{\beta}_{28\times3} \cdot [1, ln(h), ln(h)^2]'. \quad (6)$$

Where $q^m = [q(1d|h), \quad \cdots \quad q(28d|h), q(2m|h), \quad \cdots \quad q(60m|h)]'$ is a column vector containing 28 cumulative probabilities of dying that can be predicted to a given level of mortality h. Age-specific mortality rates can be recovered from the model by decumulating the predicted values of equation (6), under the assumption that the force of mortality is constant at each age interval.

One relevant aspect of this model is the capacity to handle demographic restrictions. Although the statistical fitting is achieved for a given level of mortality h, and this probability would be the preferred entry value; the model can also produce a life table matching a probability of dying at any other ages ${}_nq_x$. In this case, it is possible to find the optimal value of $h^* > 0$, solving the equation (7) by numerical methods:

$$\frac{q(x+n|h^*) - q(x|h^*)}{1 - q(x|h^*)} = {}_nq_x \, . \quad (7)$$

So far, the model meets the criteria of being adjusted from a statistical perspective, for a given value of h. However, an important limitation is that it will always reproduce the same mortality pattern that would result from averaging all life tables used for the

estimation. There is a trade-off between specificity and simplicity. For the model to be able to represent more particular patterns, it is necessary to increase the number of parameters. However, the application can be useful because it allows the researcher to incorporate more relevant information when estimating a model life table.

The shape of the mortality schedule

The model was extended to include up to three additional entry values. These values can be adjusted in order to reproduce some particular characteristics of a population when there are more values to be fitted. Additional parameters resulted from extracting information contained in the covariance of residuals at different ages, following the same approach of Wilmoth, et al., (2012). The intuition is very simple: once the model is estimated using a predictor h, the error term has everything that is unrelated to the predictor and perhaps the level of mortality. Thus, if some information is relevant to the shape of mortality that is orthogonal (or independent) to the level of mortality, that information would be in the error term. The covariance of errors at different ages allows an approximation to age patterns that systematically deviate from the general model. Thus, the mortality pattern is extracted from the covariance matrix of the residuals.

Given that the error term of estimating equation (5) is a column vector containing the errors for all equations: $\epsilon = [\epsilon(1d)', \quad \cdots \quad \epsilon(28d)', \epsilon(2m)', \quad \cdots \quad \epsilon(60m)']'$; it can be conveniently reshaped into a matrix of dimension $1{,}319 \times 28$, having as many rows as life tables used for the estimation, and as many columns as ages in the model. This matrix was used to estimate the variance and covariance of the errors at different ages, and a singular value decomposition was made. The aim of the decomposition is to reinterpret the array of variances and covariances from an orthonormal matrix U, and a diagonal matrix Σ consisting of the eigenvalues, given that: $\mathrm{U} \cdot \Sigma \cdot \mathrm{U}' = \epsilon' \cdot \epsilon/(N-k)$, and: $\mathrm{U} \cdot \mathrm{U}' = I$. The decomposition allows extracting a set of orthonormal vectors from the matrix U, that can be used as additional information in the model life table. Each vector would adjust the cumulative probabilities of dying at all ages, given an increase in the cumulative probability of dying at particular age and keeping constant the level of the mortality h.

The first three vectors of the matrix U were added to the model life table as they were extra coefficients, so that the model would have up to four parameters for the estimation and the same number of entry points that can be fitted in a life table. Considering all the above, the equation (6) is rewritten in the form:

$$ln(q^m) = \hat{\beta}_{28\times 3} \cdot [1, ln(h), ln(h)^2]' + \mathrm{U}_{28\times 3} \cdot [k_1, k_2, k_3]'. \qquad (8)$$

1.5. Results

Fitting

Table 2 shows the overall fit of the equation (5) for the three methods described and trying different predictors. There were considered fourteen predictors, and a single model was estimated for each of them. As predictors were considered some probabilities of dying that could be calculated from vital statistics or demographic surveys, and somehow related to the level of mortality at early ages. Given the multiplicity of predictors and regression methods, the Root Mean Square Error (RMSE) was the criterion to compare the predictive capacity of each model. Considering that the model consists of 28 equations and age intervals are not uniform, the reported RMSE results from weighting each equation by the length of the age interval. Comparing the rows of the table, the best predictor is the cumulative probability of dying at the exact age of 24 months $q(24m)$ regardless of the estimation method, and the same result holds for both males and females. Table 2 also shows that most suitable adjustments are resulting from using probabilities of dying that include the first month of life. Nevertheless, the neonatal mortality by itself would not best predict the mortality at all other ages. Comparing the columns of the table, it is observed that once the outliers are excluded, the variance of the estimators is reduced considerably. So a better and more adjusted fit would be simply to exclude outliers.

Table 2:Weighted root mean square error

	Female			Male		
Predictor	*(1)*	*(2)*	*(3)*	*(1)*	*(2)*	*(3)*
Neonatal [0 days, *28d*)	0.1930	0.1840	0.1840	0.1893	0.1817	0.1831
Postneonatal [*28d,12m*)	0.1929	0.1902	0.1904	0.2055	0.2027	0.2034
Second year [*12m, 24m*)	0.2532	0.2473	0.2471	0.2546	0.2512	0.2508
Third year [*24m, 36m*)	0.2837	0.2783	0.2782	0.2861	0.2781	0.2785
Fourth year [*36m, 48m*)	0.3176	0.3049	0.3051	0.3064	0.2930	0.2929
Fifth year [*48m, 60m*)	0.3308	0.3227	0.3253	0.3214	0.3137	0.3133
Interval [*5m, 21m*)	0.2000	0.1927	0.1925	0.2087	0.2055	0.2058
Interval [*3m, 24m*)	0.1888	0.1837	0.1841	0.2002	0.1982	0.1984
Child Mortality [*12m, 60m*)	0.2242	0.2194	0.2196	0.2289	0.2246	0.2244
Infant Mortality [*0d, 12m*)	0.0521	0.0480	0.0470	0.0492	0.0465	0.0460
First two years [*0d, 24m*)	0.0394	0.0374	0.0375	0.0382	0.0366	0.0367
First three years [*0d, 36m*)	0.0412	0.0398	0.0398	0.0399	0.0388	0.0388
First four years [*0d, 48m*)	0.0448	0.0433	0.0432	0.0438	0.0427	0.0427
Under-5 mortality [*0d, 60m*)	0.0486	0.0469	0.0469	0.0477	0.0465	0.0465

Note: *(1)* OLS; *(2)* Bi-weight; *(3)* Outliers.

Table 3 and Table·4 show the estimated coefficients for females and males, using $q(24m)$ as a predictor. The coefficients were estimated by OLS using the 1,319 life tables initially described, but excluding extreme observations. Bearing in mind equation (8), the first three columns reported in Table 3 and Table 4 correspond to the $\hat{\beta}_{28\times3}$ coefficients; while the three following columns correspond to the orthonormal vectors $U_{28\times3}$, which allows the life table model to adjust to particular characteristics of a population.

Table 3: Coefficients for a log-quadratic model of the cumulative probability of dying as a function of $q(24m)$

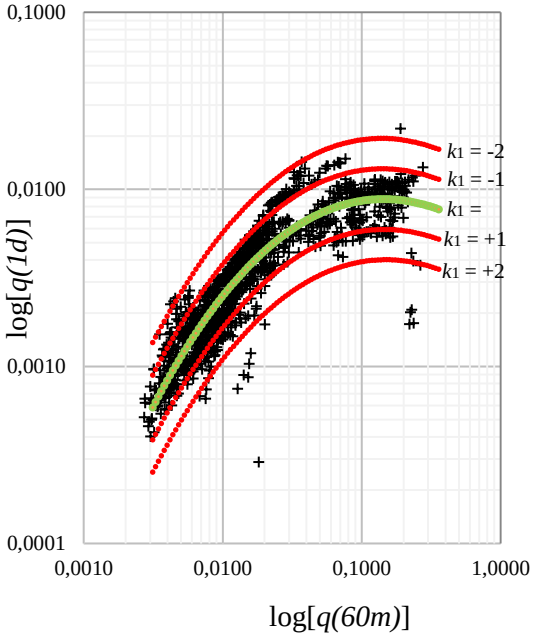
Female	β_1	β_2	β_3	U_1	U_2	U_3
q(1d)	-5.5867	-0.8065	-0.1896	-0.3945	0.6356	0.5241
q(2d)	-5.8435	-1.1021	-0.2273	-0.3538	0.2868	-0.1260
q(3d)	-5.6704	-1.0958	-0.2275	-0.3279	0.1411	-0.2051
q(4d)	-5.5146	-1.0713	-0.2258	-0.3118	0.0518	-0.2288
q(5d)	-5.2872	-0.9878	-0.2162	-0.3133	-0.0768	-0.2050
q(6d)	-5.1359	-0.9393	-0.2110	-0.3089	-0.1229	-0.1912
q(7d)	-4.9568	-0.8715	-0.2034	-0.3039	-0.1472	-0.1757
q(14d)	-3.9775	-0.4545	-0.1548	-0.2605	-0.2686	-0.0768
q(21d)	-3.4738	-0.2542	-0.1324	-0.2269	-0.2793	0.0140
q(28d)	-3.0402	-0.0772	-0.1128	-0.2077	-0.2570	0.0594
q(2m)	-2.3079	0.1984	-0.0825	-0.1561	-0.2297	0.1750
q(3m)	-1.9135	0.3264	-0.0698	-0.1180	-0.2031	0.2266
q(4m)	-1.6685	0.4018	-0.0624	-0.0912	-0.1756	0.2399
q(5m)	-1.4613	0.4709	-0.0553	-0.0755	-0.1565	0.2488
q(6m)	-1.2840	0.5305	-0.0493	-0.0631	-0.1427	0.2466
q(7m)	-1.1454	0.5757	-0.0449	-0.0536	-0.1279	0.2313
q(8m)	-1.0203	0.6172	-0.0409	-0.0458	-0.1138	0.2140
q(9m)	-0.9014	0.6579	-0.0368	-0.0387	-0.0995	0.1926
q(10m)	-0.8037	0.6910	-0.0336	-0.0328	-0.0847	0.1701
q(11m)	-0.7102	0.7244	-0.0302	-0.0272	-0.0713	0.1458
q(12m)	-0.6034	0.7663	-0.0257	-0.0234	-0.0686	0.1375
q(15m)	-0.4052	0.8427	-0.0175	-0.0135	-0.0478	0.0787
q(18m)	-0.2505	0.9027	-0.0109	-0.0074	-0.0296	0.0423
q(21m)	-0.1205	0.9531	-0.0053	-0.0034	-0.0143	0.0182
q(24m)	-	1.0000	-	-	-	-
q(36m)	0.2192	1.0801	0.0090	0.0096	0.0213	-0.0497
q(48m)	0.3465	1.1245	0.0141	0.0141	0.0320	-0.0808
q(60m)	0.4269	1.1505	0.0172	0.0176	0.0387	-0.1041

Table 4: Coefficients for a log-quadratic model of the cumulative probability of dying as a function of $q(24m)$

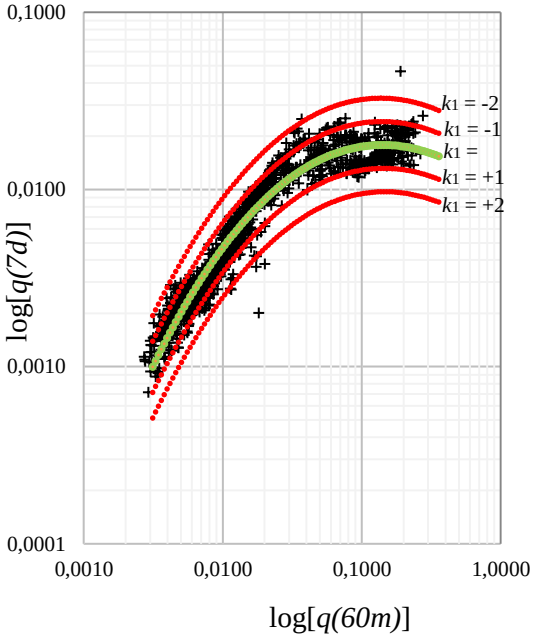
Male	β_1	β_2	β_3	U_1	U_2	U_3
q(1d)	-5.0766	-0.6925	-0.1892	-0.3912	0.6811	0.4734
q(2d)	-5.2752	-0.9794	-0.2275	-0.3446	0.2866	-0.1363
q(3d)	-5.2022	-1.0305	-0.2349	-0.3245	0.1253	-0.2674
q(4d)	-5.0516	-1.0071	-0.2330	-0.3103	0.0296	-0.2652
q(5d)	-4.8579	-0.9340	-0.2240	-0.3158	-0.0802	-0.2040
q(6d)	-4.6915	-0.8707	-0.2162	-0.3125	-0.1382	-0.1575
q(7d)	-4.5302	-0.8098	-0.2093	-0.3051	-0.1660	-0.1385
q(14d)	-3.6262	-0.4028	-0.1590	-0.2601	-0.2701	-0.0412
q(21d)	-3.1460	-0.1969	-0.1346	-0.2331	-0.2762	0.0401
q(28d)	-2.7424	-0.0228	-0.1141	-0.2123	-0.2555	0.1042
q(2m)	-2.0454	0.2542	-0.0819	-0.1632	-0.2136	0.1995
q(3m)	-1.6699	0.3824	-0.0683	-0.1225	-0.1881	0.2449
q(4m)	-1.4564	0.4488	-0.0614	-0.0932	-0.1533	0.2509
q(5m)	-1.2663	0.5156	-0.0540	-0.0747	-0.1289	0.2488
q(6m)	-1.0953	0.5782	-0.0472	-0.0622	-0.1151	0.2470
q(7m)	-0.9675	0.6235	-0.0423	-0.0526	-0.1008	0.2290
q(8m)	-0.8498	0.6667	-0.0376	-0.0445	-0.0882	0.2090
q(9m)	-0.7461	0.7044	-0.0337	-0.0375	-0.0737	0.1882
q(10m)	-0.6587	0.7363	-0.0303	-0.0315	-0.0624	0.1666
q(11m)	-0.5793	0.7661	-0.0271	-0.0268	-0.0538	0.1481
q(12m)	-0.5005	0.7967	-0.0238	-0.0226	-0.0475	0.1334
q(15m)	-0.3319	0.8661	-0.0158	-0.0137	-0.0359	0.0848
q(18m)	-0.2065	0.9167	-0.0099	-0.0078	-0.0237	0.0491
q(21m)	-0.0971	0.9609	-0.0047	-0.0035	-0.0117	0.0220
q(24m)	-	1.0000	-	-	-	-
q(36m)	0.1746	1.0646	0.0079	0.0097	0.0124	-0.0517
q(48m)	0.2668	1.0947	0.0117	0.0156	0.0191	-0.0838
q(60m)	0.3210	1.1093	0.0136	0.0193	0.0197	-0.1063

Figure 3 shows the fit of the model to the cumulative probabilities of dying at some selected ages other than $q(24m)$, given that the model was estimated using $q(24m)$ as the predictor. The figure confirms the necessity of using a log-quadratic model. Although the relationship is almost linear after the first 12 months of life, the log-quadratic form produces an adequate fit at neonatal ages. Figure 3 also shows that most observations fall within a range of $\pm 2 \cdot k_1$ on the first day of life, and $\pm k_1$ when predicting the cumulative probability of dying at the exact ages of 7 and 28 days.

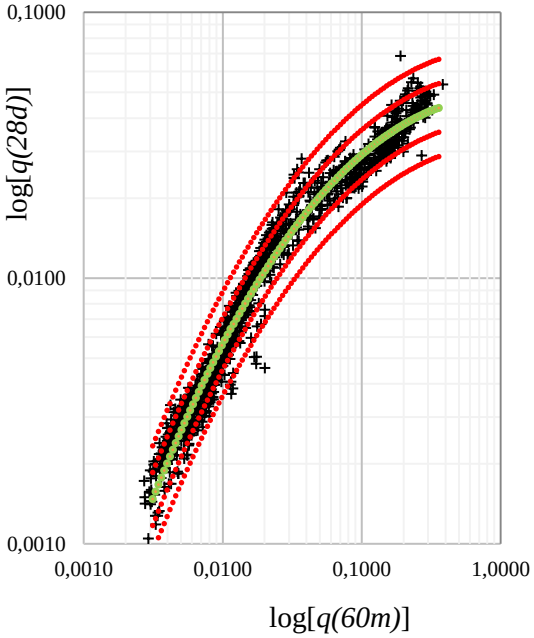
Figure 3: Cumulative probabilities of dying $q(x)$ vs. under-five mortality $q(60m)$
Log-quadratic fit using $q(24m)$ as a predictor and five values of k_1
Female



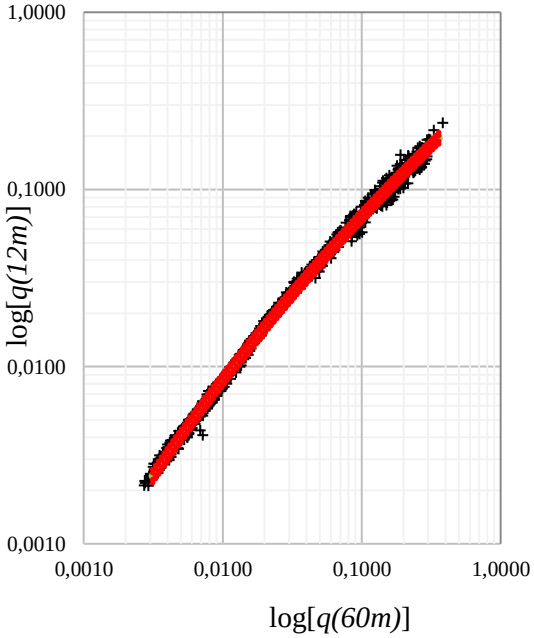
1 day *vs*. 60 months of life



7 days *vs*. 60 months of life

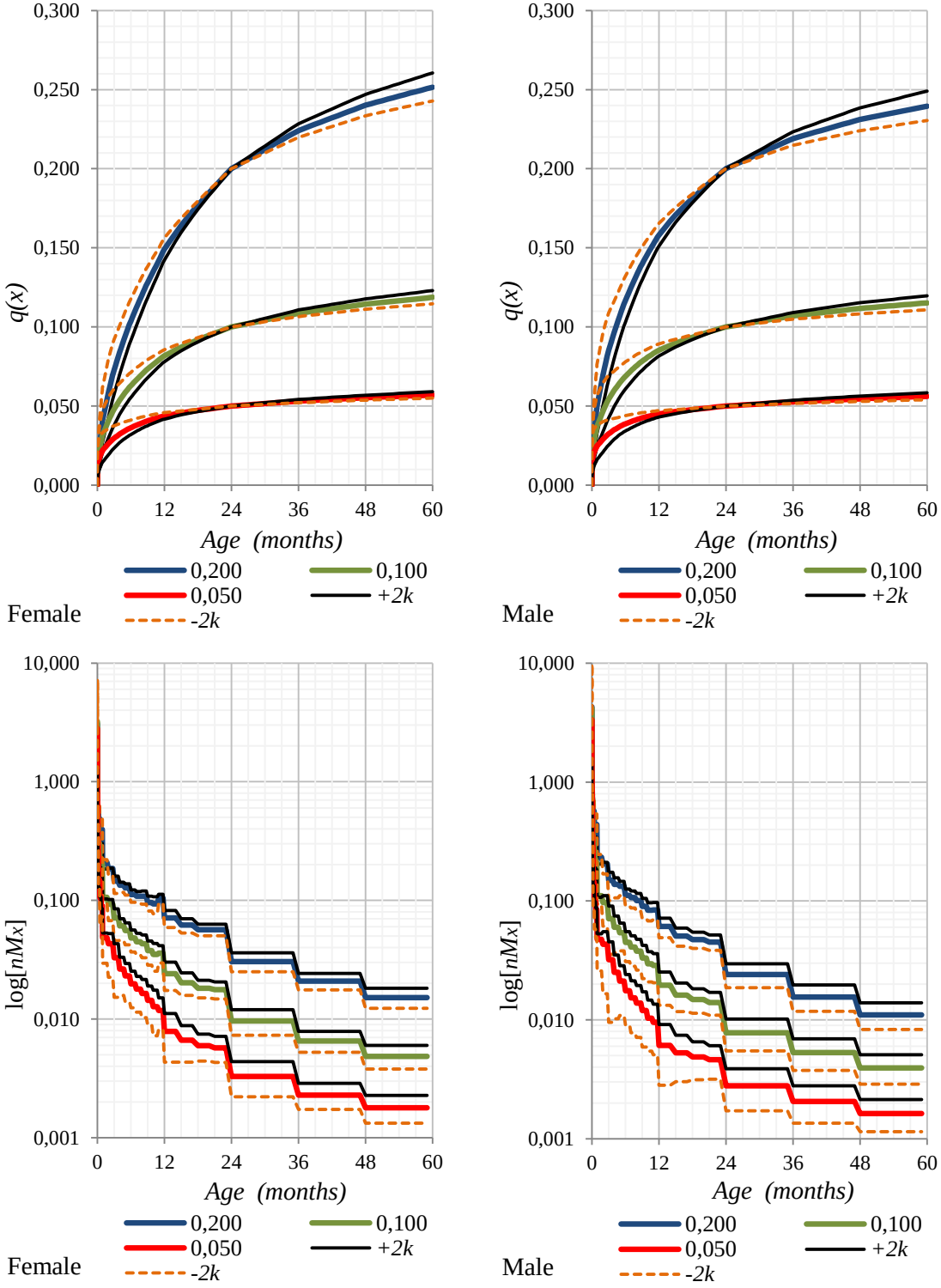


28 days *vs*. 60 months of life



12 months *vs*. 60 months of life

Figure 4: Cumulative probabilities of dying and age-specific mortality rates for different combinations of $q(24m)$ and $\pm 2 \cdot k_1$



Predicting

Figure 4 shows the cumulative probabilities of dying predicted by the model and the specific mortality rates that can be calculated from them. Different levels of mortality were combined from $q(24m)$ with three possible values of k_1. Keeping constant the value of $q(24m)$, a positive value of k_1 decreases the cumulative probabilities of dying below the age of two years, while increases the cumulative probability of dying from 2 to 5 years of age. With negative values of k_1, the effect is quite the opposite. However, the effect on the resulting specific mortality rates is ambiguous and depends on how k_1 is distributed across ages. According to the values reported in the Table 3 and Table 4, a positive value of k_1 produces a larger decrease in the cumulative probabilities of dying during the first days and weeks of life; consequently, to keep the probability of dying before the age of two constant, mortality rates for other ages must increase.

The main application of a model life table is as an indirect method. Thus, a model should have the capacity to predict complete mortality schedules using incomplete information, for example, knowing just one probability of dying. Therefore, the performance and predictive capacity of the model were evaluated for a given probability of dying below the age of five, and ignoring the value of $q(24m)$. Consequently, a value of $q(24m)$ was calculated for each country-year solving the equation (7). In this case, numerical methods were used in order to match the model to the best available information. For each sex, the first column of Table 5 shows the resulting weighted RMSE of predicting all life tables, given as the only available information the probabilities of dying at selected age intervals. The first column shows that more uncertainty would result in predicting a complete life table for a given value of neonatal mortality. Similarly, more variance would result if the mortality during the first months of life were unknown. This means that the model is sensitive to the mortality at early ages.

However, this problem could be attenuated by using a second input value. For example, assume that a researcher wants to estimate a mortality schedule using the coefficients reported in Table 3 (or Table 4) for a given value of the neonatal mortality, which is the best available information. Although Table 5 warns that the RMSE is too high, a significant improvement would result in the prediction if the model is forced to match the neonatal mortality and the probability of dying at the age of two years. In the case of females, the RMSE would be reduced from 0.1914 to 0.0281. In general, adding more reliable information reduces the RMSE as is shown in Table 5 for different age intervals. For each sex, the second column of Table 5 shows the second input value that minimizes the RMSE and the third column is the resulting RMSE of matching these two entry values. In order to match any two probabilities of dying and given the set of coefficients reported in Table 3 and Table 4, optimal values of $q(24m)$ and k_1 were calculated for each country-year solving the equation (8) by numerical methods. Considering the results

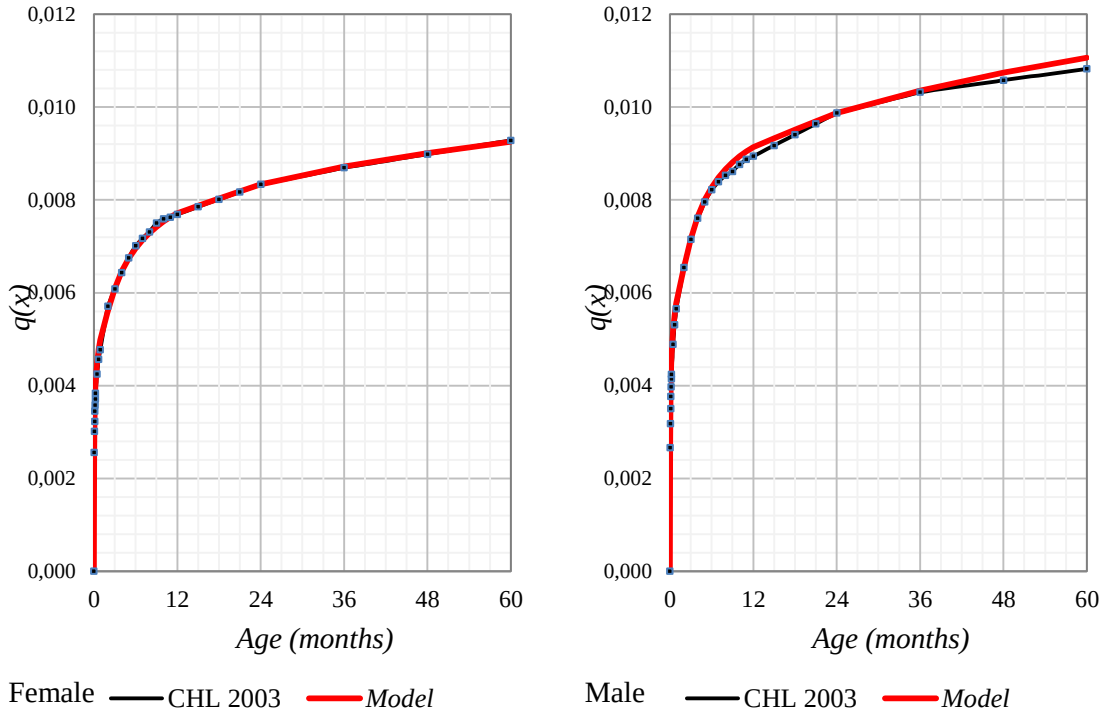
reported in Table 5, even if the model was estimated from $q(24m)$ and this is one value to be matched, a better prediction would result to use the cumulative probability of dying at the age of three months $q(3m)$ as the second entry value.

