Pedro Cisalpino Pinheiro

Mortality differentials in Brazil: an analysis using modal age at death and measures of dispersion

Belo Horizonte, MG
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Tese apresentada ao curso de Programa de Pós-Graduação em Demografia do Centro de Desenvolvimento e Planejamento Regional da Faculdade de Ciências Econômicas da Universidade Federal de Minas Gerais, como requisito parcial à obtenção do Título de Doutor em Demografia.

Orientador: Prof. Bernardo Lanza Queiroz

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Cedeplar

Curso de Pós-Graduação em Demografia da Faculdade de Ciências Econômicas

ATA DE DEFESA DE TESE DE **PEDRO CISALPINO PINHEIRO** Nº. REGISTRO 2016663922. Às quatorze horas do dia vinte e três do mês de setembro de dois mil e dezenove, reuniu-se na ***Faculdade de Ciências Econômicas*** da Universidade Federal de Minas Gerais a Comissão Examinadora de TESE, indicada "*ad referendum*" pelo Colegiado do Curso em 10/09/2019, para julgar, em exame final, o trabalho final intitulado "*Mortality differentials in Brazil: an analysis using modal age at death and measures of dispersion*", requisito final para a obtenção do Grau de *Doutor em Demografia*, área de concentração em Demografia. Abrindo a sessão, o Presidente da Comissão, Prof. Bernardo Lanza Queiroz, após dar a conhecer aos presentes o teor das Normas Regulamentares do Trabalho Final, passou a palavra ao candidato, para apresentação de seu trabalho. Seguiu-se a arguição pelos examinadores, com a respectiva defesa do candidato. Logo após, a Comissão composta pelos professores: Bernardo Lanza Queiroz, Laura Lídia Rodríguez Wong, Cássio Maldonado Turra, Marcos Roberto Gonzaga (participação por videoconferência) e Luciana Correia Alves (participação por videoconferência) se reuniu, sem a presença do candidato e do público, para julgamento e expedição do resultado final. A Comissão APROVOU o candidato por unanimidade. O resultado final foi comunicado publicamente ao candidato pelo Presidente da Comissão. Nada mais havendo a tratar o Presidente encerrou a reunião e lavrou a presente ATA, que será assinada por todos os membros participantes da Comissão Examinadora. Belo Horizonte, 23 de setembro de 2019.

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To Vanessa and Antônio

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LIST OF ABBREVIATIONS

FU – Federal Units

M – Modal age at death

MD - median age at death

IQR - Interquartile Range

SD(M+) - Standard Deviation above the mode

e° - Life expectancy

LAC – Latin American and Caribbean

BMI - Body Mass Index

HG - Hunter and Gatherers

NS - Negligible senescence

HMD - Human Mortality Database

lx – life table number of survivors by age

ndx - life table number of deaths by age

CVD - Cardiovascular Diseases

PNAD – Pesquisa Nacional por Amostra de Domicílios

DALY - Disability Lost Years

NTD – Neglected Tropical Diseases

CDR – Crude Death Rate

SSA - Social Security Administration

MIS – Mortality Information System

Datasus - Brazilian Ministry of Health Database

SEG-adj - Adjusted Synthetic Extinct Cohort

DDM - Death Distribution Methods

GGB - General Growth Balance Method

LQ – Log-Quadratic

5q0 - Probability of dying before age five

45q15 - Probability of death between age 15 and 60

TP – TOPALS

Q1 – Age indicating the point were 75% of the hypothetical cohort is still alive

Q3 – Age indicating the point were 25% of the hypothetical cohort is still alive

IHD – Ischemic Heart Disease

MMR – Mother Mortality Ratio

ASMR – Age Specific Mortality Rate

TA – Traffic Accidents

RTI - Road Traffic Incident

SUMMARY

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ABSTRACT

This dissertation aimed to analyze adult and old age mortality in Brazil and its 27 Federal units between 1980 and 2010, by sex, using the modal age at death (M), the median age at death (MD), the interquartile range (IQR), and the standard deviation above the mode (SD(M+)). The selected set of mortality measures allowed us to observe changes in different parts of the age distribution. The smoothed single age mortality rates were estimated using three different approaches: Wilmoth and colleagues (2012) Log-Quad; TOPALS (Gonzaga and Schmertmann, 2016); and Beers Osculatory Interpolation. Another goal was to understand how applied methods influenced the selected measures and the age at death distributions. Our results show that both modal age rose and the median age at death rose independently of the selected method, indicating that mortality diminished substantially in adult and older ages. Both lifespan variability measures indicated that between 1980 and 2010 mortality was compressed into a smaller age interval. Modal age at death was most influenced by the selected smoothing method. TOPALS presented the best fit in most analyzed years and captured the important features of mortality change. Sex differential in the modal age at death rose between 1980 and 2010, while MD gap reached its peak in 1991 and diminished afterwards. Both age at death variability measures also placed women in a better position. Geographic analysis showed that differences between states diminished between 1980 and 2010 considering all four measures, as well as the shape of age at death distributions. The highest modal age at deaths observed in less developed states, along with the mortality crossover at older ages indicate that old age data from both mortality information system and from Brazilian censuses might present regional quality differentials.

Key words: lifespan variability; modal age at death; sex differences; geographic differences

RESUMO

O principal objetivo desta tese é utilizar a idade modal à morte, a idade mediana à morte, a distância interquartílica e o desvio padrão para analisar a variação na mortalidade adulta no Brasil e nas Unidades da Federação, entre 1980 e 2010, para homens e mulheres. Neste sentido, três diferentes métodos foram aplicados para a suavização das curvas de mortalidade por idade simples: Log-Quad (WILMOTH et al., 2012); TOPALS (Gonzaga & Schmertamann, 2016) e a interpolação osculatória de Beers. Assim, analisamos, também, os impactos relacionados à escolha do método nas medidas analisadas e na distribuição de óbitos por idade. Tanto a idade modal à morte como a idade mediana à morte aumentaram consideravelmente no período analisado, independentemente do método de suavização. As duas medidas de variabilidade na idade à morte indicaram que os óbitos estão mais concentrados em um menor número de anos. A medida que apresentou maior influência da escolha do método foi a idade modal à morte. O ajuste do TOPALS apresentou os melhores resultados, sendo capaz de refletir importantes características do padrão de mortalidade brasileiro. Entre 1980 e 2010 a diferença entre a idade modal à morte de homens e mulheres aumentou. Em relação à idade mediana, após um aumento entre 1980 e 1991, o diferencial diminuiu nos últimos anos. As mulheres apresentam IQR e SD(M+) mais baixos que os homens. Entre as UFs, a diferença entre o valor máximo e mínimo observado nas quatro medidas diminuiu no período analisado. O valor mais elevado da idade modal à morte registrado em estados menos desenvolvidos do ponto de vista socioeconômico e o crossover da mortalidade registrado nas idades mais avançadas são sinais de que os dados dos sistemas de informação de mortalidade e/ou dos censos pode apresentar problemas nas idades mais avançadas.

Palavras chave: variabilidade na idade à morte; idade modal à morte; diferencial por sexo; diferencial geográfico

CHAPTER 1 - INTRODUCTION

Life expectancy has been rising for, at least, the last 160 years (OEPPEN & VAUPEL, 2002). Linear highest life expectancy at birth rise registered by Oeppen and Vaupel (2002) challenges perceptions on observed mortality change and on what to expect about changes in the future (LEE, 2019). In developed countries, if the same mortality reduction path continues, in the future, most children born in 2000 will live up to 100 years (VAUPEL, 2010). In contrast, most children born in the 19th century died very young (VAUPEL et al., 2011). For many researchers, the question about mortality change in the future is whether decline will be fast or slow (CRIMMINS, 2015).

Along with the expressive mortality decline, came the debate about the biological limits of longevity and, so far, no consensus has been reached (OEPPEN & VAUPEL, 2002; ROOTZÉN & ZHOLUD, 2017; COUZIN-FRANKEL, 2011; DONG et al. 2016; OLSHANKY et al., 1990; VAUPEL et al., 1998; WILMOTH, 2000; WILMOTH, 1998). Several authors argue that lifespan might have a natural limit (FRIES, 1980; OLSHANSKY et al., 1990; OLSHANSKY et al., 2005; CRIMMINS, 2015; DONG et al., 2016). Many others argue that there is not enough evidence to support the existence of a limit to life expectancy (BARBI et al., 2018; BROWN et al., 2017; HUGHES & HAKIN, 2017; LENART & VAUPEL, 2017; de BEER et al., 2017; WILMOTH, 2000). Rootzén and Zholud (2017) understand that the idea that lifespan is limited did not receive a convincing answer and, as it is based on the existing data, may be modified as better quality data become available.

The magnitude of mortality decline, and change in mortality age profile are, however, unquestionable. In more developed countries, mortality reduction since the 1960's is concentrated at older ages (HORIUCHI et al., 2013; OEPPEN & VAUPEL, 2002; WILMOTH, 2000). Consequently, we saw a deceleration in the increase of life expectancy at birth and an increase in maximum lifespan (CANUDAS-ROMO, 2008; HORIUCHI et al. 2013; VAUPEL, 2010; WILMOTH, 2000, 2002; RAU et al., 2008). In recent decades, most LAC countries, and especially Brazil, experienced accelerated decline in infant, child and adult mortality. (ARRIAGA & DAVIS, 1969; PALLONI& PINTO-AGUIRRE, 2011; BARRETO et al., 2012). Important mortality decline was also observed in older

ages, with different levels between regions (SIMÕES, 2002; IBGE, 2010; BORGES, 2017; CORREA & MIRANDA-RIBEIRO, 2017).

In that context, the objective of the present work is to examine mortality changes in adult and old ages in Brazil and in its 27 Federal Units (FU), by sex, between 1980 and 2010, using the modal age at death (M), the median age at death (MD), the interquartile range (IQR), and the standard deviation above the mode (SD(M+)). Appling these measures we are also interested in understanding and discussing mortality differentials by sex and by geographic unit. Differently from developed countries, along with other developing countries, mortality in Brazil fell very fast, and the pattern of change was more complex than the traditional epidemiological transition describes. In a short period, important mortality changes were registered in a broad age range, with relevant divergence between states and sexes.

Another aim in the present dissertation is to discuss differences resulting from the smoothing methods applied to mortality estimates. To obtain a smooth age at death distribution, we applied different strategies: the life table system, Log-Quad, developed by Wilmoth et al. (2012); TOPALS regression developed by Gonzaga & Schmenrtmann (2016); and the Beers Osculatory Interpolation method, described in Siegel & Swanson (2004). It is important to understand if and how the observed results relate to the smoothing method choice. The modal age at death (M) is one of the analyzed indicators as it reflects mortality change in older ages. Mortality is also changing on younger ages, so we will analyze how the median age at death (MD) behaved in the period. We also use two measures of mortality dispersion the Interquartile Range (IQR) and the Standard Deviation above the mode (SD(M+)) that will allow us to observe how concentrated is the age at death distributions in every considered year, by sex and Federal Unit.

We are also interested in understanding whether there is a convergence or divergence in adult mortality in Brazil. This is relevant because changes in life expectancy soon could be heavily explained by differences in adult mortality as infant and child mortality have shown signs of convergence in recent years (BORGES, 2017; DUARTE et al., 2002). There are several studies about trends

in infant and child mortality (BARUFI et al., 2012), but much less is known about adult and older age mortality in Brazil (DUARTE et al., 2002; GONZAGA & SCHMERTMANN, 2016). At the same time, divergent mortality dynamics has been observed at different geographic levels (FRANÇA et al., 2017b; BRANT et al. 2017; LEITE et al., 2015; CAMPOS et al., 2015; BORGES, 2017). Looking at mortality at a broader age range, by sex and Federal Units (FU) may help understand how those processes are evolving, it also could serve as a source of information that help the adoption of preventive public health measures.

This dissertation will contribute to the literature on mortality change in Brazil by describing how the selected measures, not very often used in Brazilian mortality literature, behaved in a period of important changes. At the same time, focusing on geographic and sex differentials will broaden the knowledge on the internal dynamics of mortality change in Brazil. The smoothing method comparison will also represent a relevant contribution to other mortality studies in Brazilian and developing contexts as it shows how the measures may be influenced by the selected methods.

In countries where mortality decline is concentrated at older ages, the modal age at death can be an important reference to understand variations not captured by life expectancy at birth (e°) (CANUDAS-ROMO, 2008). Differently from e° or the median age at death (MD), the mode is not sensitive to mortality change at younger ages (CANUDAS-ROMO, 2010). The mode is also free from an arbitrary age limit definition (KANNISTO, 2001). The rise in the modal age can indicate where in the age distribution mortality decline was observed (CANUDAS-ROMO, 2010). Canudas-Romo (2010) demonstrated that a modal age at death rise is, necessarily, a consequence of mortality decline at ages above the mode. These characteristics make M a good tool to explain the current pattern of mortality variation in high-income countries (HORIUCHI et al., 2013).

The rectangularization of survival curve and the compression of mortality processes are important frameworks used to describe patterns of mortality change (OUELLETTE et al., 2012, WILMOTH & HORIUCHI, 1999; FRIES, 1980; CANUDAS-ROMO, 2008; CHEUNG & ROBINE, 2007; CHEUNG et al., 2009). The first refers to changes in survival curve shape as the probability of surviving

to higher ages increases (OUELLETTE et al., 2012, WILMOTH & HORIUCHI, 1999). By its turn, compression of mortality describes the concentration of deaths in a smaller age interval, indicating reduced variability in the age of death. A less uncertain lifespan influences how individuals live and plan their lives (WILMOTH & HORIUCHI, 1999). Surviving to higher ages changes resources allocation (education, health, preventive behavior, savings) throughout lifecycle (OPPEN & VAUPEL, 2002; LEE, 2003).

In low mortality countries, more recently, the compression of mortality seems to have stopped and the shifting of the mortality curve may well describe the pattern of mortality change. In a shifting context, the modal age of death rises while variability remains stable (CANUDAS-ROMO, 2008; CHEUNG et al. 2009; CHEUNG & ROBINE, 2007). Even if it represents only a temporary phenomenon, Canudas-Romo (2008) highlights the importance of the identification of the shifting of the mortality curve as a different pattern related to mortality change.

In developing countries, there is growing interest in using modal age at death and/or the compression/rectagularization/shifiting of mortality dynamics as analytical tools. The present work intends to contribute to this effort. Gonzaga et al. (2018) estimated age at death variability measures for a series of Latin American and Caribbean (LAC) countries and they showed a rapid process compression of mortality for females, and stagnation for males, mainly due to an increase in external causes of deaths. Alvarez et al. (2020) found that amenable diseases dispersed along the age span are largely responsible for the lifespan variability gap between LAC and developed countries. They also highlight that external causes are the main impediment for mortality convergence between LAC and developed countries among males. Gonzaga et al. (2009) analyzed the variability in age at death in the state of São Paulo and observed two distinct periods, one with increase in variability (1980-1995) and one with decreasing variability (1995-2005). Gonzaga and Costa (2016) focused on the regional differences in Brazil between 1980 and 2010 and observed that the rectangularization of the survival curve was more expressive for women and in the South and Southeast regions. One main challenge in analyzing evolution of mortality indicators in developing countries is related to the poor data quality (HILL, 2017; WILMOTH et al., 2012), especially in countries with great regional

heterogeneity in socioeconomic and demographic indicators (GONZAGA & COSTA, 2016).

The traditional epidemiological transition theory cannot fully describe the fast mortality decline observed in many developing countries, including Brazil (OMRAN, 1971; BORGES, 2017; FRENK et al. 1991). According to Frenk et al. (1991), epidemiological transition in LAC countries is better defined as prolonged (where different types of diseases coexist) and polarized (poorer people present higher disease rates and these diseases are often from different types). Another important characteristic in such framework is the reversibility of changes, also known as counter-transition (FRENK et al. 1991; FRENK et al. 1989). Illustrating such epidemiological complexity, in Brazil (2008), the share of the disability lost years (DALY) related to infectious and parasitic diseases, maternal conditions, and nutritional deficiencies was 13.2%, non-communicable disease represented 77.2% and external causes, 9.5%, of the burden of disease (LEITE et al., 2015). At the same time as infant mortality gains are still relevant in Brazil, the older age group contributes significantly to life expectancy gains (BORGES, 2017; CORRÊA & MIRANDA-RIBEIRO, 2017).

Brazilian mortality transition, beyond its complexity, has a strong regional component. Mortality declined first in the more socioeconomically developed regions of the country - Southeast and South - (BORGES, 2017; SIMÕES, 2002; WOOD & CARVALHO, 1994; CARVALHO & WOOD, 1978). In the last decades, Northeast and North (the poorer regions) observed improvement in mortality levels leading to reduction in regional life expectancy differentials (BORGES, 2017, SIMOES, 2002). More recently, South and Southeastern regions presented moderate life expectancy gain as they already had lower infant mortality. At the same time, they registered expressive mortality decline at older ages (BORGES, 2017). Another divergent regional dynamic was observed between 2000 and 2010, while male young adult mortality diminished in Southeastern region it rose in the Northeast and North (BORGES, 2017; WAISELFISZ et al. 2013; MALTA et al., 2017). Socioeconomic inequality is an important problem in Brazil (UN, 2016; MEDEIROS et al., 2015) and may influence internal mortality differentials (PAMPEL et al., 2005; CUTLER et al. 2006; LANTZ et al., 1998; EDWARDS & TULJAPURKAR, 2005).

Mortality analysis in Brazil, as elsewhere, must also consider gender differences. Life expectancy gap between males and females in Brazil reached 7.2 in 2016 (IBGE, 2017). Since mortality started declining in 1940, males and female's mortality presented both convergence and divergence dynamics (BORGES, 2017; SIMÕES, 2002). Noymer and Van (2014) observe that in most countries from the Human Mortality Database (HMD), generally, male and female mortality co-moved - changed in the same direction, even in periods when life expectancy sex gap diverged the most. The main causes of death in Brazil are considerably different for males and females (LEITE et al., 2015) and, consequently, affect mortality differently along the age distribution. It is important to analyze each sex individually in order to capture the intrinsic features of mortality variation with time.

Kanso (2014) analyzed Brazilian mortality using the modal age at death, the median age at death and variability measures in the 1980-2010, without smoothing the data. Gonzaga & Souza (2016) analyzed compression of mortality dynamics at the level of Brazilian great regions, while Gonzaga et al. (2008) studied that same process in São Paulo state between 1980 and 2005. Other studies focused on LAC countries to understand compression of mortality and age at death dynamics (GONZAGA et al., 2018; ALVAREZ et al., 2020; SOLÍS & GARCÍA-GUERRERO, 2019). Several authors discuss Brazilian mortality changes in the last decades (BORGES, 2017; SIMOES, 2002; CORREA & MIRANDA-RIBEIRO, 2017; BRASIL, 2016; DUARTE et al., 2002). Many of them also analyze regional mortality differentials (BORGES, 2017; SIMOES, 2002; LEITE et al., 2015; ALBUQUERQUE et al., 2017; WOOD & CARVALHO, 1994; CARVALHO & WOOD, 1978), others focuses on geographic differences by specific causes (BRANT et al., 2017; CAMPOS et al., 2015; CERQUEIRA, 2014; WEISENFILZ, 2013; IPEA, 2017; MALTA et al., 2017; MALTA et al., 2016; MARTINS-MELO et a., 2018). Our contribution to that literature probably rests on the effort to use the set of described measures to analyze convergenge/divergence dynamics between sex and Brazilian states, as well as discussing the consequences of different smoothing methods. The rich literature on mortality changes in Brazil will be fundamental to discuss and make sense of our findings.

Among its main results, this dissertation shows that both modal age at death and the median age at death rose between 1980 and 2010, indicating mortality changes in a broad age range including older ages. Variability in the age of death decline for both male and female considering both IQR and SD(M(+)). Modal age of death was sensitive to the smoothing method applied, even in more recent years with. Considering TOPALS, that provided the best fit to the data, female modal age rose more than male, as well as the median age at death, widening sex gap in the period. One clear divergence between sexes was captured by the changing local mode at young adult ages for men. After the local mode rise between 1980 and 1991, it never lowered throughout the analyzed period, reflecting different dynamics at state level in young adult mortality. In the course of years, Federal Units the age at death distribution curves got closer to each other. At FU level, however, higher modal age of death was observed in Northeastern states, probably indicating content problems (reporting or registering) in mortality or population (or both) data.

With the proposed goals in mind, the present dissertation is organized in seven chapters, including this introduction. Chapter 2 presents the literature review in order to contextualize the work in a broader discussion. Chapter 3 presents the data sources, data treatment, methods applied to smooth mortality curves and to construct the selected indicators. A comparison of each smoothing method is presented in Chapter 4, considering results for every analyzed year (1980, 1991, 2000 and 2010). In Chapter 5, differences of mortality dynamics by gender are presented considering TOPALS estimates. Regional differences at Federal Units (FU) level are discussed at Chapter 6. Chapter 7 concludes this dissertation and presents a research agenda to be pursued in a close future.

CHAPTER 2 - LITERATURE REVIEW

2.1 Longevity gains

According to Wilmoth (2000), longevity gains are, probably, the most important achievement in human history. In countries like Japan, Sweden and Spain three quarters the babies born in the beginning of the current decade will reach age 75. In contrast, most children born in the 19th century died at very young ages (VAUPEL et al., 2011). In 1950, in Japan, the female probability of surviving from age 65 to age 100 was less than one in a thousand. In the beginning of the last decade, the same chance of survival was one in twenty (OEPPEN & VAUPEL, 2002). Rau and his colleagues (2008) found that the probability an 80 years old Japanese woman will live to complete her 90th birthday was more than one-half in 2002. In developed countries, if we observe the same path in mortality reduction in the future, most of the children born in 2000 will live up to 100 years (VAUPEL, 2010).

Mortality decline in the first half of the last century, in developed countries, was concentrated at young ages. Since 1950, mortality fell more expressively at older ages (HORIUCHI et al., 2013; OEPPEN & VAUPEL, 2002). Consequently, we saw a deceleration in life expectancy evolution since the 1950's and a rise in the maximum lifespan (CANUDAS-ROMO, 2008; HORIUCHI et al. 2013; VAUPEL, 2010; WILMOTH, 2000, 2002; RAU et al., 2008). Considering 30 countries from the Kannisto-Thatcher Database, Kannisto et al. (1994) found that between the 1920's and the 1930's the rate of improvement in old age mortality was close to zero. From 1930 to 1960, mortality at old ages diminished 0.5 per cent per year. Rau and his colleagues (2008) highlight - in reference to Kannisto et al. (1994) and Vaupel (1997) – that mortality decline in old ages begun accelerating in the 1970's. Since the 1970's, mortality declined about 3% every year for Japanese women above age 80 (VAUPEL et al. 1998).

Japan is a very successful example of rapid mortality reduction, also in old ages. Robine and Saito (2003) observe that there are now signs that mortality reduction at older ages slowed down. Between 1973 and 2000, the number of centenarians

in Japan rose from 266 to 13,036, only partially explained by a rise in the number of births, 100 years earlier (ROBINE & SAITO, 2003). Controlling for the size of the initial cohort, the number of centenarians rose by a factor of 12. Considering the population above 105, authors show that 64% of the increase was consequence of mortality reduction. For women, death rates for those aged 100-105 diminished from 0.5 in 1975 to 0.35 in 1998 (ROBINE & SAITO, 2003).

Although, countries performed differently when it comes to mortality reduction. At the same time as Japan, France and Switzerland showed expressive rise in life expectancy above age 80, between 1960 and 2000, in Latvia, the same indicator practically did not change (RAU et al., 2008). In the Netherlands, especially for males, the rise in $e°_{80}$ was very modest. The United Stated is another example of the different paths traced by developed countries. Since the 1980, there was a deceleration in life expectancy gains in the USA, which once was the leader in old-age mortality decline (RAU et al., 2008).

According to Tuljapurkar et al. (2000), the rate of decline in mortality depends on the balance between amount of resources allocated in that goal and their marginal effectiveness. Throughout the years, the level of resources rose and the effectiveness diminished, since mortality causes are getting more complex (TULJAPURKAR et al., 2000). For the future, those authors believe that there will be an increase in the volume of resources allocated and a growing complexity of causes. Deeg et al. (2013) argues that mortality decline in old age in Amsterdam in the 1996-2006 period was consequence of both life course factors (education, behaviour) and better disease specific survival (medical and long care improvements).

In developing countries, this trajectory was different. Since the 1950's, most of those countries started a rapid mortality decline (PALLONI & PINTO-AGUIRRE, 2011). Cutler et al. (2006) understand that mortality decline in the developing context since the end of Second World War (WWII) was consequence of the knowledge accumulated in 200 years in rich countries. In 1950, the forerunners in mortality decline in Latin America and the Caribbean (LAC) had life expectancy at age five (e_5°) of about 58.7, comparable to Norway in 1900 (PALLONI & PINTO-AGUIRRE, 2011). Palloni and Pinto-Aguirre (2011) emphasize that, since

the end of World War II, forerunners benefited from the diffusion of medical technology.

Mortality decline at older ages in LAC countries, according to Palloni and Pinto-Aguirre (2011), traced a faster path than the observed in rich countries. Life expectancy at age 60 among LAC forerunners countries grew linearly in the period between 1925 and 2000. Among the laggard LAC countries, since the 1980's, the rate of gain in $e°_{60}$ accelerated (PALLONI & PINTO-AGUIRRE, 2011). In the same sense, Borges (2017) observes that mortality decline in ages above 60 contributed substantially to the rise in life expectancy in Brazil, especially for women. At the same time, however, between 2000 and 2010, there still were significant gains in life expectancy in young ages.

Life expectancy has been rising for, at least, the last 160 years (OEPPEN & VAUPEL, 2002). There is an important debate about the biological limits of longevity and, yet, no consensus has been reached (OEPPEN & VAUPEL, 2002; CANUDAS-ROMO, 2008; OUELLETTE & BOURBEAU, 2011; DONG et al. 2016). Christensen and Vaupel (1996) argue that little success has been achieved in determining the causes of extreme longevity. In 2016, Dong et al. (2016) observed that lifespan might have a natural limit. Authors found that the maximum reported age at death in countries like France, United Kingdom, and Japan increased rapidly between 1970 and 1990 and became stable around 1995. After 1995, the linear regression applied showed diminution in maximum reported age at death. The authors argued that their results clearly indicate that duration of life is limited. Unsurprisingly, their paper received a lot of attention and many critics as well.

Among the critics Dong et al. (2016) received, Brown et al. (2017) and Rozing et al. (2017) argued the fact they used the same dataset to both generate and test their hypothesis is problematic. The sample size was also among the critics the paper received (BROWN et al.,2017). After 1995, their estimates are dependent on Jeanne Calmont[1] (BROWN et al.,2017; HUGHES & HAKIN, 2017). Similarly, Lenart & Vaupel (2017) point out that if analysis started in 1950, they would find

[1] Highest registered age at death with 122 years.

different results. Same authors also observed that above age 90 caution is required using HMD data. According to de Beer et al. (2017) the observed rise of the number of supercentenarians would – even if death probabilities remain unchanged – naturally increase observed maximum age at death. Dong et al. (2016) replied all of the critics about their results.

The inexistence of lifespan limit, as far as we can see, is the result that Barbi et al. (2017) presented in their paper analyzing a cohort of Italians. Based on a dataset of more than 3800 individuals aged 105 or over, between 2009 and 2015, the authors found that above age 105 age specific probability of dying reached a plateau with the annual probability of dying equal to 0.475. According to Barbi et al. (2017), the increasing number of long-lived people and the mortality decline across cohorts are lowering the level of the plateau or postponing the age when it appears.

Another recent work argues that human life is unlimited, although short (ROOTZÉN & ZHOLUD, 2017). According to them, the answer that life is limited has not received a convincing answer, they also argue that all conclusions are based on the existing data and may be modified as higher quality data become available. After 110, Rootzén and Zholud (2017) estimated that the annual probability of dying is equal to 0.47, results that are very close to what Barbi et al. (2017) observed.

Controversies about the existence of a limit to human life are not new. In 2011, Couzin-Frankel (2011) wrote about the heat debate on the topic focusing, mainly, on Olshansky and Vaupel. Olshanky et al. (1990) estimated the mortality reductions needed to elevate the life expectancy at birth from 80 to 120. According to them, the complete elimination of the all mortality above age 50 would increase life expectancy at birth only by 3.5 years. To reach a life expectancy of 85 years old, the upper limit Fries (1980) hypothesized, Olshanky et al. (1990) calculated that mortality would need to be reduced by 65% and 43%, for men and women, respectively. Authors thought that given the mortality changes necessary to achieve a life expectancy at birth of 85 years old, it was unlikely that on average people live beyond that limit.

In a posterior paper, Olshanky et al. (2005) presented another pessimistic view about the future tendencies in life expectancy. Authors argue that, in opposition to the position held by several authors (OPPEN & VAUPEL, 2002; KEILMAN et al., 2017; TULJAPURKAR et al. 2000; BOONGARTS, 2009), life expectancy forecast do not need to be revised. Olshanky et al. (2005) argue that extrapolation of life expectancy trends do not consider health status of people currently alive. Authors understand obesity as relevant risk for future trends in mortality estimates. In the USA, in the 1980's and 1990's, obesity rose 50% (OLSHANSKY et al., 2005). According to their estimates, if all black males had an optimal Body Mass Index (BMI), life expectancy would be from 0.30 to 1.08 years higher. Although the effect could be higher, varying from two to five years, as the young, relatively more obese, cohorts age (OLSHANSKY et al., 2005).

At the other side in the debate, Vaupel et al. (1998) understand that if there was a limit in mortality decline, countries with low mortality rates would be showing signs of deceleration in the rate of reduction. Boongarts (2009) highlighted the same point. Carey (2003) observes that, for all species with available information, the existence of life-span limit would be perceivable by the existence of a pattern in the living records, which is not the case. The existence of species specifics issues (strains, sex, biotypes among others) that influence observed life span is another argument presented by Carey (2003) against the existence of limit in life-span.

The concern with pessimism in official life expectancy forecasts is present in the work of author like Oeppen & Vaupel (2002), Tuljapurkar et al. (2000), Boongarts (2009) and, more recently, Keilman et al. (2017). In all cases, authors compare forecasted life expectancy from the past with the achieved indicator and alert for the possible impact to governments if planning was based on the official forecasts. Interestingly, Keilman et al. (2017) highlights that in Austria, among other developed countries, until the 1990's, official forecasts projected that mortality would remain constant after 2015. Same author believe that governments should be concerned with aging processes in their countries since forecasted old age population is smaller than the observed. In the same sense, Tuljapurkar et al. (2000) found a median forecasted life expectancy at birth substantially higher that official forecasts. As same authors point out, one-year

difference in life expectancy represent more than 5% difference in the dependency ratio. In 2050, their forecasted dependency ratio was 6% (U.K) and 40% (Japan) higher than the official forecasts (TULJAPURKAR et al., 2000).

Burguer et al. (2012) tried to understand the importance of mortality reduction from an evolutionary point of view. Compared to hunter and gatherers (HG) populations, at all ages, low mortality populations present expressively lower probability of death, authors highlight. Burguer et al. (2012) assert that their most remarkable finding is that hunter and gatherers mortality profile is closer to wild chimpanzees than mortality profile in Japan or Sweden in the 21st century. Until age 15, HG present mortality rates 100 times higher than that in Japan and Sweden, at all ages hunter and gatherers present mortality rates at least 10 times higher than the observed in low mortality countries in the present (BURGUER et al., 2012). Same authors emphasize that the level of mortality reductions registered in the last century for humans was greater than advances made in laboratory with other species.

From a biological perspective, aging is defined as the increase in susceptibility of dying with time (GREY et al., 2002; BAUDISH, 2011). Negligible senescence (NS), by its turn, describes the absence of increase in mortality as an organism ages (GREY et al., 2002). According to Grey et al. (2002), the absence of negligible senescence in homoeothermic (hot blooded) organisms does not mean that it would be impossible to be engineered. Among the strategies to engineer NS, authors highlight that exercises (not regular ones) have potential to restore both muscle mass and bone density. Another example they present, is to induce regrowth of lost synapses in neurons.

Boongarts (2009) observed that in most parts of developed world practically all the deaths are considered senescent (results of biological aging). Author divided deaths observed in 17 developed countries among senescent deaths and non-senescent (unrelated to aging) between 1960 and 2000. Considering age specific senescent death rates, the author found the same pattern in 1960 and 2000, the difference was a shifted to the right, indicating rising in longevity. Boongarts (2009) observes that the standard deviation of the age at deaths senescent distribution changed little in the period analyzed.

2.2 Modal age of death

In ageing analyses, a disadvantage of indicators like age specific death rates, life expectancy at some specific age, probability of dying or surviving is that they require an arbitrary selection of age limit (KANNISTO, 2001). Kannisto (2001) points out that the mode, as the most common length of life in a given mortality regime, is a good indicator for ageing and longevity and is free from arbitrary age limit definition. According to Horiuchi and colleagues (2013), the fact that longevity gains are concentrated on higher ages gives greater analytical meaning to the modal age at death.

Over time, the differences in life expectancy and modal age at death trends indicate that they are reflecting different aspects of mortality decline (CHEUNG & ROBINE, 2007). In low mortality countries, the modal age at death can be an important reference to understand variations in mortality not captured by the life expectancy (CANUDAS-ROMO, 2008). Cheung and Robine (2007) observe that although several scholars understood that the mode, in theory, represents the most central and natural characteristic of human longevity, such indicator was rarely used before Kannisto (2001).

Canudas-Romo (2010) notes that mortality reduction conducts changes in central tendency indicators (mean, median, and mode). In each one of those, the age in which mortality reductions occurs determine the pace of the trends. The choice between the mean, the median and the mode, depends on the researcher objectives (CANUDAS-ROMO, 2010). When the death distribution is symmetrical, the mean (e°) is the best indicator of a mortality situation. Although death distribution was never symmetrical, life expectancy prevailed among mortality analyses (CANUDAS-ROMO, 2010). The main reason, according to the same author, is that predictions and inferences based on the mean have fewer total errors or smaller standard deviation.

Canudas-Romo (2010) analyzed the pace in the record mean, median, and mode, based on the Human Mortality Database (HMD). Life expectancy and median age at death increased slower and more regularly since the second half of the 20th century, period that mortality gains became more concentrated in adult

and elderly population (CANUDAS-ROMO 2010; CANUDAS-ROMO, 2006). For females, until the mid-1940's, the mode stagnated (until the 1970's for males). At the other hand, at the same time, e° and the median rose substantially. Since the 1970's, the pace of increase was similar for all three measures (CANUDAS-ROMO 2010).

Canudas-Romo (2010) applied a two-phase linear regression model to estimate the pace of increase in the record modal age at death. For males and females, at the beginning of the series, the mode rose 0.5 years every 10 years. Since the 1940's, the record mode for the females rose 1.4 years, every 10 years. Considering males, the pace of increase since 1970 was 1.2 year in 10 years. Female life expectancy and median age at death decelerated, while the mode accelerated in the last decade of the 20th century (CANUDAS-ROMO 2010).

The mortality reduction impact on the measures depends on the age at which such improvement happens (CANUDAS-ROMO 2010). Canudas-Romo (2010) points out that the median will only vary if the mortality reduction is registered in the age interval before the number of survivors (lx) is equal to 0.5. By its turn, the mode to change depends that mortality improvement occurs in ages above the mode (CANUDAS-ROMO 2010). Mortality reductions in ages before the mode will make the number of survivors (and consequently the number of deaths) higher in some ages, still the number of deaths will not be greater than the amount observed at the modal age at death. Since more survivors will reach age M, consequently more deaths will be registered at this age (CANUDAS-ROMO, 2010). Therefore, a rise in M implies mortality reductions in ages above the mode. In order to obtain a higher number of deaths in a given age (above M), enough survivors have to live up to then (CANUDAS-ROMO 2010).

In a life table, the ndx column indicates the number of deaths in a synthetic cohort observed in the interval between age x and age x+1 (CHEUNG et al. 2009). According to Keilman et al. (2017), ndx has a pleasant property, even though age specific mortality rates do not have additive property (as fertility rates does), the ndx sum in all ages equals the radix of the life table, which means that one can interpret the values of ndx as the distribution of death by age. For a discrete age x, ndx is the unconditional probability of dying at exact age x, considering a life

table with radix equal to one (KEILMAN et al., 2017).The ndx column is the main variable to estimate de modal age at death. The number of deaths by age in mortality regime is given by the number of survivors up to that age multiplied by the correspondent force of mortality, $d(a,t)=l(a,t)*\mu(a,t)$ (CANUDAS-ROMO, 2008) .

The death distribution by age in a given mortality regime is bi-modal (KANNISTO, 2001; CANUDAS-ROMO, 2010). The first mode is observed at age 0, while the second mode is placed in advanced ages. In high mortality countries, the first mode is higher than the second one, although it is not the case for most countries these days (KANNISTO, 2001; HORIUCHI et al. 2013; CANUDAS-ROMO, 2008). In Russia and in other countries like Brazil (for males), the increase in mortality for young adults made identifiable another mode in those ages. However, still, it was lower than the old age mode (CANUDAS-ROMO, 2008; CANUDAS-ROMO, 2010). van Raalte and Caswell (2013) note the presence of the second mode in adulthood ages, in 1918, for France male (during WWI and year of the great flu).

2.3 Compression of Mortality, Retangularization of the Survival Curve and Shifting of the Mortality

As the probability of surviving to higher ages rises, the survival curve becomes rectangular (WILMOTH & HORIUCHI, 1999). Related to this process, the compression of the mortality makes age at death distribution more concentrated in a narrower interval, as age at death variability reduces (WILMOTH & HORIUCHI, 1999). The compression of the mortality hypothesis, originally presented by Fries (1980), assumed that there was a limit in human longevity (BERGERON-BOUCHER et al., 2015). Fries (1980) foresaw that there would be a huge mortality decline at younger ages and that the distribution of death by age would present a normal distribution with mean (mode) 85 and standard deviation equal to four (WILMOTH & HORIUCHI, 1999). Both life expectancy (mean) and the modal age at death thresholds have been crossed (VALLIN & MESLÉ, 2016; HORIUCHI et al., 2013; CANUDAS-ROMO, 2008). Age at death variability, however, is still higher than Fries (1980) suggested (OUELLETTE &BOURBEAU,

2011; WILMOTH & HORIUCHI, 1999). According to Wilmoth and Horiuchi (1999), the rectangularization of the survival curve does not mean that there is a limit in human longevity. Nevertheless, if there were a limit to life expectancy, the reduction of mortality would necessarily lead to the rectangularization of the survival curve (WILMOTH & HORIUCHI, 1999).

The main contribution of the analysis of the variability in the age of death rests on the way the individuals live and plan their lives (WILMOTH & HORIUCHI, 1999). In that sense, Tuljapurkar (2010) highlights that longer lives affect individual decisions about the level and timing of lifecycle decisions like schooling, work, savings, and retirement. At population level, same author argue that it changed the flows of labor and money, education, pensions, and health systems. Age at death variability, measured by the interquartile range (IQR), reduced expressively since mortality decline started. In rich countries, in the end of the last century, the middle half of the life table deaths was concentrated in a 15-year age interval, falling from above 60 at the end of the 19th century (WILMOTH & HORIUCHI, 1999).

Wilmoth and Horiuchi (1999) described the dynamics of the IQR variation by age group of mortality reduction. Since the second half of the 20th century, old age mortality decline contributed significantly to the IQR changes. If mortality reduction occurs in ages bellow the first quartile, IQR diminish since the first quartile will be placed in a higher age, compressing the age at death distribution. Mortality reduction in ages above the first and bellow the third quartile increases IQR, since it lengthens the age that marks the third quartile. Wilmoth and Horiuchi (1999), pointed out that once populations achieved moderately low levels of mortality, the percentiles of the distribution of ages at death raised in parallel.

Other authors (CANUDAS-ROMO, 2008; CHEUNG & ROBINE, 2007; CHEUNG et al., 2009; ALVAREZ et al., 2020) observed that the compression of the mortality curve stopped in countries with low mortality, while the modal age kept rising. The shifting of the mortality curve hypotheses, formulated by Boongarts (2005), refers to the shifting pattern of the force of mortality, while its shape do not change. In that process, while the curve is shifting, the hazard function falls, but maintains the same age profile, while the survival curve increases

(CANUDAS-ROMO, 2008; BERGERON-BOUCHER et al., 2015). Based on the analyses of six low mortality countries, Canudas-Romo (2008) observes that the shifting of the mortality curve may well describe the current pattern of mortality chance in such countries. Even if the shifting process is only a temporary phenomenon, author understands that it is relevant to the identification of different patterns and processes related to mortality change.

Cheung and Robine (2007) argue that in Japan - since the 1980's for females and 1990's for males - the standard deviation above the mode (another age at death variability metric) remained stagnated. In that country, in a 50 years period, the modal age at death rose more than 11 and 13.3 years, respectively, for men and women. Considering five low mortality countries, France, Italy, Switzerland, Japan and Sweden, Cheung et al. (2009) identified that only in the last of those the compression of mortality process was still occurring. Bergeron-Boucher et al. (2015) decomposed life expectancy gains in terms of changes in the timing of mortality and in terms of the lifespan extension. The authors, considering Swedish females and a group of countries from the HMD database argue that, before 1950, the rise in life expectancy was mainly driven by compression of the mortality process. Since then, mortality reduction in higher ages was the most important factor in the life expectancy gains.

The literature on the modal age of death and on lifespan variability considered mainly countries with low mortality (CANUDAS-ROMO, 2008; CANUDAS-ROMO, 2010; OUELLETTE & BOURBEAU, 2011; OUELLETTE et al., 2012; KANNISTO, 2000; KANNISTO 2001). Yet, there is a lot of heterogeneity in these countries. Canudas-Romo (2008) and Ouellete & Bourbeau (2011), for example, observed that the USA had the less concentrated age at death distribution. Shkolnikov et al. (2003), note that, in 1997, USA and U.K had similar life expectancy at birth, but, due to higher mortality from external causes, USA had higher age at death variability.

In the USA, the differentials in mortality between states are quite expressive. Such internal disparities may explain why USA lags behind countries with similar development levels (WILMOTH et al., 2011). Wilmoth and colleagues (2011) points out that, between 1999 and 2001, Hawaii had the highest life expectancy

at state level (79.7) and Columbia District had the lowest (72.3). Considering life expectancy at age 50, between 1950 and 2000, the difference between the best and worst achieving state diminished very modestly as it passed from 4.5 to 4.4. In the two last decades of the 20th century, the path of expected remaining year of life of the richest half of American population (above age 50) is comparable to that of the rich countries. At the other side, since the 1980's, mortality among the poorest stagnated (WILMOTH et al., 2011; EZZATI et al., 2008).

Ouellete et al. (2012) looked at retangularization and compression of mortality processes in the Canadian provinces. They observed that there was an upward trend in the modal age in all provinces. Although, the regional disparities measured by the mode remained practically the same in the period analyzed. Authors highlighted that there were reductions in life expectancies differentials between provinces in consequence of the progress in infant mortality and in infectious and parasitic diseases.

Edwards and Tuljarpurkar (2005) decomposed age at death variability by income and schooling. Individuals in underprivileged situation presented lower age at death and higher variability. However, even after the exclusion of poorer individuals, variability of the age at death in the USA was higher than in the other countries analyzed. When it comes to gender differentials, Edwards and Tuljarpurkar (2005) shows that differentials are related to different exposure to risk. Although, when they removed external causes, the age at death variability gap dropped by less than one tenth. Several authors tried to analyze sex differentials in health and mortality and observe that it might be a combination of biological, behavioral and social factors, as well as their interaction (CRIMMINS et al., 2010; PRESTON & WANG, 2006; GLEI& HORIUCHI, 2007).

Firenbaugh et al. (2014) decomposed blacks and whites age at death variability in the USA. They figured that lifespan is more uncertain among blacks in a great extent (87%) because age at death varies more for them in comparison with white dying from the same cause. Age at death varies more for blacks than for whites in almost every cause of death. Only 12% of the differences in the age at death variance are explained by differences in cause specific death rates (FIREBAUGH et al., 2014). van Raalte et al. (2018) argue that lifespan inequality is the most

fundamental of all inequalities, as all other kinds of inequality are conditional on being alive. Same authors understand that mortality inequalities, as economical differentials, should be approached understanding differences within populations. They argue that age at death variability measures should be used to supplement measures of average longevity.

There is a growing interest in analyzing lifespan variability in developing countries (GONZAGA et al. 2018; ALVAREZ et al., 2020; SOLIS & GÁRCIA-GUERRERO, 2019; ABURTO et al., 2018). Gonzaga et al. (2018) focused on 13 countries from Latin America and Caribe (LAC) to understand changes in the age at death variability. Authors found similar pattern among the analyzed countries: in most cases, a decline followed by an increase. Rising mortality from homicides and transport accidents are pointed as the main reason behind the reversal in the trend. In their study, Brazil, Argentina and Colombia age at death variability kept declining, although at a slower pace. In those cases, infant mortality decline, probably, was strong enough to compensate the effect in age at death variability cause by violent deaths at young ages (GONZAGA et al., 2018).

Alvarez et al. (2020) observe that, generally, standard deviation in age at death was lower in LAC countries with higher life expectancy. In Panamá and Mexico, authors found higher age at death variability for males and females compared to countries with similar life expectancy levels. Life expectancy was correlated to age death variability diminution towards HMD countries level (ALVAREZ et al., 2020). Solis & Garcia-Guerrero (2019) also observed a strong negative correlation between life expectancy and inequality in the age at death. Alvarez et al. (2020) highlight that amenable diseases increase lifespan variability in LAC in comparison with HMD countries, meaning that deaths from that group of causes are more dispersed along the age span. They also found that the same amenable diseases contributed to lifespan diminution (compression) in LAC, especially in Brazil and Ecuador. For males, external causes account for a large share in the variability gap between LAC and the benchmark (HMD countries).

Gonzaga and Costa (2016) considered age at death variability in Brazilian regions between 1980 and 2010. For women, compression of mortality and retangularization of the survival were more expressive, female presented a lower

IQR, especially in South and Southeast regions (GONZAGA & COSTA, 2016). In São Paulo state, between 1980 and 2005, Gonzaga et al. (2009) observe a period of rising in the age of age at death variability followed by a period of diminution. Interquartile Range was always lower for women. In 1995, the gender gap in IQR reached its highest point, 5.88. Between 1980 and 1991, São Paulo presented substantial rise in homicide deaths rates that affect, primarily, young-adult men (CERQUEIRA, 2014), that may be among the reasons behind the gap observed in age at death variability.

Central and Eastern European countries also provide an interesting view of the age ate death variability changes, given mortality dynamics observed in the last decades. Aburto and van Raalte (2018) points out that, after 1991, Russia and the countries from the Balkans experienced expressive rise in age at death variability, the analyzed indicator reached it maximum in 1994-1995. After that point, besides some fluctuation, all countries in Central Europe presented diminishing life span variability (ABURTO & van RAALTE, 2018). Shkolnokov et al. (2003) argue that - except for the period 1985-1987 (related to Gorbachev alcohol campaign), from 1964 to 2000, mortality continuously increased for ages between 15 and 69, the age group with 70 or older also increased, although at a slower pace.

2.4 Compression of Morbidity

In developed countries, mortality reduction is the main responsible for the great rise in old age population (ROBINE et al, 2009; MANTON et al., 1982). Robine and Saito (2003), for example, estimated that 55% of the increase of the centenarian population in Japan since 1972 was a consequence of mortality reduction. As old age population increases, rises the concerns with the quality of the additional number of years those individuals are expected to live, as well as worries with the costs working age population may have to carry (LEE & GOLDSTEIN, 2003; CAI & LUBITZ, 2007).

According to Fries (1980), disability and poorer life quality are linked to the eventual death of an individual. However, same author observes that as mortality

diminishes disability is also likely to decrease, consequently, morbidity would be compressed into a shorter period between the disability onset and the moment of death. As already mentioned in a previous section, Fries (1980) understood that life was biologically limited. In the process Fries (1980) named Compression of Morbidity, death and disability would both occur later in life. At that point, the author thought that health improvements in the decades to come would be concentrated on chronic diseases and on the improvement of quality of life, not on its extension.

Gruenberg (1977) presented a less optimistic view on how the additional years of life would be lived. Author argued that the net effect of medical innovations that led to mortality reductions was to raise the prevalence of some diseases and disabilities. Accordingly, the longer lives would be lived with higher occurrence of disease and disability. Kramer (1980) shared the same concerns and argue that there were two mechanisms behind the rise of disability prevalence, the increase of the population with highest disability risk (old age groups) and rise in chronic diseases duration due to mortality reduction (KRAMER, 1980).

Olshansky et al. (1991) structured the view that old age mortality reduction would lead higher prevalence of disease and disability as the extension of morbidity hypothesis. Such hypothesis would be characterized by a postponement in the age of the fatal disease onset, a decrease in fatal disease incidence and diminution of age specific death rates. At the same time, however, there would be an increase in the time between the onset of non-fatal disease and the consequential death, age specific disability rates from non-fatal diseases would rise as well (OLSHANSKY et al., 1991).

In comparison with the compression of morbidity or expansion of morbidity perspectives, Manton (1982) adopted a more moderated position (CRIMMINS & BÉLTRAN-SANCHES, 2010; ROBINE et al., 2009; MANTON et al., 1982). Manton et al. (1982) observes that besides differences in the optimism degree, authors like Fries (1980) and Gruenberg (1977) had important points in common, for example, they shared the view that chronic diseases should be the focus of public health actions. Manton (1982) proposed another way to look to the relation between a rising life expectancy at older ages and the quality of those additional

years. According to the dynamic equilibrium perspective, the duration of disease would increase with mortality reduction, but its severity would also be reduced (MANTON, 1982).

The morbidity consequences of the old age mortality reductions are, however, difficult to define, as it is hard to quantify functional disability. The manner interest variables are constructed and the methods applied in the analysis can produce different results (CAI & LUBITZ, 2007; CRIMMINS & BELTRÁN-SANCHES, 2010; NEPOMUCENO & TURRA, 2015; CAMARGOS & GONZAGA, 2015).

Cai and Lubitz (2007) analyzed disabilities trends in the USA for the population with 65 and over, between 1992 and 2003, their results do not agree integrally with the compression/expansion/dynamic equilibrium perspectives. Authors observe that life expectancy with severe disability diminished, such result fits to the compression of morbidity perspective. At the same time, however, they found an increase in the active life expectancy, which can be related to the dynamic equilibrium perspective. Cai and Lubitz (2007) did not find support to the expansion of morbidity perspective.

By their turn, Crimmins and Béltran-Sanches (2010) analyzed morbidity and mortality trends for causes like Cardiovascular Diseases (CVD) , Cancer, Diabetes, Physical Functioning, among others, in the USA and highlight that little has been done in terms of eliminating or postponing diseases and disabilities. At the same time as diseases became less lethal and less disabling, there was an extension of life with disease and with mobility functioning loss (CRIMMINS & BÉLTRAN-SANCHES, 2010). Authors argue that the compression of mortality is a compelling idea and people want to live a healthy and lengthy life, but that it may be less likely to happen.

Beltrán-Sanches et al. (2014) point out that most of the analyses concerning how additional year of life are being lived relied on the time when disability started, not on the chronic disease onset. Beltrán-Sanches et al. (2014) understand that morbidity should not be considered only when there are functional loss or disability. Authors argue that the broader conception of morbidity represent a more close representation to the impact diseases can have on individuals and in

the society as a whole. The argument is based on the fact that chronic conditions increase the likelihood of developing functional mobility or disability, raise medical costs, cause sub-clinical damage and can be associated with psychosocial burden (BELTRÁN-SANCHES et al., 2014).

In less developed countries, naturally, there are also worries on how the additional years of life expectancy would be lived at older ages, especially given their the rapid mortality transitions and the cost of an ageing society in poorer countries context. Authors like Palloni et al. (2002) and Wong et al. (2006) observes the expressive mortality reduction, particularly at very young ages, are likely to impact on frailty distribution of the old age population in LAC countries. In that sense, Palloni et al. (2002) notice the growing evidence associating early ages exposure to harsh conditions and poorer health and mortality conditions at older ages. As they highlight, health status at old ages is consequence of mortality regimes in the past.

In poorer countries, cohorts reaching age 60, between 2000-2020, lived in a more severe mortality regime as they were younger and are more likely to present lower health status compared to individuals from same socioeconomic strata in developed countries (WONG et al., 2006;WONG et al., 2011). Those turning 50 near the year 2000 were born in the 1950´s mortality context, generally with high prevalence of infectious diseases (WONG et al., 2011). Wong et al. (2006) also emphasize that LAC countries are getting old without the institutional development in a context where with less kin available for care. They argue that, in developed countries, societies aged at a slower pace and it happened after diminishing social and economic inequality. While in LAC countries, the rapid aging process is marked by huge social inequalities and frail economies. The mixed epidemiological profile, prevalence of chronic diseases that co-exists with infectious diseases, is another distinguishing feature of the LAC ageing societies (WONG et al., 2011).

Wong et al. (2011) compared disability indicators from Mexico and USA to test the hypothesis that those from a worse epidemiological context at young ages (in Mexico) would be more frail (measured by physical disability) individuals compared to same age individuals in the USA (non-Hispanic White). Results,

however, reject their hypothesis. Americans are more likely to transit to disability and are more likely to increase the number of reported disabilities (WONG et al., 2011). Among the possible reasons for the unexpected result authors listed: Mexicans under-report physical limitations; analyzed cohort is highly selective; Mexicans present lower old age mortality (that can be related to age misreporting); current epidemiological and lifestyle context.

Camargos and Gonzaga (2015) analyzed three different dimensions of the prevalence of disability in Brazil, between 1998 and 2008. For women, the prevalence of women in good health rose. The difference for males was not statistically significant (CAMARGOS & GONZAGA, 2015). Authors found that women live, compared to men, more years in active state (disability free), although, proportionally, on average men live a larger proportion of their life in better health. Between 1998 and 2008, same authors show that life expectancy in good health rose at all age groups, for both male and female. The same picture was observed considering disability free life expectancy, except for women above 80. At the other hand, if good health is considered absence of disease, in a view similar to Beltrán-Sanches et al. (2014), the number of years lived with disease rose in Brazil (CAMARGOS & GONZAGA, 2015). In the analyzed period, total life expectancy above 60 rose, but the differences in life expectancy free from chronic disease was not statistically significant.

Using a different methodology, Nepomuceno and Turra (2015) observed an increase in the number of years lived with disability for Brazilian women, between 1998-2003 and 2003-2008. For women aged 65 in active state, the number of years expected to be lived in good health fell from 17.4 to 16.1 at the same time as total life expectancy for that same group rose (NEPOMUCENO & TURRA, 2015). Their results indicate a rise in the proportion of time lived with functional disability at age 65 from 11.8% (1998-2003) to 18.8% (2003-2008).

Beltrán-Sanches and Andrade (2013) considered disability trends in São Paulo and in urban centers in México differentiating those individual with at least one year of schooling and those that never went to school. In São Paulo, the work did not find significant differences between the likelihood of transitioning to disability and/or recovering from it, considering those with some or no schooling.

Considering Mexicans, recovery chance was lower and disability was higher for those with less education. In both São Paulo and Mexico urban centers, those with more schooling presented higher life expectancy at age 60 and higher disability-free life expectancy (BELTRÁN-SANCHES & ANDRADE, 2013).

2.5 Mortality trends in Brazil

In Brazil, mortality decline started in the 1930´s (WOOD & CARVALHO, 1994; CARVALHO & WOOD, 1978; PRATA, 1992; IBGE, 2010; SIMÕES, 2002). At that time, life expectancy at birth was close to 40 years and rose to 43.6 in 1940 (WOOD & CARVALHO, 1994). In 1970, the same indicator reached 62 years old. Wood and Carvalho (1994) attributed mortality decline in Brazil at that time to both improvements in standard of living and diffusion of medical and health technologies. Compared to developed countries, Brazil - along with many others developing countries - presented a very rapid mortality decline (ARRIAGA & DAVIS, (1969); PALLONI et al., 2011; PRATA, 1992).

Between 1940 and 2010, life expectancy rose more than 30 years, the most expressive increase in a single decade was registered between 1970 and 1980 (SIMÕES, 2002; IBGE, 2010). In that period, according to Simões (2002), male and female life expectancy at birth rose more than seven years. Infant mortality decline contributed the most for such rise (SIMÕES, 2002). Several authors (SIMÕES, 2002; MERRICK, 1985; BARRETO et al. 2011; SCHRAMM et al., 2004) understand that public policies - vaccination campaigns, wider access to pipped water and sewage systems - were the main responsible for the observed decline.

It is important to note that there are few studies analyzing the onset and the first decades of mortality decline in Brazil, mainly due to data quality or even it existence at all. Carvalho (1974) argued that, at that point, the real Brazilian mortality pattern was in fact unknown. His life expectancy analysis considered infant mortality estimates based on Brass method (from census data) along with model life tables. Merrick (1985) also note that most child mortality analysis available at that point were based on indirect measures from censuses and the

PNAD[2] surveys. The first Brazilian vital statistics system was established in 1974 (MERRICK, 1985).

Merrick (1985) detected that the expansion of mother and husband educational attainment contributed significantly to infant mortality decline in Brazil between 1970 and 1976. He also found that expanded access to pipped water also played an important role, although in a lesser extent. The expansion in the distribution of households with access to pipped water attenuated infant mortality differential related to educational attainment (MERRICK, 1985). Sastry (1997) analyzed differences in infant and child mortality between rural and urban areas. In 1986, infant and child mortality was considerably higher in rural areas in Northeast and the Southeast/South regions. After controlling for household variables, the differences diminished but remained important in the Northeast. There, after adding community covariates, the urban-rural child mortality gap became statistically insignificant (SASTRY,1997).

Between 1990 and 1999, Brazilian mortality below age five declined 28% (DUARTE et al., 2002). As authors argue, there was great regional differences on both child mortality levels and observed decline. In 1999, infant mortality in the Northeastern (highest) region was 3.1 times higher than it was in the South (lowest). At state level, Alagoas (highest) child mortality was 4.2 times higher than the observed in Rio Grande do Sul (lowest) (DUARTE et al., 2002). Child mortality regional differentials diminished between 1990 and 2015 (FRANÇA et al., 2017a). In that period, França et al. (2017) demonstrate that infant mortality decline was more important in the Northeast, that had highest level in 1990.

Between 1990 and 2015, congenital anomalies mortality in infants varied little and became the leading cause in several states, indicating an epidemiological profile closer to developed countries (FRANÇA et al., 2017a). However, at the same time, same authors found that avoidable causes (communicable, maternal, neonatal, and nutritional conditions) were still the leading causes in 2015 for children with less than five years old.

[2] Pesquisa Nacional por Amostra de Domicílio (National Household Sample Survey).

The epidemiological transition theory (OMRAN, 1971) describes the process of mortality and morbidity profile change from high incidence of infectious and parasite diseases, with low life expectancy, to another with high prevalence of chronic disease with high life expectancy. Omran's (1971) transition was characterized by three phases. 'The Age of Pestilence and Famine', with high and fluctuating mortality, 'The Age of Receding Pandemics', with progressive mortality decline, and 'The Age of Degenerative and Man made Disease ', when mortality decline continues and would, then, stabilize at lower levels. Omran´s proposition was well suited to describe the epidemiological transitions in developed countries until the 1970 (MESLÉ & VALLIN, 2011).

Other authors, including Omran himself (OMRAN, 1998), added other phases to the original theory intending to aggregate epidemiological patterns not initially predicted (HORIUCHI, 1999; OLSHANSKY & AULT, 1986; RODGER & HACKENBERG, 1987). Meslé e Vallin (2011) observes that, even with additional phases, the epidemiological transition theory is constantly challenged. The theory was not able to describe integrally the transition process in many developing countries (BORGES, 2017; FRENK et al. 1991). Frenk et al. (1991) described the transition in a group of Latin America and Caribbean countries using the prolonged and polarized model. Such framework is characterized by the superposition of phases, by the reversibility of changes (counter-transition), and by a prolonged process where different types of diseases coexist. Polarization is another distinctive feature of the LAC transition, where individuals from different socioeconomic conditions often suffer from different health conditions (FRENK et al. 1991; FRENK et al. 1989).

In Brazil, Schramm et al. (2004) argue that different theoretical epidemiological transition phases happened simultaneously and the re-emergence of some infectious or parasitic diseases was registered. In that same sense, Leite et al. (2015) characterize Brazilian epidemiological profile as complex, along with the prominence of non-communicable diseases it still presents a non-negligible incidence of diseases related to maternal and perinatal conditions.

Illustrating such complexity, Schramm et al. (2004) found that (based on 1998 data) diseases from Group 2 (Non-Communicable) were responsible for 66.3%

of burden of disease in Brazil, while Group 1 diseases (infectious and parasitic diseases, maternal conditions, and nutritional deficiencies) represented 23.5% of the Disability-Adjusted Life Year (DALY). External causes (Group 3) embodied 10.2% of that indicator in that year. In 2008, Group 1 diminished to 13.2%, non-communicable disease rose to 77.2%, and external causes represented 9.5% of the burden of disease (LEITE et al., 2015).

Besides declining incidence, infectious diseases still are a public health problem in Brazil (BARRETO et al. 2011; BORGES, 2017). Between 2000 and 2010, Corrêa and Miranda-Ribeiro (2017) estimated that almost 10 percent of male life expectancy gains were related to infectious or parasitic diseases. In 2016, Martins-Melo et al. (2018) studied Neglected Tropical Diseases (NTD) – a group of 12 infectious and parasitic diseases - and observed highest rates in the less socioeconomically developed regions (North, Northeast, and Central West). Minas Gerais state (Southeast) was among those FU with highest NTD mortality. The lowest rates were registered in the Southern region (MARTINS-MELO et al., 2018).

Among NTD, the highest DALY was observed in both sides of the age distribution (MARTINS-MELO et al., 2018). The age distribution of infectious diseases is getting closer to the profile observed in high-income countries, more frequent in adults than in children (BARRETO et al. 2011). Since mortality started declining, several infectious diseases were successfully controlled (diarrhea, cholera, pertussis, diphtheria, measles), there are though cases of failure in control (dengue fever and visceral leishmaniosis) (BARRETO et al., 2011).

In 1985, cardiovascular diseases already were the most prevalent cause of death in Brazilian state capitals (PRATA, 1992). Non-communicable diseases are becoming the main public health priority in Brazil (SCHMIDT et al. 2011). Leite et al. (2015) argue that chronic diseases represented the majority of the burden of disease in all Brazilian regions in 2008. Cardiovascular ischemic disease was the main cause of DALY for the male population and the second cause for females (LEITE et al. 2015). In 2011, chronic diseases represented 72.2% of all deaths registered in Brazil (MALTA et al. 2014). Mortality decline from such illnesses – considering the age group between 35 and 59 - from 1991 to 2010

contributed significantly to male life expectancy rise (BORGES, 2017). For females, Borges (2017) argues that middle-aged adults showed a more regular and significant path between 1980 and 2010, driven mainly by reduction in the circulatory system related deaths.

Brant et al. (2017) observed that CVD in 2015 (31.2%) represented a slightly higher share of the burden of disease in comparison to 1990 (29.3%). In the same period, however, CVD mortality declined 40% (BRANT et al., 2017). Although, as the authors highlight, it was a heterogeneous decline. North and Northeast regions presented the lowest CVD decline and, consequently, the highest CVD rates. For women, CVD mortality reduction in the analyzed period was more expressive in those same regions (BRANT et al., 2017).

Between 2000 and 2010, mortality decline from cardiovascular diseases in ages above 60 represented an important share of female life expectancy rise (BORGES, 2017; CORREA & MIRANDA-RIBEIRO, 2017). The older age groups have been contributing to life expectancy gains (BORGES, 2017; CORRÊA, MIRANDA-RIBEIRO, 2017; SIMÕES, 2002). In that same decade, women above 80 years old contributed more than infants to female life expectancy gains (BORGES, 2017). Same author found that mortality decline that group is concentrated in the South and Southeast regions.