Table 5: The weighted RMSE of matching one entry value *(1)*, the second entry point that minimizes the WRMSE *(2)*, and the WRMSE of matching two entry values *(3)*

	Female			Male		
Matching	*(1)*	*(2)*	*(3)*	*(1)*	*(2)*	*(3)*
Neonatal [0 *days*, *28d*)	0.1914	$q(24m)$	0.0281	0.1951	$q(24m)$	0.0269
Postneonatal [*28d*, *12m*)	0.2124	$q(15m)$	0.0414	0.2221	$q(18m)$	0.0380
Second year [*12m*, *24m*)	0.2999	$q(15m)$	0.0590	0.3214	$q(24m)$	0.0419
Third year [*24m*, *36m*)	0.3399	$q(21m)$	0.0556	0.3317	$q(18m)$	0.0526
Fourth year [*36m*, *48m*)	0.3462	$q(18m)$	0.0795	0.3232	$q(21m)$	0.0650
Fifth year [*48m*, *60m*)	0.3619	$q(21m)$	0.0829	0.3374	$q(21m)$	0.0754
Interval [*5m*, *21m*)	0.2265	$q(21m)$	0.0285	0.2380	$q(24m)$	0.0274
Interval [*3m*, *24m*)	0.2149	$q(24m)$	0.0270	0.2273	$q(24m)$	0.0259
Child Mortality [*12m*, *60m*)	0.2639	$q(10m)$	0.0445	0.2639	$q(9m)$	0.0428
Infant Mortality [*0d*, *12m*)	0.0495	${}_{46m}q_{2m}$	0.0339	0.0472	${}_{46m}q_{2m}$	0.0322
First two years [*0d*, *24m*)	0.0375	$q(3m)$	0.0259	0.0367	$q(3m)$	0.0245
First three years [*0d*, *36m*)	0.0396	$q(3m)$	0.0247	0.0387	$q(3m)$	0.0236
First four years [*0d*, *48m*)	0.0432	$q(3m)$	0.0265	0.0427	$q(4m)$	0.0252
Under-5 mortality [*0d*, *60m*)	0.0470	$q(4m)$	0.0287	0.0467	$q(4m)$	0.0273

The capacity of the model to predict individual life tables using few input values is illustrated in Figures 5-10 for some country-years: Chile (2003), Italy (1980), United States (1940), France (1918, during the influenza pandemic), Sweden (1891), and Norway (1880). Using the estimated coefficients β and the first orthonormal vector from the covariance matrix U_1, the first four cases were fitted using only two values: the cumulative probability of dying at 24 months $q(24m)$, and the cumulative probability of dying at the age of three months $q(3m)$. In the last two cases, the model failed to reproduce the mortality pattern with these characteristics. They are both late patterns of under-five mortality. Therefore, the model was adjusted using three probabilities of dying. This is using the estimated coefficients and two orthonormal vectors: U_1 and U_3. On the one hand, the neonatal mortality $q(28d)$, the infant mortality $q(12m)$, and the child mortality ${}_{48m}q_{12m}$ were used to predict Sweden in 1891. On the other hand, $q(28d)$, $q(12m)$, and the probability of dying in the second, third or fourth year of life ${}_{36m}q_{12m}$ were used to predict Norway in 1880. Figures 5-10 show that the model effectively reproduces the cumulative probabilities of dying and the specific mortality rates calculated from them.

Figure 5: Chile, 2003
Cumulative probabilities of dying and age-specific mortality rates



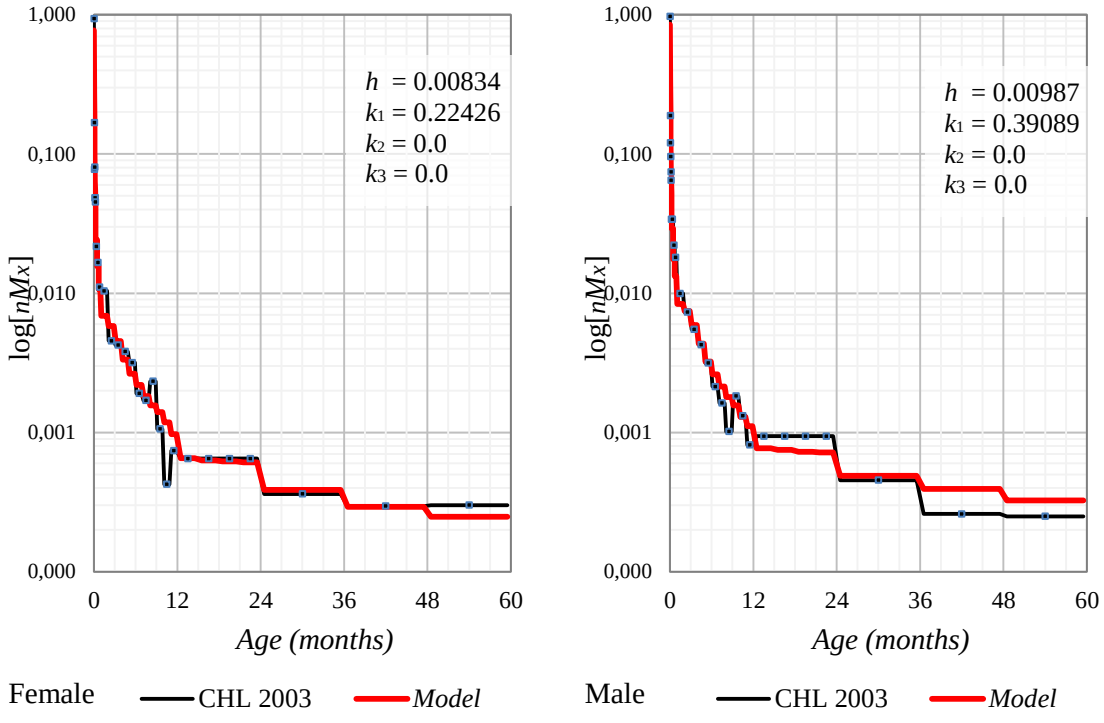
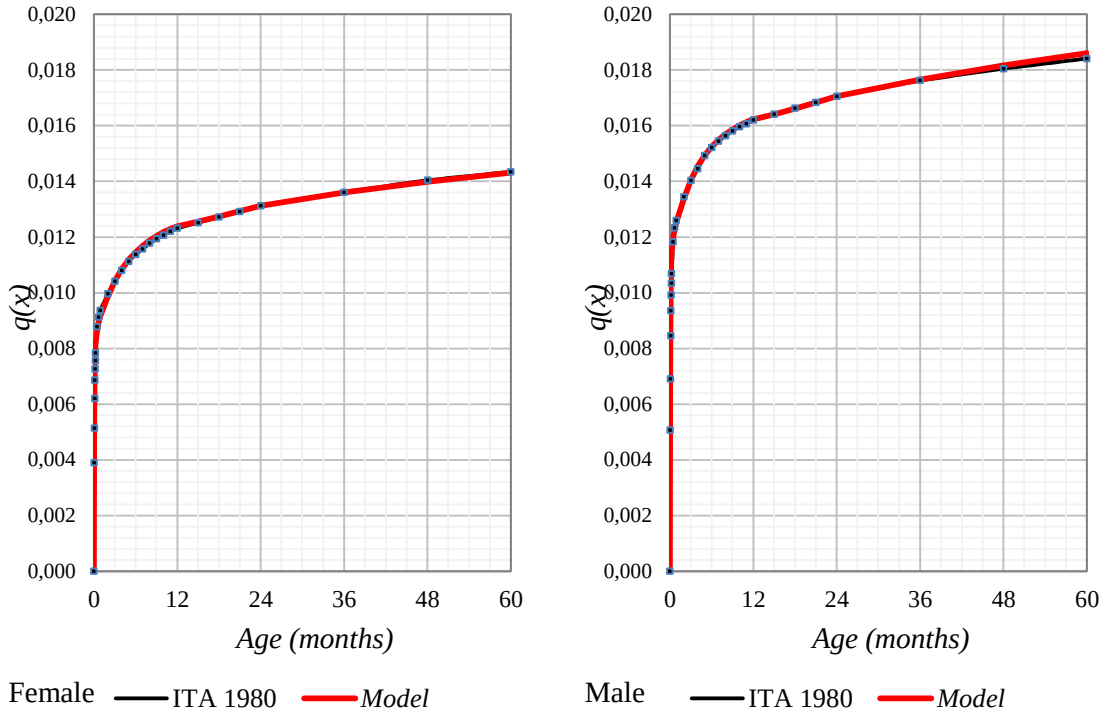


Figure 6: Italy, 1980
Cumulative probabilities of dying and age-specific mortality rates



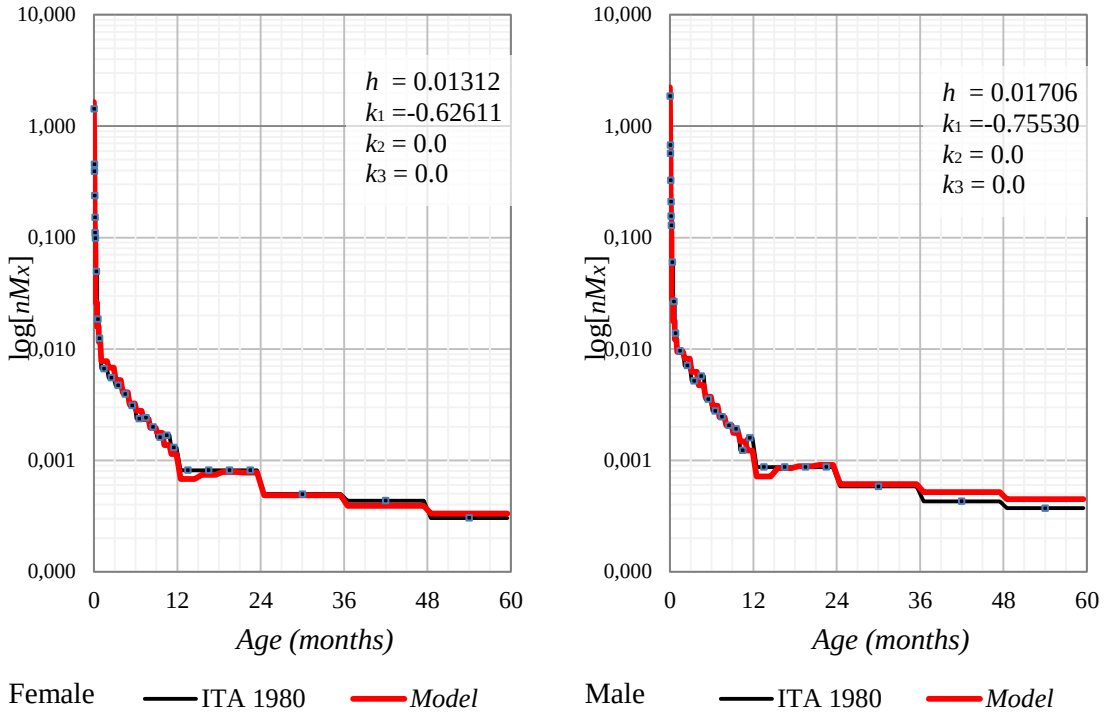
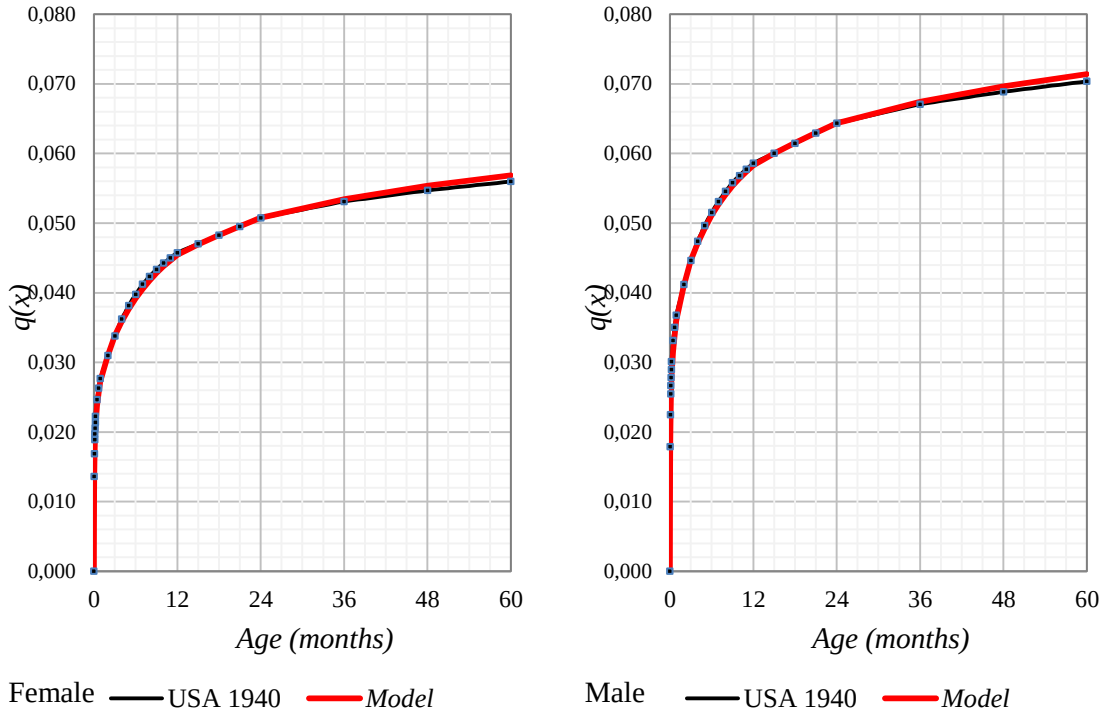


Figure 7: United States, 1940
Cumulative probabilities of dying and age-specific mortality rates



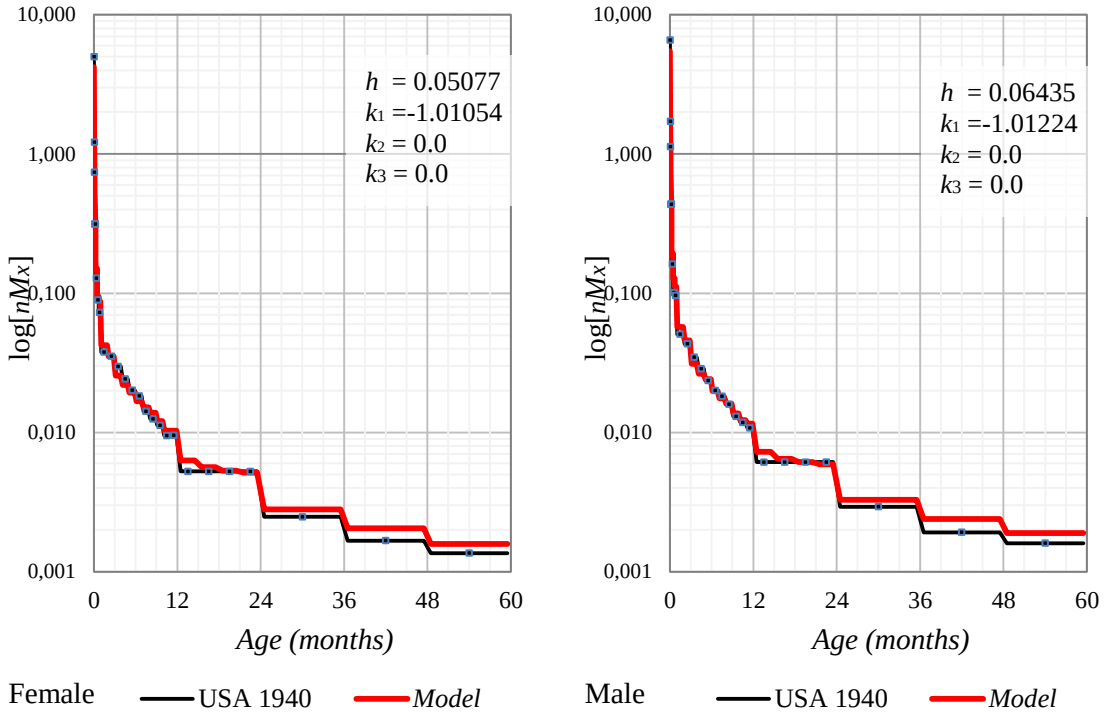
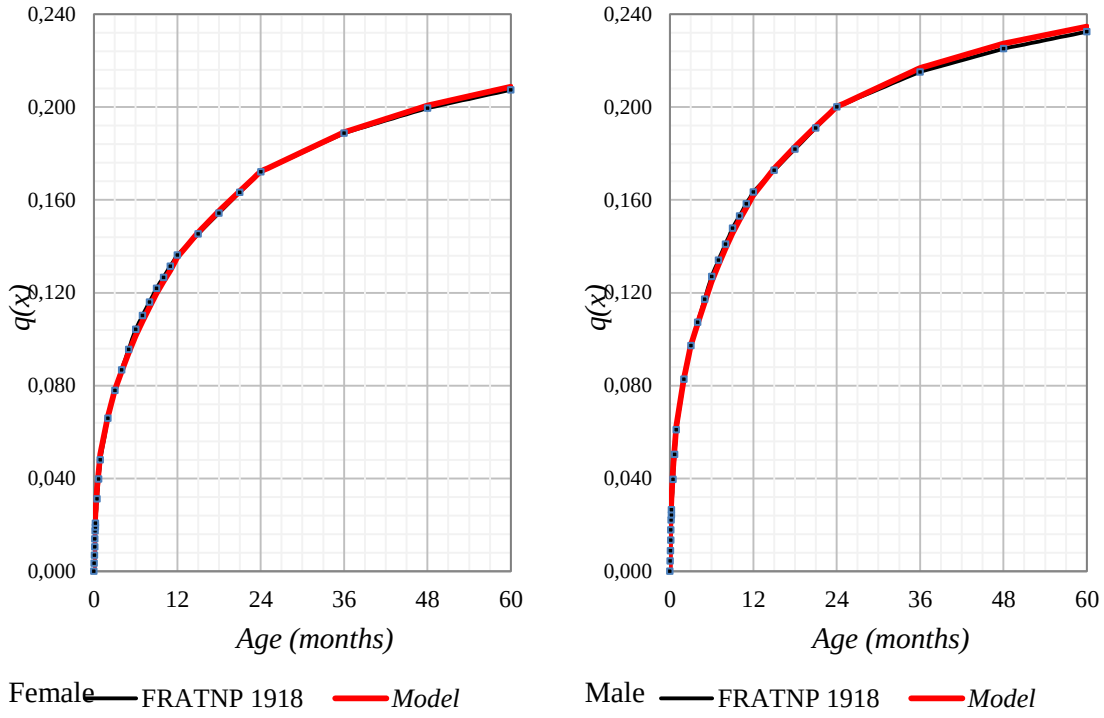


Figure 8: France, 1918
Cumulative probabilities of dying and age-specific mortality rates



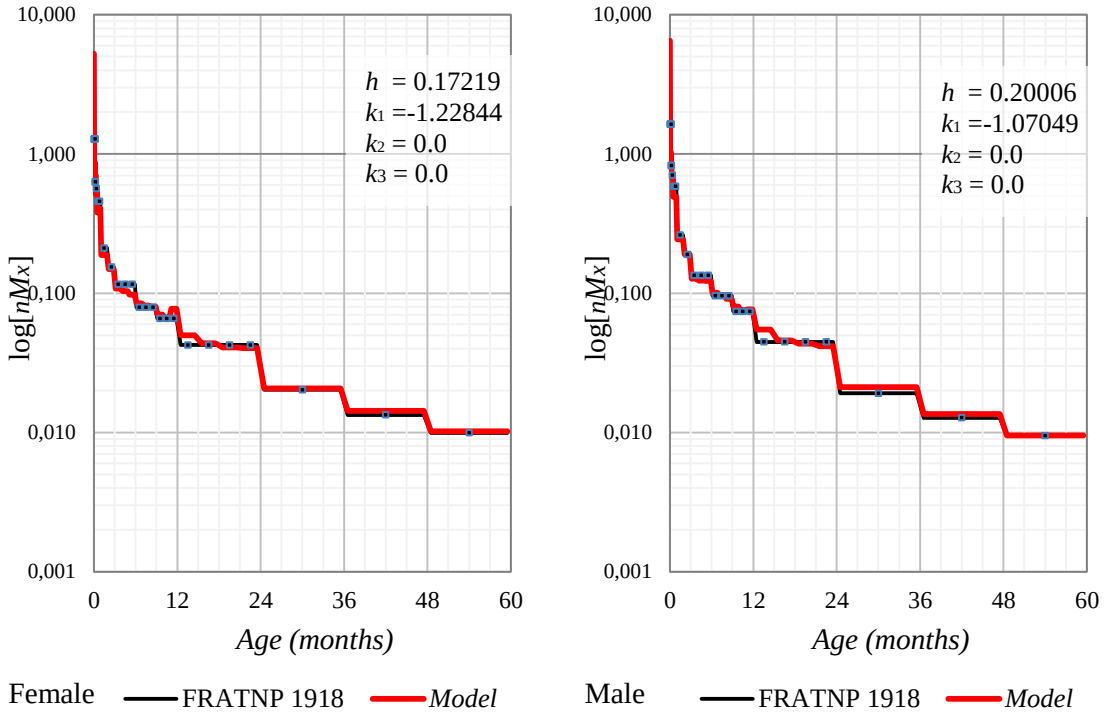
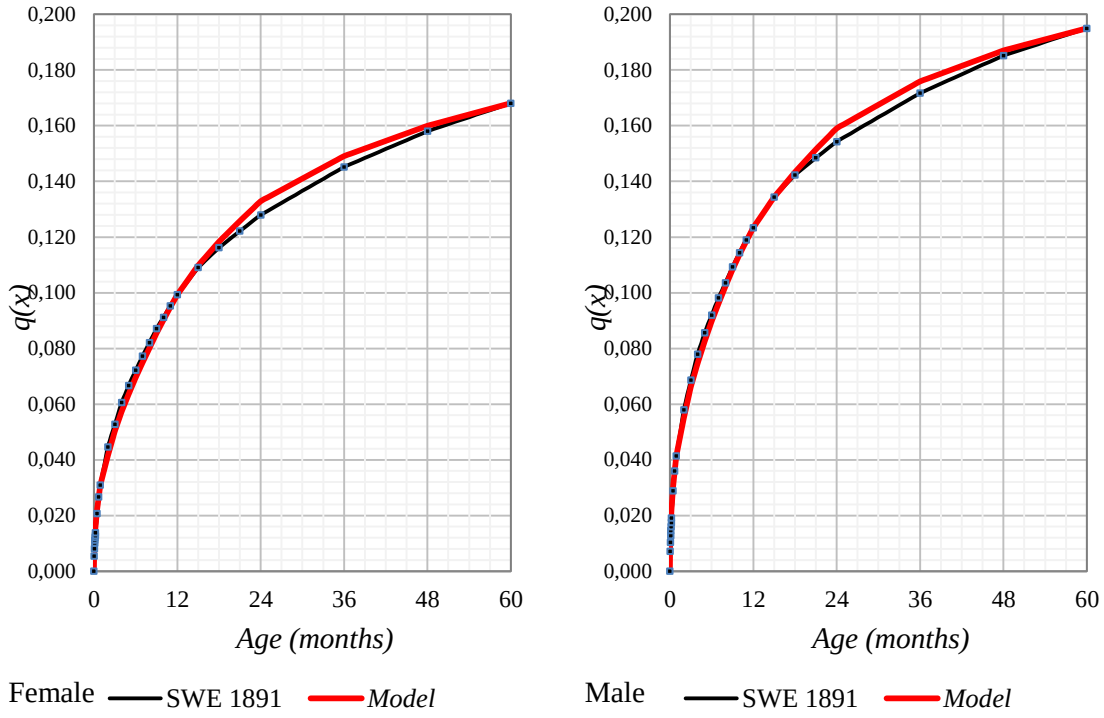


Figure 9: Sweden, 1891
Cumulative probabilities of dying and age-specific mortality rates