Along with the male external cause's, cardiovascular diseases are the main reason behind gender life expectancy differentials (BORGES, 2017; SIMÕES, 2002; BRANT et al., 2017). According to Brant et al. (2017), male and female CVD mortality decline was relatively similar, respectively, 39.8% and 41.2% between 1990 and 2015. CDV mortality rate for males was, however, considerably higher in 2015, 315.8 deaths per 100.000 inhabitants, while female rate was equal to 210.7 (BRANT et al., 2017). Considering ischemic heart disease (IHD), Daniel et al. (2005) observes that part of the sex mortality differential might be related to the fact that men usually seek assistance later after in beginning of the symptoms and they usually do not consider the symptoms as seriously as they should. In the same sense, Parahyba (2005) highlights that Brazilian old age women seek medical assistance more often than their male counterparts do, even after controlling for socioeconomic characteristics.

For the 2016 cohort, if mortality conditions were held constant, a baby girl (79.4) would live on average 7.2 years longer than a baby boy (72.2) (IBGE, 2017). In 1940, that difference was about 4.4 years and rose to 7.8 years in 2000. Excessive male mortality, however, is not a unique Brazilian feature. Mortality differences between male and female are explained by biological and behavioral factors (SCHUNEMANN et al., 2017; LUY, 2003; PRESTON & WANG, 2006). Women are less likely to engage in risk behavior; they more often seek medical assistance and do it earlier as symptoms appear. There are also anatomic and physiological reasons for the observed female mortality advantage. Female investment in health is higher in both young and middle ages (SCHUNEMANN et al., 2017; LUY, 2003). Behavioral factors explain most of the observed differential (LUY, 2003; SCHUNEMANN et al., 2017; PRESTON & WANG, 2006).

In Brazil, male mortality is higher in all ages. Besides CVD (BRANT et al., 2017), male mortality is higher in NTD (MARTINS-MELO et al., 2018), external causes (CAMPOS et al., 2015; LEITE et al., 2015) and in every group of avoidable causes Abreu et al. (2009) studied. It is very likely that behavior is also the main force behind sex gap.The second and the third more important mortality causes for males were, respectively, homicides and violence and alcohol abuse, while that same causes do appear among the 10 more important for females (LEITE et al., 2015). Same authors observe also that male mortality is higher in every Brazilian region and that the registered gap is the highest in the Northeast region.

Between 1980 and 1991, the sex differential in life expectancy at birth rose by 1.4 year, the more expressive increase observed in a single decade. (SIMÕES, 2002). That period was marked by substantial rise in young men homicide rates in Brazil (CERQUEIRA, 2014). Simões (2002) observed that young male (15-29) mortality contributed negatively to life expectancy change between 1980 and 1991. External causes are the third leading cause of death in Brazil. Among male with less than 49 years old, however, it is the leading cause of death (BRASIL, 2016).

Amid external causes, violent deaths and transport accidents are, respectively, the two most important causes (CAMPOS et al. 2015). Homicide rates rose significantly between 1980 and 2000 (CERQUEIRA, 2014). In the following

decade, however, it showed little variation (IPEA, 2017; CERQUEIRA, 2014).During the 2000-2010 period, violent death rates varied regionally (IPEA, 2017; WAISELFISZ, 2013; CAMPOS et al, 2015; MINISTÉRIO DA SAÚDE, 2015). Borges (2017) shows that in Southern and Southeastern regions young adult mortality contributed negatively to life expectancy changes in 1980-1991 and 1991-2000, while the same trend was not identified in North and Northeastern regions. Between 2000 and 2010, however, the picture was quite the opposite. In North and Northeast regions young adult mortality contributed negatively to life expectancy gains, while Southern and Southeastern negative contribution ceased (BORGES, 2017).

Waiselfisz (2013) observes that in all but one Northeastern state (Pernambuco), homicide rates rose between 2001 and 2011, while in the North the same cause of death rose 75%. Between 2005 and 2015, in six states from North and Northeast regions homicide rate rose more than 100% (IPEA, 2017). Same study indicates that in Rio Grande do Norte (Northeast), homicide rate rose 232%. At the other hand, homicides rates diminished in all Southeastern states (São Paulo, Rio de Janeiro, Minas Gerais and Espírito Santo) (IPEA, 2017).

Different mortality patterns among regions are not new in Brazil nor are exclusively related to violent deaths (WOOD & CARVALHO, 1994; BORGES, 2017; SIMÕES, 2002). Borges (2017), using estimates from different authors, illustrates how Northeastern (poorest) and Southeastern (richest) life expectancy differential evolved since 1940's. In 1950, male Southeast life expectancy at birth was 13.3 years higher than Northeast, the highest gap ever registered. In 2000, regional gap among males was 3.4 years (the lowest). Another divergence took place between 2000 and 2010, when that gap rose to 4.9. In that period, as already mentioned, several Northeastern states experienced rise in external cause deaths (BORGES, 2017; WAIZELFISZ, 2013; IPEA, 2017). From 1940 to 1960, female e° differential between Northeast and Southeast regions rose by 2.3 and reached its maximum (9.8). A period of convergence was observed from 1960 to 1980, when the regional gap was equal to 7.0. Another divergence occurred between 1980 and 1991. Since then, life expectancy differential among those regions took another convergence path and reached its minimum (3.5) in 2010 (BORGES, 2017).

Regional inequalities in Brazil are observed in socioeconomic, health and mortality measures (ALBUQUERQUE et al., 2017; BORGES, 2017; WOOD & CARVALHO, 1994; LEITE et al., 2015). Albuquerque et al. (2017) highlights that Northeast and North regions present the highest mortality rates and concentrate a large share of the poor municipalities. The distribution of Brazilian Health System services also reflects regional inequalities (ALBUQUERQUE et al., 2017). Between 2000 and 2016, the offer of health services in Northeastern and Northern expanded, and their socioeconomic conditions improved. However, none of the areas the authors analyzed in those regions was among the more developed measured by socioeconomic and health service characteristics.

The relationship between income and mortality have been discussed for a long time and there is several research relating lower socioeconomic condition to higher mortality outcomes (PAMPEL et al., 2010; CUTLER et al., 2006; EDWARDS & TULJAPURKAR, 2005; LANTZ et al., 1998). Brazilian income inequality is very high and persistent (MEDEIROS et al., 2015). According to Medeiros et al. (2015), the poorest half of the population accumulate less than 10% of total wealth. Inequality presents a very clear regional component. North and Northeast regions have the highest share of individuals living in poverty and, at the same time have higher inequality measures (IBGE, 2018). In general, states in worst socioeconomic situation present higher mortality in practically every cause of death (BRANT et al., 2017; WAISELFISZ, 2013; CAMPOS et al, 2015; FRANÇA et al., 2017a; BORGES, 2017; MARTINS-MELO et al., 2018). The 1980-2010 period registered important changes in Brazil in both mortality and socioeconomic terms. Looking how mortality measures changed at state level in that period may contribute to broaden our knowledge of the mortality dynamics in Brazil.

2.6 Age Misreporting

Booth and Gerland (2015) argue that non-sampling errors present in demographic data, generally, relates to both coverage and content. Those problems, as highlighted, are often systematic, non-compensating and usually introduce bias to the analysis. According to the same authors, those errors are

more frequent and more severe in populations less socioeconomically developed. In Latin America, for example, the number of registered deaths is a fraction of the real number of deaths and age misreporting is observed both in death registry system and censuses (PALLONI et al. 2016).

In the 1980's and 1990's, mortality crossover was an extensively discussed phenomena related to content error in demographic data (COALE & KISKER, 1990; COALE & LI, 1992; PRESTON et al., 1996; PRESTON et al., 1999; ELO & PRESTON, 1994). As Coale and Kisker (1990) present, mortality crossover is characterized by lower mortality at older ages in less developed populations, relative to a group in better conditions, at the same time as the first society presents higher mortality at younger ages. There were two main lines of explanations to such pattern: one relating mortality crossover to selection of survivors who passed through adverse conditions at younger ages, and another that understands biased data used to estimate mortality rates as the main cause behind the phenomena (COALE & KISKER, 1990).

The first reasoning recognizes that heterogeneity in susceptibility to mortality, at least in part, is the reason behind the crossover (VAUPEL & YASHIN, 1985; MANTON et al. 1987; VAUPEL et al., 1979). As frailer individuals leave the population at younger ages, the remaining more robust present lower mortality at older ages (COALE & KISKER, 1990; VAUPEL & YASHIN, 1985; VAUPEL et al., 1979; MANTON et al., 1987). In a heterogeneous population, Vaupel and Yashin (1985) observe that the cohort hazard rate is dependent on the subcohorts hazard rates, and it is also directly influenced by the proportion of the population belonging to each group. In that sense, overtime, as the frailer cohort leaves the population, given their higher mortality, the observed hazard rate will be more like the hazard rate from the more robust cohort (VAUPEL & YASHIN, 1985).

Vaupel and Yashin (1985) also argue that a crossover can also be observed considering two different populations where both the frailer and the more robust subgroups (that compound each population) present the same mortality rates. In such cases, significant differences in the proportions of each subgroup would explain the pattern. In their paper, Vaupel and Yashin (1985) present several examples of what they call ruses related to heterogeneity in transitions (deaths,

marriage, and unemployment) analyses. The main argument is that grouped individuals present differences in several aspects, and such differences affect the chances of transiting between states. Consequently, the surviving population will be different from the original group, and the observed dynamics will be naturally different from the same processes at the individual level (VAUPEL & YASHIN, 1985).

Several authors understood that heterogeneity in frailty was the main reason behind crossover process (VAUPEL & YASHIN, 1985; VAUPEL et al., 1979; PRESTON et al., 1996). Manton et al. (1987), for example, analyzed extensively mortality and morbidity differentials among African-Americans and whites in the U.S. Except for the elderly; the authors observed that the first group presented lower mortality at all ages. Manton et al. (1987) were not convinced that age misreporting alone could explain mortality crossover, and believed that heterogeneity may be among the reasons behind the crossover process.

Manton and Vaupel (1995) held another work that pointed heterogeneity among the reasons behind an observed crossover. They compared mortality above age 80 between U.S.A white population and other four developed countries. Besides its higher mortality before age 65, U.S.A presented the lowest mortality among the countries considered. Differently from the African American population, age reporting issues in death registry systems are not expressive among the white population in the U.S.A (HILL et al., 2000).

According to Coale and Kisker (1990), there are reasons to believe that harsh conditions at young and middle ages would also lead to higher mortality at older ages, not lower as the selection effect would indicate. It is plausible that the same poor environment would cause higher mortality in advanced ages (COALE & KISKER, 1990). An illustration that questions the heterogeneity perspective presented by the same authors is the fact that the exposure to events that affect health conditions (like infectious diseases or smoking) does not diminish the frailty of those not affected. Saw from that perspective, crossover in mortality was more likely to be a consequence of age misreporting (COALE & KISKER, 1990; PRESTON et al. 1996; ELO & PRESTON, 1994).

Political conditions and cultural aspects influence the level of age misreporting (COALE & KISKER, 1990). There are cases when individuals declare age improperly intending to appear younger or older, depending on the cultural importance of age in a given society (PRESTON et al., 1996). The quality of age information is strongly related to literacy levels (PALLONI et al., 2016). Another kind of problems that affect age reporting is that, very often, one individual supply information for all household members and are unable to do so properly (COALE & KISKER, 1990;HORTA, 2012).

Age misstatement is more expressive at higher ages (HORTA, 2012; COALE & KISKER, 1990; ORTEGA & GARCIA, 1986). The more common pattern begins with an upward transfer at age 60 or 70 and increases rapidly with age (COALE & KISKER, 1990). Same authors highlight that, since the previous age groups are usually larger, above 60 years old, each age group gain people from age overstatement. For example, the age group between 85 and 89 in China (1982) was 25% of the size of the previous age group (80-84) (COALE & KISKER, 1990). Therefore, age misreporting of both the death and the living reduces mortality estimates at old ages (COALE & KISKER, 1986; PRESTON et al., 1996, PRESTON et al., 1999).

Pursuing signs of age overstatement, Coale and Kisker (1990) chose a model life table that was able to project the population of interest from age *a* to be matched to the same population aged *a+T* in the T years later census. Authors observed that in countries with well-reported ages the mortality estimates required to match population T years after form a sequence in the approximated same level of in the selected model life table. At the other hand, in countries with less accurate data, as age advanced, that same exercise required sequentially the use of a lower level model life tables. Coale and Kisker (1990) argue that results got far from reliable.

The Chinese 1982 Census, analyzed by Coale and Li (1991), provides an illustrative case of the impacts of age misreporting. Given their close relation to the Chinese calendar, the Han majority population in China declare day at birth accurately, even the illiterate (COALE & LI, 1991). Compared to South Asia, Africa or Latin America, in China the preference for reporting ages terminating in

zero was very little. As the authors present, only 68 million out of one billion people were non-Han, and most of them lived in the Xinjiang Province. Interestingly, besides their minor relative importance, the non-Han tendency to overstate age had impact on old age mortality estimates in China.

Excluding Xinjiang Province from the analyses, influenced very little on life expectancy at birth estimates. At older ages, however, the impact of that omission was quite expressive (COALE & LI, 1991). The 95-99 age group male mortality rates in Xinjiang Province was 29% of the median of the other 28 provinces. Coale and Li (1991) consider such rates not plausible and understand that they affected old age mortality estimates for the whole country. The reason for such low mortality at older ages, as the authors discuss, is age misreporting pattern in the non-Han minority that represented most of the inhabitants in Xinjiang at the time of the census.

The African American lower old age mortality in the late 1970's and early 1980's, relative to white US population, was a extensively discussed crossover process (PRESTON et al.,1996; ELO & PRESTON, 1994; MANTON et al. 1987; HILL et al. 2000). According to Palloni et al. (2014), it is an extreme example of the potential damage of age misreporting in mortality estimates. As Elo and Preston (1994) pointed out, most observers attributed to heterogeneity the explanation for such phenomenon. Although, Preston et al. (1996) showed that African American age misstatement was expressive at older ages and the resulting bias affected mortality estimates.

Based on the intercensal cohort comparison, Elo and Preston (1994) found that many more African Americans were enumerated in age groups 65-69 and 70-74, especially in 1940-1960 censuses, than it would be expected considering the changes in both the size of the cohorts and mortality registered. For such age group, the main explanation relates to an incentive to overstate age given by Social Security System at the time (ELO & PRESTON, 1994). Above age 75, the number of African American enumerated fell below the number expected (ELO & PRESTON, 1994). Authors applied a test based on the redistribution of ages at death that considered a matching study developed in the 1960's –and argue that

results tend to support that age misreporting may be behind the differences between the enumerated an expected number of individuals at age 75 and over.

Another evaluation exercise taken by Elo and Preston (1994), based on model life tables, showed that crude death rate (CDR) above age 50 estimated with death counts from registry system and censuses and with the model CDR were similar. For that reason, they argue that CDR at 50 and over is the single most reliable indicator of old age mortality for African Americans. Thus, based in the CDR 50 + and a North model life table, the estimated age specific mortality rates above 50 and over displayed an age pattern of mortality completely different from the observed. There was no sign of crossover (ELO & PRESTON, 1994). According to the researchers, African American crossover observed in official statistics would mean a unique mortality pattern for that subgroup, not observed in any other country with reliable data.

In order to investigate and overcome such data problems for African American mortality estimates, Preston et al. (1996) deployed a matching exercise between individuals present on a sample of African American death certificates (1980 and 1985) that could be also identified in the 1900, 1910 or 1920 U.S.A censuses. Same procedure was made considering death data from Social Security Administration (SSA). The matching results was used to establish the correct age at death distribution and, then, to estimate more reliable mortality rates for old age African Americans (PRESTON et al. 1996).

If death certificates and early censuses age matched, 20.2% of women and 13.2% of men deaths would be enumerated in a higher age bracket (PRESTON et al. 1996). Preston et al. (1996) emphasize that, on average, age at death appears to be understated on death certificates on ages below 90 (above that age it seems to be overstated). The results is different from Coale and Kisker (1990) interpretation, but goes in the same direction other similar studies did (PRESTON et al., 1999). The matching based on SSA files presented a similar pattern.

Preston et al. (1996) list some of potential reasons behind age misreporting for observed in death certificates. For African Americans born before the 1920’s,

most of them from southern states, there was no (or inadequate) birth registry system. A second reason authors point out is a potential lack of chronological sense in that population. The deliberate understatement in the age of the descendent by the informant in order to appear younger, more frequently observed for women and the inability to appropriately report the age of the descendent were also listed.

After age at death distribution was corrected, mortality estimates showed no crossover between African American and white population below age 95 (PRESTON et al. 1996). Authors highlight that the 85+ age group was the only one in official U.S abridged life tables that presented African American mortality lower than white population. That was the same age group where uncorrected and corrected African American mortality estimates crossed over.

The number of deaths registered or the population enumerated in each age group is affected by both the direction of the misreporting and the size of each group in the underlying true distribution (PRESTON et al. 1996; PRESTON et al. 1999; PALLONI et al., 2016). The age distribution declines very rapidly at older ages, therefore there are many more people to be transferred upwards than downwards, even if the net effect of age misreporting is to understate age (PRESTON et al. 1996; PRESTON et al. 1999). As argued by Preston el al. (1999), the steeper the slope in true underlying age distribution, more people will be inaccurately reported in older ages in the presence of age misreporting.

Simulations held by Preston el al. (1999) investigated the effect of age misreporting in older age mortality estimations considering all possible kinds of misstatement in both the age distribution of the population and in the registered deaths. Authors found that net overstatement, net understatement and symmetrical misreporting of age increased the number of surviving individuals above a specific age. The first age inflated varied with the direction of the misreporting. Regarding the age distribution of deaths, the effects of the patterns of misreporting were the same, although in a lesser extent. As presented by Preston et al. (1999), differences in the impact on mortality estimates between age misreporting in the distribution of deaths and age misstatement in the

distribution of the population is due to the fact that in the first the true distribution begins a downward trajectory only above age 80.

Given that age misreporting impacts differently in both age distribution of deaths and age distribution of the living, age specific death rates will also be affected (PRESTON et al. 1999). Accordingly, Palloni et al. (2016) highlight that observed mortality will be underestimated, unless propensity to overstate age at death is expressively higher than propensity to misreport age for the living. The age at with distortions began varies with the pattern of statement. Preston et al. (1999) showed that all patterns of misstatement reduced death rates and produced crossover between true rates and mistakenly reported estimated rates. The pattern of misstatement influenced the age when the crossover was observed, biases started the earliest, around age 50, when age was overstated (PRESTON et al.,1999).

In developing countries, much less is known about the magnitude of age misreporting and its possible impacts on mortality estimates (PALLONI et al. 2016). Age misreporting, as Palloni et al. (2016) point out, relative to undercount, is harder to identify and to correct. Data usually available in most countries do not provide information about both the conditional propensity of individuals to misreport age and the conditional distribution of the difference between correct and declared age, which are the functions needed to correct age misreporting (PALLONI et al. 2016). At the other hand, in general, demographic data in such countries present severe problems related to coverage and content (PALLONI et al., 2016; Del PAPOLO, 2000; HORTA, 2012; GOMES & TURRA, 2009; QUEIROZ et al., 2017; AGOSTINHO, 2009; ROSENWAIKE & PRESTON, 1984).

According to Palloni et al. (2016), above age 40, both censuses and vital statistic in almost all Latin American Countries are affected by age overstatement. For example, in Brazil, same authors estimate that age overstatement increased life expectancy at age 60 by 1.2 year in 2005. Porto Rico had lower elderly mortality than it would be expected given their income level (ROSENWAIKE & PRESTON, 1984). Based on an intercensal cohort analysis, Rosenwaike & Preston (1984) found signs of age misreporting. Their study showed that there were almost as many intercensal deaths for the cohort 75+ (1960) as there were members in the

same cohort. At the same time, however, practically half that cohort was enumerated in the 1970 census. The authors argue that the only other (although, not plausible) explanation would be that deaths are over registered.

According to Del Papolo (2000), who analyzed quality of age information in eight LAC countries (Argentina, Bolivia, Brazil, Chile, Guatemala, México, Peru and Venezuela), the quality evolved but age misstatement is not a problem only in past censuses. Based on the Whipple Index, that measures digit preferences for the numbers 0 and 5, author observes that age declaration got better in most countries between 1970 and 1990. Age heaping (digit preference) is one sign that age has been misreported (COALE & KISKER, 1990).

In Brazil, age heaping diminished from 1970 to 2010, although, for males the indicator rose from 1991 to 2010 (HORTA, 2012). Same author found that in practically every Brazilian state the digit preference diminished between 2000 and 2010, especially in North and Northeast regions. It is important, however, to note that there was still huge regional differences. For example, Horta (2012) shows that males Whipple Index was 110.27 (moderate quality) in Amazonas, while it was 102 in Santa Catarina – the closer to 100, the better. Agostinho (2009) found a similar regional pattern based on the 2000 census.

As Horta (2012) observes, Whipple Index in the Brazilian states is closely related to how age is declared in Census. In 1970, only 65% of people enumerated in the Brazilian census informed age by the day of birth, compared to 95% in 2010 (HORTA, 2012). However, there was still relevant regional differences, as the same author showed.

Del Papolo (2000) presents age misstatement estimates for eight Latin American countries made by Grushka (1996), applying the extinct generation methodology. In Brazil, between 1980 and 1991, the ratio between the number of individuals enumerated in the censuses to the expected number of individuals based on death registry system and the age specific growth rate indicates impact of age misreporting is observable for women starting at age 55. For men, the ratio becomes larger than one (indicating over-enumeration in the census) around age 75 (Del Papolo, 2000, *apud* Grushka, 1996). Using same methodology, Gomes

and Turra (2009) investigated the number of centenarians enumerated in the 1991 Brazilian census. Authors found the number of centenarians indirect estimated to be one third of the number recorded in 1991. Part of the magnitude of the difference could be explained by imputation of age held by the Brazilian census bureau (IBGE), 65% of centenarians in the 1991 census had their age imputed (GOMES & TURRA, 2009).

Nepomuceno e Turra (2019) estimated the number of centenarians in Brazil, based on variable-r method, from 1900 to 2000. Authors found that in the past the prevalence of centenarians in Brazil was higher than in high longevity countries. In fact, prevalence of centenarians diminished between 1900 and 2010, despite the fact that life expectancy increased by more than 40 years in the same period, indicating improvement in data quality (NEPOMUCENO & TURRA, 2019). However, in 2000, the number of enumerated centenarians in the census was about 10 times higher than the number estimated. As authors argue, the estimated number of centenarians was considerably smaller than the enumerated, even smaller than the number of registered deaths above age 100.

Agostinho (2009) investigated signs of age misreporting in Brazil and its states based on the ratio between observed population and the estimated population (considering the same population 10 years younger exposed to the survival probabilities from a model life table), following Coale and Kisker (1990). For males, the survival ratio applied to estimate the population was higher than the observed. Even though, above age 80 (1970-1980) and above age 90 (1980-1990), the indicator showed signs of age misreporting (AGOSTINHO, 2009). Same author also analyzed differences between states, southern states along with São Paulo and Rio the Janeiro (Southeast) presented the lowest ratios between observed and estimated population. At the other side, in Northern and Northeastern states presented highest ratios, indicating that age misreporting might be more severe in such states.

Agostinho (2009) also targeted the quality of age information in the death registry system. Based on the Whipple Index, digit preference was not expressive for the 1999-2001 period, except from Roraima, Rondônia, Tocantins, Acre, and Amapá (North). Compared to Japan in 1980, ratios between the number of deaths above

age 70 to deaths above 60, between deaths at 80 and over to deaths at 60 and over, and the ratio between deaths above 90 to deaths above age 60, there were signs of age overstatement in most states. In states located in the South, Southeast and West-Central regions, the signs of age misstatement in death registry system was less important (AGOSTINHO, 2009).

At microregions level (comparable small area), there are signs of regional differences in the quality of age information at older ages (REIS & TURRA, 2016). Between 2000 and 2010, authors found that the number of microregions with between one and two centenarians by 10.000 people diminished. The north of Minas Gerais state, Bahia and the coastline of the Northeast region, were the regions that presented the highest prevalence of centenarians. It is important to highlight that these are not among the wealthiest regions with lowest mortality indicators. Reis and Turra (2016) also analyzed two other measures: the ratio between those aged 100+ and those with more that 85; and sex ratio in ages 100+. They argue that both measures presented values considerably higher than observed in countries with higher data quality and that are more advanced in the aging process.

Matching analyses for Latin American countries that would allow the estimation of the magnitude of age misreporting are very rare since the required data is generally not available in most countries. In Costa Rica, Ortega and Garcia (1985) investigated the accuracy in age informed in the 1984 census. One year after the census, people aged 60 and over in two Cantones (Costa Rican territorial administrative division) were interviewed and asked to show an identification document with date of birth information. Results showed that age misreporting was not that expressive, approximately 80% declared age accurately or within a one year error margin (ORTEGA & GARCIA, 1985). However, the same exercise wouldn´t allow to identify appropriately (or to capture the entire dimension of the issue) age reporting in countries like Brazil, where civil registry system was very problematic in the past (IBGE, 2014; IBGE, 2000; MELLO-JORGE et al. 1993;TURRA & WONG, 2007)

In 2014, IBGE estimated that 1% of all births were not registered. The regional distinctions in the under-enumeration of births is quite expressive, in the North

and Northeast, respectively, only 87.5% and 88.1% of the births were registered (IBGE, 2014). Although, that picture is much better that it was in a recent past. According to IBGE (2014), during the 1980's and 1990's, underegistration of live births varied between 30.3% and 17.8%. Another problem related to birth registry system in Brazil is that a substantial proportion the birth registrations happens later than the established legal period of three months. In 1974, 54.7% of births were registered within a 10-year period. That proportion diminished, in 2004, 10.2% of births were registered in a time period between four months and 10 years (IBGE, 2014).

Mello-Jorge et al. (1993) presents that the first estimative of the under-enumeration of births was made by Saade in 1947, when the author estimated that 38% of all live births in Brazil were not registered. Portela (1989) present one illustrative example of the magnitude of birth registry system problems in the municipality of Piripiri - located in the state of Piauí (Northeast), where the estimated under-coverage ratio of births was 68.7% between 1983 and 1984. Same authors observe that their result is close to other estimates made in municipalities in the North and Northeast regions. At the other hand, Mello-Jorge et al. (1993) highlight that Souza (1992) estimated that underegistered births in Maringá – located in the southern state of Paraná – represented 9% of the total all births in 1989.

Wong and Turra (2007) in a work developed for the Inter-American Development Bank highlight several efforts in the last decades to minimize the under-enumeration problems. It is interesting to note, however, that the problem of unregistered individuals was also noted in other public policies. In 2005, the Programa Nacional de Documentação do Trabalhador Rural (National Program for Rural Worker Documentation) emitted more than 140.000 documents for rural worker, among then, approximately 5% were birth certificates (WONG & TURRA, 2007). Another example, presented by the same authors, is that, in a literacy course for adults, 7.5% of the attendees did not have a birth certificate. As far as we could find out, there are no studies relating the quality of age information and the proportions of under-registered births. Although, both under-coverage and problems in the quality of the registration system are expected to influence how

age is stated, more expressively as people age and their birth is a farer event with less live testimonies and without a reliable registry.

CHAPTER 3 - DATA AND METHODS

3.1 Data

The number of deaths used in all estimates are public available at the Mortality Information System (MIS) – Sistema de Informação de Mortalidade (SIM), from Brazilian Ministry of Health Database (Datasus). MIS collects information on mortality by age, sex and cause of death in all municipalities. Using Tabwin[3] (Datasus data analysis software) the consolidated number of deaths can be downloaded by single age from 1979 to 2016 (MINISTÉRIO DA SAÚDE, 2019). We concentrate our analysis on the Federal Units (FU) level and Brazil as whole. The country is administratively divided into 26 states and the Federal District (Distrito Federal) that represent 27 Federal Units. The first analyzed year was 1980 because death counts series only starts in 1979. The last study year was 2010 (the last Brazilian census) because it was necessary to use population from censuses in the denominator.

Single age specific population for each year by sex and state was collected from the Brazilian censuses sample microdata (IBGE 1980, 1991, 2000, 2010). Single age population is also available at the IBGE website[4], but only for the two last censuses (2000, 2010). Another drawback in that data source, the open age interval is 100+ and we decided to work with 110+ as the last age group. Another possible option was to get single age population was the IPUMS website (MINNESOTA POPULATION CENTER, 2018). However, IPUMS also uses the 100+ as the open age interval. Another potential problem, their data is based on a sample of the census sample that can lead to differences in single age population that may be problematic considering smaller administrative units, especially at older ages

There were cases of both death and population (only in the 1980 Census) counts with unknown age. To deal with that situation, cases with missing age were

[3] Tabwin can be downloaded in the following link http://www2.datasus.gov.br/DATASUS/index.php?area=060805&item=3

[4] https://sidra.ibge.gov.br/home/pnadct/brasil

redistributed proportionately, respectively, considering the distribution of deaths and individuals by age in all years, for each sex.

Prior to smoothing the single age mortality curves and estimating the modal age at death (M), the median age at death (MD), the standard deviation above the mode (SD+) and the interquartile range (IQR), the first step was to adjust mortality levels by the degree of completeness of death counts registration using estimates made by Queiroz et al. (2017). One important limitations in the MIS database is the under-registration of death counts and its variability across states (QUEIROZ, et.al. 2017). Then, age specific mortality rates (for males and females) were estimated considering the sum of three year around the years when the census were held (numerator) and a measure of population exposure - developed by (Gonzaga and Schmertmann, 2016[5]) - for the same three years, in four census year (1980, 1991, 2000, and 2010). The exposure measure was developed to reduce the occurrence of specific ages with zero individuals counted in the denominator. The use of the exposure measure is also a way to diminish the impacts of digit preference. The use of a three-year sum in the numerator lessens possible random fluctuation problems with the death counts. The exposure measure is based on the idea that the number of person-year lived in the interval by individuals aged x can be measured based on census date using a surviving probability. We used the survival probabilities in from average of mortality from the Human Mortality Database (HMD) extended until age 110, as it will be more properly discussed in this Chapter.

To generate a smoothed complete mortality schedule by age we applied three methods: Log-Quad (WILMOTH et al. 2012), TOPALS (GONZAGA & SCHMERTMANN, 2016), and Beers Osculatory Interpolation (BEERS (1944), *apud* SIEGEL & SWANSON, 2004). All data was treated and analyzed using R, the two methods first mentioned have R codes public available developed by their authors. Beers osculatory interpolation was applied using the R package demotools, developed by Tim Riffe[6].

[5] The R code to estimate the exposure measure is available at http://schmert.net/topals-mortality/programs/exposure%20calculation.R
[6] https://github.com/timriffe/DemoTools

3.2 Adjusting for under-registration of death counts

To deal with the undercount of deaths we used the Adjusted Synthetic Extinct Cohort (SEG-adj) estimates produced by Queiroz and his colleagues (2017) for each Federal Unit, and estimates made by Queiroz et al. (2016) for Brazil as whole. It is important to mention that SEG-adj - and other Death Distribution Methods (DDM) - generates undercount estimates for the inter-census period 1980-1991, 1991-2000, 2000-2010, and there were four - centered around - years to correct (1980, 1991, 2000, 2010). In that sense, we corrected 1980 death counts using the adjustment factor based on the 1980-1991 estimates, the 1991 number of deceasing individuals was correct considering the 1991-2000 SEG-adj estimates. The last two census centered years (2000 and 2010) were corrected with the last available estimates (2000-2010).

SEG-adj is a Death Distribution Method (DDM) proposed by Hill et al. (2009) that combines General Growth Balance Method (GGB) (HILL, 1987) and Synthetic Extinct Cohort (SEG) developed by Bennett and Horiuchi (1981). Authors construct the age distribution of the population based on the number of deaths and the age specific growth rates (QUEIROZ et al. 2017; HILL et al. 2009). The procedure is based on the knowledge that the number of people in age x is equal to the observed number of deaths above age x, adjusted by age specific growth rates (QUEIROZ et al. 2017; HILL et al. 2009). In that sense, the number of deaths above x age combined with the specific growth rates provide an estimate of the population at age x. Thus, a ratio between the estimated population and the observed population above age x gives an assessment of the completeness of the death counts in a database (QUEIROZ et al. 2017; HILL et al. 2009).

By its turn, GGB is built upon the balancing equation from which the difference between birth rate at age x and plus and the population growth rate at age x and above gives an estimate of the mortality rate at age x and plus (QUEIROZ et al., 2017). The completeness of death registration is given by the relationship between the mortality rates estimated from two censuses as described and age specific mortality rates estimated based on death registry system. From an orthogonal regression between those two rates, the intercept gives the coverage variation between censuses and the slope of the regression gives the undercount

of deaths estimate of the death registry system (QUEIROZ et al. 2017; HILL et al. 2009). From the GGB, SEG-adj method uses the coverage variation between censuses as a first input to correct population coverage differences prior to estimating age specific grow rates (HILL et al., 2009).

All three DDM (GGB, SEG and SEG-adj) consider closed population and, thus, are sensitive to migration (QUEIROZ et al., 2017). Hill et al. (2009) suggest dealing with that issue by choosing an age group that is not that much affected by migration. Queiroz and colleagues (2017), from whom we use estimates, used the DDM [7]R package that suggests an age interval that minimizes the potential influence of migration.

Another frequent discussed problem related DDM methods is that they assume that omission is evenly distributed across the age range, and that may be a strong assumption (QUEIROZ & SAWEYR, 2012), especially considering the youngest (MURRAY et al. 2010). DDM methods also assume that age at both at death registry system and in population censuses are well declared (QUEIROZ & SAWYER, 2012), and there are clear signs that it may not be the case (HORTA, 2012; AGOSTINHO, 2009; TURRA, 2012; GOMES & TURRA, 2009). However, as Murray et al. (2010) argue it is still necessary to implement efforts to understand the magnitude of stochastic and systematic age misreporting so that kind of bias could be more properly treated.

3.3 Adjusting Mortality age profiles

3.3.1 Log-Quadratic

The first method applied for mortality estimation was the system of mortality tables developed by Wilmoth et al. (2012). The log-quadratic model (LQ) estimates a complete set of age specific mortality rates using as inputs the probability of dying before age five (5q0) and the probability of death between age 15 and 60 (45q15). The method also allows the estimation based only on the infant mortality parameter, since it is frequently observed in most countries

[7] https://cran.r-project.org/package=DDM

(WILMOTH et al., 2012). LQ do not produce an estimate of completeness of death counts coverage, but allows one to estimate full age profiles of mortality with limited or defective data. Equation 1 formalizes the model:

$$\log(m_x) = a_x + b_x h + c_x h^2 + v_x k \qquad \text{(Equation 1)},$$

Where log(mx) is the log age specific mortality rate, h = log(5q0) reflects the level of child mortality, k reflects the level of excess adult mortality and is chosen to match 45q15 or other global measure of adult mortality, the other variables are constants in the model. X represents each age group (0,1-4,5-9,10-14…110+). As Wilmoth et al. (2012) highlight, only one value of the parameters h and k are used in all ages and, in that sense, determine the level and shape of the predicted mortality.

The main disadvantage with LQ is that the constants (ax,bx,cx) in the model were constructed using 719 life-tables from countries in the Human Mortality Database (HMD)[8]. As it developers pointed out, there are only two developing countries (Chile and Taiwan) and only one large country with a non-European majority population (Japan). Naturally, the experience of those countries might not reflect the experience of less developed countries or countries more ethnically diverse. However, the method is very flexible and one could re-estimate the constants by adding other countries to the database. The main issue is that those additional countries might have data limitation and mortality age-profiles might have been estimated using indirect methods or model life-tables. According to Wilmoth et al. (2012), a tradeoff between accuracy and representativeness is unavoidable.

Canudas-Romo et al. (2015) applied LQ to study sex differentials in India and its states between 1970 and 2013 and the observed changes by age and cause. Along with two other life table system models, Hu and Yu (2014) applied log-quadratic to China data and compared the results. Sharrow et al. (2014), along

[8] www.mortality.org

with other indirect methods, also applied LQ prior to proposing a method that could capture HIV prevalence.

In our LQ application, once we corrected for the undercount of deaths, we estimated age specific mortality rates considering a three-year – centered on census year - death sum in the rate numerator. In the denominator, we considered the already mentioned population exposition estimates. From the set of age specific mortality rates, we estimated life-tables for each sex in each year of analysis (1980, 1991, 2000 and 2010). Then, the input parameters for the LQ (5q0 and 45q15) were then calculated from those life tables by sex for each year. We applied the model using the coefficients estimated by Wilmoth et al. (2012) with the HMD data. Since we will apply more than one method, we will be able to analyze and compare potential differences in the results in consequence of that choice. Once the age specific mortality rates from LQ were estimated, we constructed another set of life tables from which the analyzed functions (mx, dx, lx) were get.

3.3.2 TOPALS

The second method applied to estimate the whole mortality curve for Brazil and it states was the TOPALS (Tp) regressions developed by Gonzaga e Schmertmann (2016). Their model is based on TOPALS relational model developed by De Beer (2012). The original TOPALS model uses a linear spline to model the ratio between the observed age specific probability of death and the same measure from a standard age schedule (De Beer, 2012). If the standard age schedule presents a smooth pattern, argues De Beer (2012), multiplying that standard by a linear spline will produce a smooth age schedule.

By their turn, Gonzaga and Schmertmann (2016) propose a Poisson regression method based on a relational model. Their model “builds complete schedules of age-specific rates via mathematical adjustments to a specified standard schedule. Our version of TOPALS constructs a fitted schedule of log mortality rates at ages 0…99 by adding a linear spline function with seven parameters (α0 ... α1) to a pre-specified standard schedule. We estimate parameters by

maximizing a penalized Poisson likelihood function for age-specific deaths, conditional on age-specific exposure" (GONZAGA & SCHMERTMANN, 2016, p.632). In our application, we made a small change in their proposition, by adding an additional α parameter and changing the size of the B-spline matrix so the estimates could be taken until age 109. The position of the additional α parameter was decided after the visual inspection of the estimated age specific mortality curves in the sense that it more closely resemble the expected mortality trajectory.

$$\lambda_{110x1}(a) = \lambda^{*}_{110x1} + B_{110x8} * \alpha_{8x1} \quad \text{(Equation 2)}$$

Equation 2 presents the model, λ is the vector log mortality rates, λ* is the standard schedule, B matrix of constants in which each column is a linear B-spline function and α is vector of parameters representing offsets (GONZAGA & SCHMERTMANN, 2016). The knots were defined at ages 0,1,10,20,30,60,80,110. As Gonzaga and Schmertmann (2016) describe, α value represents additive offset to the log rate schedule at the exact ages where the knots are positioned. Between the knots, the offsets change linearly (GONZAGA & SCHMERTMANN, 2016).

De Beer (2012) presents the knots as the ages at which the successive linear segments are connected. Same author argue that the knots can be selected considering the optimal fit between the data and the linear spline (using a non-linear least square), however it would represent different knots for different countries (as he was studying). In our case, the picture is similar as we are considering different FU in different years. It would represent a different set of knots for every state in every year and for each sex.

Gonzaga and Schmertmann (2016) argues that their version of TOPALS is not sensitive to the pattern chosen, since mortality estimates change very little with the chosen schedule. However, in situation where the number of observations is small, the fit could be closer to standard. An attempt to use a standard closer to

a Brazilian mortality patterns was not effective. The Latin America Mortality Database - LAMbDA (PALLONI et al., 2014) make available mortality estimates corrected for both undercount and age misreporting for Brazil for three different periods (1985, 1995, 2005) (PALLONI et al., 2016). However, such estimates are grouped into five-year age intervals and the open-ended age group is 85+. We tried to create a single aged pattern from LAMbDA dataset using a combination of two different mortality laws, Heligman & Pollard (1980) from ages 0-95 (predicted from 85 to 95), and Kannisto (1992), from ages 95 to 110. The results, however, did not presented a good fit, especially in younger ages. For that reason the standard applied in TOPALS estimates was the average of the Human Mortality Database (HMD) in 2015, same used in Gonzaga and Schmertmann (2016), extended from age 99 to 109 using Kannisto (1992) mortality law (THATCHER et al., 1998). The application was made using the R package *MortalityLaws* developed by Marius Pascarius and Vladimir Canudas-Romo (2018).

Mortality rates for the HMD mortality pattern in the 100-109 interval were predicted using Kannisto (1992) mortality law. Similar to Thatcher et al. (1998), we used the 80 to 99 observed interval to predict the 100-109 one. Same authors argue that Kannisto model is simple, easy to fit and that the resulting extrapolation are consistent. In their evaluation, the author found that the model behaved well after age 90 for men and after age 95 for women.

$$\mu_x = c + \frac{ae^{bx}}{1 + ae^{bx}} \quad \text{(Equation 3)},$$

Kannisto model is formalized in Equation 3. μ_x represents the force of mortality, c represents the mortality risk independent of age, a and b are model parameters and x is age.

Another important feature about TOPALS is that it can handle well when there are zero deaths or people counted in a specific year or age. In their paper, Gonzaga and Schmertmann (2016) assert that TOPALS can be used in two ways: to estimate complete schedule of log mortality rates in areas where the

coverage of vital registration is not a problem and to smooth death rates prior to correcting for undercount. In our work, since the first step was to use DDM estimates to correct for potential problems with registration system, we will use the TOPALS estimates in both ways. In the cases when we observed zero deaths or population, the model will enable us to have the whole age schedule estimates. At the same time, the TOPALS will give us a smoother mortality estimate in all cases, which is fundamental to calculate the modal age at death with more precision (HORIUCHI et al. 2013).

Schmertmann and Gonzaga (2018) proposed a Bayesian regression model that smooths age specific mortality rates and accounts for uncertainty about undercount of deaths level. In their model, mortality by age was modeled using TOPALS. Similarly, Borges (2018) proposed a Bayesian probabilistic approach to count consistently population, deaths, births and migrants. TOPALS application in his work aimed to smooth mortality information across the age range and to combine two independent mortality schedules (the observed data and the chosen standard). Dyrting (2017) adapted De Beers (2012) TOPALS with Eilers and Marx (1996)'s penalized B-Splines to estimate a complete set of migration probabilities. Jubithana (2018) applied TOPALS to analyze external causes mortality in Suriname between 2004 and 2012.

As with LQ, TOPALS was applied after correcting the number of deaths by undercount estimates and considering the exposure measure constructed with population data for every census years. TOPALS was also the method chosen to compare the differences in mortality observed between males and females and to describe the age at death dynamics registered at the Federal Units level.

3.3.3 Beers Osculatory Interpolation

Another smoothing method applied was Beers Osculatory Interpolation. The aim was to offer one more comparative perspective on how the chosen method influence the estimated indicators. Siegel and Swanson (2004) observe that along with other interpolation techniques, Beers can be employed from binned age groups that suffer from reporting errors. Dealing with age information in

groups help diminish digit preference. Same authors highlight that graduation procedures fit different curves to the original 5 year age group.

According to Siegel and Swanson (2004), most interpolation techniques were designed to guarantee that the sum of the interpolated single ages to be consistent with the grouped original information. Authors argue that Beers, Sprague and Karup-King present similar results and that the choice of the interpolation method dependent on the nature of the data. The US decennial life tables for 1989 to 1991 were estimated with Beers multipliers (SIEGEL & SWANSON, 2004), that was the reason to pick Beers as the model in this comparative exercise. Differently from LQ and TP, the Beers estimates were based on the population distributed in five year age groups - constructed from the single aged population - and the grouped number of death from the moving average centered on each census year. The age groups applied were 0,1-4,5-10,10-15…..110+. The idea was to have a cruder (less methodologically intensive) comparison base. After grouping the single aged data, Beers was applied, and then single aged mortality rates were estimated. From that rates, life tables were estimated by sex for each year.

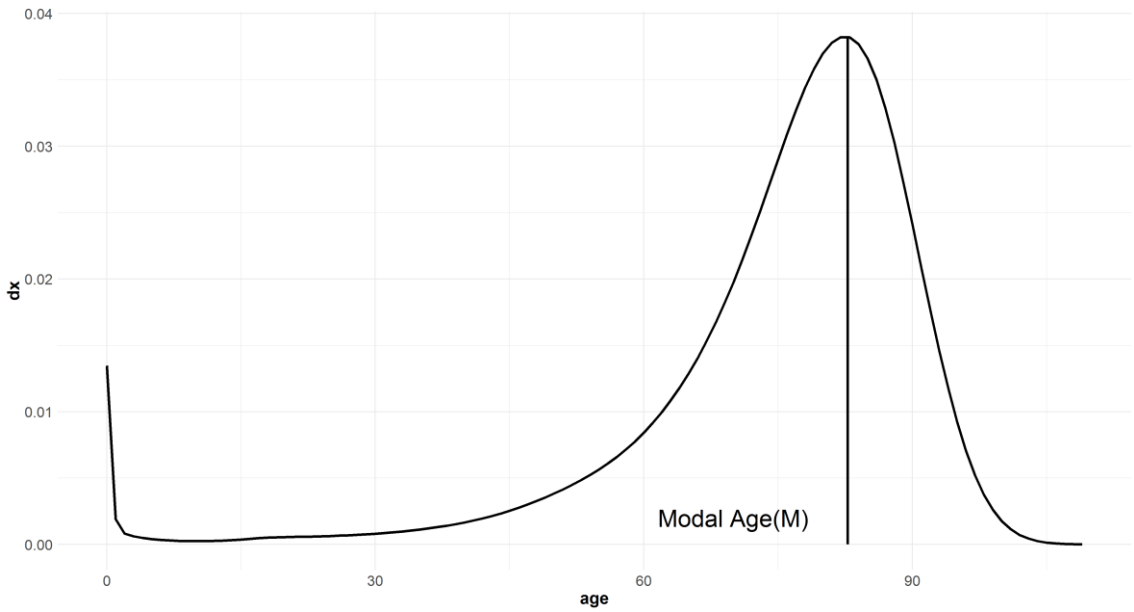
3.4 Modal Age of Death

From all mortality estimation (BEERS, TOPALS, LQ), we derived life-tables specific by sex and year. We also generated life tables without any smoothing technique. The idea is to compare how the chosen method (or the absence of one) influences the estimates. For Federal Units and gender comparison, only the measures based on TOPALS were estimated because, as will be presented in Chapter 4, it offered better results. From each year, sex and location life tables, the age at death distribution (dx) is the main function to estimate the modal age at death. According to Kannisto (2001), given the flatness in dx curve in highest ages, the mode in very sensitive to variations, this is the reason why the curve should be smoothed in cases where period age at death distribution are not unimodal and or smooth. The modal age at death (M) was calculated from dx function, according to Kannisto (2001) proposition, as Equation (4) presents:

$$\mathrm{M} = x + \frac{d_{(x)} - d_{(x-1)}}{\left[d_{(x)} - d_{(x-1)}\right] + \left[d_{(x)} - d_{(x+1)}\right]} \qquad \text{Equation (4)}$$

Where x is the age with the highest number of death in older ages, d(x-1), d(x), and d(x+1) are the number of deaths in x, and in the ages right above and below x from the age at death distribution function. The presented formula allows the mode to be estimated considering fraction of years and indeed giving more precision to the parameters (KANNISTO, 2001). We estimated M as suggested for Brazil (1980, 1991, 2000 and 2010) considering LQ, TP and Beers estimates, as well as based on life tables without any smoothing technique. Figure 3.1 illustrates the modal age at death in the age at death distribution.

Figure 3.1– Age at death distribution and the modal age at death



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

3.5 Median age at death

To analyze another mortality measure, also allowing comparison with the mode, we estimated the median age at death (MD). MD represents the age at which half of the population is still alive (lx=0.5). As life expectancy, and differently from the mode, the median age at death is sensitive to mortality changes in younger ages. The median to rise necessarily needs mortality to change in ages below the median (CANUDAS-ROMO, 2010). If mortality diminution were observed only in ages above the median, that indicator would stay the same. Given the expressive mortality variation observed in Brazil in the period considered, to see how these two measured varied will help understand more clearly the patterns of adult and old mortality change in Brazil.

3.6 Interquartile range (IQR)

IQR was calculated from the estimated life-tables. From the survival function (lx), with initial cohort size equal to 1, the age that indicates the first quartile (Q1) – when 75% of the cohort is alive (lx=0.75) - and the third quartile (Q3) - 25% of survivors form the cohort – were identified by linear interpolation. The difference between those ages (Q3 and Q1) represents the IQR.

$$IQR = Q3 - Q1 \qquad \text{Equation (5)}$$

Wilmoth and Horiuchi (1999) argue that the Interquartile range (IQR) has two appealing characteristics as a measure of the age at death variability. It has an intuitive meaning, indicates the size of the age range in which 50% of deaths occur, and it is very simple to be calculated. The fact that the IQR is measured in years is also an important characteristic (WILMOTH & HORIUCHI, 1999). Given that we are also interested in analyzing mortality change in a larger part of the age at death distribution, the two measures that comprise IQR are also measures of interest. Changes in the age where 75% and 25% of the hypothetical

population are still alive offer interesting insights in the patterns of mortality change.

Figure 3.2 – Number of survivors by age (lx) and the distance that indicate the IQR

Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

3.7 Standard deviation above the mode

Another variability measure considered was the standard deviation above the mode (SD+). As Kannisto (2001) argue the SD(M+) estimate is based on the assumption that the number of deaths is evenly distributed within each year of age. SD(M+) is calculated as the root-mean-square of the positive deviations from the mode (Kannisto, 2001). Cheung et al. (2009) present the formula to calculate SD(M+) in presented in Equation (6).

$$SD(M+) = \sqrt{\frac{\sum_{1=M}^{\omega}(X_i - M)^2}{n}} \qquad \text{Equation (6)},$$

Where Xi represents each remaining age above the mode until the last life table age (ω), M is the estimated modal age at death and n is the number of years considered.

The standard deviation above the mode is a variability measure that considers only ages above the mode. In that sense, it will help us understand age at death distribution focused on older ages, as it gives a measure life span variability in that specific part of the age distribution. Along with the IQR we will be able the have a broad picture of age at death variability in Brazil and its states, by sex, in the period between 1980 and 2010.

CHAPTER 4 - COMPARISON OF APPLIED METHODS

This Chapter discusses the observed changes in the selected indicator - Modal age at death (M), Median age at death (MD), Interquartile Range (IQR) and the Standard Deviation above the mode(SD(+)) – to analyze adult and old age mortality in Brazil, by sex, between 1980 and 2010. Besides debating the observed results with Brazilian mortality literature, another aim is to interpret how different smoothing methods affect the estimates. In that sense, three different methods were applied – TOPALS (TP), Loq-Quad (LQ), Beers Osculatory Interpolation (Beers). We also present estimates without smoothing to discuss the importance of a smoothed curve in the analysis. We also show TOPALS without correcting the number of deaths, trying to visualize differences related to the omission deaths based on DDM estimates. Section 4.1 presents the resulting differences from the applied methods, while Section 4.2 analyses how age at death distribution curves evolved in the period. Such analyses were based on TOPALS and LOG-QUAD estimates that presented insightful patterns of variation that helps understand mortality change in Brazil and differences between the methods.

4.1 Differences in applied methods by sex

4.1.1 Females

Table 4.1 shows the modal age at death (M), the median age at death (MD), the interquartile range (IQR) and the standard deviation above the mode (SD(M+)) for females, between 1980 and 2010, considering all applied methods. The modal age of death rose substantially along these decades. In each intercensal period considered (1980-1991; 1991-2000; 2000-2010), M rose, independently of the method applied. That means that mortality declined in ages above de mode (CANUDAS-ROMO, 2008; CANUDAS-ROMO, 2010). Except for TOPALS (with a 5.35 rise), the observed increase in the modal age at death was very similar considering the other estimates, varying between 3.75 and 3.98. In every year, Log-Quad modal age was lower. Beers, Unadjusted and TOPALS modal age estimates are, with the exception of 1980, very similar. This is an expected result

given that those estimates were made using, practically, the same set of data. It is interesting to note, however, that both Beers and Unadjusted estimates were always higher than TOPALS.

Table 4.1 - Mode, median, Interquartile range, Q1, Q3 and standard deviation above the mode, Brazil, females, 1980-2010

Year	Method	Q1	MD	Q3	IQR	SD(M+)	M
1980	UNADJUSTED	65.28	77.09	84.77	19.49	9.12	82.59
1991		67.67	78.59	86.01	18.34	8.31	82.78
2000		69.18	80.19	87.94	18.76	8.60	83.90
2010		71.43	81.86	89.28	17.85	7.80	86.34
1980	BEERS	65.26	77.24	85.13	19.86	7.84	82.75
1991		67.73	78.65	86.24	18.51	7.48	83.65
2000		69.20	80.23	88.02	18.82	8.09	84.51
2010		71.45	81.86	89.38	17.93	7.07	86.66
1980	LOG-QUAD	61.44	73.96	81.81	20.37	7.46	78.98
1991		66.50	76.75	83.89	17.39	7.20	80.84
2000		68.43	78.03	84.92	16.49	7.00	81.89
2010		70.09	79.23	85.92	15.83	6.80	82.96
1980	TOPALS (DDM)	65.34	77.19	84.80	19.46	9.93	80.43
1991		67.70	78.47	86.00	18.30	9.23	80.85
2000		69.25	80.08	87.84	18.60	8.62	83.81
2010		71.52	81.82	89.24	17.72	7.94	85.74

Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

Another clear sign of mortality reduction for females is the rise in the median age at death (MD) (Table 4.1). Independently of the method applied, MD rose in every decade considered. This result indicates that mortality also diminished in other parts of the age distribution, along with old age mortality change expressed by modal age rise. Between 1980 and 2010, MD increased expressively in all estimates. Differently from the observed in the modal age at death, MD rose the most considering Log-Quad. On the other hand, as observed in the modal age at death, Log-Quad presented the lowest MD in every year considered. Between Unadjusted estimates, Beers and TOPALS the differences in MD were very low, almost negligible, indicating that MD is less sensitive to the smoothness of the

curve. Kannisto (2001) and Horiuchi et al. (2013) have emphasized the importance of a smoothed age at death distribution in M estimates. An unsmoothed age at death distribution can have more than one mode. In such cases, a noisy point of the distribution, not necessarily the most frequent value, may represent the mode.

Canudas-Romo (2010) highlights that the median age at death is dependent on the number of survivors to the age when half of them are still alive. In that sense, the median increases when mortality reduction is registered in ages below that point (CANUDAS-ROMO, 2010). Same author argues that, unlike life expectancy, both the median age at death and the modal age at death have the capacity to place mortality reduction along the age range. TOPALS (DDM) estimates indicates that MD rose 4.6 years between 1980 and 2010. Between 1980 and 1991, same indicator rose from 77.2 to 78.5 meaning that mortality declined in ages below 77.2, covering a broad age range. Research indicates that mortality reduction in Brazil between 1980 and 2010 was very rapid and was observed throughout the age distribution (FRANÇA et al., 2017b; SIMÕES, 2002; BORGES, 2017; IBGE, 2010; CAMPOS et al.,2015; LEITE et al., 2015).

There are characteristics of mortality change in the period that, along with differences between methods, may help explain differences in MD comparing Log-Quad and TOPALS. As already mentioned, the first method uses 5q0 and 45q15 as inputs. In Log-Quad application, MD rose the most between 1980 and 1991, probably influenced by infant mortality reduction registered in the period (BORGES, 2017; IBGE, 2010; FRANÇA & LANSKY, 2005; FRANÇA et al.,2017a). Child mortality is the most important parameter in Log-Quad, the 1980 curve was built upon a much higher 5q0 in comparison to 1991. In that sense, mortality reduction between 1980 and 1991 implied by LQ estimates, given the importance of 5q0 in the LQ estimates, is extended to a broader age range. The wide age range in LQ reduction between 1980 and 1991 influenced the median age at death rise substantially. Considering TOPALS estimates, MD rose the most between 2000 and 2010. Probably, as TOPALS considers the entire age range and MD changes captured mortality diminution from several age groups, including older ages (BORGES, 2017; CORREA & MIRANDA-RIBEIRO, 2016; FRANÇA et al., 2017b; LEITE et al.,2015; BRANT et al., 2017).

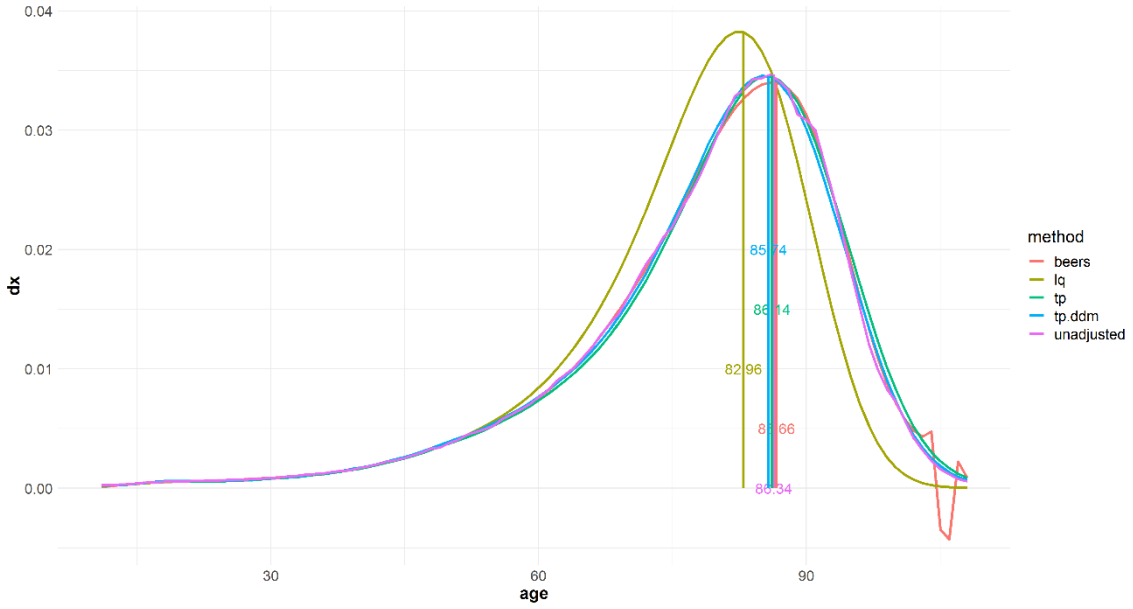
When we look at the variability in the age at death – measured by the interquartile age range (IQR) and by the standard deviation above the mode (SD(M+)), there is no sign that the compression of mortality has stopped for Brazilian females. Other authors observed the same path in female age at death variability (GONZAGA et al., 2018; GONZAGA & SOUZA, 2016; ALVAREZ et al., 2020). The standard deviation above the mode diminished sequentially along the years of interest, in all estimates. It is also interesting to note that, like it was observed in MD and M, the SD(M+) was lower than the other estimates in the Log-Quad application. Contrarily from what was observed in the MD, differences between Unadjusted, Beers and TOPALS were higher, indicating that SD(M+) is more sensitive to the chosen smoothing method.

Table 4.1 also presents the interquartile range (IQR) estimated for females, the other measure of variability considered. The estimated IQR in all applications also indicates that the compression of mortality is still in process, meaning that the age at death distribution is becoming concentrated in a narrower age range (CANUDAS-ROMO, 2008; WILMOTH & HORIUCHI, 1999; FRIES, 1980; GONZAGA et al., 2018; GONZAGA & SOUZA, 2016). As observed in the modal age at death analysis, TOPALS, Beers and Unadjusted estimated were higher than Log-Quad. Interestingly, in 1980 LQ IQR was the highest, in the subsequent years it was always the lowest. As in MD, the high infant mortality in 1980 level may have overestimated mortality in other ages, in comparison with the other methods and consequently inflated IQR in that year.

It is important to highlight that, different from the other decades, female IQR rose between 1991 and 2000. That result expresses an important feature of the indicator. As mentioned in the method section, IQR represents the difference between the age that indicates the point where 25% (Q3) of the hypothetical cohort is still alive and the age where 75% (Q1) of the cohort remains in the population. The rise observed between 1991 and 2000 indicates a steeper change in Q3 than in Q1. Between 1991 and 2000, Q1 rose less than Q3 ($\Delta IQR=\Delta Q3-\Delta Q1$), meaning that in period there was important mortality decline in ages after Q1 (first quartile) and below Q3 (third quartile) (WILMOTH & HORIUCHI, 1999).

When we look at the age at death distribution curves, another set of relevant insights can be drawn. Figures 4.1-4.4 present female age at death distribution from 1980 to 2010 and will help comprehend better the differences in the measures related to the chosen model. Age at death distributions show ages above 10, as the focus of the present dissertation is set in adult and old age mortality. As in Table 4.1, Figure 4.1 presents estimates considering TOPALS, Beers osculatory interpolation, Unadjusted, and Log-quad. To demonstrate the impact of the DDM correction in the age at death distribution the Figures also show TOPALS estimates without correcting the number of deaths.

Figure 4.1 - Age at death distribution, females, Brazil, 2010, TOPALS, TOPALS (DDM), Log-Quadratic, Unadjusted, Beers.



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

Figure 4.1 indicates that in 2010, visually, the differences between Beers, Unadjusted data, TOPALS and TOPALS without correcting from undercounting were modest. The estimated M varied very little. For example, the difference between M in TOPALS with and without DDM was only 0.40, the estimated coverage in the 2000-2010 period was 0.953 (QUEIROZ et al., 2016). Figure 4.1 also shows that the correction of the number of deaths by the same factor implies in a shift in the age at death distribution to the left.

It is also interesting to note that the curve estimated using the Beers osculatory interpolation (Figure 4.1) resulted in an age at death distribution flatter in the point that indicate the modal age at death. It is also worth noticing that Beers curve is, in comparison with the others, a little inclined to the right. Consequently, Beers M was the highest. Mortality rises with age, especially at higher ages, probably the interpolation had a little difficult capturing this feature of human mortality at the end of age at death distribution. Although, it is important to highlight that the method presented a good fit, except for the end of the distribution where the curve got disturbed.

One of the most important results present in Figure 4.1 is the difference in the age at death distributions between Log-Quad estimates and the other fitted curves. From age 11 to around age 55, all curves trace nearly the same path. From that point on, the LQ age at death distribution rises at a steeper pace and registers a higher number of deaths in the age interval that contains the modal age at death (82). LQ also presents a faster downward trajectory that help understanding the lower IQR. In consequence of the steeper slope in LQ curve, Q1 and Q3 are lower compared to the other methods and so it is the IQR for both males and females. One potential reason to the lower LQ modal age is that in the countries that generated model coefficients old age mortality was higher than observed in Brazil considering same levels of 5q0 and 45q15.

TOPALS resulted in a close fit to original data as the comparison with the Unadjusted curve fit indicates. In all estimates (both IQR and M), TOPALS outcomes were higher than LQ. One other potential explanation to the higher modal age in TOPALS (and the others) could the related to age misreporting. LQ coefficients were based on better quality data, and probably with less problems of age reporting, especially in older ages. Wilmoth et al. (2012) model was elaborated using life tables from countries known to have advanced mortality systems and, consequently, less prone to suffer from reporting problems.

Age misreporting in older ages usually underestimates mortality, independently of the direction of the bias (PRESTON et al., 1999). Preston and his colleagues identified that when ages were overstated, starting at age 55, mortality was lower than it would be if ages were correctly stated. Interestingly, the same age where

LQ start to diverge from the other curves in Figure 4.1. In Brazil, there are clear signs of the presence of age misreporting (TURRA, 2012; HORTA, 2012; PALLONI et al., 2016; AGOSTINHO, 2009; PALLONI et al., 2016). Below age 75, Brazil had proportionally higher mortality in comparison with countries known to have high good quality vital statistics, while, in older ages, mortality was lower (TURRA, 2012). Accordingly, Palloni and Pinto-Aguirre (2011) found that, in most countries in Latin American Countries, age is systematically overstated in both censuses and in death statistics. In 2005, Palloni et al. (2016) estimated that Brazilian life expectancy above age 60, if age were correctly stated, would be 1.2 year lower.