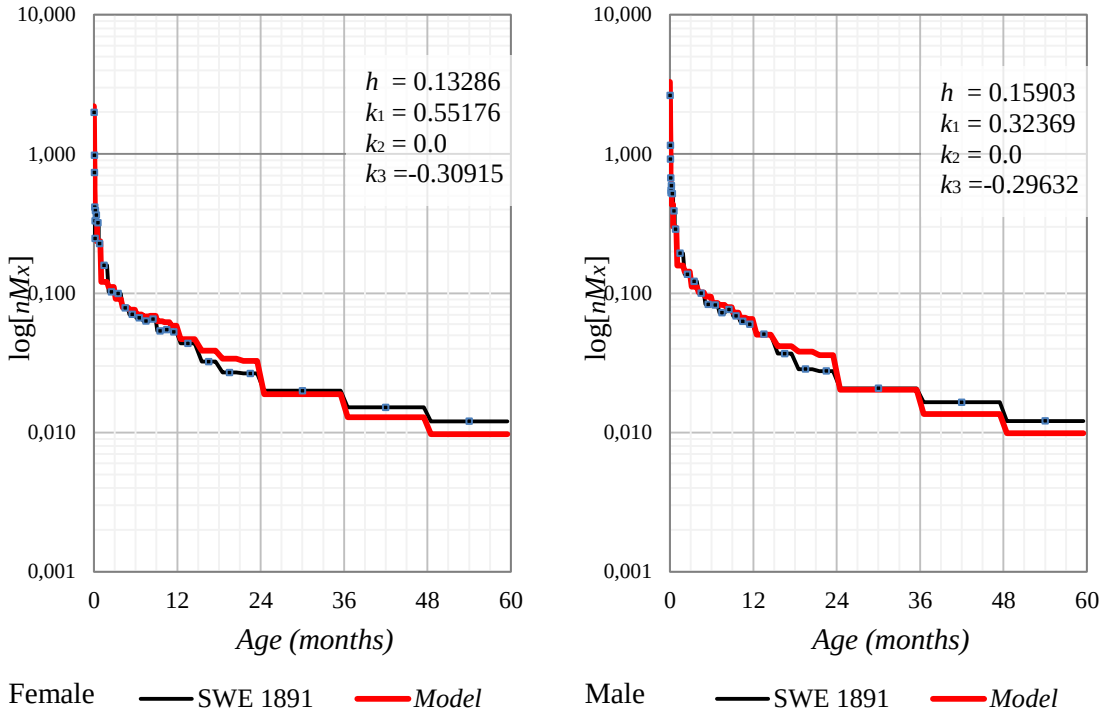
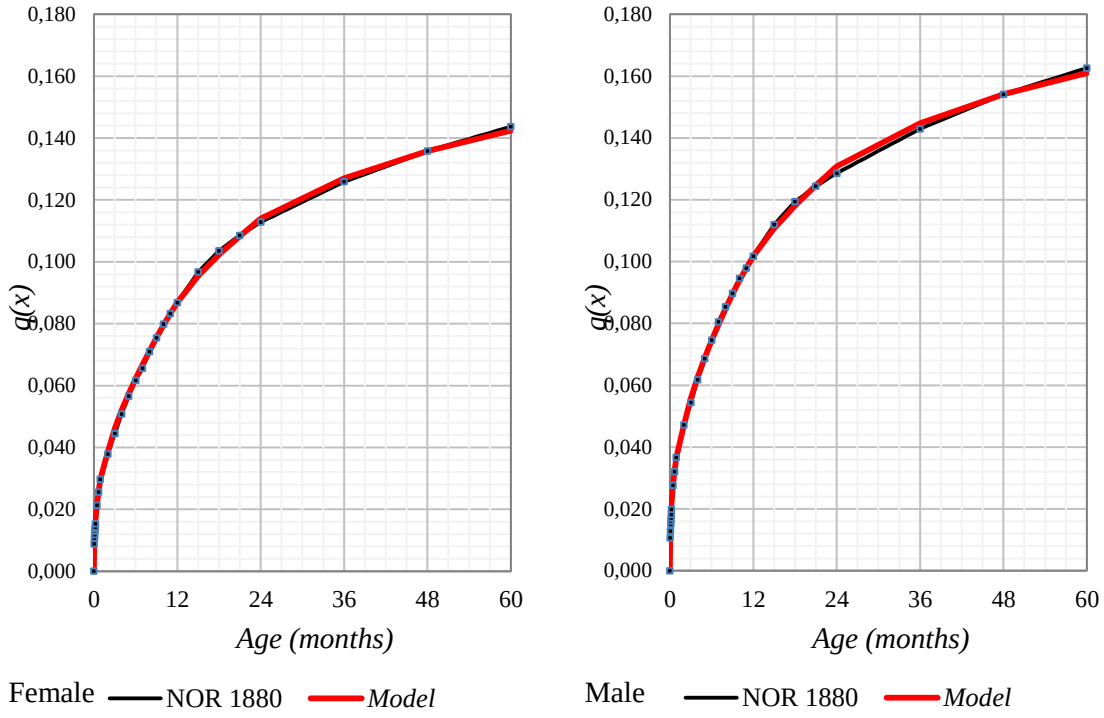
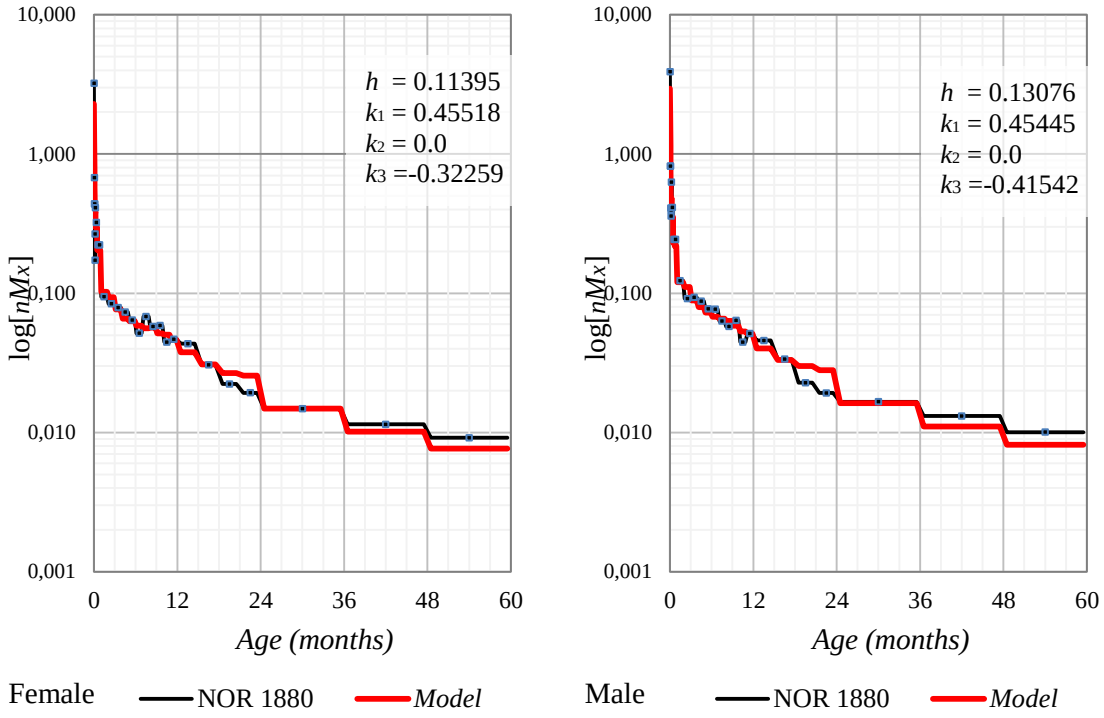


Figure 10: Norway, 1880
Cumulative probabilities of dying and age-specific mortality rates





1.6. Discussion

Using vital registrations and census enumerations, a model life table is estimated to study the age patterns of under-five mortality. From a few input values, the model allows the prediction of a cumulative probability of dying by subintervals of age. A general solution is proposed, since patterns of high and low mortality can be reproduced from incomplete information and using the same set of equations. Critical aspects of model life tables were addressed in this paper: the use of historical sources, the statistical fitting, and the prediction of actual mortality schedules. Inasmuch as the model is statistically adjusted and flexible to admit input information at different age intervals, it is a useful method for validation or indirect estimation.

Given an empirical life table and keeping constant at least one probability of dying, the model can be used to test whether the deaths of a population are more concentrated at the beginning or the end of an age interval. This allows to infer if a population has an early or late pattern of mortality using the model as a standard for comparison. Previous research has conducted a similar analysis by applying the biometric model of BP (Bourgeois-Pichat, 1950): late patterns of infant mortality have been associated to breastfeeding whereas early patterns to the artificial feeding (Knodel & Kintner, 1977). However, no inferences can be drawn from the biometric model after the first year of life. This is an important limitation considering that late patterns of under-five mortality are a substantial characteristic of high mortality populations. In this regard, the model life table proposed in this paper is a relevant material for the demographic analysis of early patterns of mortality.

The model can be used as an indirect estimation method in contexts with limited data where direct estimation is not an option. Kingkade & Arriaga (1997), and Guillot et al. (2013) propose methods for estimating infant mortality from the probabilities of dying at those age intervals in which direct estimation is more reliable. This paper contributes to the discussion of the indirect estimation of mortality at early ages proposing a more general approach: several age intervals were considered, and the model was estimated using an extended set of country-years. In addition, the model follows the approach proposed by Wilmoth et al. (2012), allowing to adjust a hypothetical life table for more than one input value by extracting relevant information from the covariance of the errors. More than one input parameter not only improves the predictions of the model but also allows to represent particular characteristics of the population under study when more information is available. Consequently, a mortality schedule can be estimated from a few probabilities of dying and be compared with the empirical distribution of deaths, in order to detect biases and omissions from unreliable statistics or that require validation such as those estimated from demographic surveys.

1.7. References

Barker, D., 1990. The fetal and infant origins of adult disease. *British Medical Journal*, Volume 301, p. 1111.

Beaton, E. & Tukey, J. W., 1974. The fitting of power series, meaning polynomials, illustrated on band-spectroscopic data. *Technometrics*, 16(2), pp. 147-185.

Beaver, M., 1973. Population, infant mortality and milk. *Population Studies*, 27(2), pp. 243-254.

Bourgeois-Pichat, J., 1950. Analyse de la mortalité infantile. *Revue de l'Institut International de Statistique/Review of the International Statistical Institute*, 18(1-2), pp. 45-68.

Brass, W., 1971. On the scale of mortality. In: W. Brass, ed. *Biological Aspects of Demography*. London: Taylor & Francis Ltd., pp. 69-110.

Brass, W., 1996. Demographic data analysis in less developed countries: 1946-1996. *Population Studies*, 50(3), pp. 451-467.

Ceesay, S. M. et al., 1997. Effects on birth weight and perinatal mortality of maternal dietary supplements in rural Gambia: 5 year randomised controlled trial. *The British Medical Journal*, Volume 315, pp. 786-90.

Coale, A. & Guo, G., 1989. Revised regional model life tables at very low levels of mortality. *Population Index*, 55(4), pp. 613-643.

Coale, A. J. & Demeny, P., 1966. *Regional Model Life Tables and Stable Populations.* Princeton: Princeton University Press.

Coale, A. & Trussell, J., 1996. The development and use of demographic models. *Population Studies*, 50(3), pp. 469-484.

Cutler, D. & Miller, G., 2005. The role of public health improvements in health advances: The twentieth-century United States. *Demography*, 42(1), pp. 1-22.

Dyhouse, C., 1978. Working-class mothers and infant mortality in England, 1895-1914. *Journal of Social History*, 12(2), pp. 248-267.

Easterlin, R. A., 2004. How beneficent is the market? A look at the modern history of mortality. In: *The Reluctant Economist: Perspectives on Economics, Economic History, and Demography*. Cambridge-New York, NY: Cambridge University Press, pp. 101-138.

Ewbank, D. C., Gomez De Leon, J. C. & Stoto, M. A., 1983. A reducible four-parameter system of model life tables. *Population Studies,* 37(1), pp. 105-127.

Gabriel, K. R. & Ronen, I., 1958. Estimates of mortality from infant mortality rates. *Population Studies,* 12(2), pp. 164-169.

Galley, C. & Woods, R., 1999. On the distribution of deaths during the first year of life. *Population: An English Selection,* Volume 11, pp. 35-59.

Garenne, M., 1982. *Variations in the age pattern of infant and child mortality with a special reference to a case of study in Ngayokheme (rural Senegal),* PhD Dissertation in Demography: University of Pennsylvania.

Godfrey, K. et al., 1996. Maternal nutrition in early and late pregnancy in relation to placental and fetal growth. *British Medical Journal,* Volume 312, pp. 410-414.

Guillot, M., Lim, S.-j., Torgasheva, L. & Denisenko, M., 2013. Infant mortality in Kyrgyzstan before and after the break-up of the Soviet Union. *Population Studies,* 67(3), pp. 335-352.

Hogan, H. R., 1976. *Age patters of infant mortality,* PhD Dissertation in Economics: Princeton University.

Huffman, S. & Lamphere, B., 1984. Breastfeeding performance and child survival. *Population and Development Review,* Volume 10 (Supplement, Child Survival: Strategies for Research), pp. 93-116.

Jelliffe, D. B. & Jellife, E. E. P., 1979. Early infant nutrition: breast feeding. In: M. Winick, ed. *Nutrition: Pre- and Postnatal Development.* New York: Plenum Press, pp. 229-259.

Kingkade, W. W. & Arriaga, E. E., 1997. Mortality in the new independent sates: Patterns and impacts. In: J. L. Bobadilla, C. Costello & F. Mitchell., eds. *Premature death in the New Independent States.* Washington, DC: National Academy Press, pp. 156-183.

Knodel, J. & Kintner, H., 1977. The impact of breast feeding patterns on the biometric analysis of infant mortality. *Demography,* 14(4), pp. 391-409.

Lamberti, L. et al., 2011. Breastfeeding and the risk for diarrhea morbidity and mortality. *BMC Public Health,* 11(Suppl 3).

Ledermann, S. & Breas, J., 1959. Les dimensions de la mortalité. *Population (French edition),* 14e(4), pp. 637-682.

London, A, 1993. The impact of advances in medicine on the biometric analysis of infant mortality. *Biodemography and Social Biology*, 40(3-4), pp. 260-282.

McCormick, M., 1985. The contribution of low birth weight to infant mortality and childhood morbidity. *The New England Journal of Medicine*, 312(2), pp. 82-90.

McKeown, T., 1983. Food, infection, and population. *The Journal of Interdisciplinary History*, 14(2), pp. 227-247.

McKeown, T. & Record, R. G., 1962. Reasons for the decline of mortality in England and Wales during the nineteenth century. *Population Studies*, 16(2), pp. 94-122.

Moore, S. E. et al., 1999. Prenatal or early postnatal events predict infectious deaths in young adulthood in rural Africa. *International Journal of Epidemiology*, Volume 28, p. 1088–1095.

Moore, S. E. et al., 1997. Season of birth predicts mortality in rural Gambia. *Nature*, Volume 388, p. 434.

Murray, C. J. L. et al., 2003. Modified logit life table system: Principles, empirical Validation, and application. *Population Studies*, 57(2), pp. 165-182.

Palloni, A. & Millman, S., 1986. Effects of inter-birth intervals and breastfeeding on infant and early childhood mortality. *Population Studies*, 40(2), pp. 215-236.

Prentice , A. et al., 1987. Increased birthweight after prenatal dietary supplementation of rural African women. *The American Journal of Clinical Nutrition*, Volume 46, pp. 912-25.

Preston, S. H., 1972. Influence of cause of death structure on age-patterns of mortality. In: T. N. E. Greville, ed. *Population Dynamics.* New York: Academic Press, pp. 201-250.

Preston, S. H. & Haines, M. R., 1991. *Fatal Years: Child Mortality in Late Nineteenth-Century America.* Princeton, NJ: Princeton University Press.

Rollet, C., 1997. The fight against infant mortality in the past: An international comparison. In: *Infant and Child Mortality in the Past.* Oxford, England: Oxford University Press, pp. 38-60.

Roseboom, T. et al., 2001. Effects of prenatal exposure to the Dutch famine on adult disease in later life: an overview. *Molecular and Cellular Endocrinology*, Volume 185, pp. 93-98.

The Lancet, 1988. Infant mortality. 12 November, pp. 1117-1118.

United Nations, 1955. *Age and Sex Patterns of Mortality: Model Life-Tables for Under-Developed Countries,* New York, NY: United Nations, Department of Social Affairs - Population Branch.

United Nations, 1982. *Model Life Tables for Developing Countries,* New York, NY: United Nations, Department of International Economic and Social Affairs.

Victora, C. et al., 2016. Breastfeeding in the 21st century: epidemiology, mechanisms, and lifelong effect. *The Lancet,* Volume 387, p. 475–90.

Victora, C. G. et al., 1988. Influence of birth weight on mortality from infectious diseases: A case-control study. *Pediatrics,* 81(6), pp. 807-811.

Wilmoth, J. et al., 2012. A flexible two-dimensional mortality model for use in indirect estimation. *Population Studies,* 66(1), pp. 1-28.

Chapter 2

Age patterns of under-five mortality and the quality assessment of the Demographic and Health Surveys

Abstract

Under-five mortality is still a major problem in many areas of the world lacking reliable records for a proper monitoring. Therefore, indirect estimation has been the keystone of the demographic analysis in countries with incomplete, imperfect, or nonexistent vital records. Specifically, complete maternity histories are used to estimate mortality at early ages and the *Demographic and Health Survey* (DHS) is the main source of data. However, given its self-reported nature, this information is susceptible to some errors: underreported children ever born and misreported ages at death. Age misreporting obscures the study of mortality patterns based on survey data. Yet, errors are most evident when many populations are analyzed together and are compared to model life tables. This paper examines 252 public-domain surveys and proposes an empirical strategy to study mortality patterns at early ages. When survey estimates are contrasted to a model life table, particular characteristics can be identified. For a given level of mortality, deaths could be more concentrated in the early days and months of life (early pattern), or after the first year of age (late pattern). This paper shows that populations with high levels mortality are more likely to show late patterns of under-five mortality. Data quality issues regarding misreported ages at death are also analyzed. Particularly, this paper proposes a simple solution to the problem of heaping at the age of 12 months and the consequent underestimation of the infant mortality.

Key words: Infant and child mortality; under-five mortality; complete maternity histories; Demographic and Health Survey; demographic estimation; indirect methods; model life tables; age patterns of mortality at early ages.

2.1. Introduction

One of the major difficulties of monitoring the progress of reducing under-five mortality is the lack of vital records in those areas where it remains high. In contexts where vital records are either unreliable or non-existent, the analysis of basic demographic statistics is based on indirect and retrospective methods addressed to take advantage of information that can be collected from censuses and surveys (Brass, 1996). Given certain assumptions, indirect estimates of mortality at early ages can be calculated from the number of children ever born and the number of survivors, both collected in population censuses (Brass & Coale, 1968). Similarly, surveys allow a deeper inquiry and research for some questions, resulting in the possibility of calculating infant and child mortality in greater detail and fewer assumptions using complete maternity histories (Hill, 1991). Considering that women at reproductive ages report the date of birth of their offspring, and in the case of deceased children provide the age at death, direct estimates of the probabilities of dying can be assessed from a Lexis diagram (Rutstein S. O., 1984; Somoza, 1980). This is a conventional method that was applied in the *World Fertility Survey* and has been adapted to estimate infant and child mortality in the DHS (Mahy, 2003). However, the reliability of the estimates greatly depends on the quality of the reported life events. Since survey estimates are defined for extended periods of exposure to the risk of death, reporting bias should not be dismissed. Surveys inquire about vital events that occurred several years ago and the information collected may be susceptible to errors of recall and approximation (Hill, 1991). Not because surveys are systematically collected means that they are perfect data (Trussell & Menken, 1984). In addition to sampling errors that were not of minor importance in the first phase of the DHS (Curtis, 1995), the impressions that could bias the estimates of mortality at early ages are the underreported children ever born and the misreported ages at death.

Although census and survey estimates are practical solutions and sometimes the only available information, these are not a substitute for vital registration. On the one hand, surveys do not replace the legal character of the vital registration, thus certain benefits depending on proper documentation cannot be assured (AbouZahr, Savigny, Mikkelsen, Setel, Lozano, & Lopez, 2015). On the other hand, errors in the reporting of life events hinder the study of mortality patterns. Traditionally, under-five mortality patterns have been investigated by scattering the infant and the child mortality and making a contrast to models life tables (Bicego, et al., 1991; Guillot, et al., 2012; Sullivan, et al., 1994). Although some regions adjust to the pre-established ranges by conventional models, others characterize by an excess of child mortality relative to the level infant mortality (Guillot, et al., 2012). However, these inferences sometimes rely on values that have been calculated from surveys and it has been a documented fact that misreported ages at death could underestimate infant mortality and overestimate child mortality (Hill, 1991;

Rutstein, 1984). Therefore, one of the main limitations in the study of mortality patterns using this approach is that comparisons are concentrated only on infant and child mortality, without taking into consideration other relevant ages to explain the population differences in the level and shape of the mortality at early ages.

Using a Lexis diagram and complete maternity histories, this paper estimates mortality schedules for a set of 252 *Demographic and Health Surveys* of public domain. Estimates were calculated directly under the assumption of a synthetic cohort, but due to the fact maternity histories are self-reported data, the reliability of the estimates was examined. Mortality schedules were compared to a model life table for under-five mortality fitted to the vital records of 24 countries and using a broad set of sub-intervals of age. These comparisons allowed to evaluate the consistency of the estimates made from the survey data. Misreported ages at death were detected when all surveys were analyzed altogether and compared to model predictions. This allowed the identification of some probabilities of dying that were not severely affected by reporting bias. Since differences in environment and the socioeconomic background may produce different patterns of under-five mortality, the model life table was also used as an indirect method to study the age pattern of under-five mortality from the most reliable information.

2.2. Data

The main source of information is the *Demographic and Health Survey* (DHS). Particularly, the *complete maternity histories* of 252 public-domain surveys were analyzed. The countries and years of collection are listed in Table 1. In addition to other relevant characteristics, each record of the *births recode* includes the date of birth, and if deceased, the age at death. Dates of birth were reported by years and months. However, the age of death was reported in days when it occurred in the first 28 (neonatal), in months for the deaths of children under two years, and in years for those older than two. This information allows the calculation of death probabilities for different age intervals and at specific time periods. Records with incomplete information were not included and the estimates were adjusted to the expansion factors of the survey. For each survey, mortality schedules were calculated following the same approach of Somoza (1980) and Rutstein (1984), which has been specially developed for the estimation of infant and child mortality from retrospective sources such as the *World Fertility Survey* and the DHS. Using a Lexis diagram, the method is intended to produce period life tables of five calendar-years of exposure. However, if calculations are independent for each sex, the recommended period of exposure is ten years.

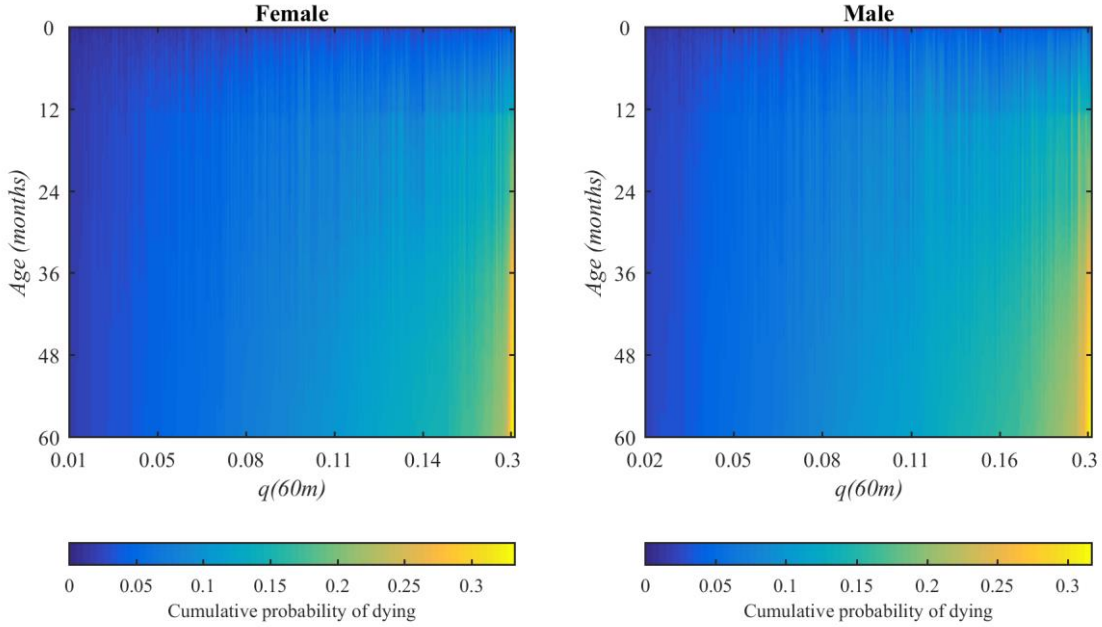
Table 1: DHS used for the analysis and the number of life tables per country

Central Asia	
Kazakhstan 1995, 1999	2
Kyrgyz Republic 1997, 2012	2
Tajikistan 2012	1
Uzbekistan 1996	1
Latin America & Caribbean	
Bolivia 1989, 1994, 1998, 2003, 2008	5
Brazil 1986, 1991, 1996	3
Colombia 1986, 1990, 1995, 2000, 2005, 2010	6
Dominican Republic 1986, 1991, 1996, 1999, 2002, 2007, 2013	7
Ecuador 1987	1
Guatemala 1987, 1995, 1998-99	3
Guyana 2005, 2009	2
Haiti 1994-95, 2000, 2005-06, 2012	4
Honduras 2005-06, 2011-12	2
Nicaragua 1998, 2001	2
Paraguay 1990	1
Peru 1986, 1991-92, 1996, 2000, 2007-08, 2009, 2010, 2011, 2012	9
Trinidad and Tobago 1987	1
North Africa/West Asia/Europe	
Albania 2008-09	1
Armenia 2000, 2005, 2010	3
Azerbaijan 2006	1
Egypt 1988, 1992, 1995, 2000, 2003, 2005, 2008, 2014	8
Jordan 1990, 1997, 2002, 2007, 2009, 2012	6
Moldova 2005	1
Morocco 1987, 1992, 2003-04	3
Tunisia 1988	1
Turkey 1993, 1998, 2003	3
Ukraine 2007	1
Yemen 1991-92, 2013	2
South & Southeast Asia	
Bangladesh 1993-94, 1996-97, 1999-00, 2004, 2007, 2011, 2014	7
Cambodia 2000, 2005, 2010, 2014	4
India 1992-93, 1998-99, 2005-06	3
Indonesia 1987, 1991, 1994, 1997, 2002-03, 2007, 2012	7
Maldives 2009	1
Nepal 1996, 2001, 2006, 2011	4
Pakistan 1990-91, 2006-07, 2012-13	3
Philippines 1993, 1998, 2003, 2008, 2013	5
Sri Lanka 1987	1
Thailand 1987	1
Timor-Leste 2009-10	1
Vietnam 1997, 2002	2
Sub-Saharan Africa	
Angola 2011	1
Benin 1996, 2001, 2006, 2011-12	4
Burkina Faso 1993, 1998-99, 2003, 2010	4
Burundi 1987, 2011	2
Cameroon 1991, 1998, 2004, 2011	4
Central African Republic 1994-95	1
Chad 1996-97, 2004, 2014-15	3
Comoros 1996, 2012	2
Congo 2005, 2012	2
Congo Democratic Republic 2007, 2013-14	2
Cote d'Ivoire 1994, 1998-99, 2005, 2011-12	4
Ethiopia 2000, 2005, 2011	3
Gabon 2000, 2012	2
Gambia 2013	1
Ghana 1988, 1993, 1998, 2003, 2008, 2014	6
Guinea 1999, 2005, 2012	3
Kenya 1989, 1993, 1998, 2008-09, 2014	5
Lesotho 2004, 2009, 2014	3
Liberia 1986, 2007, 2009, 2013	4
Madagascar 1992, 1997, 2003-04, 2009	4
Malawi 1992, 2000, 2004, 2010	4
Mali 1987, 1996, 2001, 2006, 2013	5
Mozambique 1997, 2003, 2011	3
Namibia 1992, 2000, 2006-07, 2013	4
Niger 1992, 1998, 2006, 2012	4
Nigeria 1990, 2003, 2008, 2010, 2013	5
Rwanda 1992, 2000, 2005, 2007-08, 2010, 2014-2015	6
Sao Tome and Principe 2009	1
Senegal 1986, 1992-93, 1997, 2005, 2008-09, 2010-11, 2012-13, 2014, 2015	9
Sierra Leone 2008, 2013	2
South Africa 1998	1
Sudan 1989-90	1
Swaziland 2006-07	1
Tanzania 1991-92, 1996, 1999, 2004-05, 2007-08, 2010	6
Togo 1988, 1998, 2013-14	3
Uganda 1988-89, 1995-96, 2000-01, 2006, 2009, 2011	6
Zambia 1992, 1996, 2001-02, 2007, 2013-14	5
Zimbabwe 1989, 1994, 1999, 2005-06, 2010-11	5
Total	252

Using the births reported in the last fifteen years and the deaths reported in the last ten years before the survey was conducted, two life tables were computed form each survey. This is one for each sex. Life tables were calculated in months of age during the first two years of life and in years after the age of two. Since neonatal deaths were reported in days of age, probabilities of death were calculated in days for the first week of life and in weeks for the first 28 days of life. These values were calculated from the cumulative probability of dying in the first month of life and using proportionality assumptions. First, it was assumed that exposure to the risk of dying is proportional to the length of the age interval. Second, it was assumed that the specific mortality rate is proportional to the number of deaths reported in that subinterval.