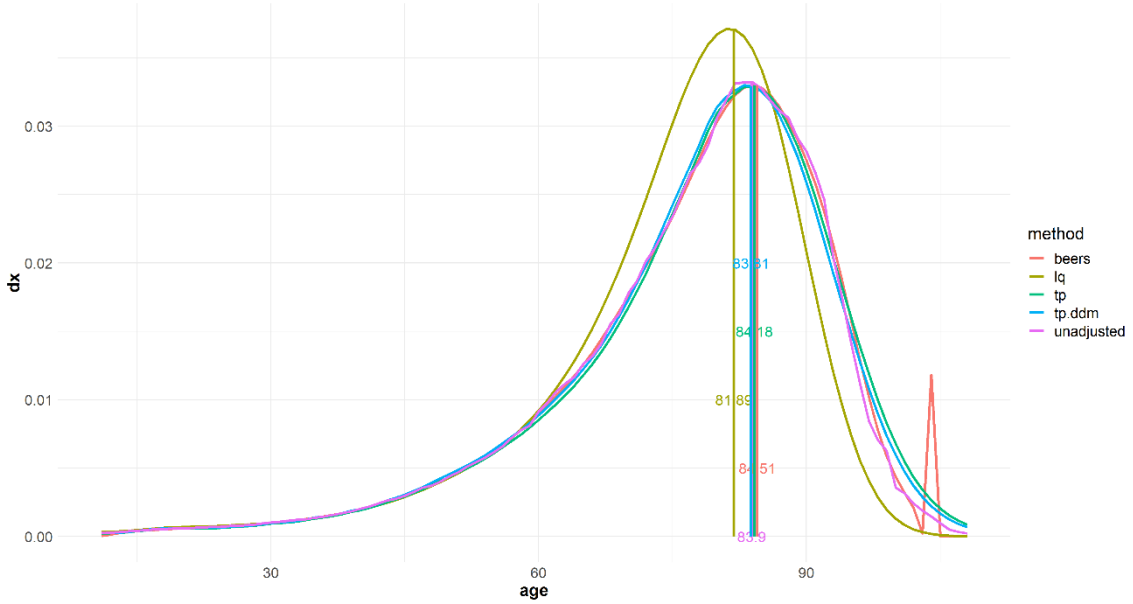
As age misreporting underestimates mortality at older ages (PRESTON et al., 1999; 1996; PALLONI et al., 2016), and since there are several signs that age is misreported in Brazil, a higher number if survivor would reach higher ages (given the underestimated mortality) and, consequently, the modal age of death would be higher than the expected if age was properly stated. LQ only uses information from the original dataset until age 60 (from 45q15), so it is, potentially, less influenced by age misreporting. Costa Rica example may help understand the potential effects of age exaggeration. Glei et al. (2019) observe that in 1950, when registry systems were less reliable, Costa Rican unadjusted life expectancy at age 80 was considerably higher than the observed in all HMD countries. Authors argue that it is probably related to age exaggeration and/or undercount of deaths. In 2013, Costa Rica men tied with the second highest e_{80} in the HMD database (GLEI et al., 2019). They also show that old age mortality in Costa Rica was considerably lower to the observed in Sweden and stood below the adjusted LAMBdA estimates.

Lower modal age observed in LQ estimates could also be related to the fact that the number of deaths in all ages was corrected by a unique factor. Death registry system coverage varies with age and quality is worst for infants and for the elderly (QUEIROZ & SAWYER, 2012; HILL et al., 2009). In that sense, it would be necessary to correct the number of death after a certain age by a larger factor. At the other hand, old age information also is more likely to present sampling problems (MURRAY et al., 2010). Since the highest considered age from the data

is 60, LQ is potentially less influenced by an eventual omission of death related to the correction factor used.

Figure 4.2 presents the same set of curves described in Figure 4.1 for the year 2000. The results are similar to the previous Figure. TOPALS (with and without DDM), Beers and the curve estimated with Unadjusted data presented similar fit. It is hard to differentiate them on the plot (Figure 4.2). The fact that Beers estimate is the highest gives an indicator that the curve is slightly leaned to the right, but it is hard to visualize in Figure 4.2. Another feature of Beers curve is that, as in Figure 4.1, the final years of the distribution are instable. The differences between Log-Quad and TOPALS, as with the other curves, are very similar to what was shown in Figure 4.1.

Figure 4.2 – Age at death distribution, females, Brazil, 2000, TOPALS, TOPALS (DDM), Log-Quadratic, Unadjusted, Beers

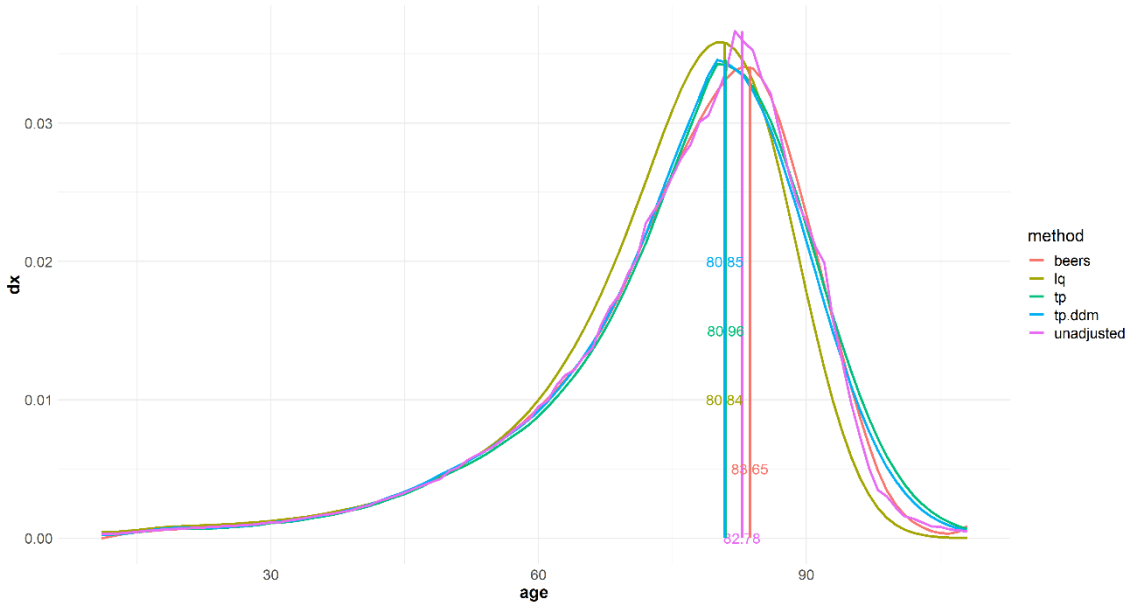


Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

Compared to Figure 4.1 and Figure 4.2, 1991 age at death distribution curves for females are more divergent (Figure 4.3). The Unadjusted curve is more volatile compared to the others and it implied in a considerably higher modal age at

death, compared to TOPALS. Figure 4.3 offers a great opportunity to visualize more clearly the skewness to the right in the Beers curve. It is interesting to note that the TOPALS fit, in this case, presented a slight skewness to the left that contrasts with the right inclination observed in the Beers curve. Another interesting feature in Figure 4.3 is that, as in the two previous Figures, starting at age 55, LQ curve rises at a steeper pace than the others do. However, differently from the previous years the difference between the estimated modal age at death using TOPALS and LQ was very little.

Figure 4.3 – Age at death distribution, females, Brazil, 1991, TOPALS, TOPALS (DDM), Log-Quadratic, Unadjusted, Beers

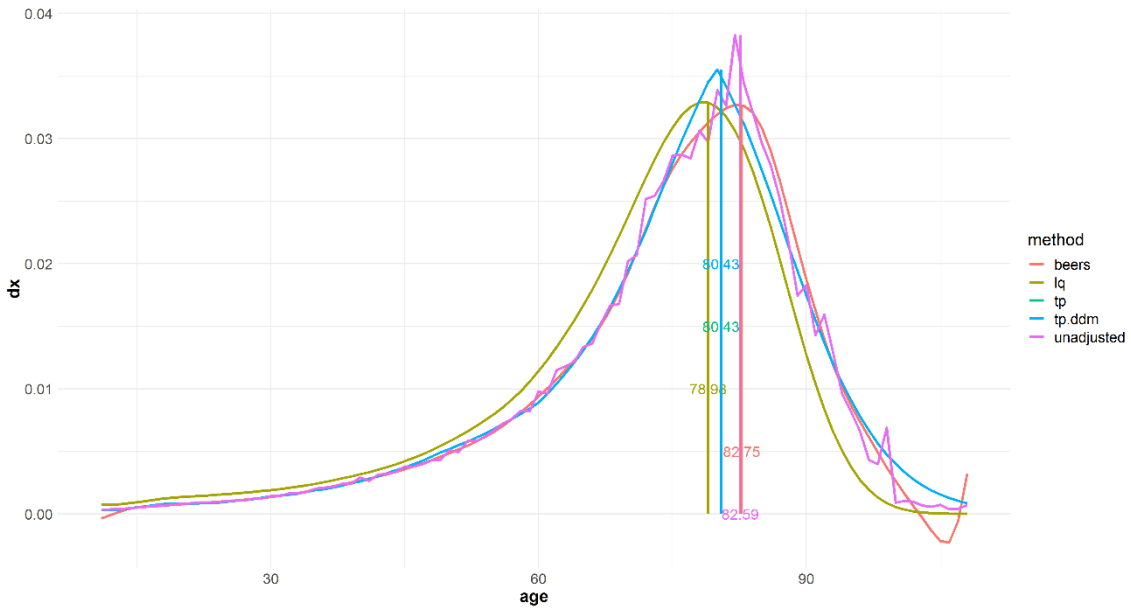


Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

In Figure 4.4, the adjusted curves presented more divergent patterns, as in Figure 4.3. Again, the Unadjusted curve was much more instable than the other years, indicating that both death and population databases improved in the 1980-2010 period (LIMA & QUEIROZ, 2014; HORTA, 2012; AGOSTINHO, 2009). TOPALS fits at the top of the age at death distribution was sharper that the previous years.

Probably, data was more problematic, especially at older ages. Agostinho (2009) found a very strong single age concentration index age 80, for females in 1980, that might relate to the observed result. Another potential explanation to the unexpected shape in TOPALS age death distribution for females in 1980 might be related to the position of the knots in the model. One of the knots was positioned at the exact age 80, it might be the case that for women in 1980 and 1991 (as the slightly skewness to the left may suggest) that the estimation would present a better fit if that knot was positioned elsewhere. Although, as mentioned in Chapter 3, we opt to use the same positions for the knots to compare the same analyzed population in different points in time. In 2000 and 2010, the fit was very good.

Figure 4.4 – Age at death distribution, females, Brazil, 1980, TOPALS, TOPALS (DDM), Log-Quadratic, Unadjusted, Beers



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

The tendency in the Beers osculatory interpolation to present a right leaned curve is also very clear in Figure 4.4. Same Figure shows a fuzzier result in the end of the Beers age at death distribution. Beers osculatory interpolation can lead to

negative results, as we can see in the right end of the estimated age at death distribution.

Log-Quad age at death distribution (Figure 4.4), differently from the other years analyzed, presented a higher number of deaths in all ages. Probably, that result is a consequence of the way Log-Quad was developed. The main parameter in LQ estimates is 5q0, while 45q15 is used to adjust mortality at adult ages (WILMOTH et al., 2012). In 1980, Brazil still had high infant mortality levels (FRANÇA & LANSKY, 2005) that may have led the LQ to estimate higher mortality levels in other ages. The higher level of LQ curve in 1980, naturally, directly relates to the higher IQR registered in 1980, as well as to the shaper decline in MD as time passed by. As in the other studied years, the divergent path between the LQ and other curves is also observable. In this case, however, it started earlier, around age 50.

4.1.2 Males

Table 4.2 brings the same set of indicators presented in Table 4.1 for male population. These indicators were estimated using the same methods, for the same years. Modal age at death was higher in TOPALS, Beers and the Unadjusted dataset, same as observed with female analysis. It is interesting to note, however, that the Unadjusted estimates presented the highest M in all years. Female results indicated Beers estimates as the highest, given a small skewness to the right. Unadjusted modal age estimates in Table 4.2 also present an unexpected result, between 1980 and 2010, the modal age at death diminished modestly. Probably, that results from the fact that the curve was not smoothed, especially in 1980 (KANNISTO, 2001; HORIUCHI et al., 2013).

Except from the Unadjusted curve, modal age at rose between 1980 and 2010. Compared to females (Table 4.1), it was more modest rise. Which implies a smaller diminution in old age mortality (CANUDAS-ROMO, 2008). Beers and Log-Quad M rise in the period was close to three years, while same rise in TOPALS was close to two years. The differences between male and female estimates will be further discussed in Chapter 5. In Table 4.2, it is also worth

noticing that, between 1980 and 1991, M diminished very modestly, from 78.97 to 78.87.

The median age at death analysis offers another interesting picture of the pattern of mortality change in Brazilian male population. In all estimates, the median age at death rose between 1980 and 2010. Such rise, however, was lower than observed in the female population. As observed in female MD analysis, LQ estimates showed the most expressive rise in the median age at death and the differences observed between the other methods was modest. MD variation indicates, however, an important feature of male mortality change in Brazil. Except from LQ estimates, the observed rise in MD was shy in the 1980-1991 period.

Between 1980 and 1991, despite important diminution in infant mortality rates, young adult male mortality rose (BORGES 2017; SIMÕES, 2002; CAMARANO et al., 1997; CERQUEIRA, 2014; FRANÇA et al., 2017). This picture is better illustrated comparing males and female MD change in that same decade. TOPALS female MD rose 1.28 against a 0.14 rise in male population. It is important to remember that in that same period infant and child mortality declined expressively, naturally, for both sexes (MERRICK, 1985; SIMÕES, 2002; BORGES, 2017; FRANÇA & LANSKY, 2005; FRANÇA et al., 2017).

The compression of the mortality process is still in course for the Brazilian male population, independently of the method chosen. Results that goes in the same direction Gonzaga et al. (2018) observed. In all cases, IQR diminished in every intercensal period. The 1980-1991 period, however, presented only a modest fall in the interquartile range, compared to women. As already mentioned, young adult mortality rise may have decelerated male IQR decline. Gonzaga et al., 2018 hypothesized that infant mortality reduction kept the declining tendency in male age at death variability in Brazil, despite the external cause mortality increase. Alvarez et al. (2020) argue that external causes contribute to the gap in lifespan variability between LAC and developed countries.

The other age at death variability measure (SD(+)) applied indicated a more erratic dynamic, except for LQ that showed a continuous diminution in SD (M+).

Considering the whole period (1980-2010), SD (M+) diminished modestly, indicating that mortality was compressing (WILMOTH & HORIUCHI, 1999; FRIES, 1980; CANUDAS-ROMO, 2008). Between 1991 and 2000, however, standard deviation above the mode rose from 8.76 to 9.19, implying an increase in the age at death variability at older ages. In the last analyzed decade (2000-2010), that the compressing dynamic was registered again, as SD (M+) fell from 9.24 to 8.99. The unexpected rise in age at death variability between 1991 and 2000 should be interpreted carefully, as data is more problematic at the end of the distribution. The same caution should be adopted when analyzing the rise in the modal age between 1980 and 1991, especially considering the initial years of the series.

Table 4.2 - Mode, median, Interquartile range, Q1, Q3 and standard deviation above the mode, Brazil, males, 1980-2010

year	method	Q1	MD	Q3	IQR	SD(M+)	M
1980	UNADJUSTED	55.94	71.45	81.05	25.11	8.4	82.34
1991		56.91	71.47	81.27	24.36	7.56	82.3
2000		59.30	73.16	82.97	23.67	8.54	82.24
2010		61.81	75.22	84.48	22.67	8.67	82.22
1980	BEERS	55.82	71.45	81.20	25.37	8.87	77.97
1991		56.98	71.55	81.32	24.34	8.56	78.69
2000		59.36	73.19	83.07	23.72	9.13	80.24
2010		61.88	75.3	84.55	22.68	9.05	80.74
1980	LOG-QUAD	53.88	68.32	77.67	23.79	8.59	75.04
1991		59.11	70.94	79.47	20.36	8.33	76.33
2000		61.17	72.24	80.45	19.28	8.31	77.13
2010		62.85	73.41	81.40	18.55	7.93	77.97
1980	TOPALS (DDM)	56.08	71.29	80.84	24.75	9.19	78.97
1991		56.86	71.43	81.05	24.19	8.76	78.87
2000		59.16	73.24	82.94	23.77	9.24	79.81
2010		61.86	75.24	84.48	22.62	8.99	80.93

Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

Changes in Q1 and Q3 are also illustrative to understand the patterns of male mortality change reflected in the IQR. Between 1980 and 1991, TOPALS male Q1 rose only 0.78, a modest rise compared to the 2.38 observed with the female

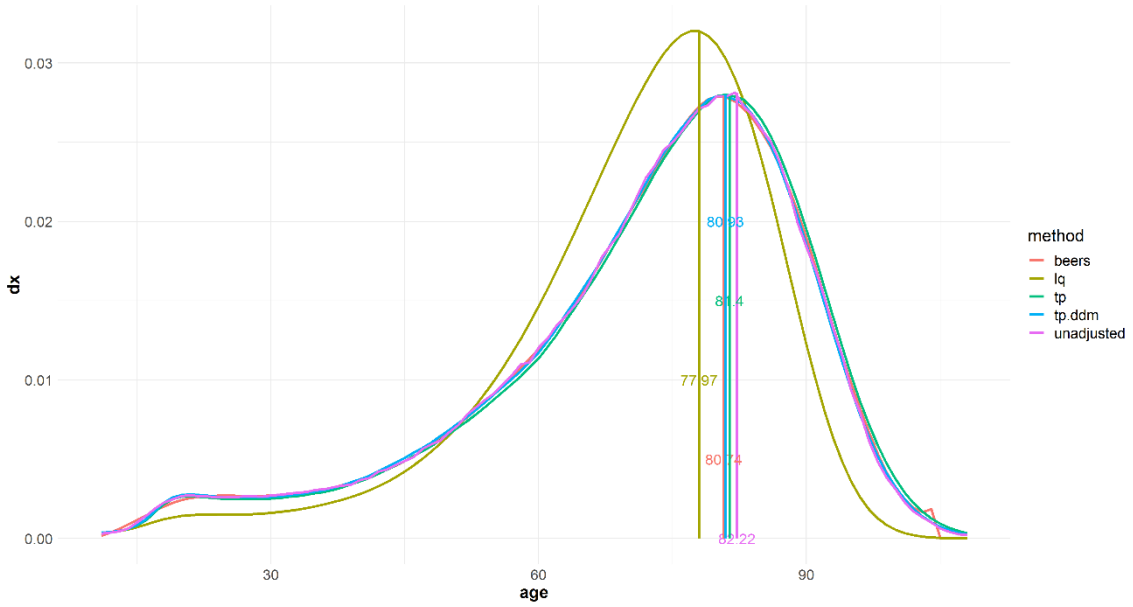
data. Probably, the young adult mortality rise attenuated the impact of infant mortality reduction on the age at which 75% of the initial cohort was still alive.

A close look at Q1 estimates also offer interesting insights about the differences resulting from the method of choice. In 1991, Q1 was lower in TOPALS, Beers and in the Unadjusted analysis. It is interesting to observe that the deceleration of change in Q1, IQR and MD in these same estimates. As it will get clearer in the plot analysis, LQ had more difficult capturing the changes in young adult mortality. In 1980, LQ Q1 was the lowest. Between 1980 and 1991, same indicator rose substantially (from 53.88 to 59.11), while in the applications a much more modest change was observed (0.97 on average). Another sign that the differences that generated that same diverging pattern is concentrated in young ages is that - in all years – Q3 was always higher in TOPALS and others compared to LQ. The slope of the age at death distribution for males is substantially steeper in LQ, as it was also shown for females in the previous section.

Figure 4.5 (2010) presents the age at death distribution curves for males. Log-Quad and the other methods presented very different results at young adult ages. The divergent path in the dx curves started around age 14, when Unadjusted, TOPALS and beers curves rises sharply and reach the local mode (CANUDAS-ROMO, 2008; KANNISTO, 2001) around age 20. The evolution of the male age at death distribution will be more properly discussed in the next section (4.2). At this moment, the main interest rest in the method comparison.

Approximately at age (14), LQ curve also presents an upwards slope, but meek compared to all other estimates. Another interesting feature in that part of the mortality curve is that Beers rises at a slower pace and peaks in a higher age compared to TP and Unadjusted curves. As already highlighted, in the end of the age at death distribution, probably, the number of deaths rises faster with age in young adults age groups than the interpolation process could capture. After the local mode, TP and Unadjusted curves decline slightly and take another upward trend around age 30. After rising at age 14 both Beers and LQ maintain a flat pace (at different levels) before rising again approximately at age 30.

Figure 4.5 – Age at death distribution, Males, Brazil, 2010, TOPALS, TOPALS (DDM), Log-Quadratic, Unadjusted, Beers.



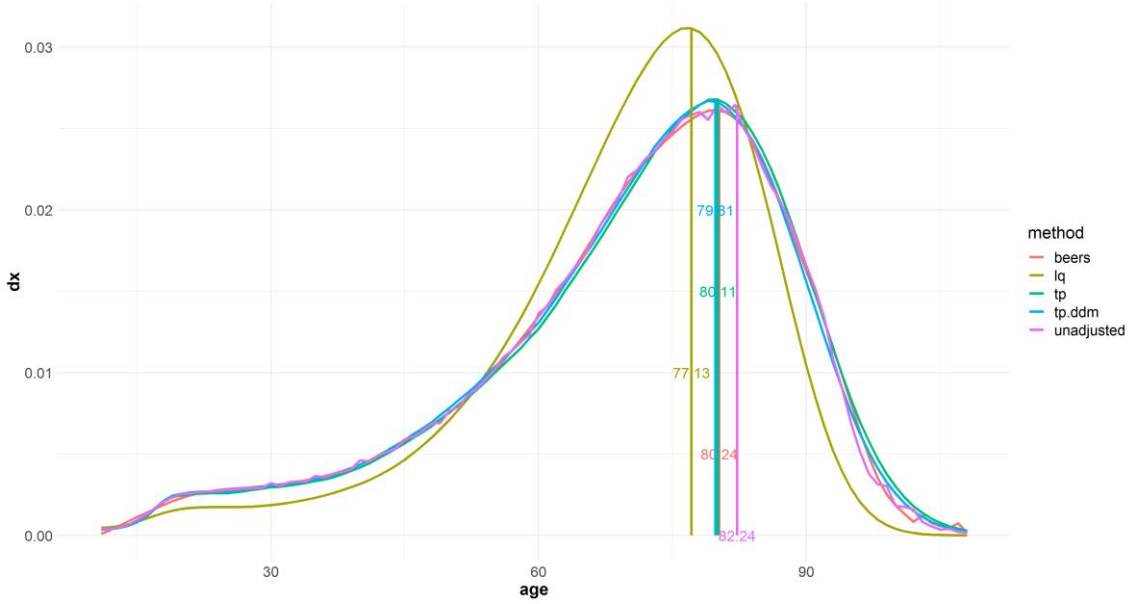
Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

The LQ estimate was not able to capture the young adult mortality hump for males. Young-adult age at death distribution for the LQ is the lowest. Probably, since LQ coefficients are based on HMD data (few non-European countries), and given its input parameters (5q0 and 45q15) (WILMOTH et al., 2012), the model could not capture the excess mortality observed in young adult ages. Among other models, Sharrow et al. (2014) observed that Log-Quad could not fit the HIV mortality hump in Lesotho, LQ produced a high flat pattern in age specific mortality that match an overall level of mortality. It seems that we are handling with same kind of problem with the young-adult male mortality LQ estimates.

In most parts of the age range, it is hard to distinguish between Unadjusted, Beers and both TOPALS curves. Except from the Unadjusted estimates, their modal age at death are very similar. Unadjusted curve (in purple) presented a tiny spike at the top of the age at death distribution that resulted in a modal age more than one year higher than the other methods that have shown a similar fit. Yet again,

it demonstrates the importance of a smoothed age at death distribution on M calculation (KANNISTO, 2001; HORIUCHI et al., 2013).

Figure 4.6 – Age at death distribution, Males, Brazil, 2000, TOPALS, TOPALS (DDM), Log-Quadratic, Unadjusted, Beers.



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

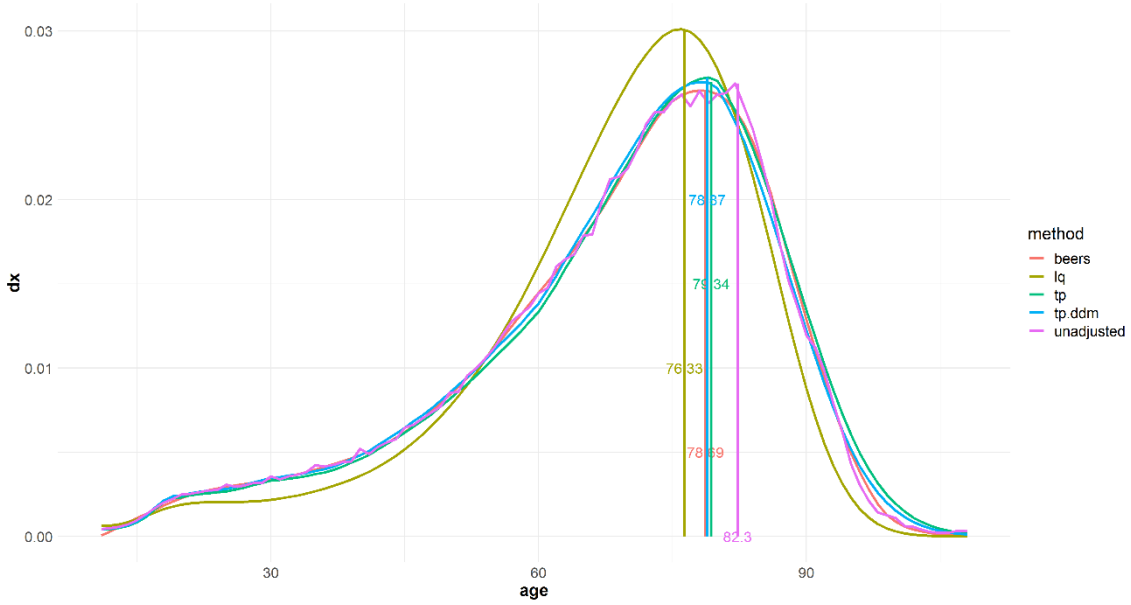
Compared to Figure 4.5, the estimated age at death curves for males in 2000 present a similar situation (Figure 4.6). At young adult ages, the differences related to the methods of choice are the same as was described in Figure 4.5. The LQ could not capture the excessive mortality in young adult ages and presented a flatter pattern. Around age 50, LQ rises fasters than the other curves and reaches the mode at younger ages. Beers has also presented a flatter pattern, but at a higher level than the LQ curve. The local mode in TOPALS estimates was, again, close age 20. At the end of dx curve, Unadjusted, in 2000 and as in 2010 (Figure 4.7), was sharp, leading to a higher modal age at death.

Compared to 2010, the unsmoothed curve in 2000 was even more unstable. Another feature in Figure 4.6, which was not so clear in 2010 estimates (Figure 4.7), is that Beers curve (like registered in the female analysis) was leaned to the

right at the end of the age at death distribution. Consequently, Beers modal age at death was higher than TOPALS. The differences between TOPALS curves estimated with and without DDM correction, as in the other examples, is a shift in the distribution to the right and, therefore, a higher (slightly) modal age.

At first sight, the already discussed differences in the slope between LQ and the other method is salient in Figure 4.7 (1991). Interestingly, Unadjusted modal age of death is the highest again with a very clear spiky at top of the age at death distribution. Unadjusted estimates evolved between 1991 and 2010, indicating that the quality of both age at death and age information in censuses improved with time. Beers curve at the top of the distribution presented a lower concentration of deaths in the modal age and a flatter pattern at the top of the curve. In this case, it isn´t clear if Beers curve is inclined to the right.

Figure 4.7 – Age at death distribution, Males, Brazil, 1991, TOPALS, TOPALS (DDM), Log-Quadratic, Unadjusted, Beers.



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

Just as in Figure 4.5 and 4.6, LQ could not capture the excessive mortality observed in male young adult ages. However, there are differences in the curves compared to the other years, especially considering TOPALS. As in the other years, TOPALS rises faster than the other curves at age 14 but, differently, the distribution does not decline after reaching the local model. Consequently, the

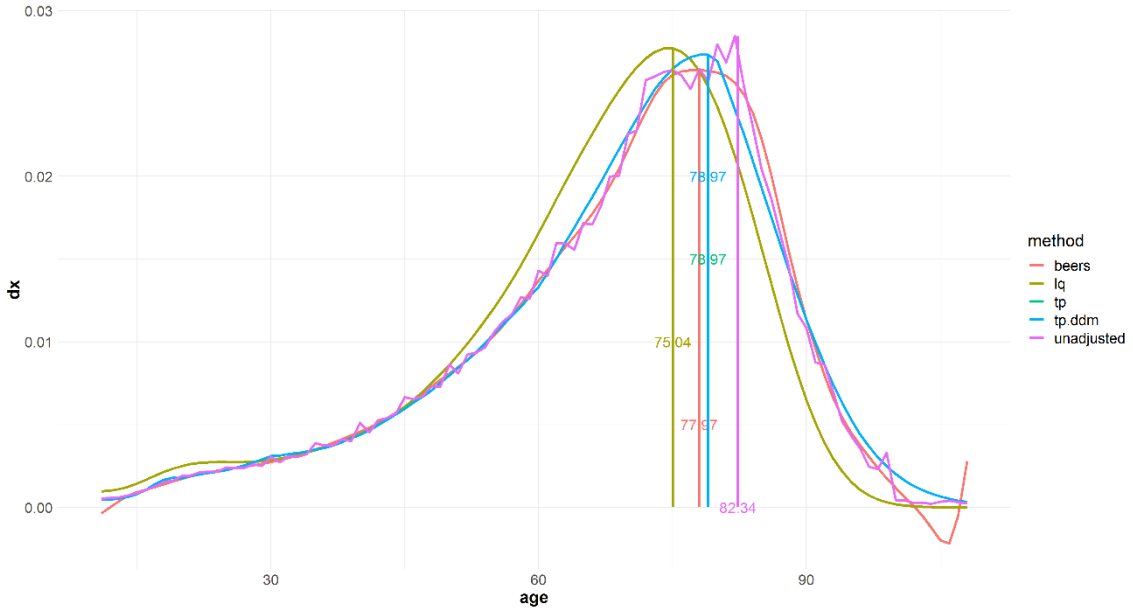
TOPALS curves trace a path similar to the observed using Beers estimates. The decomposition of life expectancy gain by age and causes of death held by Borges (2017) indicate that some causes of deaths (external causes and others) contributed negative to life expectancy gains in Brazil, between 1980 and 1991, in a wide age range, more expressively, between 15 and 45.

Another interesting result present in Figure 4.7 is the moderate break in the TOPALS age at death distribution at age 60. That characteristic was also present in the other years but is more easily seen in Figure 4.7. Such pattern could be explained by age misreporting related to digit preference (HORTA, 2012). As the population exposure measure attenuated digit preference problems in the censuses, it might represent an indication that Mortality Information System also suffers with age reporting problems (AGOSTINHO, 2009; PALLONI et al., 2014). The fact that the break is less perceptible in the older curves could be seen as an indication that both censuses and mortality information systems quality improved in the period (HORTA, 2012; LIMA & QUEIROZ, 2014; QUEIROZ et al., 2017).

Another potential explanation to the observed break, as well already discussed at exact age 80 for females, is that the knot positioned at age 60 in the model estimates might not be the best fit to the model considering 1991 data. As de Beers (2012) discuss, it is possible to determine the optimal position of the knots that would represent a different set of knot for each year, location, and sex.

As observed with females (Figure 4.4), 1980 LQ curve level was higher than observed in the other estimates from the beginning of the curve (Figure 4.8). It was discussed in the previous section that 5q0 is the main parameter estimating mortality schedule in LQ (WILMOTH et al., 2012) and, as child mortality levels were considerably higher in 1980, the estimated curve registered a greater number of deaths in that ages, compared to the other estimates. In contrast, it is interesting noticing that young adult mortality rise between 1980 and 1991 was not captured in Log-Quad estimates for 1991. The steeper rise in age at death distribution in LQ estimates, as in all other years and both sexes, is also present in Figure 4.8.

Figure 4.8 – Age at death distribution, males, Brazil, 1980, TOPALS, TOPALS (DDM), Log-Quadratic, Unadjusted, Beers.



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

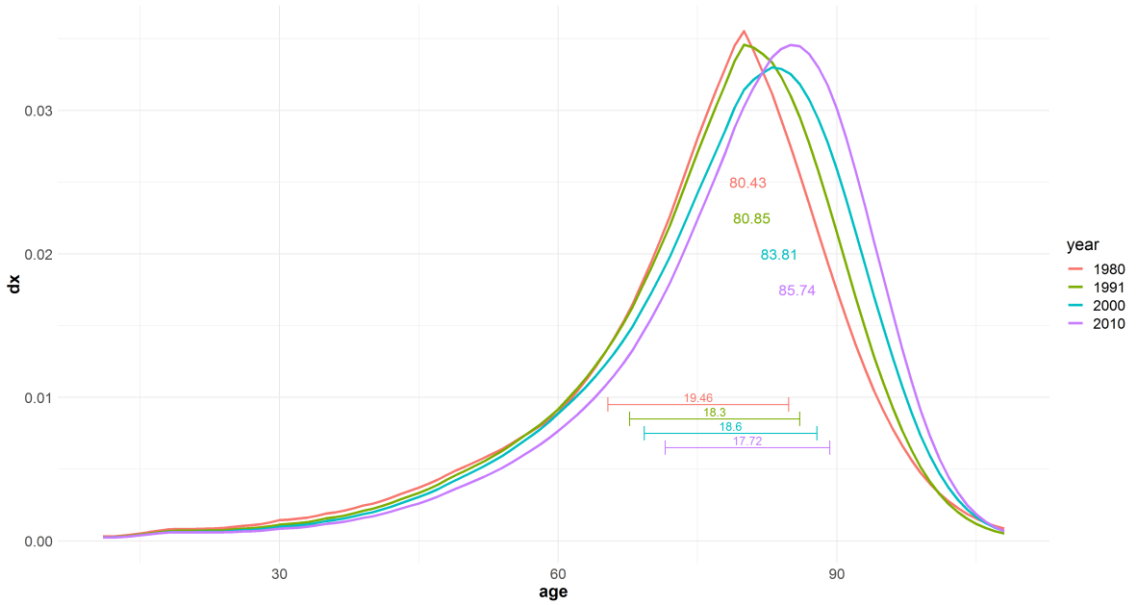
Another already seem feature of the 1980 age at death distribution is the highest modal age estimated with Unadjusted data and caused by a spiky top of the dx curve. The flatter top of age in the Beers curve is also worthy noticing, as well as the problems at the end of the curve in the same method application.

4.2 Age at death distribution between 1980 and 2010

4.2.1 Females (TOPALS)

The clear shift to the right in Figure 4.9 helps visualize the rise in modal age at death for females, between 1980 and 2010. In the period, female M rose 5.31 years, indicating an average rise of 1.77 year each decade. Canudas-Romo (2008) observed a less expressive rise (1.4) path for females in developed countries after 1940. As observed with males, the 1980-1991 period represented the most modest rise in M. Borges (2017) found that the relative contribution from the 80+ group was meek in that same decade.

Figure 4.9 - Age at death distribution, Brazil, Males, 1980-2010, TOPALS



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

From the start (11) until age 25, it is hard to differentiate the curves. From age 25 on, however, a more pronounced decline in perceivable, especially comparing the 1980 and the 2010 curves. From 1990 to 2015, World Health Organization (WHO, 2015) estimated a 3.5% average annual decline in Maternal Mortality Ratio (MMR). Besides the registered decline, Brazil is still considered a country with high MMR (LAURENTTI et al., 2004). It is clear in Figure 4.9 the rise in Q1 between 1980 and 1991, as already mentioned, that period registered important mortality decline at younger ages (SIMÕES, 2002; BORGES, 2017; FRANÇA & LANSKY, 2005; FRANÇA et al., 2017a).

It is interesting to observe the change in the curves between 1991 and 2000. There was a minor perceptive diminution in the number of deaths by age from age 38 to around age 60 and a more expressive movement downward from age 60 on. Brant et al. (2017), highlights that female CVD mortality declined almost 40% between 1990 and 2015. Borges (2017) identified an ascendant life

expectancy gains between 1991 and 2000, the open ended age interval (80+) was the second most import relative contribution. After infants, the 50+ group presented the highest life expectancy gains between 1991 and 2000 (SIMÕES, 2002). That observed pattern may explain why Q1 rose less that Q3 (1991-2000) and, consequently, IQR rose in that decade. According to Figure 4.9, M rose the most between 1991 and 2000. As already mentioned, it is important to note that 1991 curve leaned to the left at top the age at death distribution that might have affected M estimate.

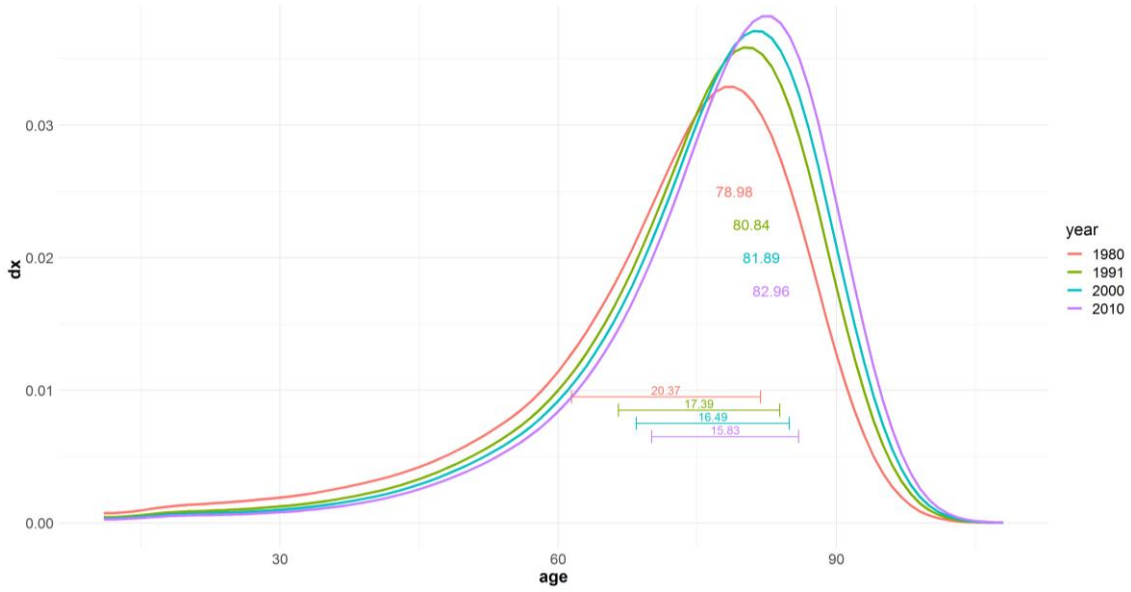
The 2000-2010 curves indicates another expressive rise in M. In that period, the 80+ age group contributed more than the first age group (0-1) to life expectancy rise (BORGES, 2017). As in the previous decade, the contribution to life expectancy gains rose with age (after age 5), more sensitively after age 60. Contribution to life expectancy from ages 60 to 79 was higher that the gains registered between 0 and five years old (CORRÊA & MIRANDA-RIBEIRO, 2017). In 2010, Kanso (2014), using unsmoothed data, estimated a slightly higher modal age at death (86.5) in comparison with TOPALS, but very close to the Unadjusted modal age (86.34), presented in Table 4.1. In the same year, she observed a closer IQR of 17.9, close to TOPALS estimate (17.72). Same author estimated a considerably higher IQR in 1980 (21.0) that may be related to differences in the adjustment of the data.

4.2.2 Females (Log-Quad)

The female pattern of variation in the age at death distribution curves (LQ) showed a regular rise in modal age of death that may not resemble the mortality change in old ages (BORGES, 2017; SIMÕES, 2002; CORRÊA & MIRANDA-RIBEIRO, 2017; FRANÇA et al, 2017b; MERRICK, 1985; FRANÇA & LANSKY,2005). Differently from the Figure 4.9 (TOPALS), and from what other studies suggest (BORGES, 2017; SIMÕES, 2002; BRANT et al., 2017; FRANÇA et al., 2017b), modal age at death rose the most between 1980 and 1991. Since the parameters required in LQ application are 5q0 and 45q15, and the mode is not contained in that interval, it seems that it is harder to the model to capture nuances out of its inputs range. At the other hand, given the quality of data used

to estimate model coefficients, the slope of the curves at older ages are more likely to be closer 'real' pace of rise of the age at death distribution without age reporting issues. In that same period, for example, Figure 4.10 seems to suggest an expressive mortality diminution in practically every age of the distribution, considerably different from the other estimates.

Figure 4.10 - Age at death distribution, Brazil, Males, 1980-2010, Log-Quad

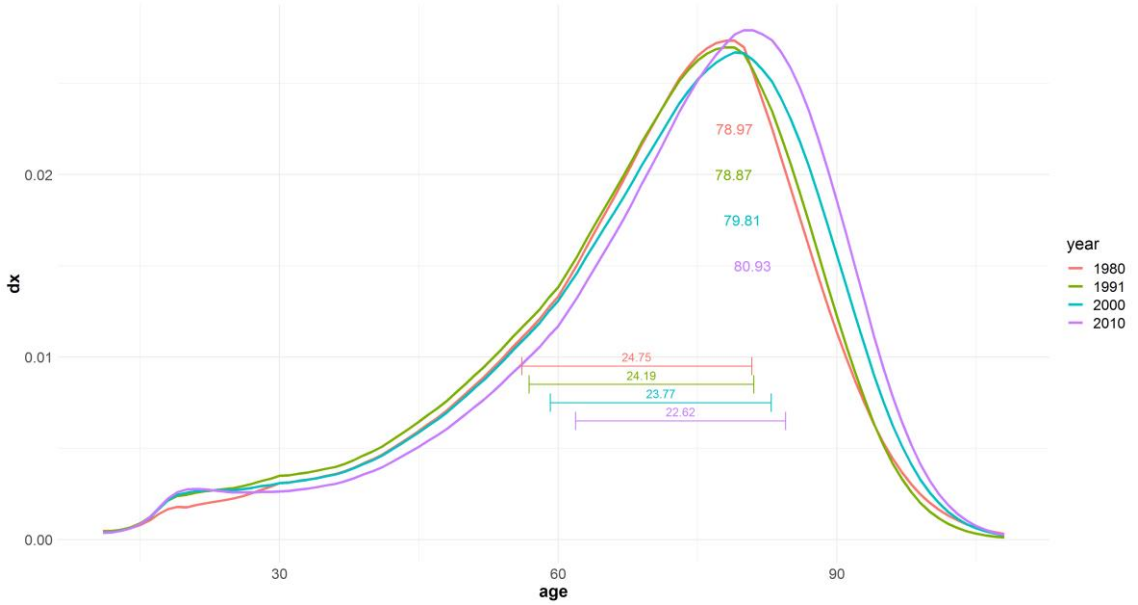


Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

4.2.3 Males (TOPALS)

The three decades between 1980 and 2010 were marked by important mortality changes (PALLONI & PINTO-AGUIRRE, 2011; SIMÕES, 2002; BORGES; 2017; FRANÇA et al. 2017b). Male mortality, especially considering young adult ages, presents particular characteristics of change. As already mentioned, modal age at death rose between 1980 and 2010 (Table 4.2), indicating that mortality diminished in ages above the mode (CANUDAS-ROMO, 2008; CANUDAS-ROMO, 2010). Figure 4.11 gives a clear view of how the age at death distribution curves shifted to the right.

Figure 4.11 - Age at death distribution, Brazil, Males, 1980-2010, TOPALS



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

In TOPALS estimates, male modal age at death rose from 78.98 to 80.93 (Figure 4.11). Same Figure shows, however, that between 1980 and 1991 M did not rise. Actually, it diminished modestly. That diminution is, probably, consequence of fit of the model between those two years or related to the quality of the data, since there is no evidence of a mortality rise in older ages. At the other hand, it expresses the fact that old age mortality practically did not change in the period. Borges (2017) observes a modest contribution from males above 80 to life expectancy gains between 1980 and 1991, and a negative contribution from that age group from diseases from the digestive system and other causes. Another indicator of modest mortality reduction at old ages is given by the shy change in Q3, age that indicate when 25% of the initial cohort is still alive (Table 4.2). In Figure 4.11, Q3 is represented at the final point of the tiny bars, Q1 the beginning, and IQR represents their difference.

From 1991 to 2000, modal age rose by approximately one year and, between 2000 and 2010, more than one year. The decomposition held by Borges (2017)

indicated that the open age group (80+) contributed more substantially to life expectancy gains in the periods 1991-2000 and 2000-2010. In Figure 4.11, is also interesting to note that the number of deaths in the age that contains the mode rose between 2000 and 2010, indicating death more concentrate around the mode. In 2010, 2.79 percent of deaths in the hypothetical cohort happened at age 80

From 1980 to 1991, a distinguished rise in the number of death is perceivable in Figure 4.11. From age 14 to age 65, the green curve (1991) moved upwards. Such rise was more expressive between 14 and 30 years old. As already mentioned, in that period a rise in external cause mortality was registered, affecting mainly male in adult ages (BORGES, 2017; SIMÕES, 2002; CAMARANO et al., 1997; DUARTE et al.,2002).

In Figure 4.11, a small peak in the 1980 curve at age 30 is probably related to a knot positioned at age 30. It is likely that both in 1980 and in 1991 another position to that knot would be better suited in the model estimation. In Figure 4.11, it is clear how the young adult mortality rise influenced Q1. Between 1980 and 1991, the rise in Q1 was the smallest registered, besides relevant infant mortality diminution (FRANÇA & LANSKY, 2005; SIMÕES, 2002; BORGES, 2017; FRANÇA et al.,2017).

Once the local mode that captures male excess mortality in young ages rose, from 1980 to 1991 (Figure 4.9), it stabilized and did not diminish any more, besides the diminution in the proportional distribution of deaths in other ages. Probably, the main reason is Brazil's internal mortality disparities. In most states in the Northeast and North the male homicide rates rose between 2000 and 2010 (WAISELFISZ, 2013; IPEA, 2017; CERQUEIRA, 2014). At the same time, in the Southeast region (especially in São Paulo and Rio de Janeiro), there was a reduction in the concentration of death of young-adults in the same period (WAISELFISZ, 2013). These internal tendencies will be broadly discussed in in Chapter 6.

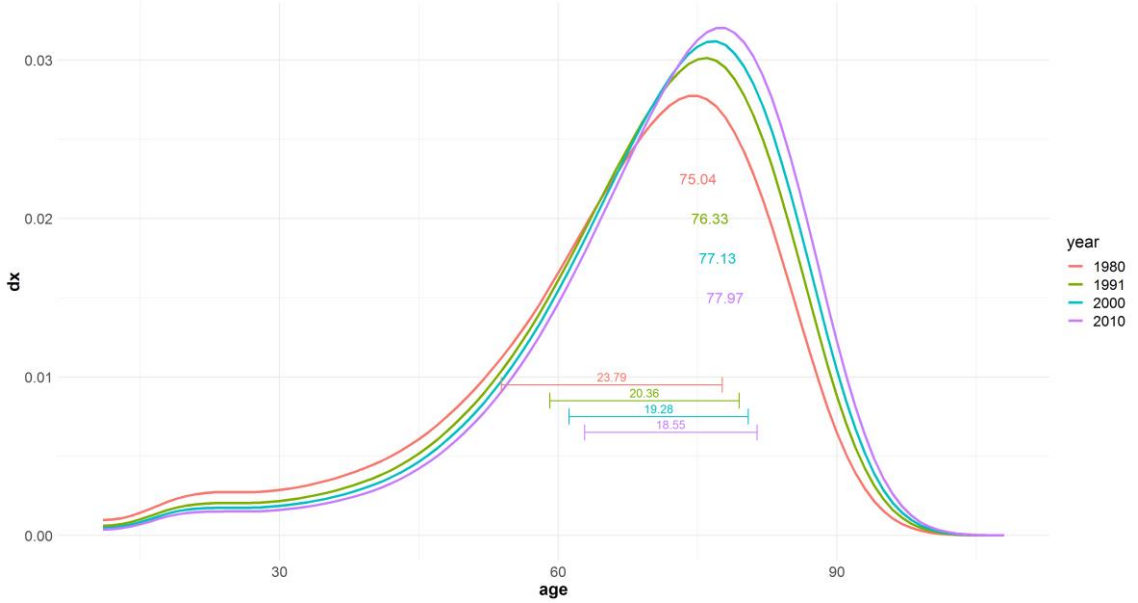
From 1991 to 2010, the local mode did not change, although, after that point, adult mortality diminished consistently, as Figure 4.10 shows. Borges (2017)

argue that male mortality from chronical (age 35 to 59) diseases contributed to life expectancy gains in that same period. Brant et al. (2017) estimated that between 1990 and 2015 CVD mortality diminished approximately 40%. As observed in old age mortality, the mortality decline for adult males was more expressive between 2000 and 2010. In the 2000-2010 period, the contribution to life expectancy rise made by the 60-75 age groups was proportionally higher than the rise brought by infant mortality reduction (CORRÊA-MIRANDA-RIBEIRO, 2016). Kanso (2014) observed in 2010 a modal age of death equal to 81.7, slightly higher than ours (80.93). For the same year, her IQR estimate (22.6) was the same as our using TOPALS (22.62). However, in 1980, hers IQR estimate was about two years higher than ours (26.7). Differences in IQR in that year could be related to different adjustments in the dataset such as the exposure measure used in the population, the DDM correction factor applied, or the related to the smoothing method. Relative to the modal age, it is more likely that the observed difference is a consequence of using a unsmoothed curve, her estimate was closer to the Unadjusted M (82.2).

4.2.4 Males (Log-Quad)

In Figure 4.12, age at death distribution curves from Log-Quad estimates are presented for the same period. It is clear that LQ the curves are well behaved and there are no signs of reporting issues. However, it is also clear that the method has problems capturing the nuances of the pattern of mortality change in Brazil. For example in young adult ages, the rise in external cause mortality was not properly captured. In that sense, Q1 rose the most between 1980 and 1991, a very different picture to what was observed and discussed with TOPALS. At the other hand, as LQ is less sensitive to age misreporting, the age at death distribution at older ages could be good benchmark in comparison with other estimates. Possibly, the old age mortality measures are less affected by biases related to poor age reporting.

Figure 4.12 - Age at death distribution, Brazil, Males, 1980-2010, Log-Quad



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM),

Figures 4.1-4.8 showed that the smoothing model was more important in the first years of the series. For both males and females, Beer, TOPALS and the unsmoothed data presented similar results in 2010 and 2000 (mainly). However, even in 2010, as male analyses showed, the modal age of death was still sensitive to the model selected. Especially in the first two years, Beers dx curves presented a small skewness to the right that influenced modal age estimation. It seems that TOPALS estimates, for females in the beginning of the series, could be improved by the selection of another set of ages where the knots are positioned.

Log-Quad, in all years, differed the most from other models. It was not able to capture young adult rise observed between 1980 and 1991. As well, the model could not capture the shape of the distribution of death in those same ages. Another problem in LQ estimates was that it is heavily influenced by 5q0, as the change in the 1980 and 1991 curves showed. As infant mortality diminished expressively in the period (FRANÇA & LANSKY, 2005; FRANÇA et al., 2017a),

the model estimated an expressive mortality decline in a broader age range, in comparison with the other estimates. That change was very clear in Figures 4.10, as well as in the MD and IQR estimates for the same period.

At the other hand, LQ is the less influenced by age misreporting or by the differences in the death registry system coverage by age. In all estimates, modal age of death was lower in LQ. It is possible, since there are signs of age misreporting and indication that death coverage is lower at higher ages (QUEIROZ & SAYWER, 2012; TURRA, 2012; AGOSTINHO, 2009), that the true modal age of death could be closer to what LQ showed, or somewhere in between (PRESTON et al., 1999; PRESTON et al., 1996; ELO & PRESTON, 1994; PALLONI et al., 2016).

The selected indicators were able to demonstrate important features of adult and old age mortality in Brazil the analyzed period, in accordance with other mortality studies. The rise in the modal age of death indicates mortality reduction in older ages. The observed path in that indicator reassemble changes discussed by other authors. It was clear that mortality fell more expressively for females in older ages an that the decline started earlier (BORGES, 2017; SIMOES, 2002; BRANT et al., 2017; CORRÊA, MIRANDA-RIBEIRO, 2017; FRANÇA et al., 2017b; LEITE et al., 2015).

The intrinsic features of mortality change were also captured by the different measures of variability. IQR was sensitive to mortality changes in different parts if the distribution. The rise in young adult mortality between 1980 and 1991 (WAISENFIZ, 2013; SIMÕES, 2002; CAMARANO et al.,1997) was reflected in both IQR and MD. Standard deviation above the mode and IQR gave clear indication that 1980-2010 registered compression of mortality (WILMOTH & HORIUCHI, 1999; GONZAZA et al. 2018). Especially for males, in LAC and in Eastern Europe external cause mortality influenced age at death variability (ALVAREZ et al.,2020; GONZAGA et al., 2018; ABURTO & van Haalte, 2018).

The age at death distribution, especially TOPALS, was undoubtedly a very good tool to capture mortality changes in the period. The curves reassembled the rise and stagnation in the young adult male mortality. It also showed the diminution of

adult and elderly mortality, more importantly between 2000 and 2010, also observed in the other researches (BORGES, 2017; SIMOES, 2002; BRANT et al., 2017; CORRÊA, MIRANDA-RIBEIRO, 2017; FRANÇA et al., 2017b). That is the reason why in the next Chapters it will be the model of choice in sex and regional comparison.

Differences between male and female mortality in the 1980-2010 period will be discussed in the following Chapter. The focus is placed on sex differentials and on distinguish features of male and female mortality evolution. Besides the already discussed mortality measures, we will present age specific mortality rates and survival curves to broaden the set of analytical tools.

CHAPTER 5 - SEX DIFFERENCES

Life expectancy at birth difference between males and females in Brazil was 7.2 years in 2016 (IBGE, 2017). In 1940, the estimated gap was 4.4 (SIMÕES, 2002). In the course of last decades in Brazil, sex mortality dynamics presented convergence and divergence paths (SIMÕES, 2002; BORGES, 2017). From 1940 to 2000, life expectancy gap rose to 7.78 and diminished afterwards (SIMÕES, 2002; IBGE, 2017). At least since 1980, male mortality has always been higher in every single age and the excessive mortality is observed in all Brazilian regions (LEITE et al., 2015).

Differences in external cause mortality explain an important part of the mortality differences between males and females (CAMPOS et al., 2015; MALTA et al., 2017). Although, another causes of death also play important roles. For example, males have higher mortality by ischemic heart disease, related to alcohol consumption, stroke, and neglected tropical diseases (LEITE et al., 2015; FRANÇA et al., 2017; BRANT et al., 2017; MARTINS-MELO et al., 2018,CAMPOS et al., 2015).

Male excessive mortality has been registered in both developed and developing countries (CULLEN et al., 2015; SCHUNEMANN et al., 2017; LUY, 2003; PRESTON & WANG, 2006; GRUSHKA, 2014; MARTÍN et al., 2010; CLARK & PERK, 2012). Explanations are usually divided into cultural (behavioral) and non-cultural aspects (biological) (CULLEN et al., 2015; SCHUNEMANN et al., 2017; LUY, 2003). Schunemann et al. (2017) estimated that health preferences and health investments explained about 70% of gender gap mortality. Several other authors argue that the behavioral component is responsible for the largest share of the differences (CULLEN et al., 2015; LUY, 2003; PRESTON & WANG, 2006; LUY & GAST, 2014).

The present Chapter discusses sex differentials in mortality observed in Brazil during the 1980 - 2010 period. TOPALS was the chosen method to describe the observed differences as its results offered the best fit to the data and captured more precisely changes and intrinsic features of the mortality decline in Brazil (Chapter 4). Sex differences and patterns of changes will be discussed

considering age at death distribution curves (dx), the number of survivors by age (lx), and age specific mortality rates (mx). The selected set of curves, along with what has been discussed in the literature, offers relevant insights on how that process evolved in Brazil. There are important sex differentials in mortality in Brazil and elsewhere. We understand that the applied analytical tools will help understand characteristics and the specificities of sex differentials in the studied period.

Between 1980 and 2010, modal age of death (M) gap between males and females more than doubled. In 1980, M for males and females was, respectively, 78.97 and 80.43 (a 1.48 gap). By 2010, that difference was 4.81. Mortality gains in old ages were more expressive for females and started earlier (BORGES, 2017). As Canudas-Romo (2010) argues, an increase in modal age of death is related to mortality decline in ages above the mode. Relative to age at death variability measures, women were also in a more privileged position, in all years, considering interquartile range (IQR). In 1980 and 1991, male presented lower SD (M+) than females, in the last two analyzed years, however, that situation was the opposite, which is the expected result given sex mortality differentials. Probably, higher standard deviation above the mode for females in the first year of the series may be related to the fit of the model in for females in those years, as discussed in the previous chapter.

IQR gap rose between 1980 and 1991 and diminished afterwards, however it was always higher for males. It is a reflection of the male excessive mortality all along the age distribution and by several causes of death (FRANÇA et al., 2017; MARTINS-MELO et al., 2018;LEITE et al., 2015; CAMPOS et al., 2015). Differences between the two variability measures (IQR and SD (M+)) reflect the fact that both of them look at different points of the survival function. IQR considers the first and the third quartile and, therefore, is also influenced by mortality changes at younger ages. By its turn, SD(M+) is only concerned with what happens in ages after the mode. Older ages are more prone to present data problems (HORTA, 2012; QUEIROZ & SAWEYR, 2012; AGOSTINHO, 2009).

The dynamic in the median age of death gap (MD) was similar to the observed with IQR. During the first decade (1980-1991), MD gap rose from 5.9 to 7.04 and

reduced afterwards to reach 6.58 in 2010. It is interesting to note that, besides the registered decline, in 2010, the sex difference in the age at which 50% of the hypothetical cohort have died (MD) was still higher than it was in 1980.

Between 1980 and 2010, there were moments of convergence and moments of divergence between the sexes in the indicators considered, at the same time it is important to keep in mind that the analyzed mortality measures for males and females took the same path. In most years, when female M rose, male modal age at death rose as well. The same dynamic was observed in the median age at death. Noymer and Van (2014) analyzed year-by-year life expectancy in 40 countries from the HMD database and observed that male and female life expectancy co-move (move in the same direction) as the norm. They found that most countries, even considering a more divergent time interval, presented significant co-movement. Even when the differences in life expectancy rose (divergence), co-movement was frequently observed (NOYMER & VAN, 2014). Our analysis provide only a few point in time but also indicate co-movement. Noymer and Van (2014) argue that overall mortality environment influences male and female mortality in the same direction.

From 1980 to 1991 (Figure 5.1A and Figure 5.1B), gap between age specific mortality rates at young adult ages rose substantially. That period, as already mentioned, registered expressive male mortality rise in ages between 15 and 30 (BORGES, 2017; SIMÕES, 2002; CAMARANO et al., 1997). Path change in male mortality is also reflected in both IQR and MD. França et al. (2017b) argue that in all Brazilian states violent death rates are higher than expected in comparison with countries with similar socioeconomic levels. Traffic accidents (TA) also play an important role in the excessive male mortality. DALY rates from TA was four times higher for males in comparison with females in 2008 (LEITE et al., 2018). After the rise observed between 1980 and 1991, age specific death rates differential did not diminish in young adult ages (Figure 5.1). Malta et al. (2017), in that sense, observes that external cause mortality between 1990 and 2015 was practically stable, mainly in consequence of different dynamics between states and regions. Internal differences will be discussed in Chapter 6.

For females, at the other hand, 15-30 age group contributed modestly to life expectancy gains between 1980 and 1991 (SIMÕES, 2002; BORGES, 2017; CAMARANO et al., 1997). Despite measurement difficulties, maternal mortality declined in the past decades in Brazil and might have helped increasing young adults gender gap (VICTORA et al., 2011; SILVA et al., 2016). It is important to highlight, however, that maternal mortality in Brazil is excessively high in comparison with developed countries (VICTORA et al., 2011).

Figure 5.1 shows a slope change in 20-30 age interval after 1980 (Panel A). Passed age 23, there is an mx diminution before it take another rising path after age 30, more clearly in 2010 (Panel D). It is interesting to note that there was consistency in the age that registered the highest age specific mortality gap. From 1980 to 2000, the difference between female and male ASMR reached the highest value at age 23, and age 22 in 2010.

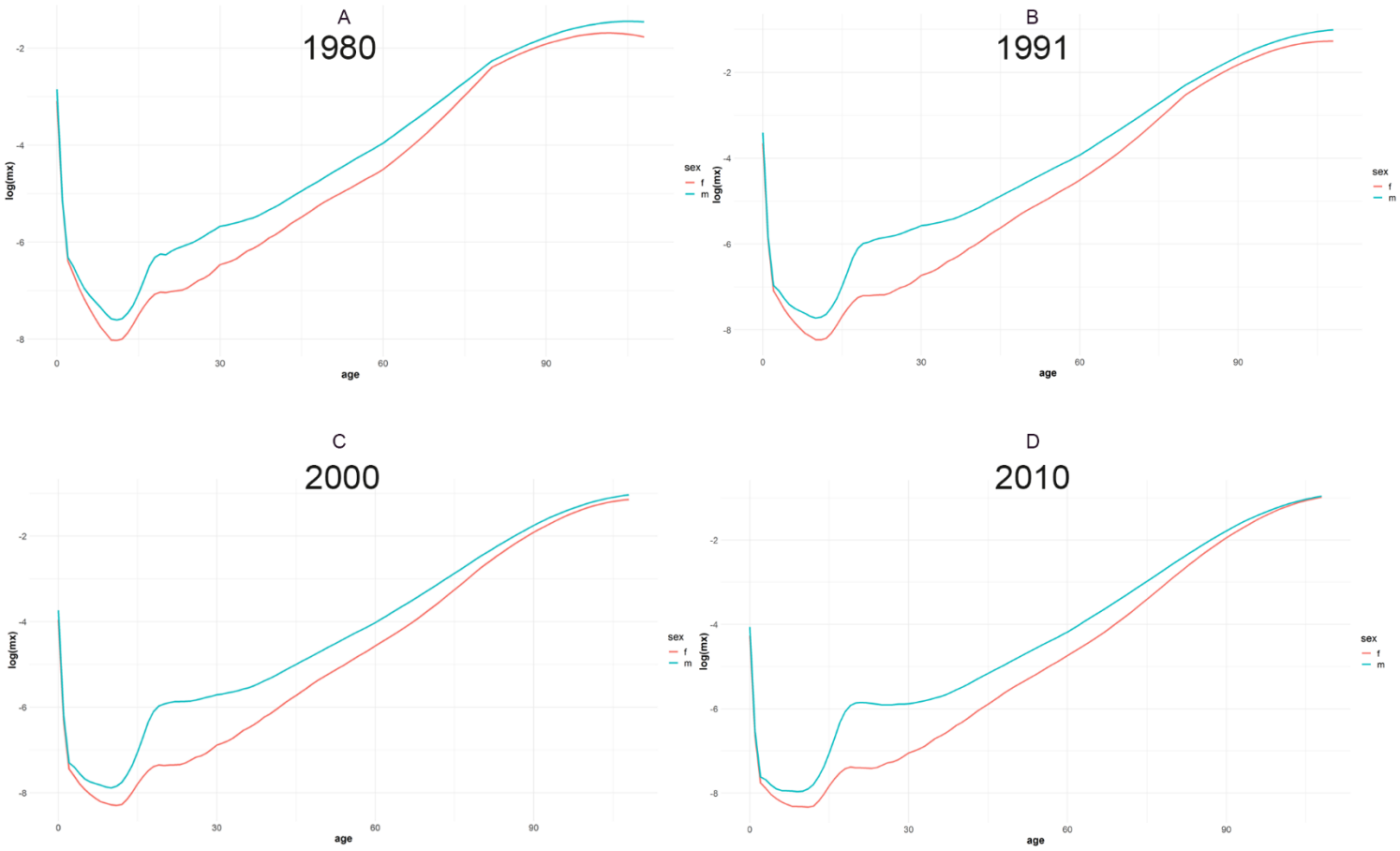
Especially at older ages, there are problems in the 1980 (Figure 5.1A) estimates that may also explain the unusual shape of female age at death distribution in that same year (Figure 4.4). For both males and females, after age 80, there is strong downwards change in the shape of the ASMR curves. It is likely that the observed mortality after that age reflects both census and death registry system quality problems (HORTA, 2012; LIMA & QUEIROZ, 2014; AGOSTINHO, 2009).