A general feature of the country-years included in the study sample is the wide range of mortality levels. For example, Figure 1 shows that in the first quintile of under-five mortality, the probability of dying in the first five years of life is less than 0.05, whereas in the fifth quintile of the distribution this probability is not less than 0.15, and in extreme cases it is higher than 0.30.

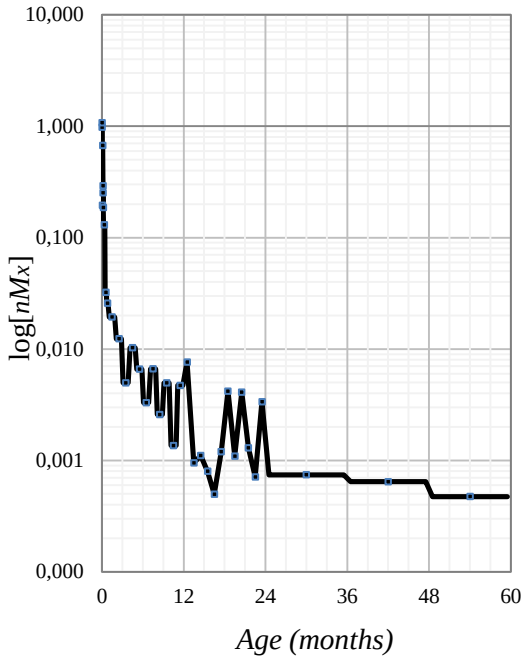
Figure 1: Cumulative probabilities of dying using all life tables sorted by $q(60m)$



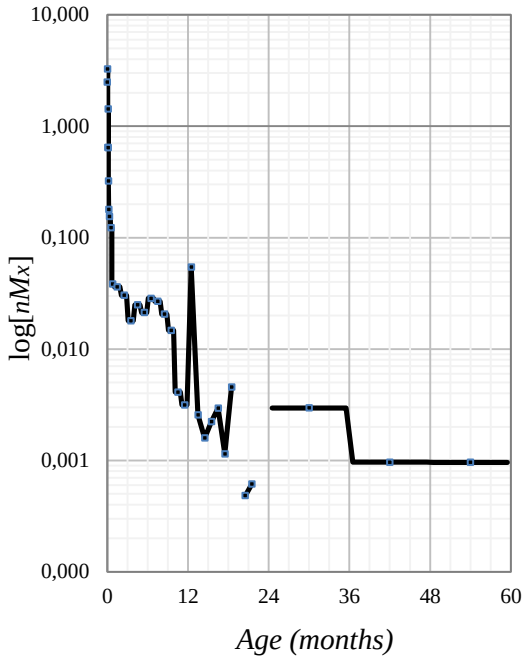
For a selection of country-years, Figure 2 shows age-specific mortality rates that were calculated from the complete maternity histories assuming that the force of mortality is constant at each subinterval of age. The fluctuations and discontinuities of age-specific mortality rates draw attention. These are characteristics that are not observed in the

mortality patterns calculated from vital records, which is the main limitation in the study of mortality patterns using self-reported data.

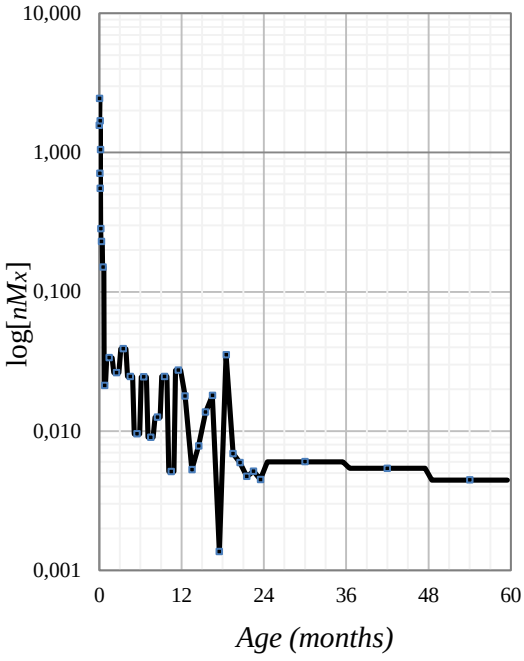
Figure 2: Age-specific mortality rates for males from selected country-years



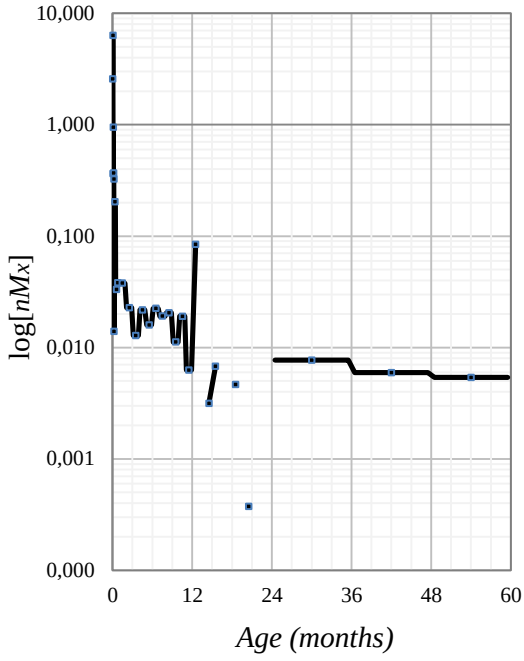
Colombia, 2010



Yemen, 2013



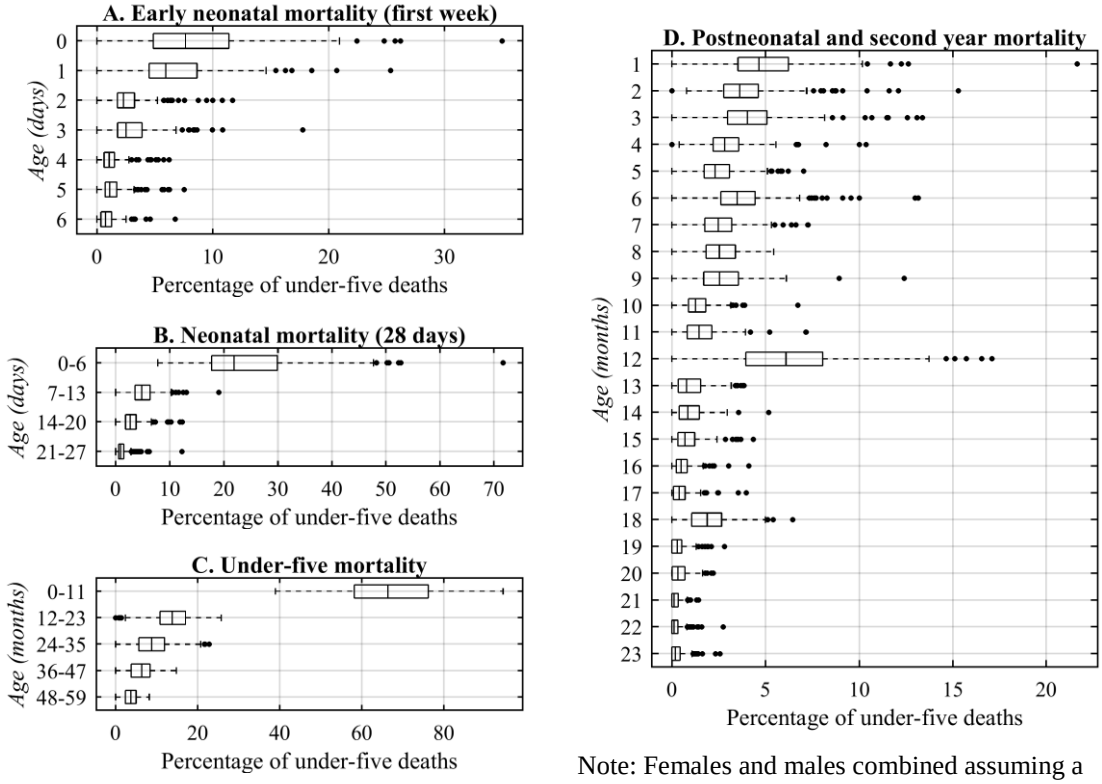
Senegal, 2015



Ghana, 2014

Complete maternity histories are self-reported data, thus errors in the calculation of probabilities of dying could be related to underreported births and the inaccuracy in reporting the age at death. Even more, these sources of errors can also be interdependent: respondents who experienced the early death of their children might be less likely to report their birth. A similar aversion would also exist in reporting the children not living with the mother at the time of the survey. These errors affect estimations in two ways. On the one hand, the underreporting of births due to early mortality creates bias in the estimates of the probability of dying during the first months of life. On the other hand, misreported ages at death also introduce bias in the calculation of the probability of dying by heaping the distribution of deaths at some specific ages while diminishing the number of deaths at adjacent ages. This possibility was examined comparing all life tables. Figure 3 shows the distribution of deaths at specific age intervals. In order to make them comparable, deaths were standardized by the total number of under-five deaths. In overall, life tables show a decrease in the percentage of deaths as age increases. At early neonatal ages (the first week of life), the proportion of deaths occurring on the first day of life is greater (boxplot A). During the first month of life, most deaths are grouped in the first week (boxplot B). The majority of under-five deaths occur in the first year of life (boxplot C).

Figure 3: Boxplots for the distribution of life table deaths by days and months of age



Note: Females and males combined assuming a sex ratio at birth of 105 males per 100 females.

However, the heaping in the proportion of deaths at the ages of 6, 12 and 18 months is a significant limitation of DHS estimates (boxplot D). According to Figure 3, the percentage of deaths at 12 months of age is unusually higher than the percentage of deaths at adjacent ages (11 or 13 months). In a very small magnitude, heaping also occur at the age of six and 18 months. These groupings could bias some results, such as the infant mortality. If ages at death were approximated by excess, the deaths of children under one year old but close to turning one, will weigh on the mortality that occurs in the second year of life. Improving on this limitation is the main objective of this paper.

2.3. Empirical validation of the DHS estimates

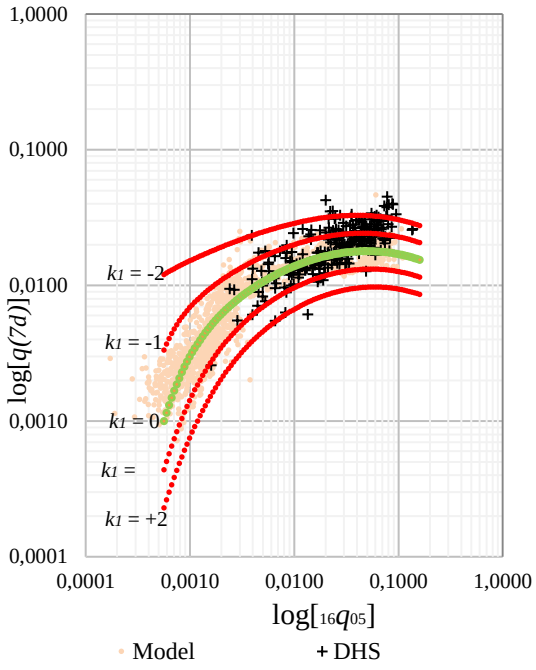
DHS estimates were compared to a model life table for under-five mortality. Given a probability of dying within a defined age range, the model allows to infer the probabilities of dying at all other ages. The implicit advantage of this comparison is to identifying those ages whose probabilities of dying differ systematically from a prediction based on vital records. These differences are explained by two competing arguments. On the one hand, DHS estimates might not fit the model because of the reporting bias. Hence, the model would provide an adequate standard to correct survey estimates. On the other hand, given that the model was largely based on European data, systematic differences might result of distinct mortality patterns. However, the model life table of under-five mortality has two advantages. First, the model has the capacity to reproduce patterns of high and low mortality, thus indirect estimations would not rely on extrapolations. Second, the model is flexible to adapt the mortality pattern to the particular characteristics of the study population. This is particularly useful when more reliable information is available and the model is demanded to match at least two entry values.

Although the model was estimated using $q(24m)$ as a predictor, the probability of dying at the age interval $[5m, 21m)$ was chosen as the input value to be matched using the model as standard for comparison. This probability of dying, which in conventional notation is defined as: ${}_{16}q_{05}$, is independent of the deaths occurring during the first 5 months of life, thus not affected by potential biases in the report of early mortality. In addition, the age interval includes the probabilities of dying at 6, 12 and 18 months of age which are affected by misreported ages at death, but also the probabilities of dying at adjacent ages. Therefore, ${}_{16}q_{05}$ is less conditioned to the inaccuracy in reporting ages at dying. Using all the surveys, Figure 4 shows the dispersion between the probability of dying in the interval $[5m, 21m)$ and the probabilities of dying at some relevant age intervals: the early neonatal mortality (first week), the neonatal mortality (first month), the infant mortality (all children under one year), and the under-five mortality (children

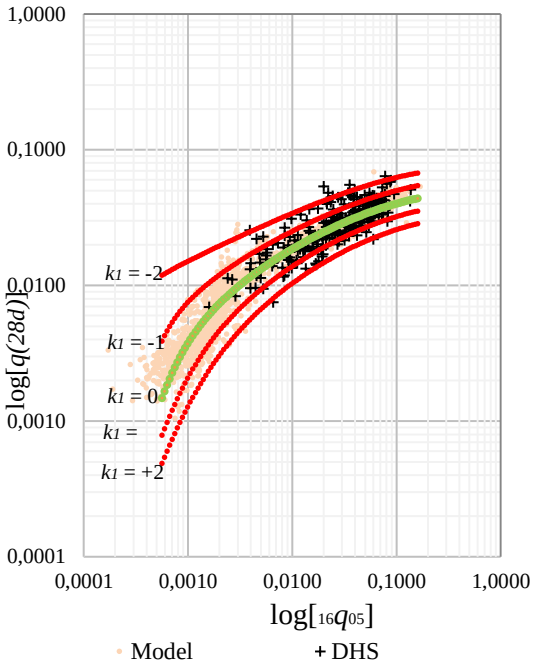
younger than 5 years). Figure 4 also shows log-quadratic estimates from a model life table based on vital records, using as a predictor the cumulative probability of dying at the age of two years $q(24m)$, and a flexible second parameter k_1 modeling the shape of the mortality. Four deductions can be made from Figure 4. First, early neonatal mortality calculated from the surveys could be greater than the one predicted by the model. Second, with few exceptions, neonatal mortality is estimated at a feasible range of data, $k_1 = \pm 2$. Third, given the clustering of points below the $k_1 = +2$ line, it might be possible that surveys underestimate infant mortality. Fourth, surveys allow a reasonable calculation of the under-five mortality.

Although a surveys based analysis allows a reasonable estimate of neonatal mortality, and under-five mortality, some biases occur at other age intervals. Figure 5 shows that the probabilities of dying in the fourth trimester $[9m, 12m)$, are systematically lower than those predicted by the model. However, these are partially compensated with higher probabilities in the fifth quarter $[12m, 15m)$. Biases continue as age increases and mortality decreases, and the most prominent discrepancies are observed in the second year of life. Figure 5 shows that the probability of dying at ages $[15m, 18m)$ and $[21m, 24m)$, are underestimated in most surveys. This suggests that the probabilities of dying calculated from the DHS might have some bias when the age at death is reported inaccurately or approximately. Considering the misreported ages at death, the conventional approach to calculate the infant mortality, the child mortality, and the under-five mortality is to use broad age intervals: first month of age, 1-2 months, 3-5 months, 6-11 months, 12-23 months, 24-35 months, 36-47 months, and 48-59 months. By doing this, it is possible to reduce the variability in the number of deaths reported by single months of age. However, these intervals do not avoid that some deaths that occurred just before the first year of life were counted as early child mortality (second year of life), when the ages were reported by making an approximation by excess.

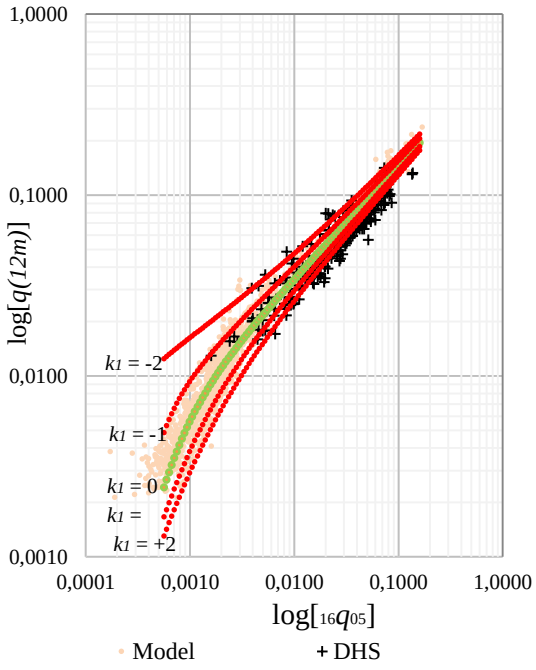
Figure 4: Cumulative probabilities of dying $q(x)$ vs. the probability of dying at the age interval $[5m, 21m)$. Log-quadratic estimates are from vital records using $q(24m)$ as a predictor, and five values of k_1
Female



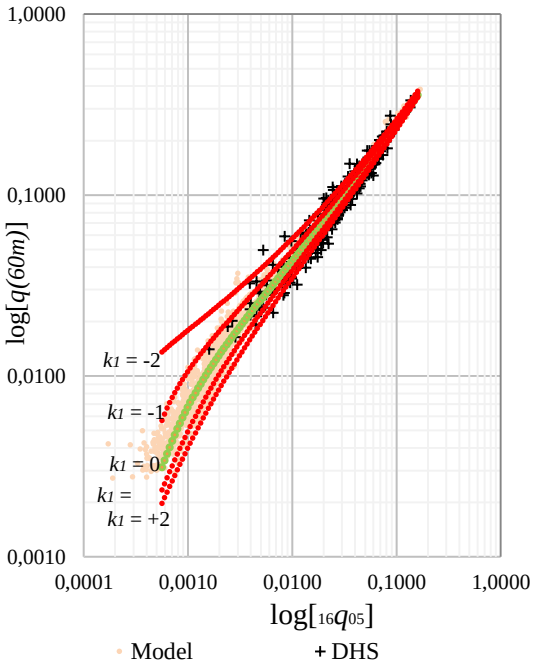
$q(7d)$



$q(28d)$



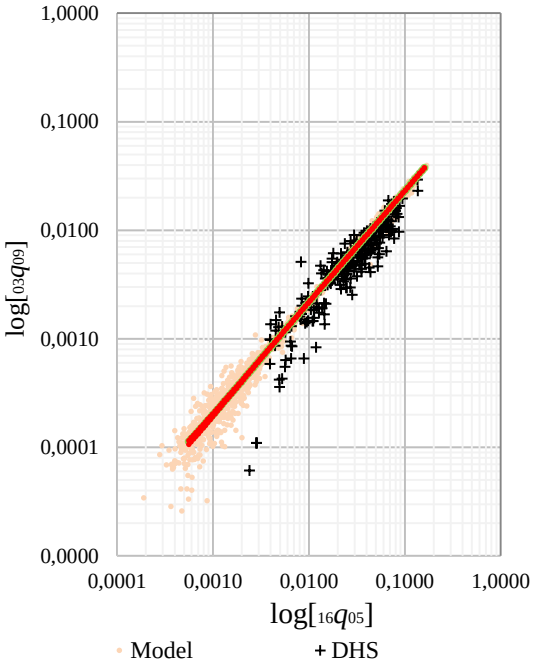
$q(12m)$



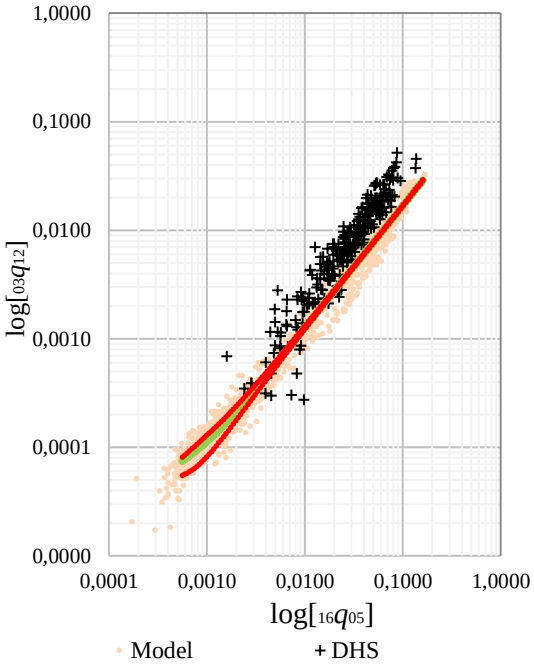
$q(60m)$

Figure 5: Probability of dying at selected age intervals vs. the probability of dying at the age interval $[5m, 21m)$. Log-quadratic estimates are from vital records using $q(24m)$ as a predictor, and five values of k_1

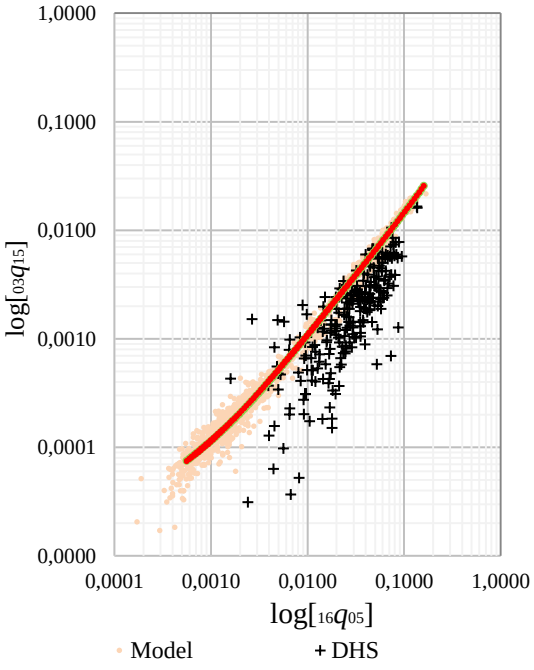
Female



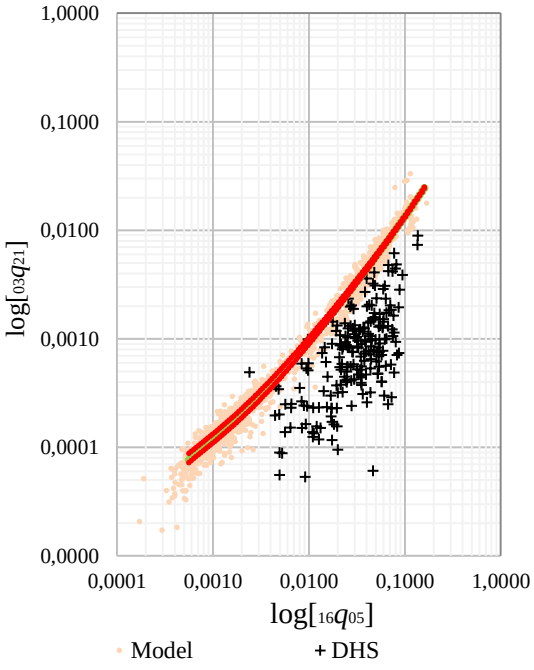
Age interval [9*m*,12*m*)



Age interval [12*m*,15*m*)



Age interval [15*m*,18*m*)



Age interval [21*m*,24*m*)

2.4. Estimation of the mortality patterns at early ages

A conventional approach to study mortality patterns using DHS estimates is to contrast the dispersion of infant mortality and child mortality with model life tables. In particular, DHS estimates are compared to the *North*, *South*, *East*, and *West* families of the regional model proposed by Coale & Demeny (1966). From these comparisons, it is usually established if the countries investigated in the DHS follow similar patterns to those pre-established from the empirical life tables. This allows the graphical assessment of late mortality patterns (early), meaning those where child mortality is higher (lower) than the predicted by a given level of mortality, for example infant mortality. However, in complete maternity histories, infant mortality may be underestimated due to the fact that ages of death are self-reported data. In consequence, there is a need to study mortality patterns from the probabilities of dying operating at different ages.

This paper aims to an alternative approach, in which estimates made from DHS are compared to a model life table for under-five mortality. The model includes the following age intervals: *(1*) days within the first week; *(2*) weeks to complete the first month of life of 28 days longer; *(3)* months of age to complete the first year of life; *(4)* trimesters of age within the second year of life; and *(5)* years to complete the first five years of life. The first advantage to use a model like this is to consider some ages that are relevant to the mortality patterns, but not contemplated in conventional models; for example, the neonatal mortality. The second advantage is the possibility to adjust additional parameters in order to reproduce observable characteristics of the population to be studied, and make an indirect estimation of a complete mortality schedule at early ages.