The fit of the model improved in the course of the years, especially at older ages, indicating that data quality also got better. Another apparent problem in female age specific mortality rates in 1980 (Figure 5.1A) is that from age 60 to age 80 the curve rises at an unexpected steeper pace, that could be related to age reporting problems in both (deaths and population) databases. Agostinho (2009) found high single age concentration index at exact age 80 for females in 1980. Another potential explanation is that the knots positioned at those ages in TOPALS estimation did not offer the best fit considering the 1980 data. In both ages (60 and 80) a knot was positioned. In 1980, more clearly, the fit would probably be better if the knots were positioned in another set of ages.

Figure 5.1 indicates that men present higher mortality rates all along the age distribution. After young adult ages, from age 35 to 59, female life expectancy

gains were more constant over time (BORGES, 2017). Above 50, for example, Simões (2002) underscores that excessive male mortality rose in the 1970-2000 period. However, in a lesser extent if compared to young adult ages. According to Martins-Melo et al. (2018), gender differences in mortality from neglected tropical diseases (NTD) were higher after age 50.

Figure 5.1 - Age specific mortality rates, Male X Female, 1980-2010, Brazil, TOPALS



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

Borges (2017) argues that cardiovascular disease (CVD) is also important in understand gender mortality gap in Brazil. Same author points out that women presented higher life expectancy gains in the 60+ age group. Between 2000 and 2010, mortality decline from diseases related to the circulatory system

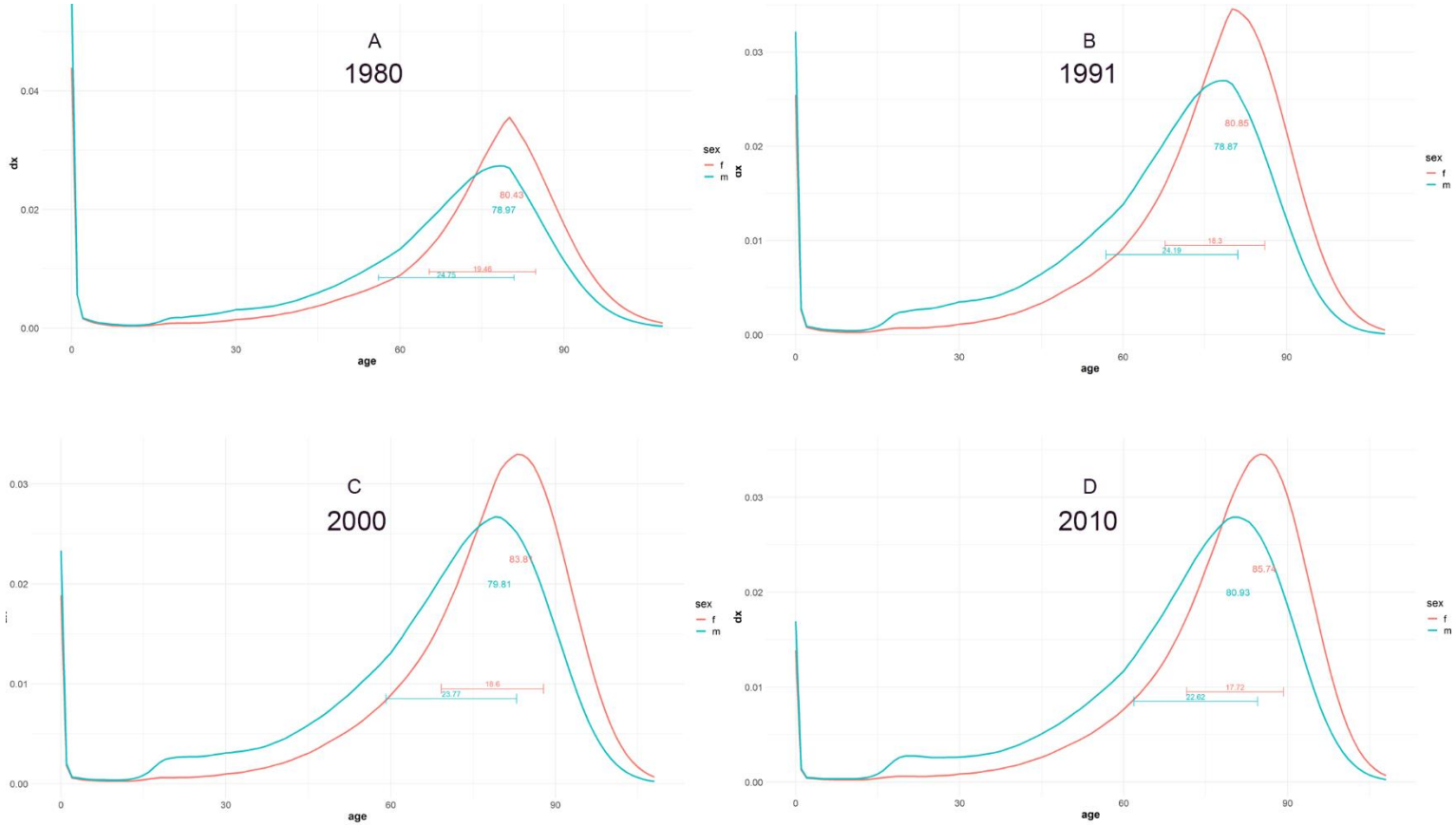
represented 27.1% of total female life expectancy gains (4.21). For men, that same cause represented a smaller proportion (20.5%) of the observed rise in e° (4.44) (CORREA & MIRANDA-RIBEIRO, 2017). Most mortality diminution from cardiovascular disease was registered above 50 years old. In relative terms, male and female CVD mortality decline between 1990 and 2015 was similar, respectively, 39.8% and 41.2% (BRANT et al., 2017). Brant et al. (2017) highlight, however, that male DALY rates (315.8/100.000) were considerably higher than females (210.7/100.000).

The pattern of mortality change (Figure 5.1) clarify how the differences in the age at death distribution evolved between 1980 and 2010 (Figure 5.2). Clearly, dx curves changed substantially in the analyzed period. Age at death distributions in Figure 5.2 are bi-modal (KANNISTO, 2001; CANUDAS-ROMO, 2008; HORIUCHI et al., 2013). In 1980 (Panel A), for both sexes, infant mode was higher than the late age mode, while in 1991 (Panel B) the first mode concentrated a higher number of life table deaths only for males. In 2000 (Panel C) and 2010 (Panel D), the late mode was higher for both sexes.

The change in ASMR between ages 20 and 30 (Figure 5.1), naturally, resemble in the age at death distribution (Figure 5.2). For males, a local mode in young adult ages in clear in Figure 5.2. In 1980 (Panel A) and 1991 (Panel B), more clearly, there are signs that the concentration of deaths in exact age 30 could be related to digit preference, as a little unusual peak in the curve is perceivable for both sexes. That concentration of deaths in age 30 (Figure 5.2, Panels A and B) could also be related to the position of the knot at that age in TOPALS that did not resulted in the best fit for these cases.

It is interesting to note how male local mode (age 21) actually got younger in 2010 (Figure 5.2D). In that year, the number of deaths rises fast from ages 14 to age 21, diminishes until age 27 and take another path upwards. Remarkably, in that same year, the first age with a higher proportion of deaths compared to the local mode age (21) is age 33. Between 2000 and 2010, Moura et al. (2016) observe that male mortality decline was smallest in the 20-29 age group and highest in the 40-49, considering the 20-59 age interval.

Figure 5.2– Age at death distribution (dx), Male X Female, 1980-2010, Brazil, TOPALS



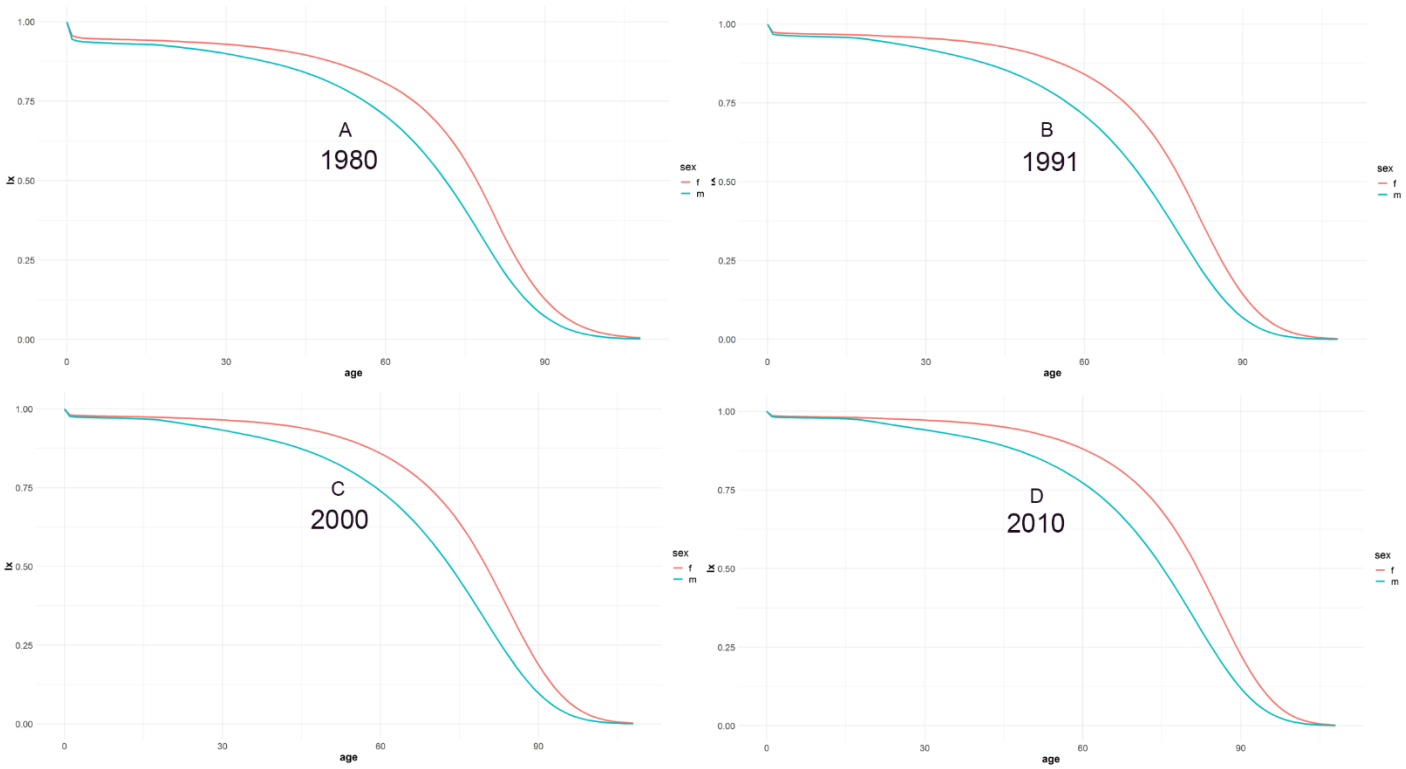
Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

The rise in excessive mortality in young adult ages in the 1980-1991 (Figure 5.2A and Figure 5.2B) period is impressive. The dx life table function can be understood as the proportional distribution of deaths by age (KEILMAN, 2017), male and female curves plotted together help visualize the amount of the male hypothetical cohort that leaves the population at very young ages, compared to women. Q1 is also a good indicator to access the magnitude of that difference, the lowest gap in Q1 was 9.26 years, in 1980. It means that, when gender gap was the lowest, the first quarter of males left the population almost 10 years earlier than females.

Another interesting feature in Figure 5.2, the number of deaths was only higher for female after age 70, explained by a greater number of women reaching ages with higher mortality. Female dx exceeded male by age 75, in 1980 (Panel A),

and close to 80 years old in 2010(Panel D). Figure 5.3 presents the number of survivors by age covering the same period and helps understand why male and female age at death distribution crossover (Figure 5.2). In 2010, only 37% of the original male hypothetical cohort was alive at age 80, while at the same age, 56% of the female cohort was still around. Naturally, more women were exposed to higher mortality risks intrinsic to older ages.

Figure 5.3– Number of survivor by age (lx), Male X Female, 1980-2010, Brazil, TOPALS



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

For males, from ages 14 to 30, compared to 1980 curve (Figure 5.3A), 1991 (Panel B) survival curve took a steeper downward trajectory. Reflecting the observed mortality rise in that age group. It is also clear in Figure 5.3 that the survival curve become more rectangular as survival probabilities rose between 1980 and 2010, a process already identified in Brazil and elsewhere (WILMOTH & HORIUCHI, 1999; GONZAGA & COSTA, 2016; GONZAGA et al., 2018). The

expressive infant mortality reduction registered in Brazil between 1980 and 2010 is also very clear. Survival curves show a higher proportion of the hypothetical cohort surviving the first year from 1980 (Figures 5.3A) to 2010 (Figures 5.3D).

In all estimates, as the set of curves in Figure 5.3 indicates, the number of male survivors by age is always lower. As already mentioned, hypothesis about sex mortality differentials are generally divided into biological – female mortality advantage is related to anatomic and physiological factors - and non-biological - culture and society are behind male excessive mortality (LUY, 2003; RODGER et al., 2010; SCHUNEMANN et al., 2017). Female advantage has also been associated to the fact that women seek medical assistance more often, and male excessive mortality have also been attributed to social stress (LUY, 2003).

The great variance in mortality gender differentials between countries along time indicates that the observed gap is not solely explained by biological factors (SCHUNEMANN et al., 2017). Wisser and Vaupel (2014) argue that that the biological characteristics account for a small part of the difference. Sex prenatal and infant mortality differentials cannot fully be attributed to behavioral factors, and might be related to biological effects (LUY, 2003). In Figures 5.1, 5.2 and 5.3 it is very clear that male mortality was higher in every age, including infants. In Mexico, García-Sancho et al. (1989) studied sex differences in infant and children mortality and found male excessive mortality in all Mexican states.

Luy (2003) compared mortality differences from nuns and monks from a Bavarian Catholic community where male and female lived by similar rules and vows (same lifestyle and environmental risks). Differently from what was observed in the general German population (where male mortality decline stagnated between 1950 and 1970), nuns and monks life expectancy at age twenty-five rose by practically the same amount (LUY, 2003). That meant, according to the author, that biological factors could not explain the observed rise in mortality gender gap in the general population. Luy and Gast (2014) highlight other populations with more homogeneous lifestyle and social environment where sex gap is also lower.

Schunemann et al. (2017) modeled the health deficit accumulation for males and females, and then they re-ran the model applying to males the estimated female

preference parameters. They found that 70% of life expectancy gap was explained by behavioral differences. Preston & Wang (2006) found that cohort-smoking patterns for those aged less than 40 impacted severely their mortality outcomes and influenced mortality gender gap in the USA. The pattern of mortality sex differentials in the USA was mainly explained by smoking habits (PRESTON & WANG, 2006). Luy and Gast (2014) highlight that it is an open debate if sex differentials in life expectancy are primarily caused by factors leading to lower female mortality of factors leading to male excessive mortality. Although, same authors argue that empirical evidence suggest that the second hypothesis is more plausible.

In Brazil, several studies indicate that behavior, in a great extent, explains mortality differences between males and females. At young adult ages, when gender mortality differences reach their maximum (Figure 5.1), external causes are the main reason to the gap. For each female external cause related death, eight men die (MOURA et al., 2015). Homicides and traffic related accidents are the most important among external causes for males (CAMPOS et al., 2015). Chandran et al. (2012) highlights that in 2008 almost 90% of registered motorcycle accident victims were males, and that the 20-29 age group present the highest rates. In Figure 5.1, it gets very clear the extent of the difference in the number of deaths in those ages between males and females. In every year, the highest difference between mx was around 22 years old.

Male mortality was higher in all parts of the age distribution (Figures 5.1-5.3). Female mortality advantage has also been associated to the fact that women seek medical assistance more often (LUY, 2003). Abreu et al. (2009) compared mortality differences by sex from avoidable causes in Brazil (avoidable given improvements in diagnosing and early treatment; avoidable given treatment and medical assistance improvements; and ischemic heart disease (IHD)) between 1983 and 2005. In all cases, male mortality was higher, especially after age 45. It is important to underscore, as authors argue, that avoidable causes represented a more expressive share of female deaths.

Abreu et al. (2009) estimated that males have 117% higher chances of dying from IHD compared to women. IHD represented the highest DALY rate in male

population (LEITE et al., 2015). According to Daniel et al. (2005), compared to women, male seek medical assistance later after pain starts and tend to diminish or deny symptoms. Parahyba (2006) argue that, even after controlling for socioeconomic conditions, Brazilian women above 60 years old seek medical assistance more often than men. In Figure 5.3 (A-D), male survival curve present a much steeper downwards slope close to age 50.

There are signs, however, that ischemic heart disease in females is less deadly given their hormonal characteristic (DANIEL et al., 2005; NIKIFOROV et al.,1998). Body Mass Index (BMI), socioeconomic characteristics, treatment of associated diseases, stress levels may also play a role in gender mortality differences from that cause (DANIEL et al., 2005). IHD may be one of the main reasons why male mortality is substantially higher, even passed young adult ages when external causes play the most important role.

After ischemic heart disease, the second more important cause of death in male population was homicides and violence, and in third, abuse and dependency of alcohol (LEITE et al., 2015). It is interesting to note that, as Leite et al. (2015) argue, both homicides and alcohol abuse are not within the ten most important causes of death for females. Wisser and Vaupel (2014) observes that in most countries homicides, suicides and drug abuse are among the main behavioral reasons behind male excessive mortality. According to Leite et al. (2015), the fact that homicides are the second largest cause of lost years of life makes it one of the main public health issue in Brazil.

Belon et al. (2012) analyzed mortality differences in Campinas (2004-2008) considering socioeconomic strata, sex and age. Considering male population, the 20-29 age group presented the highest strata gap (the ratio between the lower and the higher socioeconomic strata), and external causes showed greater social inequality in mortality (BELON et al., 2012). An interesting result brought by the same authors is that, except from external causes, females presented higher ratios between lower and the higher socioeconomic strata. Drummond Jr. & Barros (1999) found similar results for São Paulo municipality, except for the 15-24 age interval.

Belon et al. (2012) highlights that, compared to women, men have higher mortality in all ages and causes of death analyzed. In São Paulo, women from the lowest socioeconomic strata presented lower mortality than men from the wealthiest group in 1991 (DRUMMOND JR. & BARROS, 1999). In Campinas (2004-2008), the same picture is shown, lower strata females aged 20 to 64 have lower mortality than male better off in the socioeconomic distribution.

In developed countries, as gender roles in societies got more similar, mortality gap between males and females diminished and became more closely explained by biological characteristics (CULLEN et al., 2015). In developing countries, at the beginning of mortality decline (Brazil, Mexico, Thailand), gender gap rose (CULLEN et al., 2015). Same authors argue that in developed countries gender gap also rose at the beginning of mortality transition, although in a slowly rhythm. Between 2000 and 2010, Cullen et al. (2015) found signs that the sex differential diminished in the group of countries that experienced transition more recently (After 1970). Except from the measures located at the end of age distribution (M and SD(M+)), the results described in this Chapter indicate that sex differential diminished in the 1991-2010 period.

Grushka (2014) found out that in Argentina the 1960-1970 registered mortality stagnation only for males and, consequently, life expectancy gap rose. In 2010, the e° difference between males and females in that country was around seven years, close to the difference estimated for Brazil in 2016 (IBGE, 2017; GRUSHKA, 2014). In Cuba, sex differential in mortality are lower that observed in Brazil or Argentina. Martín et al. (2010) estimated a 3.8 difference in e° in 2006. Although, as observed elsewhere, accidents and alcohol related diseases represented the most dramatic cases of male excessive mortality (MARTIN et al., 2010).

Countries where epidemiological transitions started later (in the 1990-2000 period) presented higher levels of the M/F ratio, meaning lower mortality differentials between males and females (CULLEN et al., 2015). Likewise, in poorest countries, they found higher levels of M/F and even cases where that ratio was higher than one. Considering 195 countries, Clark and Perk (2012) found the lowest gender gap in the Middle East and in Africa. Cullen et at. (2015)

found that, among USA counties in 2010, poverty and proportion with lower education were negatively associated with their gender gap mortality measure. Same authors observe, in the sense, that the logarithm of income per capita and an occupational similarity index rose along with the M/F ratio. Clark and Perk (2012) found the higher income inequality leads to wider sex mortality gap.

Brant et al. (2017) highlight that in Brazilian less developed regions (North and Northeast) female mortality from CVD diminished more expressively in comparison with their male counterpart. For males, the second highest external causes mortality was registered in the Northeast region. Interestingly, the same region had one of the lowest external causes DALY rate for women (CAMPOS et al., 2015). Brazil is a very heterogeneous country, and clearly is it likely that we can identify internal differences in mortality gap between states. In the next chapter, the mortality dynamics in the Brazilian Federal Units (UF) will be presented. Although not focused on a gender gap perspective, inter-state dynamics might help broaden understanding on how mortality evolved for both males and females.

In accordance with the literature (CORRÊA & MIRANDA-RIBEIRO, 2017; BORGES, 2017. SIMÔES, 2002), old age female mortality declined more than male, as the modal age at death sex gap rise indicated. Especially between 1980 and 1991, both IQR and MD sex gaps rose influenced by the rise in young adult mortality (CERQUEIRA, 2014; BORGES, 2017; CAMARANO et al., 1997; DUARTE et al., 2002). Afterwards, MD gap diminished but in 2010 it was higher that it was in 1980. Potential issues with the model fit in 1980 and 1991, as females higher values indicate, may have influenced SD (M+) differentials. In 2000 and 2010, years with better data and better model fit for females, male SD (M+) was higher.

In all analyzed years, the highest age specific mortality gap was observed in young adult ages, at the early twenties. That difference in mostly explained by external causes. However, as the analyzed curves made clear, in all ages male mortality is higher. After age 40, the gap in number of deaths by age starts increasing again. In this part of the distribution, cardiovascular diseases play an important role (ABREU et al.,2009; BRANT et al., 2017; FRANÇA et al., 2017b).

Certainly, there are also important differences in mortality changes and differentials at lower geographic unit level. In the next Chapter, we will discuss mortality differentials at Federal Units and broaden the discussion about the mortality changes measured by the selected set of indicators applied in the present dissertation.

CHAPTER 6 - GEOGRAPHIC DIFFERENCES

The Human Development Report 2016 (UN, 2016) placed Brazil as the 10th highest Gini coefficient in the world. Income inequality is high and stable (MEDEIROS et al., 2016). Medeiros et al. (2016) highlight that the wealthiest 5% of Brazilian population concentrate half of total income. Regionally, income distribution vary expressively (MONTEIRO-NETO, 2014; IBGE 2018; SIGNOR & MOURA, 2018). In 2015, the wealthiest region (Southeast) concentrated half of total available income (SIGNOR & MOURA, 2018). It is interesting to note that the poorest regions (Northeast and North) are the most internally unequal, presenting the highest Gini coefficient in that same year (SIGNOR & MOURA, 2018).

Naturally, such differences are also seem as mortality measures. As Schramm et al. (2004) argue, poverty is reflected in Brazilian health assistance and health prevention. More developed regions concentrate advanced health services and the spatial distribution of public health services reproduces regional inequalities (SIMÕES, 2002; ALBUQUERQUE et al., 2017). However, between 2000 and 2016, both income and health services distribution improved (ALBUQUERQUE et al., 2017).

From 1940 to 2010, female life expectancy gap between the wealthiest region (Southeast) and the poorest (Northeast) diminished from 7.5 to 3.5 (BORGES, 2017; SIMÕES, 2002). França et al. (2017b) argue that mortality reduction at state level (between 1990 and 2015) was widespread and more relevant among children. Same authors argue that mortality decline was not uniform and reached Northeast and North regions in a lesser extent. As Simões (2002) observes, since 1940, Southeast region registered infant mortality diminution, while in the Northeast, at the other hand, it declined more expressively after 1980. Infant mortality differences between states are, however, still very high (BORGES, 2017).

At state level, mortality changes varied substantially (BORGES, 2017; SIMÕES, 2002; FRANÇA et al., 2017; CAMPOS et al., 2015; LEITE et al., 2015). Cardiovascular disease (CVD) mortality reduction was very heterogeneous

considering Federal Units (FU) (BRANT et al., 2017). The dynamics in external causes also presented different pattern of change (CAMPOS et al., 2015). In states from the Southeast region (more expressively São Paulo and Rio de Janeiro), homicide rates rose from 1980 to 1991 and diminished afterwards. In Northern and Northeastern states, homicides mortality rose more importantly after 2000 (WAISENFIZ, 2013). A somewhat similar pattern was also observed in the traffic accidents mortality (MORAIS NETO et al., 2012). States from the North and Northeast regions, among a few others, presented higher rates of neglected tropical diseases (dengue, visceral leishmaniosis, Chagas, schistosomiasis, among others) (MARTINS-MELO et al, 2018).

Inter-state mortality differences are also influenced by data quality, which varies substantially between states (QUEIROZ et al. 2017; AGOSTINHO, 2009; HORTA, 2012). For example, States from the Northeast and North regions present higher levels of undercounting of deaths (QUEIROZ et al., 2017) as well as higher levels of digit preference (HORTA, 2012; AGOSTINHO, 2009). França et al. (2017b) argue that health inequalities might remain hidden by differences in data quality at subnational level.

In that context, this Chapter presents TOPALS estimates[9] for Brazilian states from 1980 to 2010 and discusses how the age at death distribution at state level evolved, considering both sexes. We aim to describe, as well, how young adult male mortality varied regionally, the inter-state differences in the modal age at death and the mortality crossover at older ages observed in some Federal Units. The aim is to discuss changes along the age at death distribution in the analyzed period and try to relate them to regional mortality literature in Brazil.

6.1 FUs Age at death distribution

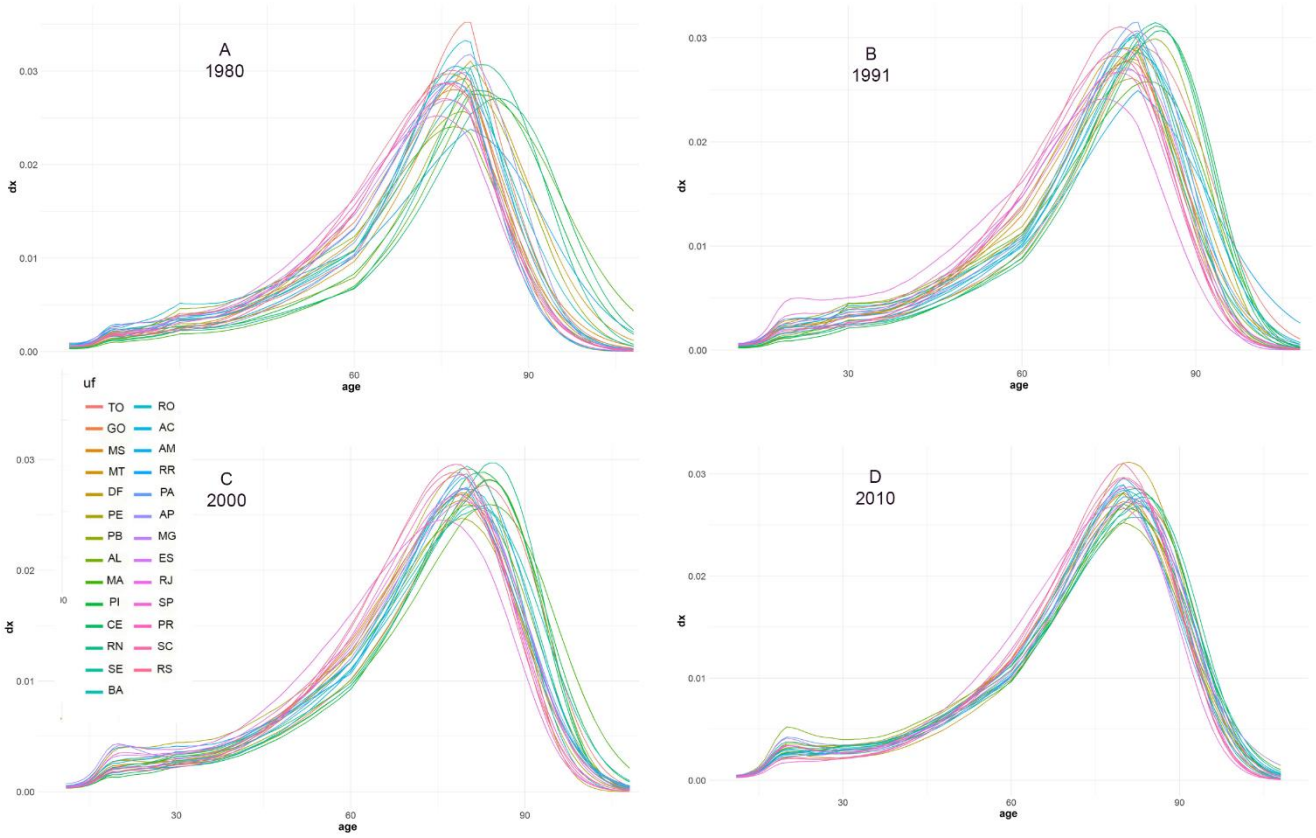
6.1.1 Male

Figure 6.1 shows male age at death distribution for each Brazilian state, from 1980 to 2010. At first sight, it gets very clear how state level dx distribution got

[9] In the annexes, all estimates are presented as tables as well

more similar with time. Compared to 1980, 2010 curves are closer to each other, in practically every age. Interestingly, however, 2010 curves seems to be more heterogeneous at young adult ages. Between 1980 and 2010, the difference from the highest and the lowest modal age of death diminished from 10.43 to 4.83. That difference must be considered with caution since data quality also improved in the period (HORTA, 2012; QUEIROZ et al., 2017; AGOSTINHO, 2009; LIMA & QUEIROZ, 2014). However, it is very clear the converging dynamic of mortality at state level (SIMÕES, 2002; BORGES, 2017; FRANÇA et al., 2017; DUARTE et al., 2002).

Figure 6.1 - Age at death distribution by state, Male, 1980-2010, Brazil



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

In 1980 (Panel A), FUs age at death distributions are, notably, very different. In most states, dx curve rises fast from age 11 (first year in the dx distribution) to

age 20. There is a clear a peak at age 30 in some states. That concentration of deaths at exact age 30, at some level, might be related to digit preference. Another potential explanation is that in 1980, for some states, data required a different set of knots in TOPALS estimation. At least to our knowledge, there is no particular reason to think why exact age 30 would present a higher probability of dying than age 31 or 32, or how to explain a fast rise from age 29. Since 1980, quality of age information improved substantially (HORTA, 2012; AGOSTINHO, 2010; LIMA & QUEIROZ, 2014), and that excessive number of deaths at age 30 diminished as time passed. The exposure measure applied to population information diminished, at least partially, digit preference in census data. In the registered number of deaths, besides correcting from undercounting, no other treatment was applied.