Considering that the complete maternity histories allow a reasonable calculation of the neonatal mortality $q^s(28d)$ and the under-five mortality $q^s(60m)$ as shown in Figure 4, a model life table was adjusted using as the most reliable information these two probabilities of dying. In addition to the fitness of the neonatal mortality estimates, there are also some other arguments for choosing this probability of dying as an input value. Inasmuch as the first month of life is the one with the highest mortality rate regardless of the historical context or the economic background, neonatal deaths are a more frequent event. Hence, compared to other ages of equal length, survey estimates of the neonatal mortality are less affected by problems related to sample size.

From equation (1) and using numerical methods, the optimal values of h and k_1 (the level and shape of the mortality) were calculated in order to match the DHS estimates.

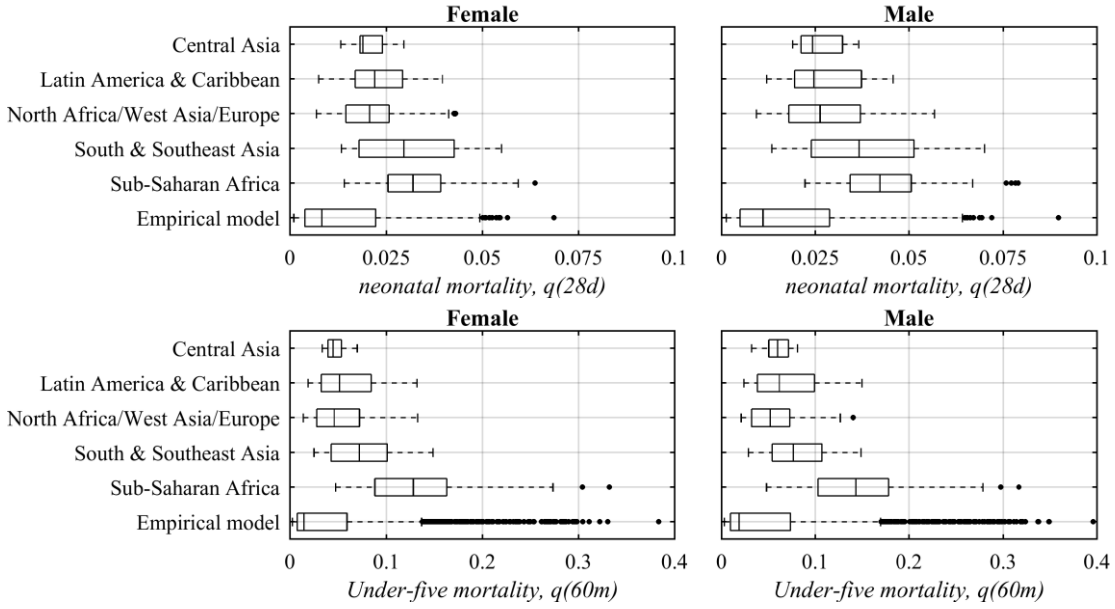
$$\begin{bmatrix} ln(q^s(28d)) \\ ln(q^s(60m)) \end{bmatrix} = \begin{bmatrix} \hat{\beta}_{1,28d} & \hat{\beta}_{2,28d} & \hat{\beta}_{3,28d} \\ \hat{\beta}_{1,60m} & \hat{\beta}_{2,60m} & \hat{\beta}_{3,60m} \end{bmatrix} \cdot \begin{bmatrix} 1 \\ ln(h) \\ ln(h)^2 \end{bmatrix} + \begin{bmatrix} U_{1,28d} \\ U_{1,60m} \end{bmatrix} \cdot k_1. \quad (1)$$

The panel A from Figure 6 shows boxplots of the neonatal and the under-five mortality, grouping the data according to the regions defined by the DHS program. The distributions of the country-years used to fit the log-quadratic model are also shown. Although the model was estimated using records from 24 low-mortality countries, the use of historical statistics allows a wide range of data. Thus, estimates from populations with higher mortality levels, such as those observed in Sub-Saharan Africa or South and Southeast Asia, are based on true experiences rather than extrapolations.

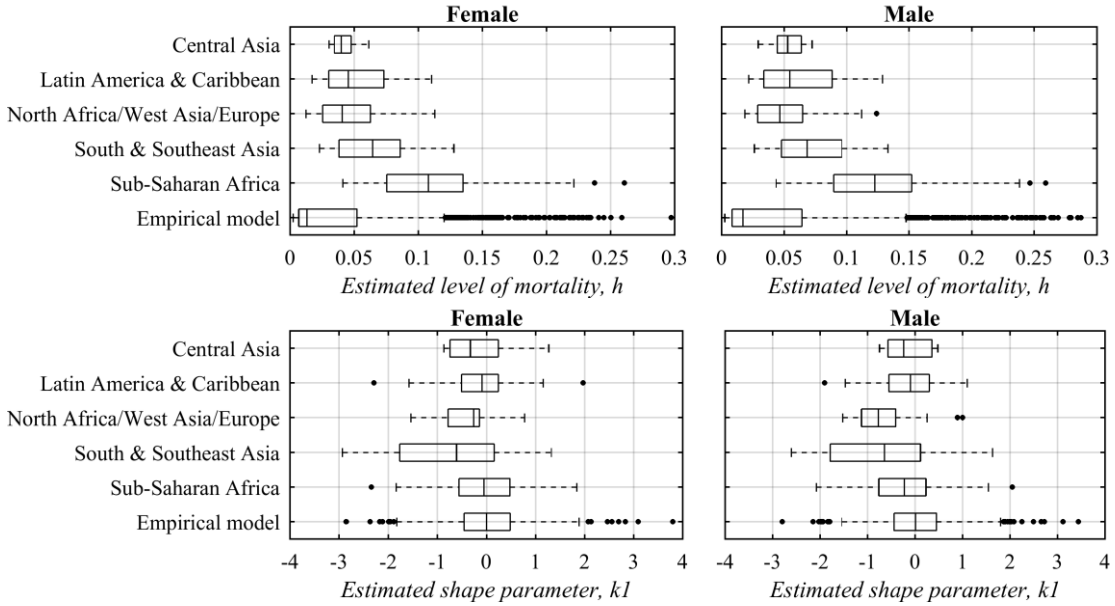
The distribution of the key parameters modeling the level and shape of the mortality for a given values of the neonatal and the under-five mortality are shown on panel B of Figure 6. On the one hand, the parameter h allows to compare the level of mortality of different populations, assuming that they follow the same mortality pattern. Thus, on average, the level of mortality estimated from DHS is higher than the mortality level of the country-years used to estimate the life table model. On the other hand, the parameter k_1 allows to compare the mortality pattern of different populations, assuming that they have the same level of mortality. Hence, a positive value of k_1 decreases the cumulative probabilities of dying before the age of two, while it increases the cumulative probabilities of dying from 2 to 5 years of age. As shown in the boxplots, negative values of $k_1$were estimated in the majority of surveys from South and Southeast Asia. This indicates that, compared to the model, the cumulative probabilities of dying are higher before the second year of life, although lower than those predicted by the model after this age. This result is assuming that the entire mortality pattern could be inferred from neonatal mortality and under-five mortality alone.

Figure 6: Boxplots for the matching values and the key parameters of the model

Panel A: *neonatal mortality and under-five mortality*

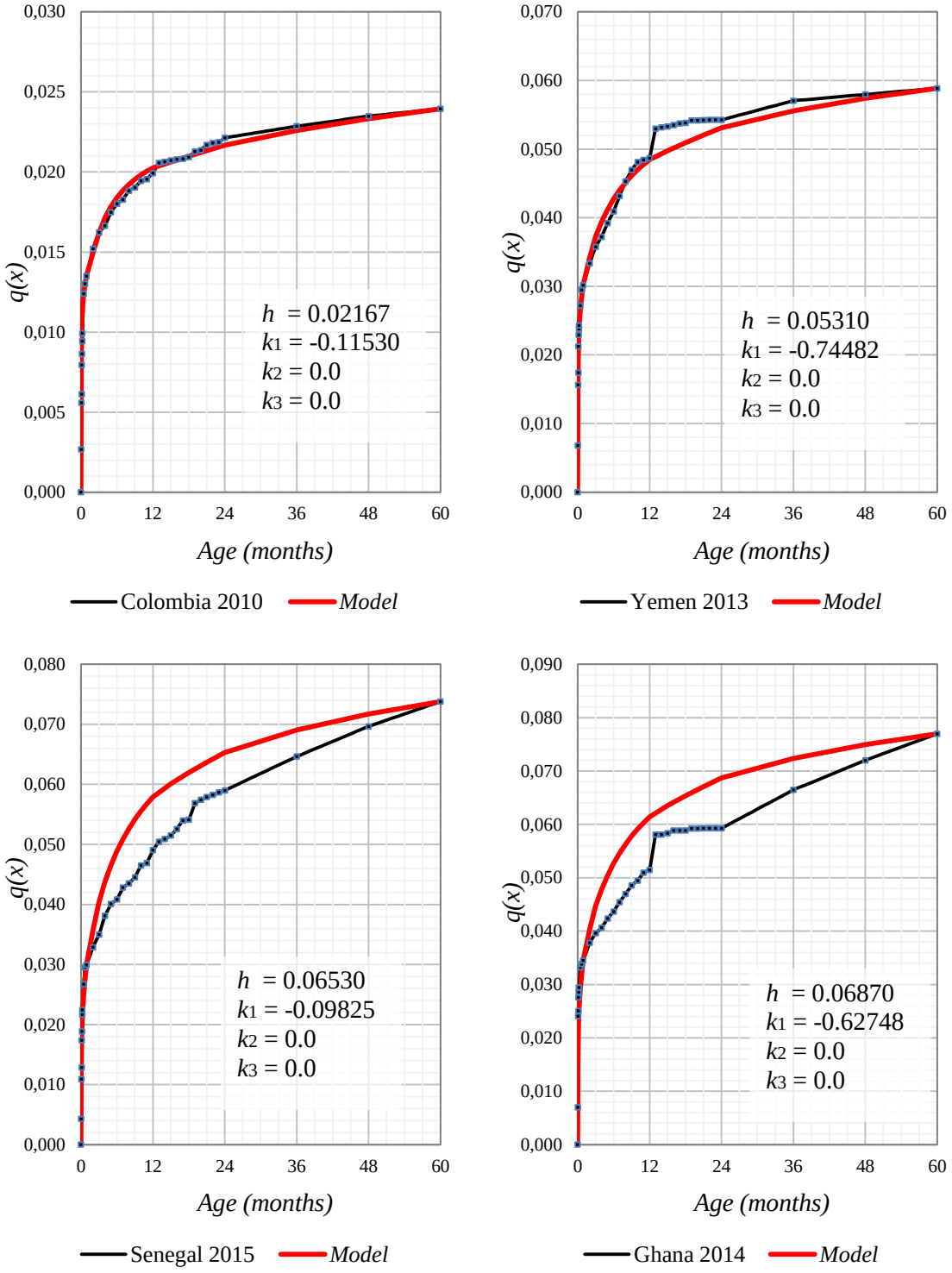


Panel B: *key parameters of the model*



Note: Central Asia (6 life tables); Latin America and the Caribbean (46); North Africa, West Asia, and Europe (30); South and Southeast Asia (39); Sub-Saharan Africa (131); and the empirical model (1,319).

Figure 7: Cumulative probabilities of dying for males from selected country-years DHS estimates vs. model life table predictions using $q(28d)$ and $q(60m)$ as matching values



Using the equation (2) and given the optimal values of the parameters h and k_1, cumulative probabilities of dying were calculated for each survey in the analysis. The DHS estimates $q^s(x)$ were then contrasted with those resulted from the model $q^m(x|h^*, k_1^*)$, as shown in Figure 7 for selected populations. Keeping constant the neonatal and the under-five mortality, the contrast allows to identify those populations that are characterized by late patterns of under-five mortality, such as Senegal in 2015, and Ghana in 2014; in contrast to others that are satisfactorily adjusted to the model such as Colombia in 2010; or those exhibiting early patterns, such as Yemen in 2013.

$$ln(q^m) = \hat{\beta}_{28\times 3} \cdot [1, ln(h), ln(h)^2]' + \mathrm{U}_{28\times 1} \cdot k_1. \quad (2)$$

Differences in mortality patterns were quantified as the difference in the logarithm of the area under the curve $q^s(x)$ and the area under the curve $q^m(x|h^*, k_1^*)$, as shown in equation (3). The logarithmic function produces relative differences between the DHS estimates and the model predictions. Thus, populations having different levels of mortality can be compared.

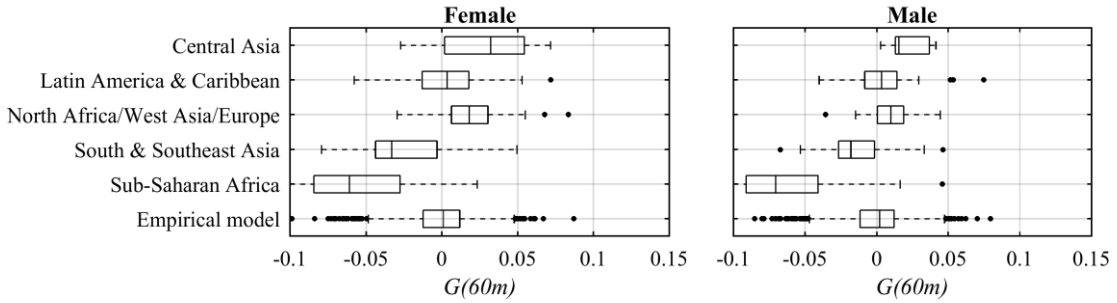
$$G(60m) = ln\left[\frac{\int_0^{60m} q^s(x)dx}{\int_0^{60m} q^m(x|h^*,k_1^*)dx}\right]. \quad (3)$$

The intuition behind the equation (3) is quite simple. For a given level of mortality, negative values of $G(60m)$ indicate late patterns and suggest a lower mortality within the first months of life that is compensated with an excess of mortality during childhood. While the positive values of $G(60m)$ indicate an early pattern and suggest that, compared to the model, there is a higher mortality within the first months of life. In light of the foregoing, the level and shape of the mortality schedule are somehow related. Using all surveys and ignoring clusters by country or region, the correlation between the parameter h and the function $G(60m)$ is estimated to be -0.497 for females and -0.609 for males. This result suggests that some populations with high mortality are characterized by late patterns.

Figure 8 shows boxplots for the differences in mortality patterns, i.e. $G(60m)$. The results suggest regional differences in the pattern of under-five mortality. Keeping constant the neonatal and the under-five mortality, there are substantial differences between the model and the DHS estimates. Compared to the model, in most of the country-years conforming South and Southeast Asia, and Sub-Saharan Africa, not only high levels of mortality were observed (Figure 6, panel B), but also late patterns were estimated (Figure 8). Conversely, the majority of country-years from the regions of Central Asia, North Africa, West Asia, and Europe (DHS) exhibited early patterns of

under-five mortality. Whereas in Latin America and the Caribbean there was observed an even distribution of late and early patterns of under-five mortality.

Figure 8: Boxplots for the differences in mortality patterns



Note: Central Asia (6 life tables); Latin America and the Caribbean (46); North Africa, West Asia, and Europe (30); South and Southeast Asia (39); Sub-Saharan Africa (131); and the empirical model (1,319).

Although the model was not intended to estimate a mortality pattern for each DHS survey, it was used to quantify significant departures from a standard mortality schedule. For a given value of the neonatal and the under-five mortality, this comparison allows to infer the concomitance of the level and the pattern of mortality.

2.5. Smoothing the heaping at the age of 12 months

As a method of indirect estimation, the model was used to estimate infant mortality $q(12m)$ from the cumulative probabilities of dying at 9 and 18 months of age. This is by solving equation (4) for the values of h and k_1, and assuming that these probabilities, $q^s(9m)$ and $q^s(18m)$, are both reliable and reasonably calculated from the DHS.

$$\begin{bmatrix} ln\left(q^s(9m)\right) \\ ln\left(q^s(18m)\right) \end{bmatrix} = \begin{bmatrix} \hat{\beta}_{1,9m} & \hat{\beta}_{2,9m} & \hat{\beta}_{3,9m} \\ \hat{\beta}_{1,18m} & \hat{\beta}_{2,18m} & \hat{\beta}_{3,18m} \end{bmatrix} \cdot \begin{bmatrix} 1 \\ ln(h) \\ ln(h)^2 \end{bmatrix} + \begin{bmatrix} \mathrm{U}_{1,9m} \\ \mathrm{U}_{1,18m} \end{bmatrix} \cdot k_1. \quad (4)$$

Once the parameters of the model were calculated, infant mortality was projected from equation (5). Similarly, the model was used to predict the cumulative probability of dying at all other ages adjacent to the 12 months of life in order to smooth the data around.

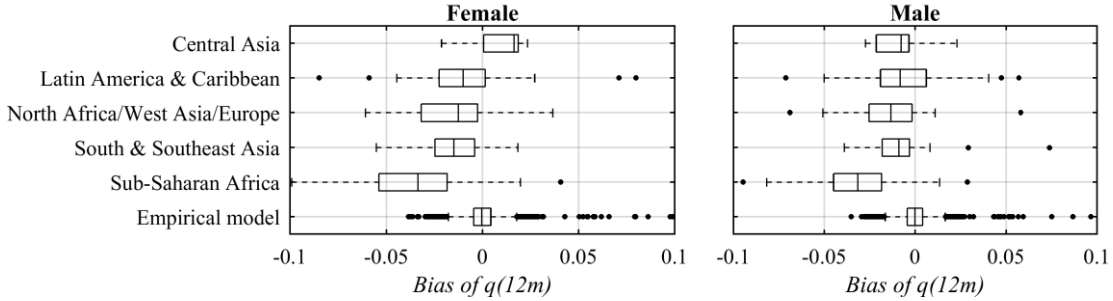
$$ln\left(q^m(12m)\right) = \begin{bmatrix} \hat{\beta}_{1,12m} & \hat{\beta}_{2,12m} & \hat{\beta}_{3,12m} \end{bmatrix} \cdot \begin{bmatrix} 1 \\ ln(h) \\ ln(h)^2 \end{bmatrix} + \mathrm{U}_{1,12m} \cdot k_1. \quad (5)$$

Nevertheless, the bias of the DHS is assumed to depend on the punctual estimation of the infant mortality and was calculated by equation (6).

$$Bias = \ln[q^s(12m)] - \ln[q^m(12m)] \quad (6)$$

Figure 9 shows boxplots for the bias of the infant mortality estimates. In most cases, the model predicts a higher value than that calculated directly from surveys. This indicates that the DHS estimates might underestimate the value of infant mortality, regardless of gender and for the majority of countries-years included in the analysis. Figure 9 also allows determining the magnitude of the bias. Although there are extreme observations and cases of overestimation, in most cases the bias does not exceed the 5 percent of infant mortality. Consequently, there would be a greater concern in the measurement of infant mortality in the region of Sub-Saharan Africa, where the level of infant mortality and the size of the bias are the highest. However, it should also be considered that this difference might be exacerbated by the already mentioned differences in the age-patterns of mortality of Sub-Saharan populations and those used to estimate the empirical model.

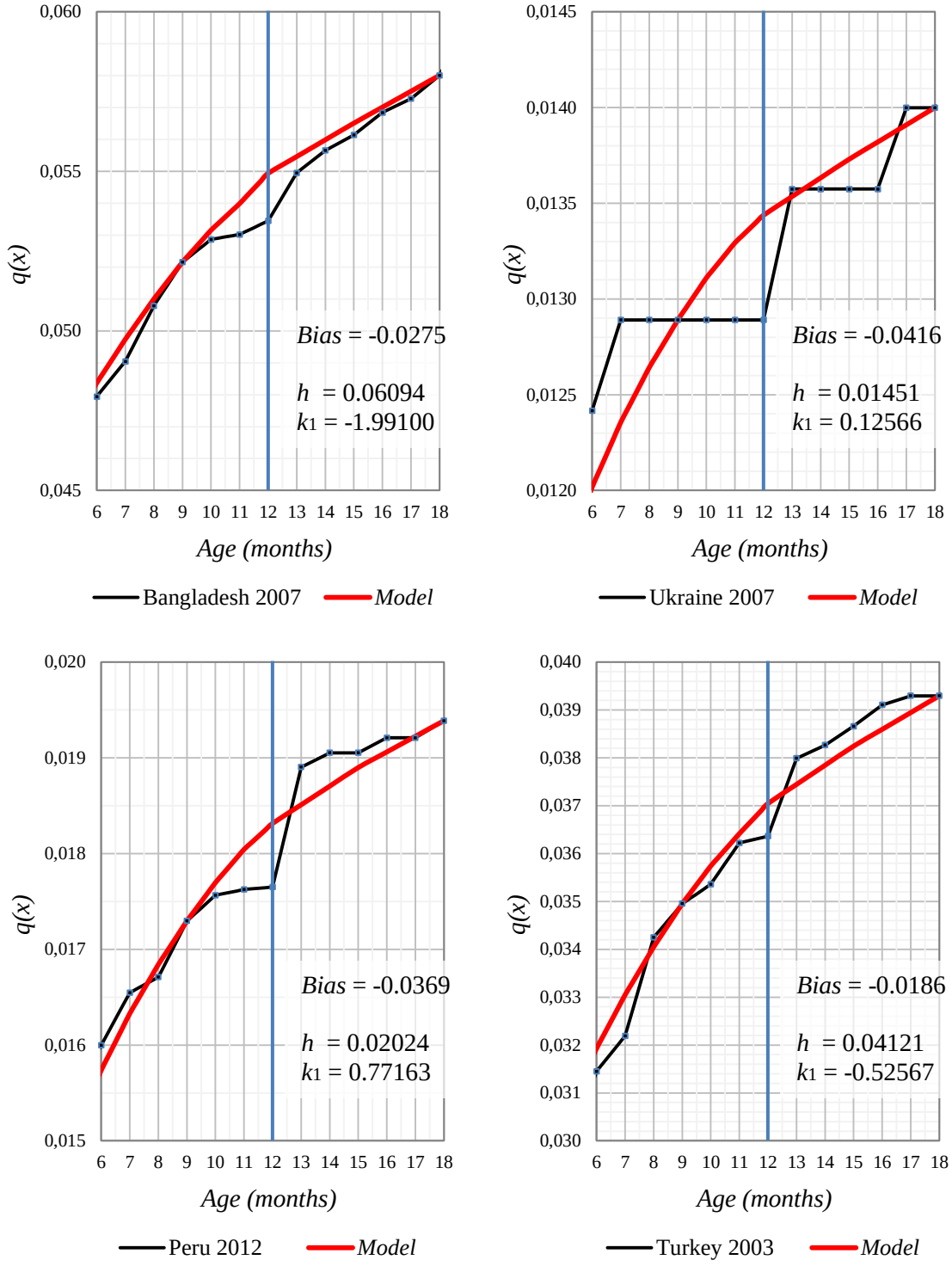
Figure 9: Boxplots for the bias of the infant mortality estimates



Note: Central Asia (6 life tables); Latin America and the Caribbean (46); North Africa, West Asia, and Europe (30); South and Southeast Asia (39); Sub-Saharan Africa (131); and the empirical model (1,319).

For a selection of countries-years, Figure 10 shows the cumulative probability of dying at ages adjacent to the 12 months of age. For each case, DHS estimates were compared to model predictions. In order to assess the bias of survey estimates, model predictions help to smooth the cumulative probability of dying and provide an indirect estimation of the infant mortality. The Figure 10 shows the discontinuity in the cumulative probability of dying at the age of 12 months. In particular, the discontinuity is a result of ages at death which are rounded by excess, diminishing the number of deaths occurring in the eleventh and twelfth month of life. Therefore, misreported ages at death lead to an underestimation of infant mortality and an overestimation of mortality in the second year of life. However, the indirect estimation based on model life tables is a relevant method to detect and correct the bias of survey estimates.

Figure 10: Cumulative probabilities of dying for females from selected country-years DHS estimates vs. model life table predictions using $q(9m)$ and $q(18m)$ as matching values



2.6. Discussion

This paper analyzes the age patterns of under-five mortality estimated from the Demographic and Health Survey by retrospective methods. The resulting mortality schedules were compared to model predictions, by taking advantage of an empirical model life table. The model includes relevant ages for the indirect estimation of the level and shape of the mortality at early ages. The analysis shows that DHS estimates of neonatal and under-five mortality fall on a range of feasible values that were estimated from vital records. Hence, these probabilities were used to infer the probabilities of dying at other ages. For a given level of neonatal and under-five mortality, a complete mortality schedule was estimated for each survey allowing to contrast the cumulative probabilities of dying. Although this approach is not intended to correct the possible errors of the DHS estimates, it was used to quantify the relative differences between model predictions and survey estimates. These differences help to illustrate how different are the mortality patterns that characterize each of the regions defined in the DHS. In this regard, this paper also provides evidence of the concomitance of late patterns and high levels of mortality at early ages. Hence, this paper contributes to the discussion in Guillot, *et al*. (2012) on the need to examine DHS estimates using detailed age groups and model life tables with the same age intervals.

This paper also examines quality issues of the DHS regarding early mortality estimates. At some ages, disparities could be more related to reporting bias and not necessarily to actual mortality patterns. In particular, ages at death are affected by rounding errors in retrospective sources, resulting in some heaping, for example, at the age of 12 months. This finding is consistent with the results of early assessments that conclude in a possible underestimation of the infant mortality and a likely overestimation of the child mortality. In this regard, this paper contributes to the discussion by proposing an indirect method to assess the bias of surveys estimates.

2.7. References

AbouZahr, C. et al., 2015. Towards universal civil registration and vital statistics systems: the time is now. *The Lancet,* Volume 386, pp. 1407-1418.

Bicego, G., Chahnazarian, A., Hil, K. & Cayemittes, M., 1991. Trends, age patterns and differentials in childhood mortality in Haiti (1960-1987) . *Population Studies,* 45(2), pp. 235-252.

Brass, W., 1996. Demographic data analysis in less developed countries: 1946-1996. *Population Studies,* 50(3), pp. 451-467.

Brass, W. & Coale, A. J., 1968. Methods of Analysis and Estimation. En: *The Demography of Tropical Africa.* Princeton, NJ: Princeton University Press, pp. 88-150.

Coale, A. J. & Demeny, P., 1966. *Regional Model Life Tables and Stable Populations.* Princeton: Princeton University Press.

Curtis, S., 1995. *Assessment of the quality of data used for direct estimation of infant and child mortality in DHS-II surveys,* DHS Occasional Papers No. 3: Calverton, Maryland. Macro International Inc. http://www.measuredhs.com/pubs/pub_details.cfm?ID=161 .

Guillot, M., Gerland, P., Pelletier, F. & Saabneh, A., 2012. Child mortality estimation: a global overview of infant and child mortality age patterns in light of new empirical data. *PLOS Medicine,* 9(8), pp. 1-15.

Hill, K., 1991. Approaches to the measurement of childhood mortality: a comparative review. *Population Index,* 57(3), pp. 368-382.

Mahy, M., 2003. *Childhood mortality in the developing world: a review of evidence from the demographic and health surveys,* DHS Comparative Reports No. 4: Calverton, Maryland. ORC Macro. http://www.measuredhs.com/pubs/pub_details.cfm?ID=450.

Rutstein, S. O., 1984. *Infant and child mortality: levels, trends, and demographic differentials,* World Fertility Survey Comparative Studies No 43: Voorburg, Netherlands. International Statistical Institute.

Somoza, J., 1980. *Illustrative analysis: infant and child mortality in Colombia,* WFS Scientific Report No.10: Voorburg, Netherlands. International Statistical Institute.

Sullivan, J. M., Rutstein, S. O. & Bicego, G. T., 1994. *Infant and child mortality,* DHS Comparative Studies No. 15: Calverton, Maryland. Macro International Inc. http://www.measuredhs.com/pubs/pub_details.cfm?ID=23.

Trussell, J. & Menken, J., 1984. Estimating levels, trends, and determinants of child mortality in countries with poor statistics. *Population and Development Review,* Volume 10 (Supplement on Child Survival: Strategies for Research), pp. 325-346.

Chapter 3

Health dynamics and survival expectations of mature adults in rural Malawi[1]

This chapter is co-authored with Hans-Peter Kohler[2] and Iliana V. Kohler[3]

Abstract

This paper investigates the reciprocal interaction of physical and mental health using a sample of mature adults aged 45+, who participated in the *Malawi Longitudinal Study of Families and Health* between 2006 and 2012. Four continuous assessments of a *12-item Short-Form Health Survey* were administrated to measure the physical and mental health of the participants. This information was supplemented by survival expectations, that were elicited from the self-reported distribution of probabilities of dying, using numerical scales and different lengths of exposure. Structural equation models were estimated assuming intertemporal relationships between physical health, mental health, and the formation of survival expectations. These models identify different pathways that have been discussed from theoretical and empirical approaches showing evidence of the concomitancy of physical and mental health issues, and the relevance of expectations about life. As an identification strategy, the effect of mental health on physical health was assumed to be lagged by two years, while it requires some adaptation and the adjustment of behaviors. Given these characteristics, this paper quantifies a significant impact of mental health on the prospective physical health, and provides evidence on the differentiated adaptation pathways for men and women.

Key words: Malawi Longitudinal Study of Families and Health; physical and mental health; subjective expectations; *Short*-Form Health Survey; longitudinal panel; Structural Equation Models.

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[2] Frederick J. Warren Professor of Demography and Professor in Sociology, University of Pennsylvania.
[3] Research Assistant Professor of Population Studies. Population Studies Center, University of Pennsylvania.

3.1. Introduction

Chronic, progressive, and debilitating forms of physical illness have psychosocial consequences affecting the quality of life and wellbeing. Although physical limitations can cause some interference with physical activities, work, and income; physical limitations also affect mental health domains such us the perception of the self and the future; the interaction with the others; and the satisfaction with life (de Ridder, et al., 2008; Powdthavee, 2009; Jokela, et al., 2014). Individuals suffering of physical illness also report a diminished quality of life (Connell, et al., 2014). Given that physical problems impose an additional deal of stress, some psychological disorders, such as depression, are expected to be more prevalent in individuals suffering of physical illness and disability (Sharpe & Curran, 2006). Indeed, longitudinal studies have shown evidence of the linking pathways from physical to mental health through the inflammation hypothesis of depression: persistently high levels of inflammation markers are significant predictors of depressive symptoms 5 to 10 years later (Kivimäki, et al., 2014).

Since physical illness may have psychological impacts, a process of adaptation is desirable. This process implies a behavioral response that will depend on the individual perception of a poor health, the expected gains to maintain a health status, and the barriers to adapt behaviors (Sharpe & Curran, 2006). To preserve the functional status, the psychological balance, and the satisfaction about life are three of the multiple aspects for an adaptive process to be successful (Stanton, et al., 2007; de Ridder, et al., 2008). Although permanent and disabling forms of physical illness would have a minimal impact on mental health, failure in adapting to the disease has a negative effect on the prospective status of physical health by making the course of the disease even worse. This effect would be exacerbated by a poor adherence or a complete lack of treatment, and the adoption of unhealthy behaviors that might result of a lack of optimism and negative expectations about life.

In this paper, structural equation models are used to investigate the reciprocal interaction of physical and mental health, as an intertemporal process that is linked to survival expectations. Individuals form expectations about future life events and adapt behaviors in consequence. Expectations might have an impact on the prospective status of physical health, but the perception of the self and the future is related to the mental health of individuals as depressed individuals tend to be more pessimistic about their lives. The empirical exercise was implemented from the *Malawi Longitudinal Study of Families and Health*. In particular, the analysis is focused on the cohort of mature adults (45+ in 2012) who participated between 2006 and 2012. Four assessments of a *12-item Short-Form Health Survey* (SF12) were administrated, using a formulary adapted to the context of Sub-Saharan Africa. Participants also reported subjective expectations about cumulative

death probabilities for different lengths of exposure: 1, 5, and 10 years; and using numerical scales. The self-report of these events allows the elicitation of survival expectations. Subjective expectations about the length of life or survival probabilities are measurements of the wellbeing of the individuals, as healthy individuals are more confident about the future and expect to live more years.

The cohort of mature adults in rural Malawi is a relevant case to the study health dynamics and the expectations about life. Compared to other age groups, mature adults in Malawi are projected to grow faster and the number of years lived with limitations is expected to increase, given that the process of epidemiological transition will rise the prevalence of non-communicable diseases (Payne, et al., 2013). Indeed, chronic conditions increase the chances to develop mental disorders (Prince, et al., 2007), and mental disorders have a considerable impact on the Global Burden of Disease (Collins, et al., 2013; Whiteford, et al., 2013). Specifically, symptoms of depression constitute a major cause for disability and severe depression is associated with higher risk of mortality (Prince, et al., 2007; Roiser, et al., 2012). This is not a problem of less importance since mental disorders are equally affecting developed and less developed societies through a loss of productivity and job absenteeism (Lim, et al., 2000; Dewa & Lin, 2000; Dewa, et al., 2007; Canavan, et al., 2013). However, much worse is the problem in less developed societies that have precarious mechanisms of social security and health care.

3.2. Theory and evidence

To some extent, the association of physical and mental health can be more reciprocal than causal. Research based on semi-structured interviews suggest that mental disorders have a potential negative effect on physical health due to insomnia, eating disorders, and the side effects of some medications (Connell, et al., 2014). One leading argument explaining the reciprocal interaction of physical and mental health domains is the comorbidity of physical and psychological disorders. In a sample of mature adults, negative perceptions of the health status predict a short-term increase in depressive symptoms, although depressive symptoms only have a moderated effect on the reported health status (Meeks, et al., 2000). A second argument is the linking pathways from mental to physical health through behavioral responses and cognitive distortions, as they can increase the risk of mortality and reduce the length of life (Cohen & Rodriguez, 1995). On the other hand, major depression has been linked to viral infections through a decreased immunity and delays in seeking for an appropriate medical help (Coughlin, 2012).

Not all individuals adapt in the same way to the changes in their health status, hence background characteristics are also important. A longitudinal study supports the fact that

symptoms of depression at older ages are associated with a higher risk of mortality through cardiovascular disease in the case of males; while females report higher scores of depression but cope with a lower risk of mortality (Scafato, et al., 2012). Selection is also a plausible mechanism in explaining the different pathways of adaptation. For example, given that patients with schizophrenia are less likely to use health services, lower detection rates of cancer and ischemic heart disease are the leading explanation behind the early mortality associated to this mental health condition (Crump, et al., 2013). However, this is also evidence of the significance of behaviors, individual characteristics, and social support that is behind the health dynamics. In a three-year window after a cardiac transplant, a prospective study found lower mortality rates in a group of patients that scored higher in a prescreening assessment of health behaviors and psychosocial support, compared to a group of patients with lower scores but similar scores of mental health and the same severity of the disease (Chacko, et al., 1996). These evidences suggest a complex interaction between physical and mental health.

Depressive symptoms are related to pessimistic views about future life events. The higher the symptoms of depression are, the larger is the negative bias in predicting future outcomes (Strunk, et al., 2006; Strunk & Adler, 2009). The cognitive model of depression establishes that depressed individuals give dedicated attention and more efficient processing to negative information. Thus, the negative bias leads to the reinforcement of maladaptive attitudes and behaviors. For example, evaluating graphical material, depressed individuals have an inaccurate perception of emotions as they show more sensitivity in recognizing sad faces compared to happy faces (Roiser, et al., 2012). The cognitive model of depression has an underlying neurobiological mechanism explaining how individuals process their emotions and adapt their behaviors in response to negative life experiences: *i)* negative stimuli produce more and lasting reactivity of the *amygdala* of depressed individuals; *ii)* an abnormal response of the *nucleus accumbens* of depressed individuals imposes more difficulties in adapting to positive rewarding behaviors (Disner, et al., 2011).

Negative experiences are also related to symptoms of depression. A twin study shows that stressful life events cause major depression, although emphasizes that some individuals predisposed to depression also have a greater propensity to cope with stressful situations (Kendler, et al., 1999). One of the issues associated with stress and depression is the potential effect on the physical health of individuals. Psychological stress induces to an adjustment process of the body, commanded by two interacted mechanisms, and producing two outcomes: the release of epinephrine and norepinephrine, and the release of glucocorticoids (Gunnar & Quevedo, 2007). Epinephrine and norepinephrine conduct the immediate and necessary response for survival (flight-or-fight response), whereas one of the functions of glucocorticoids is to suppress the stress response (Sapolsky, et al.,

2000). A constant activation of the stress response makes the cost of the suppressive mechanism to exceed its benefit, producing a physical suboptimal adaptation: the *allostatic load*, which is related to abnormal levels of basal cortisol (Gunnar & Quevedo, 2007). The allostatic load has been associated to other physiological responses such as hypertension, hypercholesterolemia, and the accumulation of adipose tissue in arms and hips. At mature ages, the allostatic load is related to a decline in the physical and cognitive functioning of individuals, and increases the risk of mortality due to cardiovascular diseases (Seeman, et al., 1997).

From a demographic perspective, there is more concern about the age and sex differences related to the association of depression and stressful life events. On the one hand, it has been conjectured that risks of anxiety and depression decrease with age, as negative emotions become less frequent at older ages, individuals gain more control over stressful events, or early life events induce to psychological immunization (Jorm, 2000). On the other hand, based on neurobiological arguments and considering that stressful life events accumulate over the lifespan, it has been hypothesized that the risk of depression is an increasing function of age. However, some empirical evidence shows no substantial correlation between age and hospital admissions to treat a severe depression (Kessing, et al., 2003). Under normal conditions, the stress-sensitivity of humans shows important changes over the lifespan. Minimal levels of cortisol are detected in newborns that increase within the first months of life, followed by a period of hyposensitivity during the early childhood and most of the childhood, and then a transition to the adult-patterns of sensitivity at the puberty (Gunnar & Quevedo, 2007). Therefore, negative experiences might be related to the age-specific responsiveness. In this regard, a study based on a retrospective inventory of stressful events reported by adults aged 55-85 shows that early life events are associated with lower levels of basal cortisol, whereas late life events with higher levels (Gerritsen, et al., 2010). Sex differences in depression have been also examined. The empirical evidence of experimental designs has shown that the association of depressive symptoms and the negative bias anticipating undesirable life events is higher in the case of women (Strunk, et al., 2006; Strunk & Adler, 2009). The neurobiological mechanism has been the leading argument conceptualizing age differences; the adaptive behavior, on the other hand, has been one explanation of the different pathways of men and women.

The perceptions of the self and future life events are linked to mental health. Although the classical model of mental health assumes a neutral balance (objective evaluation) of positive and negative results, it is a normal human condition that future oriented individuals give more attention to desirable outcomes (Taylor & Brown, 1988). Since failure and frustration are emotions that individuals want to avoid, it has been hypothesized that the lack of optimism and lower expectations are rational strategies to

prevent disappointment (van Dijk, et al., 2003). Indeed, illusion and false expectation are particularly deleterious in situations of powerlessness and uncertainty. However, optimism is a valuable feature of individuals when they have the chance to control and to affect future outcomes (Peterson, 2000). An adaptive behavior would be behind the formation of positive expectations, as healthy individuals reinforce their optimism and adjust their perceptions after the realization of wanted outcomes (Korn, et al., 2014). From a biological perspective, the optimism bias is a result of information that is asymmetrically updated because desirable outcomes prevail. In this process, the dopamine has some influence on positive expectations by diminishing the effect of unexpected negative information (Sharot, et al., 2012).

Expectations about future life events would also be related to how individuals internalize the experiences of the others, and their relative position in a society. From a sociological perspective, the optimism bias in reporting a longevity expectation is influenced by the socioeconomic status through the self-confidence about the future (Mirowsky & Ross, 2000). As decision-makers, individuals face uncertainty about their prospective health status and use available information to infer future outcomes (Hurd, 2009). This information might include beliefs, past experiences, and the experience of other individuals. But also the self-confidence that wealth can provide, thus the hypothesis of the socioeconomic status has been one leading explanation of the differences of men and women.

Empirical evidence shows that a negative health shock, such as a new diagnosis, has an impact on mortality expectations and the self-reported health status, whereas a mortality shock, such as the death of a close relative, modifies mortality expectations only (Hurd & McGarry, 2002). Although the perception about health and longevity is as subjective expectation, it is relevant when behaviors and choices are adjusted in consequence (Hurd & McGarry, 1995). In the context of the HIV epidemic, a theoretical model of the optimal choice of sexual partners indicates that pessimistic expectations about the likelihood of being infected increase risky sexual behaviors (Auld, 2003). Furthermore, empirical evidence shows that individuals modify the expectations of being infected after they were informed about their seropositive status and the seropositive status of their spouses (Delavande & Kohler, 2012).

Since optimism emerges from the fulfillment of expectations, positive expectations about longevity are highly beneficial when individuals have the autonomy to adopt behaviors that extend the length of life. Indeed, perceptions and emotional states about health problems guide behaviors that finally affect the health status (Salovey, et al., 2000). Consequently, survival expectations are related to physical and mental health. On the one hand, healthy individuals have a positive perception of the self and are more optimistic

about things that they actually have the control to change (Taylor & Brown, 1994). On the other hand, the illusion of control and positive expectations might have an impact on the behaviors and choices related to the individual's wellbeing. Inasmuch as some symptoms have a psychological origin (are psychogenic), the placebo effect is one example of how positive expectations about the treatment might have an impact on the prospective health status (Eknoyan, et al., 2013). Given that expectations might adjust behaviors before affecting final outcomes, the association is more likely to be intertemporal rather than simultaneous. Furthermore, the linking mechanism of expectations and behaviors, that reinforce the optimism of individuals, is also relevant for the adaptation to the disease.

3.3. Data

The sample used for the analysis comes from the *Malawi Longitudinal Study of Families and Health* (MLSFH; formerly, *Malawi Diffusion and Ideational Change Project*). This longitudinal cohort study began in 1998 with a random sample of 1,745 ever-married women aged 15-49, and 1,519 spouses, who were living in the rural area of three major districts: Balaka in the south, Mchinji in the center, and Rumphi in the north. Initially, the target population of the MLSFH has been extended to incorporate changes in the family composition of the original sample. In 2001, participants were interviewed again, and 331 new spouses were added to the study (254 females - 77 males). Back in 2004, the original sample was extended to include 1,531 adolescents (731 - 800), and 198 new spouses (21 - 177). For the first time in the study, participants interviewed in 2004 were voluntarily tested for HIV. Blood tests to detect HIV were also administrated in 2006, 2008, and 2012. In 2006, the sample was extended to include 529 new spouses (295 - 234). Ever since 2006, the SF12 questionnaire (*12-Item Short-Form Health Survey*) has been administered. In 2008, the sample was extended to include 826 parents of participants interviewed in previous rounds (559 - 267), and 350 new spouses (234 - 116). The follow-up of 2010 included 299 new spouses (193 - 106). In 2012, the MLSFH defined as a target population of mature adults, aged 45 and above, who were eligible in 2008 and 2010 or added in 2008; thus no additional participants were included in 2012 and 2013.

Table 1: Cohort profile of mature adults in the MLSFH

	2006	*2008*	*2010*	*2012*	*2013*
Eligible Individuals	956	1,002	1,455	1,455	1,324
(-) Temporarily Absent / Refusal	145	7	6	78	51
(-) Dead	-	-	-	39	23
(-) Lost of Follow-Up	-	-	-	92	0
(+) New Additions	46	453	-	0	0
Total Interviewed	857	1,448	1,449	1,246	1,234

Note: New additions in 2010 are not part of the cohort profile of mature adults since they were not interviewed in 2008.

The analysis was focused in four consecutive assessments with equal time intervals from 2006 to 2012. It was selected a subsample of 1,455 eligible adults, aged 45+ in 2012, who were interviewed in 2008 and 2010, as is shown in Table 1. For each round of data collection, the number of eligible individuals is equal to the actual number of interviews in the previous round in addition to the number of missing interviews, when individuals refused to participate or were temporarily absent. Due to lack of follow-up, a total of 92 respondents were not included for the analysis. Although these 92 individuals were interviewed in 2008 or 2010, follow-ups were neither conducted in 2012 nor in 2013; thus, they were classified as attrition. However, individuals who were not interviewed in 2012, but in 2008, 2010, and 2013, were included in the analysis and their data in 2012 are assumed to be missing at random.

The analysis is conditional to survivors living in the areas of data collection. In consequence, 39 cases were not included due to mortality of the participant, and 16 additional cases due to permanent migration, as they were documented in the rounds of 2012 and 2013. Similarly, some cases were not included in the analysis due to incomplete information: 15 cases were excluded because GPS location of the respondent was unknown, 8 cases were excluded because the HIV status was unknown, and 11 cases were excluded because incomplete information. In sum, the sample analyzed in this paper consists of 542 males and 732 females; this is a total of 1,274 participants. Major concern would exist with 366 respondents added in 2008 who are included in the analysis and whose information in 2006 is missing. However, if these new additions in 2008 were missed at random in 2006, no significant differences would exist in the estimations if the data collected in 2006 where completely excluded from the models (According to Table 1, the total number of new additions in 2008 is equal to 453. However, 87 respondents were excluded because incomplete information, selection, or attrition.)

Table 2: General descriptive statistics of the sample

	Male		Female	
	Included	Excluded	Included	Excluded
Age	60.43	63.25	59.09	65.92
	0.46	*1.42*	*0.42*	*1.28*
Widowed (at least once)	0.05	0.05	0.31	0.56
	0.01	*0.02*	*0.02*	*0.05*
Divorced (at least once)	0.05	0.10	0.23	0.21
	0.01	*0.03*	*0.02*	*0.04*
Years of schooling	4.35	3.76	2.50	1.81
	0.14	*0.39*	*0.10*	*0.21*
HIV prevalence	0.06	0.20	0.05	0.11
	0.01	*0.05*	*0.01*	*0.04*
Observations	542	84	732	106

Mean/*standard error of the mean*

On average, respondents included in the analysis at the time of the last interview were 59.66 years old, reported 3.28 years of formal education, and had a prevalence of HIV estimated to be 5.47%. Other individual characteristics indicate that about 20.01% of the participants had reported to be widowed at least once, and the 15.38% had reported at least one divorce. Given the initial characteristics of the MLSFH, as well as the addition of new participants in the study, the group of females is overrepresented in the sample. As it can be deduced from Table 2, males excluded from the analysis have, on average, a higher prevalence of HIV (significant at 95% level of confidence) compared to males included in the analysis. Considering the potential effect of the HIV infection on the overall mortality rate, the difference in prevalence is related to the selection of the survivors. On the other hand, females excluded from the analysis are, on average, significantly older and have reported a higher prevalence of widowhood, and less years of formal education compared to the group of females included in the analysis. Table 2 also leads to infer that the group of males included in the analysis is significantly older compared to their female counterparts, reports more years of formal education, and shows less prevalence of marital experiences ending in divorce or widowhood.

The items used to measure the physical health of the respondents are directly related to the degree of physical limitation and the interference of pain to work and to perform typical activities. Similarly, the items used to measure the mental health are related to symptoms of depression, anxiety, lack of energy, lack of motivation, and the interference of emotions to work and to perform typical activities. Although these measurements do not replace the clinical diagnosis of depression or physical disability, they are convenient

measurements for a general population as they encompass a variety of related symptoms. Physical health and mental health were measured using the inventory of a *12-item Short-Form Health Survey* (SF12). Specifically, the analysis is concentrated on 10 items of the SF12 questionnaire that were used to compute six scales of the SF12 methodology: Physical Functioning, Bodily Pain, Role Physical, Mental Health, Vitality, and Role Emotional. For the purposes of the analysis, the scales of General Health and Social Functioning were not included, as they can be equally related to physical and mental health issues. Scales were computed from self-reported data and have a range that goes from 0 to 100. Henceforth, a value equal to zero in one of the six dimensions is indicative of a severe compromise, and is potentially associated with a diminished health status.

Two additional variables were used to measure survival expectations within the next ten years: the probability of surviving the first five-year term, and the probability of surviving the second five-year term given that no mortality would occur in the first term. These probabilities were calculated directly from the subjective expectations of mortality. Participants of the MLSFH reported the increase on the cumulative probability of dying, in response of an increase in the time of exposure to the risk of mortality of one, five, and ten years after the date of the survey. Data were collected empirically, asking the participants to report an additional number of beans on a scale from 0 to 10. Once the length of time has been defined, a reported value equal to 10 indicates that the chance of dying is very likely, or above 95%, thus the survival expectation must be minimal and lower than 5% (Kohler, et al., 2015). For the purpose of the analysis, the variables of survival expectations were adjusted to a scale of 0 to 100.

Table 3 shows the mean values and the standard errors for each round of data by gender. Compared to males, females report lower scores in the group of variables describing health, and lower scores of survival expectations. Inasmuch as females report lower scores of the mental health domain and survival expectations, gender differences in rural Malawi are consistent with the literature of depression and pessimist bias in the anticipation of stressful life events. Although almost all differences in mean values are significant at a level of 5%, there are three exceptions: the survival expectations in 2006 and 2008, and the score of the role emotional in 2006.

Since data summarized in Table 3 correspond to an unbalanced panel, changes on mean values from one round to another might be affected by the composition of the sample. If that were the case, the most substantial change is expected to happen from 2006 to 2008 with the addition of the new sample of parents. However, data collected in 2010 draw particular attention. Compared to 2008 and 2012, data reported in 2010 show a drastic decline regardless of the gender. Given that the cohort of study has been defined as all mature adults interviewed in 2008 and 2010, this break cannot not be related to changes

in the sample composition. Even if the drastic decline of 2010 would be explained by the extraordinary conditions of this particular year, these conditions did not produce a similar disruption in the case of survival expectations, which decreased round after round of data.

Table 3: Descriptive statistics of the items used to measure physical health, mental health, and survival expectations (by sex)

	Male				Female			
	2006	*2008*	*2010*	*2012*	*2006*	*2008*	*2010*	*2012*
Physical Functioning (PF)	91.43	90.46	80.50	85.40	87.12	82.49	71.64	74.01
	1.05	*0.95*	*1.25*	*1.16*	*1.13*	*1.00*	*1.17*	*1.15*
Bodily Pain (BP)	91.10	84.70	80.26	80.89	86.07	77.18	72.20	71.18
	1.06	*1.12*	*1.16*	*1.21*	*1.11*	*1.00*	*1.11*	*1.17*
Role Physical (RP)	88.20	84.00	72.50	84.81	79.06	74.64	59.05	71.57
	1.68	*1.60*	*1.88*	*1.49*	*1.90*	*1.59*	*1.80*	*1.65*
Mental Health (MH)	86.69	82.15	76.93	80.35	81.05	73.23	69.76	72.73
	0.98	*0.93*	*0.92*	*0.92*	*1.04*	*0.85*	*0.85*	*0.89*
Vitality (VT)	84.90	81.98	74.33	75.99	78.98	71.74	66.83	66.45
	1.14	*1.01*	*1.12*	*1.09*	*1.12*	*0.95*	*1.00*	*0.99*
Role Emotional (RE)	89.89	92.32	83.82	88.98	86.77	83.52	73.83	81.33
	1.55	*1.14*	*1.54*	*1.30*	*1.61*	*1.36*	*1.58*	*1.39*
Probability of surviving at the time interval $[t, t+5)$	59.34	58.58	54.26	46.86	56.09	56.44	49.66	39.96
	1.27	*1.14*	*1.11*	*1.16*	*1.15*	*1.02*	*0.96*	*0.94*
Probability of surviving at the time interval $[t+5, t+10)$	56.84	60.52	55.12	42.85	55.53	58.74	49.09	36.06
	1.68	*1.48*	*1.43*	*1.53*	*1.51*	*1.37*	*1.31*	*1.30*
Observations	356	541	541	531	429	729	731	707

Mean/*standard error of the mean*

The self-perception of the probability of surviving decreases as the individual becomes older. However, compared to a life table, subjective expectations of mortality usually overestimate the probabilities of dying in rural Malawi (Delavande & Kohler, 2009; Kohler, et al., 2015). Although reported data of survival expectations are underestimated on the aggregated, subjective probabilities are informative of the individual differences in the perception of future life events. In this regard, a positive expectation about longevity would be relative to all individuals in sample. Insofar as subjective expectations were collected in multiple rounds and using exactly the same questions, data are also

informative of the intertemporal changes in responding to the questionnaire. If individual idiosyncrasies are controlled for as a fixed term, for example, being always optimistic about life; then, longitudinal data allow to investigate the response of survival expectations to changes on health. One particular aspect of the self-reported data of mortality expectations is that once the probabilities of survival are decreased in two independent time intervals, individuals tend to be more pessimistic about the near future.

3.4. Measuring physical health, mental health, and expectations

A conventional approach to the measurement of physical and mental health using population surveys is to calculate the Physical Composite Score (PCS12), and the Mental Composite Score (MCS12). In the practice, the scores are calculated as two different linear combinations of the eight scales defined by the SF12 survey. As latent variables, PCS12 and MCS12 are calculated through analytic rotation, thus a covariance structure between physical health and mental health is dictated by the method: it can be equal to zero in the case of orthogonal algorithms, or a maximum value in the case of oblique algorithms of analytic rotation. Subject to this caveat, the choice of the method would respond to the necessity to assume physical and mental health as independent or endogenous covariates. Since previous assessments of data are not relevant for this estimation, the conventional approach is not suitable to model dynamic interactions when individuals have been interviewed for several years. A second caveat in the calculation of PCS12 and MCS12 is the dilemma of using estimated coefficients from a standard population, for example, the US general population; and then, applying these coefficients to investigate males and females aged 45+ in rural Malawi. In spite of the implicit advantage of comparability of using pre-estimated coefficients, there is a trade-off between comparability and accuracy of the results. Since errors in predicting key parameters should be minimized for an estimation to be fitted, the specificity of the mature adults in rural Malawi is also a desirable condition. Moreover, if these parameters have to be calculated for a study population and multiple rounds of data are available, there is an opportunity to assess the complex interactions between physical and mental health that has been documented in the literature.

An alternative approach to the measurement of health status using the items of a SF12 survey and multiple rounds of data should consider the endogenous but also dynamic nature of physical and mental health. Furthermore, for a better understanding of how this complex interaction works, other domains affecting the health and wellbeing of individuals, such as the expectation of future life events, should also be considered. In order to avoid a prior imposition of any correlation between physical and mental health, independent items were used to measure each health domain.

Physical health is measured using three items: the scales of *Physical Functioning* (PF), *Bodily Pain* (BP), and *Role Physical* (RP). Particularly, for each assessment of data there was defined a latent variable of physical health p, that is not observed directly but through its effect on the scales of PF, BP, and RP, as shown by equations 1.1-1.3. As an identification restriction, the latent variable was always assumed to have a standard normal distribution, hence $p_t \sim N(0,1)$ for all t. From this perspective, the variable of physical health is related to the degree of physical limitation while performing moderate and strenuous physical activities in a typical day of activity, the degree of physical limitation and difficulty while performing work-related activities, and the interference of pain in work-related activities and household duties. If these symptoms were entirely absent, the scales of PF, BP, and RP would be equal to 100, and the score of the physical health would be the highest.

$$\mathrm{PF}_{i,t} = \alpha_{\mathrm{PF},t} + \beta_{\mathrm{PF}} \cdot p_{i,t} + e_{\mathrm{PF},i,t}, \qquad e_{\mathrm{PF},t} \sim N\left(0, \sigma^2_{\mathrm{PF},t}\right). \tag{1.1}$$

$$\mathrm{BP}_{i,t} = \alpha_{\mathrm{BP},t} + \beta_{\mathrm{BP}} \cdot p_{i,t} + e_{\mathrm{BP},i,t}, \qquad e_{\mathrm{BP},t} \sim N\left(0, \sigma^2_{\mathrm{BP},t}\right). \tag{1.2}$$

$$\mathrm{RP}_{i,t} = \alpha_{\mathrm{RP},t} + \beta_{\mathrm{RP}} \cdot p_{i,t} + e_{\mathrm{RP,i},t}, \qquad e_{\mathrm{RP},t} \sim N\left(0, \sigma^2_{\mathrm{RP},t}\right). \tag{1.3}$$

Mental health is approached through the scales *Mental Health* (MH), *Vitality* (VT), and *Role Emotional* (RE). As is shown by equations 2.1-2.3, for each assessment included in the analysis, there was defined a latent variable of mental health $m_t \sim N(0,1)$, which is affecting the scales of MH, VT, and RE. According to the questions of the SF12-form used to calculate these items, the latent variable of mental health is a proxy of the difficulties finding moments of calm and peace, the lack of energy to perform daily activities, the feelings of discouragement and depression, and the degree of emotional limitation to perform work-related activities and duties during a typical day of activity.

$$\mathrm{MH}_{i,t} = \alpha_{\mathrm{MH},t} + \beta_{\mathrm{MH}} \cdot m_{i,t} + e_{\mathrm{MH},i,t}, \qquad e_{\mathrm{MH},i,t} \sim N\left(0, \sigma^2_{\mathrm{MH},t}\right). \tag{2.1}$$

$$\mathrm{VT}_{i,t} = \alpha_{\mathrm{VT},t} + \beta_{\mathrm{VT}} \cdot m_{i,t} + e_{\mathrm{VT},i,t}, \qquad e_{\mathrm{VT},i,t} \sim N\left(0, \sigma^2_{\mathrm{VT},t}\right). \tag{2.2}$$

$$\mathrm{RE}_{i,t} = \alpha_{\mathrm{RE},t} + \beta_{\mathrm{RE}} \cdot m_{i,t} + e_{\mathrm{RE},i,t}, \qquad e_{\mathrm{RE},i,t} \sim N\left(0, \sigma^2_{\mathrm{RE},t}\right). \tag{2.3}$$

Participants of the MLSFH reported cumulative probabilities of dying starting from the date of the interview, and considering three periods of exposure to the risk of dying: one year, five years, and ten years. Given that data were collected using a bounded numerical scale, this is relevant information to calculate the probabilistic expectation of staying alive at two independent time intervals of five-year length each: the *probability of surviving the first five-year term* (SE^{I}), and the *probability of surviving the second five-year term* ($\mathrm{SE}^{\mathrm{II}}$). A latent variable of survival expectations was defined $s_t \sim N(0,1)$, as a

continuous latent variable affecting the self-perception of survival within the next ten years of life, using two subintervals of time as is shown by equations 3.1 and 3.2.

$$\mathrm{SE}^{\mathrm{I}}_{i,t} = \alpha_{\mathrm{SE}^{\mathrm{I}},t} + \beta_{\mathrm{SE}^{\mathrm{I}}} \cdot s_{i,t} + e_{\mathrm{SE}^{\mathrm{I}},i,t}, \qquad e_{\mathrm{SE}^{\mathrm{I}},i,t} \sim N\left(0, \sigma^2_{\mathrm{SE}^{\mathrm{I}},t}\right). \tag{3.1}$$

$$\mathrm{SE}^{\mathrm{II}}_{i,t} = \alpha_{\mathrm{SE}^{\mathrm{II}},t} + \beta_{\mathrm{SE}^{\mathrm{II}}} \cdot s_{i,t} + e_{\mathrm{SE}^{\mathrm{II}},i,t}, \qquad e_{\mathrm{SE}^{\mathrm{II}},i,t} \sim N\left(0, \sigma^2_{\mathrm{SE}^{\mathrm{II}},t}\right). \tag{3.2}$$

Described measurements entail specifying assumptions to be discussed. On the one hand, there was assumed that intercepts α, and the variance of the measurement errors σ^2, vary over time; whereas factor loadings β, are constrained to be the same values from 2006 to 2012. This condition implies a constant covariance between observed and latent variables, as long as the variance of the latent variables will not change over time. On the other hand, latent variables p, m, and s, are assumed to be standard: expected values are always equal to zero and variances are constant over time. Consequently, latent variables show the health status of an individual compared to other individuals in the sample. In summary, although measurement errors are free to change at each round of data, the effect of the latent variables in predicting survey items is always the same. Insofar as uncorrelated errors reduce the number of parameters to be estimated, measurement errors e, are assumed to be independently and normally distributed; hence: $Cov\left[e_{\mathrm{H},t}, e_{\mathrm{K},r}\right] = 0 \; \forall \; \mathrm{H} \neq \mathrm{K}, t \neq r$. Arguably, a parsimonious solution can be relaxed in order to reach a better fit; nevertheless this possibility is not explored in the paper.

Table 4: Reliability coefficients of physical health, mental health, and survival expectations

	Cronbach's alpha				Spearman–Brown coefficient			
Physical Health	*2006*	*2008*	*2010*	*2012*	*2006*	*2008*	*2010*	*2012*
All mature adults	0.841	0.764	0.805	0.818	0.874	0.794	0.833	0.832
Male	0.823	0.746	0.806	0.812	0.853	0.770	0.833	0.821
Female	0.848	0.763	0.797	0.811	0.883	0.798	0.826	0.829
Mental Health	*2006*	*2008*	*2010*	*2012*	*2006*	*2008*	*2010*	*2012*
All mature adults	0.716	0.729	0.708	0.774	0.757	0.753	0.757	0.803
Male	0.687	0.727	0.697	0.728	0.732	0.735	0.735	0.755
Female	0.729	0.709	0.701	0.787	0.769	0.746	0.758	0.820
Survival Expectations	*2006*	*2008*	*2010*	*2012*	*2006*	*2008*	*2010*	*2012*
All mature adults	0.771	0.789	0.826	0.785	0.789	0.807	0.846	0.806
Male	0.783	0.795	0.827	0.807	0.801	0.811	0.843	0.825
Female	0.761	0.783	0.822	0.760	0.779	0.802	0.845	0.785

Since an alternative approach is proposed, the internal consistency of key measurements is therefore examined. Table 4 shows the Cronbach's alpha and the Spearman-Brown coefficient of physical health, mental health, and survival expectations. Theoretical values of positive correlated items lie between zero and one. The higher is the item correlation or the number of items, the greater is the value of the reliability coefficients. The Cronbach's alpha has been defined as a lower-bound estimate of the reliability (Cronbach, 1951), and the Spearman-Brown coefficient is a particular case of the Cronbach's alpha under the assumption of standardized items. Table 4 shows adequate values of internal consistency. Considering the reduced number of items used for each health domain, reliability coefficients show that the content of the items is homogeneous. Similar results were found in individual estimates by sex and year of data collection.

3.5. Estimation of health dynamics

Three models were estimated in order to assess health dynamics in rural Malawi, assuming that changes in physical health have a direct effect on mental health, while the reciprocal effect is lagged by two years. The intuition behind the assumption is that changes in mental health might have an effect on physical health after a process of adaptation in which individuals make subjective evaluation of future life events, and adjust their behaviors in consequence. Thus, the formation of survival expectations could

play a leading role in the health dynamics. Some possible pathways were addressed on each model.

Measurements and health dynamics were estimated simultaneously through Maximum Likelihood (ML), using the statistical package Mplus7.4. In particular, the scales of Physical Functioning (PF), Bodily Pain (BP), Role Physical (RP), Mental Health (MH), Vitality (VT), Role Emotional (RE), and Survival Expectations (SE^{I} and SE^{II}), were assumed to be continuous variables. Although some of them have a broad number of categories that would not make it possible to estimate the model as one of multiple categorical variables, the dispersion of the data shows important discontinuities. In this case, some sacrifice of efficiency is expected to happen on the estimation. The ML estimation of a Structural Equation Model (SEM) has some advantages: reported variables might have some degree of measurement error; and the model is estimated without sacrificing the observations that have missing values in the endogenous variables (Alison, 2003). This means that no imputation is necessary and the model simply maximizes over the conditional expectation of the endogenous variables.

Model 1: survival expectations depend on mental health status
This model assumes that survival expectations are part of the health dynamics, as they depend directly on the mental health of individuals. This specification allows to investigate the pass-through of the health status to the subjective expectations of future life events, controlling (somehow) for fixed characteristics. Although this model assumes a one-way effect from health to expectations, it addresses the question of to what extent healthy (unhealthy) individuals are more optimistic (pessimistic) about their length of life.

The identification strategy suggests that shocks affecting each domain are independent, but physical health and mental health are linked dynamically. As is shown by equations 4.1-4.3, negative shocks of physical health ε_p, may have some direct effect on mental health that is attenuated or intensified by a proportion γ_1; nevertheless, the shocks that affect mental health ε_m, have an effect on physical health after a process of adaptation. This response is captured by the coefficient γ_2. Additionally, all shocks affecting mental health also affect expectations in a γ_3 proportion. The effect of fixed characteristics of individuals is modeled from a latent variable v, which is time invariant but changes each equation in a fixed proportion δ. For convenience, this variable follows a standard normal distribution, hence: $\mathrm{v}\sim N(0,1)$.

$$m_{i,t+2} = \gamma_1 \cdot p_{i,t+2} + \delta_m \cdot \mathrm{v}_i + \varepsilon_{m,i,t+2}, \qquad \varepsilon_m \sim N(0, \sigma_m^2). \tag{4.1}$$

$$p_{i,t+2} = \gamma_2 \cdot m_{i,t} + \delta_p \cdot \mathrm{v}_i + \varepsilon_{p,i,t+2}, \qquad \varepsilon_p \sim N\left(0, \sigma_p^2\right). \tag{4.2}$$

$$s_{i,t+2} = \gamma_3 \cdot m_{i,t+2} + \delta_s \cdot \mathrm{v}_i + \varepsilon_{s,i,t+2}, \qquad \varepsilon_s \sim N(0, \sigma_s^2). \tag{4.3}$$

The system of equations 4.1-4.3 has a path diagram shown in Figure 1 and has the following interpretation: on the one hand, individuals with diminished physical health due to disability and bodily pain would also experience some level of frustration and emotional discomfort in their lives, which are related to symptoms of depression and feelings of discouragement. This association is mainly explained by the concomitancy of physical illness and mental health disorders. On the other hand, individuals who reported lower scores of mental health were also more likely to report a diminished physical health two years later. The path diagram shows the effect of individual fixed characteristics and the effect of mental health on the survival expectations.

Additional restrictions were imposed to reach identification. These restrictions are shown in equations 4.4- 4.6. They are the result of assuming the same variance for all latent variables. The implicit advantage of the assumption is to estimate standardized coefficients.

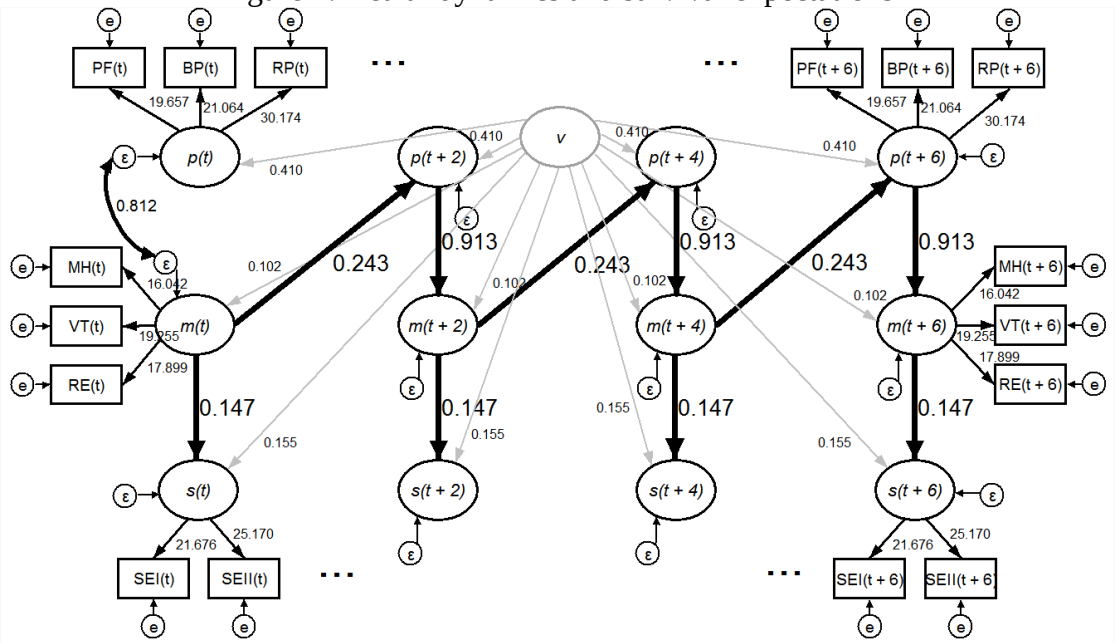
$$1 = \gamma_1^2 + \delta_m^2 + \sigma_m^2. \tag{4.4}$$

$$1 = \gamma_2^2 + \delta_p^2 + \sigma_p^2. \tag{4.5}$$

$$1 = \gamma_3^2 + \delta_s^2 + \sigma_s^2. \tag{4.6}$$

Considering that the latent variable that meets all fixed characteristics of individuals is not correlated with exogenous variables, since they are not included in the model; then, the estimated coefficients are interpreted as a random effects model rather than one of fixed effects. Hence, the model does not corrects the bias of omitting other variables, having a substantial effect on the health of individuals and survival expectations. However, as in a model of random effects, a significant value of δ allows to evaluate the relevance of modeling individual heterogeneity from the error term; and the proportion resulting from: $\delta^2/(\delta^2 + \sigma^2)$ is indicative of the fraction of the error term that is explained by fixed characteristics not included in the model.

Figure 1: Health dynamics and survival expectations



According to the path diagram, physical health p has a contemporaneous effect on mental health m, while mental health has a lagged effect on physical health. In addition, survival expectations s are affected by mental health. Following a convention in SEM, the latent variables are denoted by ovals, whereas the directly observed are shown in rectangles. Males and females combined. Physical Functioning (PF), Bodily Pain (BP), Role Physical (RP), Mental Health (MH), Vitality (VT), Role Emotional (RE), and Survival Expectations (SE^{I} and SE^{II}).

Table 5 shows the estimated coefficients of the model described in equations 4.1-4.6. According to some indicators, the model is adequately fitted to the data: Comparative Fit Index $CFI = 0.935$, Tucker-Lewis Index $TLI = 0.933$, Root Mean squared Error of Approximation $RMSEA = 0.042$. Although it is not possible to satisfy the Chi-square test $\chi^2_{(481)} = 1{,}564.348$ ($p = 0.000$), this could be a result of using a numerous sample $N = 1{,}274$. In general, the effect of physical health on mental health is strong and significant. For the population in sample, an increase in one standard deviation (sd) in physical health has an impact of $0.913\ sd$ on the mental health status. While the reciprocal effect, which is expected to occur two years later is estimated to be $0.243\ sd$. In addition, it is estimated that an increase of one sd on mental health, would increase survival expectations in $0.147\ sd$, given that survival expectations do affect physical and mental health.

Table 5 also reports independent coefficients for men and women. In this case, the model was re-estimated assuming the same factor loadings of the measurement component and the same distribution of errors. Thus, health assessments were measured equally and both groups responded to similar health shocks. Identifying restrictions resulted from the

weighted average of males and females, using the size of each group in the sample. Considering that women are overrepresented in the sample, the weighted average guaranties the core assumption of the model, that all latent variables are distributed as a normal standard; which is a condition that should be satisfied individually for each group in the analysis. Given the assumptions of the model, the six coefficients involved in the health dynamic were statistically different for men and women, as the null hypothesis of equal coefficients was rejected by a Wald test, $w = 44.262$ $(6\ df, p = 0.0000)$. However, individual testing did not rejected the null hypothesis that mental health has the same effect on survival expectation, $w = 0.008$ $(1\ df, p = 0.9299)$, and physical health has the same effect on mental health regardless of the gender, $w = 0.071$ $(1\ df, p = 0.7900)$. Nevertheless, significant differences do exist between men and women in how mental health affects the prospective status of physical health, $w = 16.964$ $(1\ df, p = 0.0000)$.

Considering all the above, this model provides some evidence about how men and women in rural Malawi have different health pathways, although they respond to health shocks that are similar. The difference is mainly explained by the lagged effect from mental to physical health. Additionally, this model shows that the formation of expectations is not different when it plays no role in determining the health status of individuals. However, this model does not explain to what extent survival expectations affect their health status. As a robustness check, alternative specifications were also considered. In particular, those in which survival expectations would affect the current or prospective status of mental health. However, controlling by physical health, survival expectations have no significant effect. Although the mental health status can determine the formation of survival expectations, this result suggests that pessimism about future life events does not necessarily imply a diminished mental health status. A completely different approach would be to argue that survival expectations affect the physical health of individuals to the extent that pessimism (optimism) about life may affect some behaviors, which is the purpose of the following model.

Table 5: Survival expectations depend on the mental health status

		Male	*Female*
γ_1	0.913 *0.011*	0.926 *0.026*	0.916 *0.020*
γ_2	0.243 *0.035*	0.382 *0.049*	0.145 *0.038*
γ_3	0.147 *0.028*	0.172 *0.040*	0.167 *0.033*
δ_m	0.102 *0.030*	0.092 *0.039*	0.070 *0.038*
δ_p	0.410 *0.039*	0.207 *0.061*	0.549 *0.037*
δ_s	0.155 *0.047*	0.258 *0.050*	0.049 *0.053*
σ_m^2	0.156 *0.017*	0.147 *0.017*	
σ_p^2	0.773 *0.021*	0.734 *0.020*	
σ_s^2	0.954 *0.010*	0.942 *0.011*	
$Var[\varepsilon_{m,2006}]$	0.990 *0.006*	0.994 *0.004*	
$Var[\varepsilon_{p,2006}]$	0.832 *0.032*	0.809 *0.028*	
$Cov[\varepsilon_{m,2006}, \varepsilon_{p,2006}]$	0.812 *0.024*	0.813 *0.022*	
Observations	1,274	542	732

Coefficient/*standard error*. General model: $CFI = 0.935$, $TLI = 0.933$, $RMSEA = 0.042$, and $\chi^2_{(481)} = 1{,}564.348$ ($p = 0.000$). Model of independent coefficients for males and females: $CFI = 0.909$, $TLI = 0.913$, $RMSEA = 0.047$, and $\chi^2_{(1035)} = 2{,}506.410$ ($p = 0.000$).

Model 2: Survival expectations as a predictor of future health

The direct effect of physical health on mental health γ_1 remains, but the reciprocal effect is assumed to materialize through the survival expectations $\gamma_3 \times \gamma_4$. This model accounts for the effect of subjective expectations as a predictor of the prospective health status γ_4, giving that mental health still has a contemporaneous effect on survival expectations γ_3. Everything else works as in the first model: physical and mental health are assumed to be correlated at the first round of data since no prior information was available; the shocks affecting each health domain are independent; and fixed characteristics where assessed as a latent variable. Hence, the system of equations that describe the structure of the model is:

$$m_{i,t+2} = \gamma_1 \cdot p_{i,t+2} + \delta_m \cdot \mathrm{v}_i + \varepsilon_{m,i,t+2}, \qquad \varepsilon_m \sim N(0, \sigma_m^2). \tag{5.1}$$

$$p_{i,t+2} = \gamma_4 \cdot s_{i,t} + \delta_p \cdot \mathrm{v}_i + \varepsilon_{p,i,t+2}, \qquad \varepsilon_p \sim N\left(0, \sigma_p^2\right). \tag{5.2}$$

$$s_{i,t+2} = \gamma_3 \cdot m_{i,t+2} + \delta_s \cdot \mathrm{v}_i + \varepsilon_{s,i,t+2}, \qquad \varepsilon_s \sim N(0, \sigma_s^2). \tag{5.3}$$

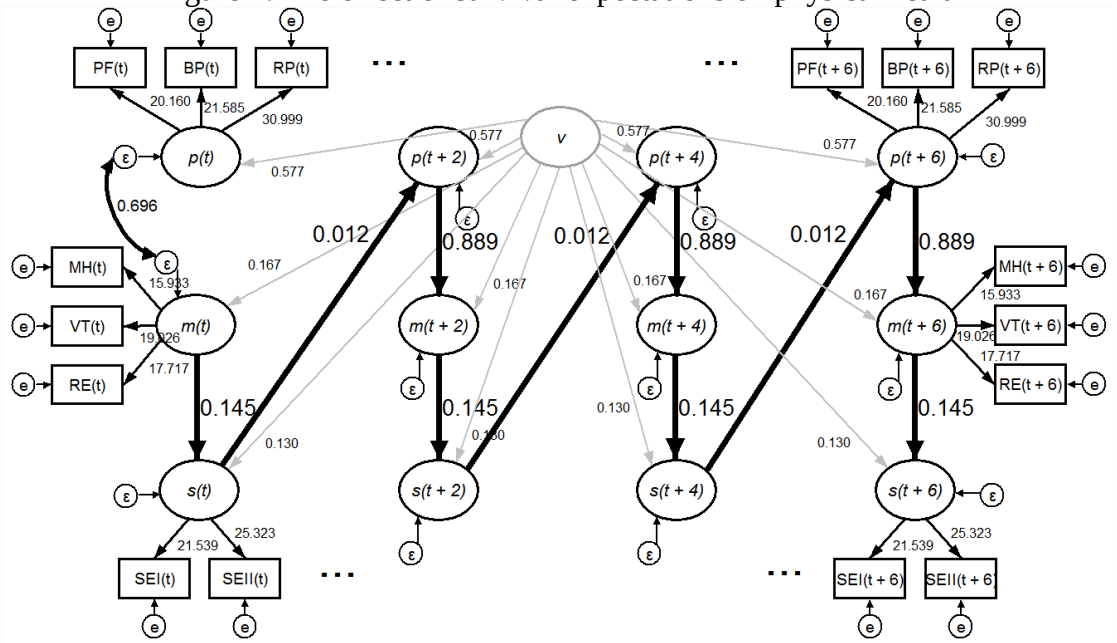
$$1 = \gamma_1^2 + \delta_m^2 + \sigma_m^2. \tag{5.4}$$

$$1 = \gamma_4^2 + \delta_p^2 + \sigma_p^2. \tag{5.5}$$

$$1 = \gamma_3^2 + \delta_s^2 + \sigma_s^2. \tag{5.6}$$

The system of equations 5.1-5.6 has a path diagram representation shown in Figure 2.

Figure 2: The effect of survival expectations on physical health



Males and females combined. Physical Functioning (PF), Bodily Pain (BP), Role Physical (RP), Mental Health (MH), Vitality (VT), Role Emotional (RE), and Survival Expectations (SE^{I} and SE^{II}).

Table 6 shows the estimated coefficients from equations 5.1-5.6. This model shows similar goodness of fit: $CFI = 0.931$, $TLI = 0.929$, $RMSEA = 0.043$. Estimated values indicate that an increase of one sd in survival expectations is associated to an increase of about 0.012 sd in the physical health two years later. This is a minimal and insignificant amount, when the model is fitted for males and females combined. Thus, negative perceptions about the self and the future would not have a deleterious effect on physical health of mature adults in rural Malawi. However, another conclusion can be reached when estimated coefficients are independent for men and women. In particular, Table 6 shows that survival expectations could itself have an effect on the physical health of men. An increase of one sd on expectations would increase physical health in 0.062 sd after two years. This is a very small effect that is different than zero only at the 10% level of significance. For women, the same coefficient is estimated to be -0.028 sd, but it is not statistically different from zero. Overall, the six coefficients involved in the dynamics of health were statistically different for men and women in the sample, $w = 41.531$ (6 $df, p = 0.0000$). However, individual testing suggested that the effect of survival expectations on physical health might not be the source of pronounced differences between men and women in rural Malawi, $w = 3.740$ (1 $df, p = 0.0531$); given that, as is shown in Table 6, the effect of mental health on survival expectations is virtually the same.

With all the above, this model also provides evidence on the differences in health pathways of mature men and women in rural Malawi. These differences could be related to the role of survival expectations as a predictor of future health. However, the effect of expectations on health is not of a considerable extent. Given this result, an alternative specification was considered assuming that survival expectations do not have a direct effect on the health status of individuals, but are correlated through the error term. This specification implies that negative (positive) shocks not only diminish (increase) the health status, but also turn individuals to be more pessimistic (optimistic) about their lives. This is the leading argument of the following model.

Table 6: Survival expectations as a predictor of future health

		Male	*Female*
γ_1	0.889	0.853	0.921
	0.120	*0.027*	*0.020*
γ_3	0.145	0.144	0.145
	0.028	*0.045*	*0.034*
γ_4	0.012	0.062	-0.028
	0.024	*0.034*	*0.032*
δ_m	0.167	0.213	0.108
	0.018	*0.033*	*0.034*
δ_p	0.577	0.471	0.661
	0.025	*0.031*	*0.025*
δ_s	0.130	0.203	0.080
	0.040	*0.056*	*0.052*
σ_m^2	0.182	0.180	
	0.017	*0.017*	
σ_p^2	0.667	0.658	
	0.020	*0.020*	
σ_s^2	0.962	0.957	
	0.006	*0.007*	
$Var[\varepsilon_{m,2006}]$	0.972	0.976	
	0.008	*0.008*	
$Var[\varepsilon_{p,2006}]$	0.668	0.657	
	0.020	*0.020*	
$Cov[\varepsilon_{m,2006}, \varepsilon_{p,2006}]$	0.696	0.700	
	0.022	*0.021*	
Observations	1,274	542	732

Coefficient/*standard error*. General model: $CFI = 0.931$, $TLI = 0.929$, $RMSEA = 0.043$, and $\chi^2_{(481)} = 1{,}625.253 (p = 0.000)$. Model of independent coefficients for males and females: $CFI = 0.905$, $TLI = 0.909$, $RMSEA = 0.048$, and $\chi^2_{(1035)} = 2{,}574.874$ $(p = 0.000)$.

Model 3: Survival expectations as an endogenous covariate

Physical health and survival expectations are assumed to be correlated through the error term, and both depend on the previous assessment of mental health. This implies that shocks that improve or worsen the physical health of individuals, also affect expectations regarding the length of their lives and vice versa. As a new feature of the structure, survival expectations are also assumed to depend on previous assessments of the same variable, allowing to investigate the persistence of negative (or positive) evaluations of the self and the future. At the first round of data, all variables are assumed to be correlated through the error term, and everything else has the same definitions of previous models. Equations 6.1-6.4 describe the general characteristics of the model, which estimation relies on identifying restrictions 6.5-6.7. These result of assuming normalized latent variables.

$$m_{i,t+2} = \gamma_1 \cdot p_{i,t+2} + \delta_m \cdot \mathrm{v}_i + \varepsilon_{m,i,t+2}, \qquad \varepsilon_m \sim N(0, \sigma_m^2). \tag{6.1}$$

$$p_{i,t+2} = \gamma_2 \cdot m_{i,t} + \delta_p \cdot \mathrm{v}_i + \varepsilon_{p,i,t+2}, \qquad \varepsilon_p \sim N\left(0, \sigma_p^2\right). \tag{6.2}$$

$$s_{i,t+2} = \gamma_4 \cdot s_{i,t} + \gamma_5 \cdot m_{i,t} + \delta_s \cdot \mathrm{v}_i + \varepsilon_{s,i,t+2}, \qquad \varepsilon_s \sim N(0, \sigma_s^2). \tag{6.3}$$

$$Cov\left[\varepsilon_p, \varepsilon_s\right] \neq 0 \tag{6.4}$$

$$1 = \gamma_1^2 + \delta_m^2 + \sigma_m^2. \tag{6.5}$$

$$1 = \gamma_2^2 + \delta_p^2 + \sigma_p^2. \tag{6.6}$$

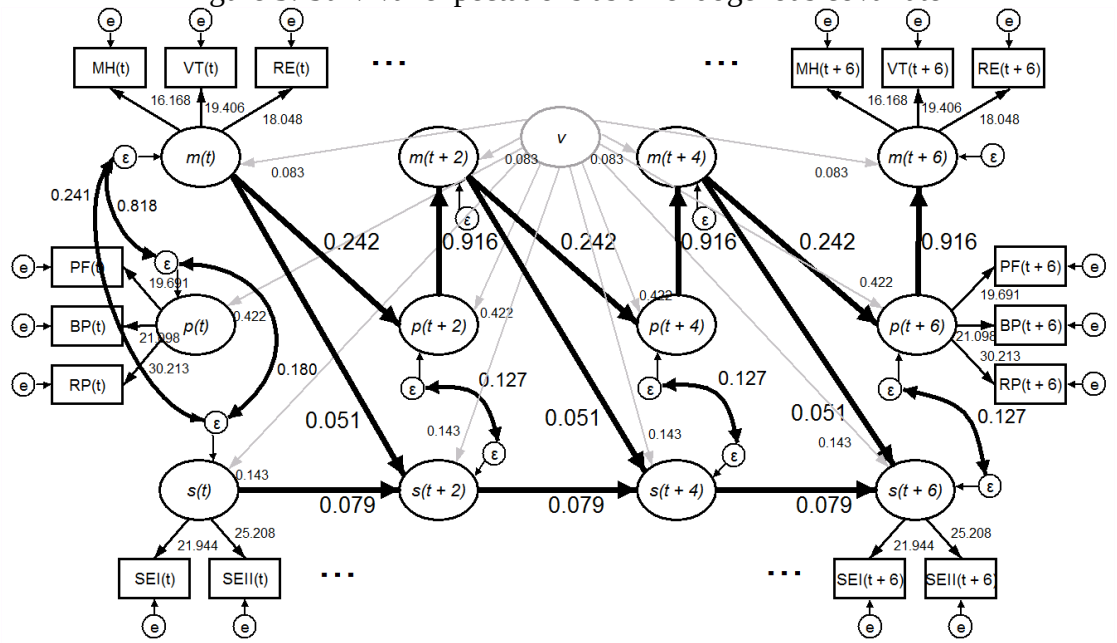
$$1 = \gamma_4^2 + \gamma_5^2 + \delta_s^2 + \sigma_s^2. \tag{6.7}$$

In addition to the intertemporal dynamics between physical and mental health that was suggested in the previous two approaches, the intuition behind this model suggests that expectations can be persistent at a coefficient γ_4; or may respond to the past experiences of the mental health at a coefficient γ_5. The system of equations 6.1-6.7 have a path diagram shown in Figure 3.

Table 7 shows the estimated coefficients from equations 6.1-6.7. The goodness of fit is similar to the previous models: $CFI = 0.936$, $TLI = 0.933$, $RMSEA = 0.042$. The estimated values suggest that, physical health and survival expectations have an estimated covariance of 0.127 units. If the variance of the residuals are estimated at 0.763 and 0.971 respectively, then they have an estimated correlation of 0.147 units. Given the assumptions of the model, this value is a measure of the concomitance between physical health and survival expectations of mature adults in rural Malawi. Regarding the other parameters, the persistence of expectations was estimated to be 0.079 sd; while the

lagged effect of mental health on expectations was estimated to be $0.051\ sd$, an amount that is no different from zero at 5% significance.

Figure 3: Survival expectations as an endogenous covariate



Males and females combined. Physical Functioning (PF), Bodily Pain (BP), Role Physical (RP), Mental Health (MH), Vitality (VT), Role Emotional (RE), and Survival Expectations (SE^{I} and SE^{II}).

Table 7 also shows independent coefficients for men and women. After this model it could be established that the seven coefficients involved in the dynamics of health were statistically different for men and women, $w = 34.149$ ($7\ df, p = 0.0000$). However, this result was addressed by the difference in only three of the coefficients: the effect of mental health on physical health γ_2, $w = 18.253$ ($1\ df, p = 0.0000$); the unobserved individual variation in the equation of physical health δ_2^2, $w = 25.937$ ($1\ df, p = 0.0000$); and, the unobserved individual variation in the equation of survival expectations δ_3^2, $w = 4.215$ ($1\ df, p = 0.0401$). One noticeable characteristic of this model is how expectations are formed differently in men and women. Although previous assessments of the mental health were the most important for males; in the case of females, survival expectations are persistent, to some extent, and seem to be uncorrelated to previous assessments of mental health. However, these differences were only significant at levels above 5%, given that, $w = 5.735$ ($2\ df, p = 0.0568$). The other variables did not show significant differences for men and women, $w = 0.809$ ($2\ df, p = 0.6674$).

With all the above, this model also provides evidence of the different health pathways of males and females in Rural Malawi. These differences are related to the effect of mental

health on the prospective physical health status, given that shocks affecting physical health also impact the survival expectations, and vice versa. This model includes two mechanisms by which expectations are formed. Is not dismissed the fact that there are differences between men and women on what is the most prevalent mechanism. The evidence suggests that for women survival expectations are persistent, thus lasting effects are observed after two years, while for men expectations respond to changes in mental health.

Table 7: Survival expectations as an endogenous covariate

		Male	*Female*
γ_1	0.916	0.932	0.914
	0.011	*0.025*	*0.020*
γ_2	0.242	0.396	0.148
	0.034	*0.050*	*0.039*
γ_4	0.079	0.004	0.101
	0.024	*0.045*	*0.031*
γ_5	0.051	0.118	0.017
	0.029	*0.044*	*0.035*
δ_m	0.083	0.082	0.067
	0.035	*0.040*	*0.038*
δ_p	0.422	0.191	0.546
	0.034	*0.071*	*0.037*
δ_s	0.143	0.267	0.111
	0.045	*0.065*	*0.045*
σ_m^2	0.154	0.145	
	0.017	*0.017*	
σ_p^2	0.763	0.734	
	0.020	*0.021*	
	0.011	*0.016*	
$Cov[\varepsilon_p, \varepsilon_s]$	0.127	0.124	
	0.024	*0.022*	
$Var[\varepsilon_{m,2006}]$	0.993	0.995	
	0.006	*0.004*	
$Var[\varepsilon_{p,2006}]$	0.822	0.814	
	0.029	*0.029*	
$Var[\varepsilon_{s,2006}]$	0.980	0.963	
	0.130	*0.016*	
$Cov[\varepsilon_{m,2006}, \varepsilon_{p,2006}]$	0.818	0.819	
	0.025	*0.022*	
$Cov[\varepsilon_{m,2006}, \varepsilon_{s,2006}]$	0.241	0.240	
	0.046	*0.046*	
$Cov[\varepsilon_{p,2006}, \varepsilon_{s,2006}]$	0.180	0.189	
	0.047	*0.046*	
Observations	1,274	542	732

Coefficient/*standard error*. General model: $CFI = 0.936$, $TLI = 0.933$, $RMSEA = 0.042$, and $\chi^2_{(481)} = 1.547.021 (p = 0.000)$. Model of independent coefficients for males and females: $CFI = 0.910$, $TLI = 0.913$, $RMSEA = 0.047$, and $\chi^2_{(1035)} = 2{,}487.797$ ($p = 0.000$).

3.6. Discussion

Structural equation models were used to assess the reciprocal interaction of physical health, mental health and survival expectations. This paper provides evidence on the different health pathways of men and women in rural Malawi. Differences greatly rely upon the effect of changes in mental health over physical health. A higher value was estimated for men, which means they are more vulnerable to changes in their mental health status; a result that is related to the different ways men and women form survival expectations. If survival expectations are correlated to the physical health, expectations of men are more related to the previous assessment of the mental health while women's expectations are persistent and rely on past expectations.

There has been concern about whether or not individuals can make accurate judgments about the lethal risks they are exposed to (Lichtenstein, et al., 1978). The problematic is even bigger when it comes to ask about probabilities to a population with low levels of schooling. However, a variety of methods and protocols have shown consistency, especially if they allow the elicitation of ranges and distributions rather than precise values (Attanasio, 2009; Delavande, et al., 2011). If possible, using scales that allow arithmetic calculation and asking for expectations about issues that concern to individuals (Manski, 2004). The MLSFH is one of those successful cases that have elicited subjective expectations using methods that allow quantifying probabilities. Although participants often overestimate the mortality risk, being a longitudinal study, more consistency is achieved in identifying how the participants have changed their expectations over time. These changes may be associated with changes in health status and that is one of the effects that this paper tries to quantify.

The differences between men and women may respond to variables that were not included in the model; for example, the socioeconomic status. That would be an important omission considering that it is related to confidence in the future (Mirowsky & Ross, 2000), and a better physical and mental health (Link & Dohrenwend., 1993; Phelan, et al., 2010). Nevertheless, the estimates presented in this paper attempted to discount for the effect ascribed to unobserved fixed characteristics. This effect keeps some relation to the individual endowments that did not changed in the years analyzed. Indeed, considering the age group and the schooling levels of the participants, less concern would exist in the potential effect of socioeconomic status. However, other individual characteristics did change; for example, the experience of divorces and the mortality of loved ones. Such events could affect both the health of the participants and their expectations about future life events. This concomitant effect was incorporated into the model as a residual correlation between physical health and survival expectations. However, they could be incorporated explicitly in a future analysis, using an inventory of

catastrophic events that, besides the death of household members, include unanticipated changes in employment or loss of economic support.

Deprivation, as a lack of economic resources, produces psychological stress, which finally has a negative impact on the overall wellbeing. Thus, individuals living in poor economic conditions are expected to report poor health outcomes (Das, et al., 2007; Tampubolon & Hanandita, 2014). Paradoxically, people living in poverty get sick with more frequency, but are less liable to report sickness; indeed, there is less complain about life and health, nevertheless a consistent report of a heavy burden of psychological stress (Banerjee & Duflo, 2007). The particular context of less developed areas would provide a complementary perspective to help to identify the deep causes of non-communicable diseases (Ebrahim, et al., 2013). Since mental health is related to poverty and wellbeing, special attention should be paid to the evidence from less developed countries. Thus, the analysis of health dynamics related to non-communicable diseases in less developed areas of the world has scientific significance. Most dynamic approaches on health and mortality use evidence from developed countries, where longitudinal data allowing to investigate these perspectives are available. Conversely, the evidence from low-and-middle income countries is limited, and hence this paper makes an important contribution to understand the link between physical and mental health.

3.7. References

Alison, P. D., 2003. Missing data techniques for structural equation modeling. *Journal of Abnormal Psychology*, 112(4), p. 545–557.

Attanasio, O. P., 2009. Expectations and perceptions in developing countries: their measurement and their use. *The American Economic Review*, 99(2), pp. 87-92.

Auld, M. C., 2003. Choices, beliefs, and infectious disease dynamics. *Journal of Health Economics*, 22(3), p. 361–377.

Banerjee, A. V. & Duflo, E., 2007. The economic lives of the poor. *The Journal of Economic Perspectives*, 21(1), pp. 141-168.

Canavan, M. E. et al., 2013. Psychological distress in Ghana: associations with employment and lost productivity. *International Journal of Mental Health Systems*, 7(9), pp. 1-9.

Chacko, R. C., Harper, R. G., Gotto, J. & Young, J., 1996. Psychiatric interview and psychometric predictors of cardiac transplant survival. *American Journal of Psychiatry*, 153(12), pp. 1607-1612.

Cohen, S. & Rodriguez, M. S., 1995. Pathways linking affective disturbances and physical disorders. *Health Psychology,* 14(5), pp. 374-380.

Collins, P. Y. et al., 2013. Grand challenges in global mental health: integration in research, policy, and practice. *PLoS Medicine,* 10(4), pp. e1001434. doi:10.1371/journal.pmed.1001434.

Connell, J., O'Cathain, A. & Brazier, J., 2014. Measuring quality of life in mental health: Are we asking the right questions?. *Social Science & Medicine,* Volume 120, pp. 12-20.

Coughlin, S. S., 2012. Anxiety and depression: linkages with viral diseases. *Public Health Reviews,* 34(2), pp. 1-17.

Cronbach, L. J., 1951. Coefficient alpha and the internal structure of tests. *Psychometrika,* 16(3), pp. 297-334.

Crump, C., Winkleby, M. A., Sundquist, K. & Sundquist, J., 2013. Comorbidities and mortality in persons with schizophrenia: a Swedish national cohort study. *American Journal of Psychiatry,* 170(3), pp. 324-333.

Das, J. et al., 2007. Mental health and poverty in developing countries: revisiting the relationship. *Social Science & Medicine,* 65(3), pp. 467-480.

de Ridder, D., Geenen, R., Kuijer, R. & van Middendorp, H., 2008. Psychological adjustment to chronic disease. *Lancet,* 372(9634), p. 246–255.

Delavande, A., Giné, X. & McKenzie, D., 2011. Measuring subjective expectations in developing countries: A critical review and new evidencE. *Journal of Development Economics,* Volume 94, p. 151–163.

Delavande, A. & Kohler, H.-P., 2009. Subjective expectations in the context of HIV/AIDS in Malawi. *Demographic Research,* 20(article 31), p. 817–875. doi:10.4054/DemRes.2009.20.31.

Delavande, A. & Kohler, H.-P., 2012. The Impact of HIV Testing on Subjective Expectations and Risky Behavior in Malawi. *Demography,* 49(3), p. 1011–1036.

Dewa, C. S. & Lin, E., 2000. Chronic physical illness, psychiatric disorder and disability in the workplace. *Social Science & Medicine,* 51(1), pp. 41-50.

Dewa, C. S., Lin, E., Kooehoorn, M. & Goldner, E., 2007. Association of chronic work stress, psychiatric disorders, and chronic physical conditions with disability among workers. *Psychiatric Services,* 58(5), pp. 652-658.

Disner, S. G., Beevers, C. G., Haigh, E. A. P. & Beck, A. T., 2011. Neural mechanisms of the cognitive model of depression. *Nature Reviews: Neuroscience,* 12(8), pp. 467-477.

Ebrahim, S. et al., 2013. Tackling non-communicable diseases in low- and middle-income countries: is the evidence from high- income countries all we need?. *PLoS Medicine,* 10(1), p. e1001377. doi:10.1371/journal.pmed.1001377.

Eknoyan, D., Hurley, R. A. & Taber, K. H., 2013. The neurobiology of placebo and nocebo: how expectations influence treatment outcomes. *Journal of Neuropsychiatry & Clinical Neurosciences,* 25(4), pp. vi-254. doi: 10.1176/appi.neuropsych.13090207.

Gerritsen, L. et al., 2010. Early and late life events and salivary cortisol in older persons. *Psychological Medicine,* 40(9), p. 1569–1578.

Gunnar, M. & Quevedo, K., 2007. The neurobiology of stress and development. *Annual Review of Psychology,* Volume 58, pp. 145-173.

Hurd, M. D., 2009. Subjective probabilities in household surveys. *Annual Review of Economics,* Volume 1, pp. 543-562.

Hurd, M. D. & McGarry, K., 1995. Evaluation of the subjective probabilities of survival in the health and retirement. *The Journal of Human Resources,* 30(Special Issue on the Health and Retirement Study: Data Quality and Early Results), pp. S268-S292.

Hurd, M. D. & McGarry, K., 2002. The predictive validity of subjective probabilities of survival. *The Economic Journal,* 112(482), pp. 966-985.

Jokela, M., Hakulinen, C., Singh-Manoux, A. & Kivimäki, M., 2014. Personality change associated with chronic diseases: pooled analysis of four prospective cohort studies. *Psychological Medicine,* 44(12), pp. 2629-2640.

Jorm, A. F., 2000. Does old age reduce the risk of anxiety and depression? A review of epidemiological studies across the adult life span. *Psychological Medicine,* 30(1), pp. 11-22.

Kendler, K. S., Karkowski, L. M. & Prescott, C. A., 1999. Causal relationship between stressful life events and the onset of major depression. *American Journal of Psychiatry,* 156(6), pp. 837-841.

Kessing, L. V., Agerbo, E. & Mortensen, P. B., 2003. Does the impact of major stressful life events on the risk of developing depression change throughout life?. *Psychological Medicine,* 33(7), pp. 1177-1184.

Kivimäki, M. et al., 2014. Long-term inflammation increases risk of common mental disorder: a cohort study. *Molecular Psychiatry,* 19(2), p. 149–150.

Kohler, H.-P.et al., 2015. Cohort profile: the Malawi Longitudinal Study of Families and Health (MLSFH). *International Journal of Epidemiology,* 44(2), pp. 394-404.

Korn, C. W. et al., 2014. Depression is related to an absence of optimistically biased belief updating about future life events. *Psychological Medicine,* 44(3), pp. 579-592.

Lichtenstein, S. et al., 1978. Judged frequency of lethal events. *Journal of Experimental Psychology: Human Learning and Memory,* 4(6), pp. 551-578.

Lim, D., Sanderson, K. & Andrews, G., 2000. Lost productivity among full-time workers with mental disorders. *Journal of Mental Health Policy and Economics,* 3(3), pp. 139-146.

Link, B. G. M. C. L. & Dohrenwend., B. P., 1993. Socioeconomic status and depression: The role of occupations involving direction, control, and planning. *American Journal of Sociology,* 98(6), pp. 1351-1387.

Manski, C. F., 2004. Measuring expectations. *Econometrica,* 72(5), pp. 1329-1376.

Meeks, S., Murrell, S. A. & Mehl, R. C., 2000. Longitudinal relationships between depressive symptoms and health in normal older and middle-aged adults. *Psychology and Aging,* 15(1), pp. 100-109.

Mirowsky, J. & Ross, C. E., 2000. Socioeconomic status and subjective life expectancy. *Social Psychology Quarterly,* 63(2), pp. 133-151.

Payne, C. F., Mkandawire, J. & Kohler, H.-P., 2013. Disability transitions and health expectancies among adults 45 years and older in Malawi: A cohort-based model. *PLoS Medicine,* 10(5), p. e1001435. doi:10.1371/journal.pmed.1001435.

Peterson, C., 2000. The future of pptimism. *American Psychologist,* 55(1), pp. 44-55.

Phelan, J. C., Link, B. G. & Tehranifar, P., 2010. Social conditions as fundamental causes of health inequalities: theory, evidence, and policy implications. *Journal of Health and Social Behavior,* 51(supplement), pp. S28-S40.

Powdthavee, N., 2009. What happens to people before and after disability? Focusing effects, lead effects, and adaptation in different areas of life. *Social Science & Medicine,* 69(12), p. 1834–1844.

Prince, M. et al., 2007. No health without mental health. *Lancet,* 370(9590), p. 859–877.

Roiser, J. P., Elliott, R. & Sahakian, B. J., 2012. Cognitive mechanisms of treatment in depression. *Nature: Neuropsychopharmacology Reviews,* Volume 37, pp. 117-136.

Salovey, P., Rothman, A. J., Detweiler, J. B. & Steward, W. T., 2000. Emotional states and physical health. *American Psychologist,* 55(1), pp. 110-121.

Sapolsky, R. M., Romero, M. & Munck, A. U., 2000. How do glucocorticoids ifluence stress responses?. *Endocrine Reviews,* 21(1), pp. 55-89.

Scafato, E. et al., 2012. Changes in severity of depressive symptoms and mortality: the Italian Longitudinal Study on Aging. *Psychological Medicine,* 42(12), pp. 2619-2629.

Seeman, T. E. et al., 1997. Price of adaptation—allostatic load and its health consequences: MacArthur Studies of Successful Aging. *Archives of Internal Medicine,* 157(19), pp. 2259-2268.

Sharot, T. et al., 2012. How dopamine enhances an optimism bias in humans. *Current Biology,* 22(17), p. 1477–1481.

Sharpe, L. & Curran, L., 2006. Understanding the process of adjustment to illness. *Social Science & Medicine,* 62(5), p. 1153–1166.

Stanton, A. L., Revenson, T. A. & Tennen, H., 2007. Health psychology: psychological adjustment to chronic disease. *Annual Review of Psychology,* Volume 58, p. 565–592.

Strunk, D. R. & Adler, A. D., 2009. Cognitive biases in three prediction tasks: A test of the cognitive model of depression. *Behaviour Research and Therapy,* 47(1), pp. 34-40.

Strunk, D. R., Lopez, H. & DeRubeis, R. J., 2006. Depressive symptoms are associated with unrealistic negative predictions of future life events. *Behaviour Research and Therapy,* 44(6), pp. 861-882.

Tampubolon, G. & Hanandita, W., 2014. Poverty and mental health in Indonesia. *Social Science & Medicine,* Volume 106, pp. 20-27.

Taylor, S. E. & Brown, J. D., 1988. Illusion and well-being: A social psychological perspective on mental health. *Psychological Bulletin,* 103(2), pp. 193-210.

Taylor, S. E. & Brown, J. D., 1994. Positive illusions and well-being revisited: separating fact from fiction. *Psychological Bulletin,* 116(1), pp. 21-27.

van Dijk, W. W., Zeelenberg, M. & van der Pligt, J., 2003. Blessed are those who expect nothing: Lowering expectations as a way of avoiding disappointment. *Journal of Economic Psychology,* 24(4), pp. 505-516.

Whiteford, H. A. et al., 2013. Global burden of disease attributable to mental and substance use disorders: findings from the Global Burden of Disease Study 2010. *Lancet*, 382(9904), pp. 1575-1586.