Another potential sign of digit preference reflected in the age at death distribution is the change in the slope the curves at age 60. In most curves plotted in Figure 6.1 (Panel A), the slope change at age 60 is visible and, in some cases, such change is very abrupt. The path change in that curves is more severe in states in green and blue tones, located, respectively, at Northeast and North regions - the less socioeconomically developed (IBGE 2018; SIGNOR & MOURA, 2018). Quality of age information is related to literacy levels (PALLONI et al., 2014). The average income of an employed person in the Northeast and North regions (2017) represented, respectively, 77% and 69% of the average income at national level (IBGE, 2018). More generally, the shape of the curves in 1980 also indicate that, in some states, there might be problems with the quality of the data that generated them. Although, the position of the knots in the 1980 estimation may have also influenced the observed shape.

Compared to 1980, 1991 (Figure 6.1B) curves present a more similar shape. In that year (1991), the number of deaths registered at young ages rises close to age 20 in some states. In Rio de Janeiro and São Paulo, more intensively, mortality from external causes rose between 1980 and 1991, caused mainly by violent deaths (WAISENFIZ et al. 2013; CERQUEIRA et al., 2017; BORGES, 2017; SIMÕES, 2002). In many FUs there still is a peak in the dx curves at age 30 (Figure 6.1B). After 1991, the concentration of deaths at young adult ages did not diminish. Actuality, more states presented an increase at the local mode of

the age at death distribution from 1980 to 2010. From 1990 to 2015, males between 15 and 19 presented the lowest mortality reduction (FRANÇA et al., 2017b).

In 1991, apparently, there are two clusters of age at death distribution, one represented by green and brown tones (Northeastern and Central-Westerns) and one identified by pink tones (Southeastern and Southern). Curiously, the first group presents higher modal age at death. Given their socioeconomic and health indicators (IBGE 2018; SIGNOR & MOURA, 2018; BORGES, 2017; SIMÕES, 2002; FRANÇA et al., 2017), we would expect higher modal age at death to be observed in the wealthiest regions. As observed in 1980, in 1991, the abrupt change the age at death distribution at age 60 was more frequently observed in Northern and Northeastern states (green and blue tones).

When we look at state level age at death distributions in 2000 (Figure 6.1C), it is clear that the curves present a more similar pattern. In 2000, the gap between highest male modal age at death and lowest diminished (8.77). Male life expectancy at birth gap between the Southeast and Northeast regions reached the lowest point in 2000 (SIMÕES, 2002; BORGES, 2017). In that same year, the concentration of deaths at 30 (Figure 6.1C) diminished substantially. Another sign of improvement in the information quality, the abrupt change in slope at age 60 also lessened. It also means a better fit in model estimation. The two clusters of dx curves observed in the previous years are still present in the 2000 estimates, but they are closer to each other. As in 1991, curves from states in the Southern and Southeastern (pink tones) and from Northeastern and Central-Westerns (green and brown tones), roughly, represent two different groups.

In 2010 (Figure 6.1A), curves are even more close to each other, indicating that mortality differentials between states are converging and, at the same time, that data quality improved in the period. Whipple Index diminished substantially, indicating that census digit preference decreased with time (HORTA, 2012; AGOSTINHO, 2009). In their death registry system coverage analysis, Queiroz et al. (2017) showed that between 1980 and 2010, at state level, SIM coverage also improved. In 2010, that peak at age 30 is practically gone and the slope change at age 60 is less frequent and less marked. However, it is important the

underscore that there are still important inter-state differences in quality of the information (HORTA, 2012; AGOSTINHO, 2009; QUEIROZ et al., 2017).

6.1.2 Young adult ages

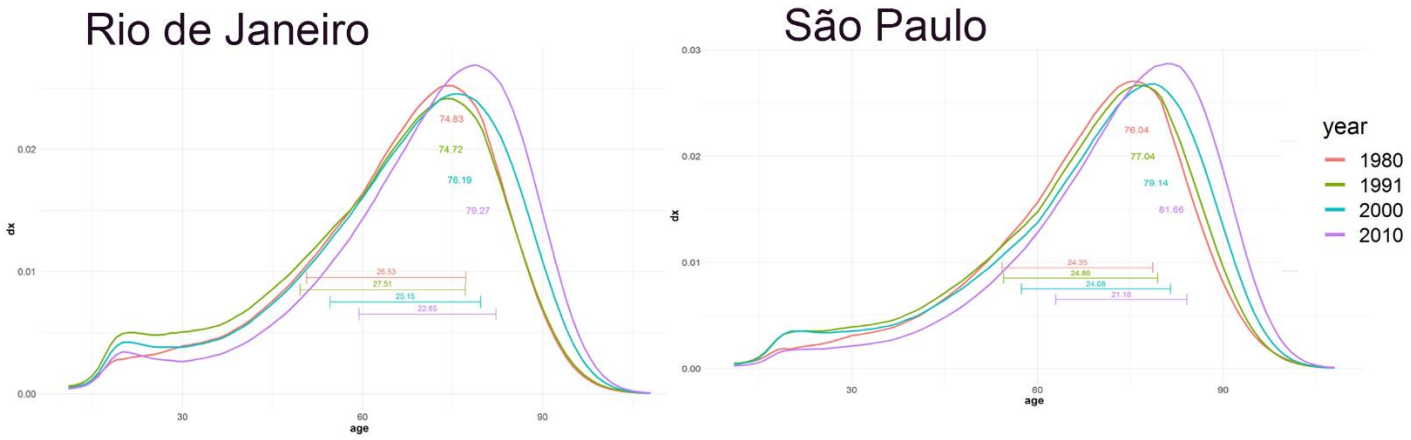
Comparing age at death distribution curves at young ages (Figure 6.1) it is clear that the local mode got younger and, in 2010, it concentrates a higher number of deaths. Between 1980 and 2010, the local mode rose for most Federal Units. In Rio de Janeiro and São Paulo, after a rise between 1980 and 1991, the number of deaths diminished in the 2000-2010 period. Mortality change in such ages presents a strong regional component (MALTA et al. 2017; CAMPOS et al., 2015; WAISENFIZ et al. 2013; CERQUEIRA et al. 2017). Malta et al. (2017) argue that homicides rates, in Brazil, were stable in the 1990-2015 period, mainly because of great regional differences. Same authors observe that, from ages 1 to 49, violent deaths are the main cause of death for males. It is also important noticing that in all Brazilian states the level of violent deaths was at least twice as high as it would be expected given their Sociodemographic Index (SDI) (FRANÇA et al., 2017b)

To illustrate the pattern of change in male age at death distribution at young ages, according to male mortality literature in Brazil, four states from different regions were selected. The dx curves from Rio de Janeiro and São Paulo (Figure 6.2), and Pará and Bahia (Figure 6.3) were chosen to demonstrate two different dynamics in the pattern of change of young adult mortality along the 1980-2010 period. Pará is located at the North region and Bahia at Northeast. São Paulo and Rio de Janeiro are both located at the Southeast.

The two sets curves presented in Figure 6.2 indicate that, from 1980 to 1991, the number of young adult deaths rose. In Rio de Janeiro and in São Paulo, there was a very clear increase in life table deaths registered at the 15-30 age interval. In Rio de Janeiro, the age that indicate the point where 75% (Q1) of the cohort was still alive actually diminished between 1980 and 1991. In São Paulo, Q1 practically did not change, while Q3 rose, consequently IQR rose in that same

decade. Gonzaga et al. (2009) also captured a rise in IQR in São Paulo from 1980 to 1990.

Figure 6.2 – Age at death distribution, Males, Rio de Janeiro and São Paulo, 1980-2010



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

Between 1991 and 2000, in Rio de Janeiro (Figure 6.2), the concentration of deaths at the local mode diminished and the late modal age at death rose. Q1 rose more substantially than Q3, representing an IQR diminution. In São Paulo, in the same period, the local mode remained practically at the same level. Compared to 1991, São Paulo dx curve (2000) declined after age 25. In contrast with Rio de Janeiro, between 1991 and 2000 IQR in São Paulo diminished more modestly.

In both states, from 1991 to 2000, a rise in modal age at death is perceivable at the end of the distribution. From 2000 to 2010, M rose even further. Clearly, indicating mortality diminution at ages above the mode (CANUDAS-ROMO, 2010). França et al. (2017b) observe that Southeast region presented higher ischemic heart disease mortality decline between 1990 and 2015, the leading cause of death for males (LEITE et al., 2015). Rio de Janeiro and São Paulo, specifically, were among states with sharpest cancer mortality decline and within the group with declining diabetes related deaths (FRANÇA et al., 2017b).

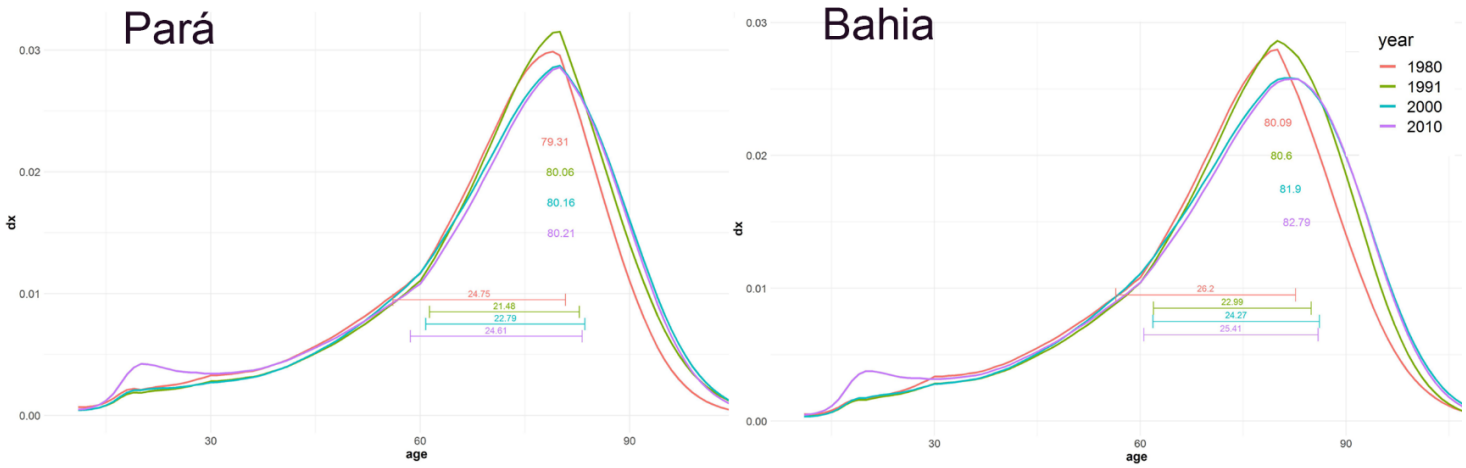
In 2010, Figure 6.2 indicates a lower proportion of deaths in young adult ages compared to the same number registered in 1980 (São Paulo). In Rio Janeiro, 2010 registered a higher number of deaths than 1980 only from ages 17 to 23. There was also a clear reduction in the number of registered deaths compared to 1991. From 2001 to 2011, homicide rates in São Paulo and Rio de Janeiro diminished, respectively, 67.7% and 43.9% (WAISELFISZ, 2013). Young adults male are also the group that dies more often in traffic accidents (TA) (OMS, 2013; MORAIS-NETO et al., 2012; MORAIS-NETO et al., 2016; CHANDRAN et al., 2008). In Rio de Janeiro, between 2000 and 2010, TA mortality diminished. In São Paulo, it presented only a minor rise. Compared to other states, however, at lower levels (MORAIS-NETO et al., 2012). The reduction in the concentration of death in the young adult age group was also influenced by observed dynamic in that cause of death.

As in Rio de Janeiro, in Pará and Bahia (Figure 6.3), when young adult mortality rose the most (2000-2010), Q1 (indicated as the beginning of the horizontal lines in Figure 6.3) reduced. Which means that the age when 75% of the cohort was still alive diminished in consequence of the additional deaths. Probably, the main explanation for such pattern is the rise in external cause mortality. According to Waiselfisz (2013), between 2001 and 2011, homicide rates in Bahia and Pará rose, respectively, 223.6% and 165.8%. Traffic accident mortality may also play an important role here. Between 2000 and 2010, Morais Neto et al. (2012) found a 46.1% increase in traffic related deaths in Bahia and 32.9% in Pará. Among external causes in Northeast and North, homicides and traffic accidents deaths are the two leading ones (CAMPOS et al., 2015; MALTA et al. 2017).

Figure 6.3 shows an impressive rise in young adult mortality in the 2000-2010 period. In both Pará and Bahia, from 1980 to 2010 the number of death at 30 also diminished. Compared to São Paulo and Rio de Janeiro, in Pará and Bahia (located in less developed regions), the change in the slope of the curves at age 60 is more expressive. In same states, more clearly in 1980 and 1991, there are also signs of digit preference at age 80. It could also be consequence of model estimation, as already discussed. However, Figure 6.3 indicates signs of improvement in data quality with passing years. In Bahia, death distribution methods (DDM) (SEG-adj) indicate that SIM coverage estimates rose from 0.77

(1980/1991) to 0.91 (2000/2010), in Pará the estimated coverage in the same period did not improve (from 0.80 to 0.77) (QUEIROZ et al.2017). In the same period, the Whipple Index diminished (AGOSTINHO, 2009; HORTA, 2012).

Figure 6.3 – Age at death distribution, Males, Pará and Bahia, 1980-2010



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

Compared to the Southeastern states, late modal age at death rose more modestly both in Pará and Bahia, indicating that old age mortality decline was less important in those states. Not only at ages above the mode, in São Paulo and Rio de Janeiro there is a very clear shift in dx curves in adult ages, even more important in the last studied decade (2000-2010). In contrast, adult mortality decline in Bahia and Pará (represented in Figure 6.3) was not so representative. From 1990 to 2015, Brant et al. (2017) the estimated decline in cardiovascular diseases was higher in the more developed regions (South and Southeast) and in Distrito Federal. At the other hand, same authors observe that in North and Northeast the decline was less expressive and states from those regions have the higher cardiovascular mortality rates.

Borges (2017) showed that, between 1980 and 1991, in Southeast and South (less markedly) mortality rose in ages between 15 and 40. Besides the already mentioned rise in external causes mortality, that same period was also marked

by an increase in mortality by HIV that affected mainly males between 25 and 39 years old (BORGES, 2017). HIV was the most important cause of death for males from the wealthiest neighborhoods in São Paulo (municipality) (DRUMMOND-JR & BARROS, 1999). In Northeast and North regions, the massive negative contribution from young adults (15-29) only came in the 2000-2010 period (BORGES, 2017). In 12 of all 27 Brazilian Federal Units, most of them in the North and Northeast regions, the risk of death for males aged 15-49 rose between 2000 and 2010 (FRANÇA et al., 2017b).

It is important to mention that the increase in violent deaths in the last decade considered here (2000-2010) was not exclusive to Northeastern and Northern states. In Minas Gerais (Southeast) and Paraná (South), age at death distribution (not shown here) also indicates a rising concentration of deaths in young adult groups from 2000 to 2010. From 2001 to 2011, Waiselfisz (2013) estimated a 50.7% increase in homicides rates in Paraná and 66.0% in Minas Gerais. Malta et al. (2017) also captured the rising violent mortality in those states.

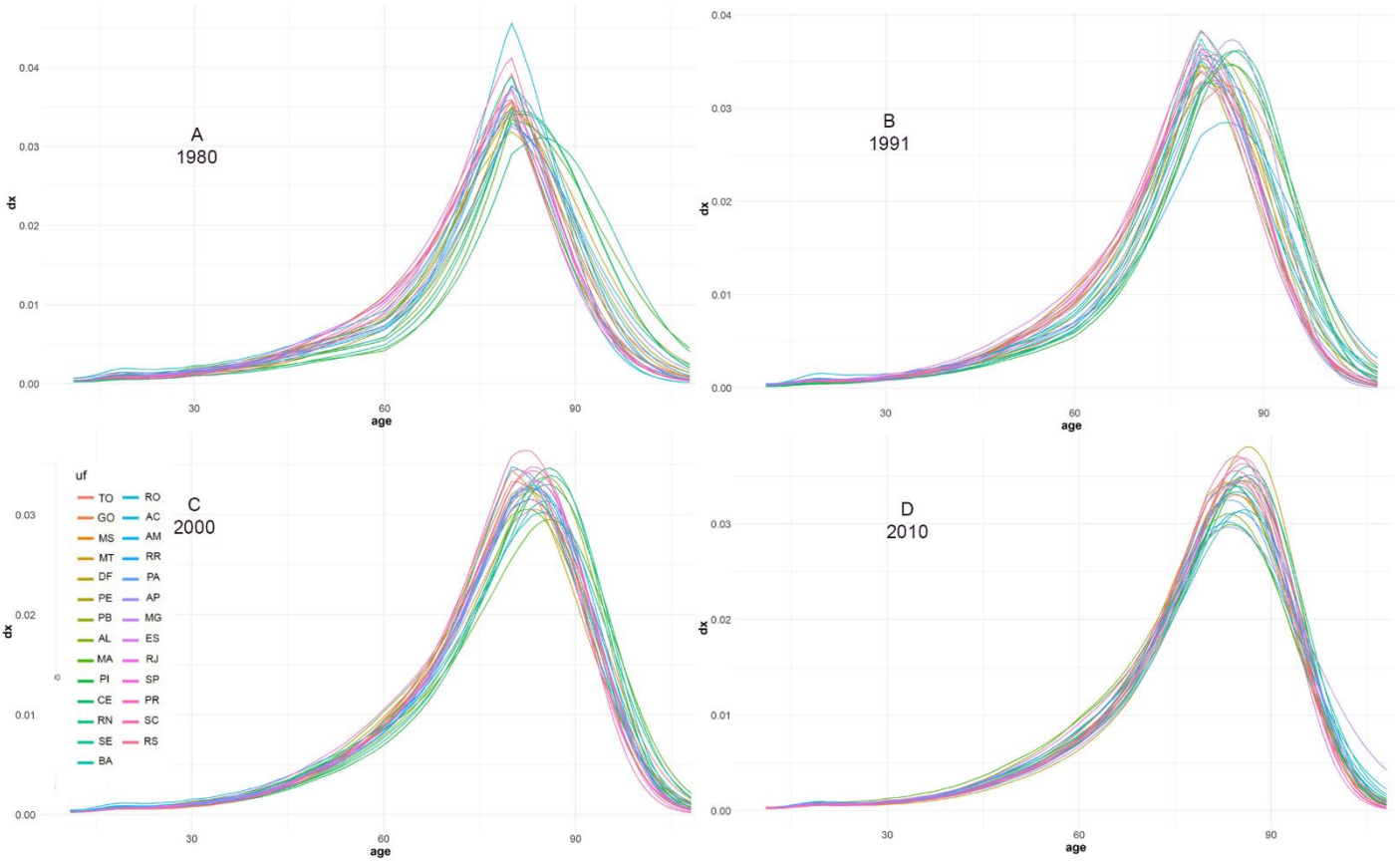
Moura Neto et al. (2012) observes that mortality by transport accidents (AT) increased in most Brazilian states between 2000 and 2010, and the highest increases were observed in Northeastern states (MOURA NETO et al. 2012). Based on 2013 National Health Survey (Pesquisa Nacional de Saúde-PNS), Moura Neto et al. (2016) point out that 3.1% of sample declared being involved in a Road Traffic Incident (RTI) in the previous year. Among regions, however, there were important differences, 4.8% of the North sample declared being involved in a RTI in the previous 12 months, twice as high as observed in the Southeast region (MOURA NETO et al., 2016).

6.1.3 Female

As observed with the male age death distribution in Brazilian states, from 1980 to 2010, the curves are closer to each other. Apparently, female (Figure 6.4) dx curves are less heterogeneous. Inter-region differences in the distribution of the most important causes of deaths was less relevant for women in comparison with men (LEITE et al., 2015). The gap between the highest and the lowest female

modal age at death diminished from 5.18 (1980) to 3.33 (2010), considerably lower than what was registered considering male population. Mortality reduction from cardiovascular diseases (1990-2015) was expressive for females (BRANT et al., 2017). In that same direction, Borges (2017) argue that, in general, female mortality reduction in the North region presented larger gains to life expectancy than males.

Figure 6.4 – Age at death distribution by state, Female, 1980-2010, Brazil



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

However, as already discussed in previous Chapters, apparently, 1980 female age at death distribution seems more problematic at older ages. It would be necessary a more profound investigation (beyond the scope of this thesis) to understand the reason behind that shape, but, among possible reasons, there are signs of a strong digit preference at age 80. In 1980, Agostinho (2009) found a very high simple age concentration index at exact age 80 for Brazilian females.

In 2010 (Figure 6.4), however, there are, practically, no clear signs of such preference. It is possible that another set of knots could lessen the sharpen pattern of the curve at age 80 in 1980 and 1991.

Figure 6.4 also (as observed for males) indicates the presence of two clusters of dx distributions, more clear in 1991. Those two groups are mainly represented by green and brown tone curves (Northeastern and Central-Western) and pink tone (Southeastern and Sothern). In 2010, the clusters are not distinguishable. One interesting feature in 2010 curves (Figure 6.4D) is that the differences between highest and lowest M are not as high (1.49 year lower) as male analysis showed.

It is also important to notice that the inclination change at age 60 is less marked for females. In 1980 (Panel A), the slope change is perceivable (in a lesser extent) in states colored in green and blue tones (Northeast and North). The single age concentration index at exact age 60, for both males and females, in 2000, was considerably higher in Northeast and North regions (AGOSTINHO, 2009). In Figure 6.4, in a lesser extent than registered with males, a peak in the number of deaths at age 30 can also be seem, especially in 1980 and 1991. The convergence dynamic in the age at death distribution between states is very impressive. Comparatively, in 2010, the FU age at death distribution are much more similar to each other.

Simões (2002) argue that mortality change for females was more balanced than the observed for males. Naturally, differences related to external causes also make female mortality less heterogeneous. Male mortality rates from that group of causes of death was five time as high as the observed for women (CAMPOS et al., 2015). It is also interesting to note that at young adult ages, from 1980 to 2010, female mortality contributed positively to life expectancy gains (SIMÕES, 2002; BORGES, 2017), it is difficult to distinguish the curves at reproductive ages in the age at death distribution (Figure 6.4).

6.2 Modal age at death by state

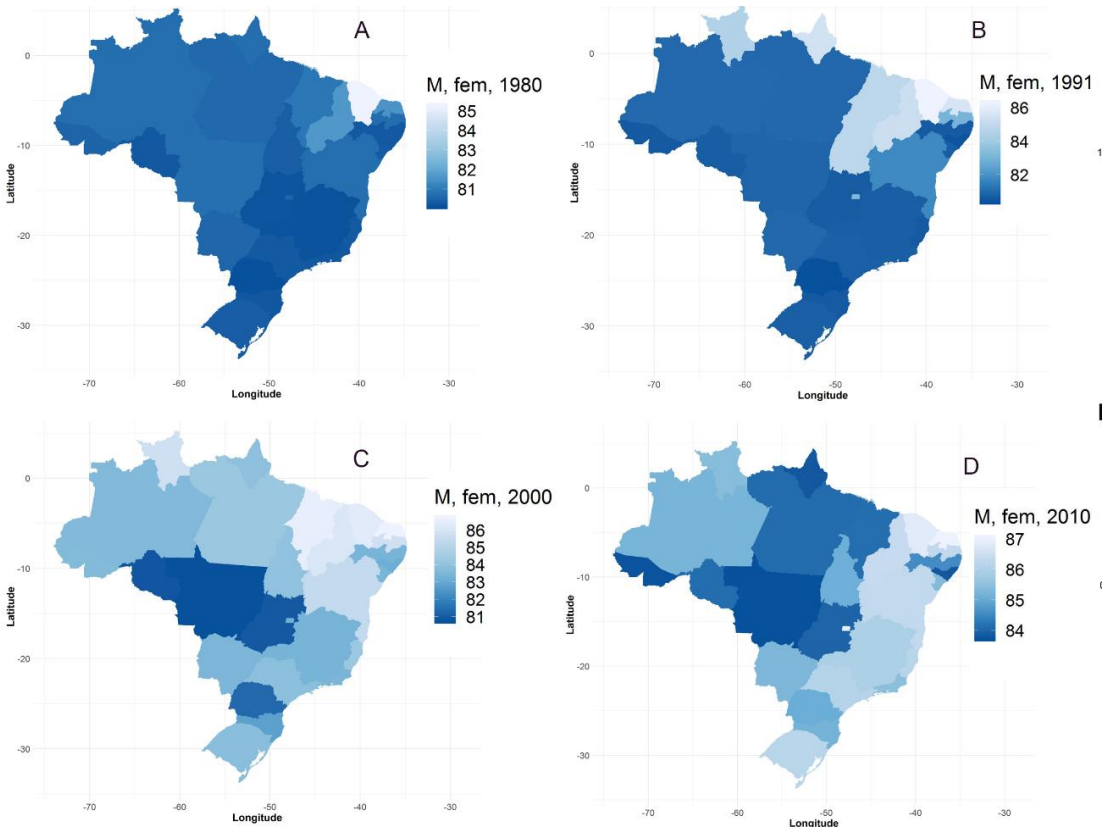
An interesting way to visualize the differences in the modal age at death (M) between states is to plot them in a choropleth map. Figure 6.5 presents the

estimated female M for every year in analysis. Surprisingly, Northeastern states presented higher modal age of death in every analyzed year. In 1980 (females), among the highest five modal age at death, only one (Central-Western) was not located in the Northeast region. The same picture was observed in 1991, the only two non-Northeastern states amongst those with higher M were Amapá and Tocantins (North). For females, all top five M in 2000 were observed in Northeastern states and, in 2010, the only exception was Distrito Federal. Along the analysis period, the regional pattern of highest achieving modal age at death changed. In 1980 and 1991, clearly, South and Southeastern states were those in darker blue in Figure 6.5 (the darker the lower M). In 2010, those states are among those with higher M, although none of them was among the five top achievers.

It is unexpected that Northeastern states presented the highest modal age of death since it is the poorest Brazilian region (MONTEIRO NETO, 2014; IBGE, 2018; SIGNOR & MEDEIROS, 2018). The poverty measure suggested by the World Bank to countries with income level similar to Brazil - below US$5.5 a day - indicates that, in 2017, 44.8% of Northeastern population lived below that line, and in the Northern region 43.1% were in that same condition. Comparatively, in the South region 12.8% lived with less than US$5.5 a day. At state level, the highest proportion of individuals living in poverty was observed in Maranhão (54%) and the lowest in Santa Catarina (8.5%), respectively a Northeastern and a Southern state (IBGE, 2018).

According to IBGE (2018), the three states with lowest average labor income were located at the Northeast regions, respectively, Maranhão, Piauí and Alagoas. In Northeast and North regions, Signor and Moura (2018) show that, at least since 1976 (except for 1986), average labor income have always been below national average. Several authors indicate that individuals in a more difficult socioeconomic condition present higher mortality (PAMPEL et al., 2005; CUTLER et al. 2006; LANTZ et al., 1998; EDWARDS & TULJAPURKAR, 2005). Edwards and Tuljapurkar (2005) observe that those in a lower socioeconomic strata present lower average lifespan and greater age at death variability.

Figure 6.5 – Modal age at death by state, Females, 1980-2010, Brazil



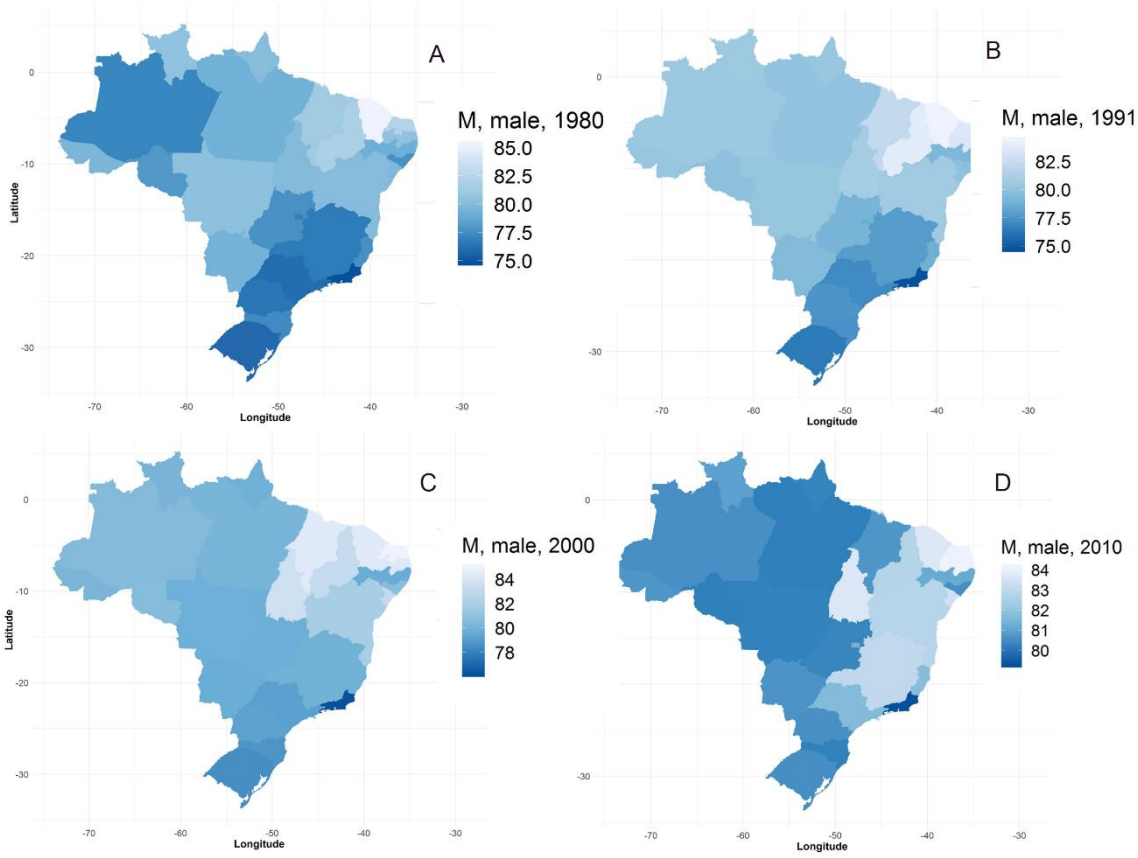
Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

As observed for females, Northeastern states registered the highest modal ages of death in every analyzed year among males (Figure 6.6). In 1980, with the exception of Roraima (North), the five highest modal age at death were recorded in that same region. All five highest male M in 1991 were located at Northeast region. In 2000 and 2010, the only exception was Tocantins (North). Figure 6.6 makes it very clear how the highest modal age at death were always higher in Northeastern states. It is interesting to note that, in 1980 and 1991, states in South and Southeast regions were those with lower modal age at death. In 2010, the highest M were still registered in the Northeast region, however, Southeastern states are clearly colored in a lighter blue, indicating that, comparatively, modal age at death rose in these states.

Considering male and female modal age at death analysis, the highest achieving states were, unexpectedly, located at states in the Northeast and North regions

region, even in the last year of analysis (2010). Data quality improved in the analyzed period, and, however, in 2010 the highest M was still observed in the Northeast. Not only the highest violent deaths rates were observed in states from the North and Northeast regions (WAISENFIZ, 2013; IPEA, 2017; MORAIS-NETO, 2012), they also presented lower CVD (BRANT et al., 2017), diabetes and cancer (FRANÇA et al., 2017) mortality diminution. In Northeast and North regions – along with Goiás, Minas Gerais and Distrito Federal, the incidence of neglected tropical diseases was also higher (MARTINS-MELO et al., 2018).

Figure 6.6 – Modal age at death by state, Males, 1980-2010, Brazil



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

In that sense, we would expect the highest modal age at death to be observed in wealthier and more developed states. Some sort of selection effect, with more

robust individuals that survived throughout lifetime in more harsh conditions and would have lower mortality at older ages, could be a possible explanation (VAUPEL & YASHIN, 1985; VAUPEL et al., 1979; MANTON et al., 1987). However, our main hypothesis behind that phenomenon is related to age misreporting problems that may affect both the death registry system and the censuses, that kind of problem affect more severely old age mortality estimates (HORTA, 2012; COALE & KISKER, 1990; ORTEGA & GARCIA, 1986). There are examples in the literature of content errors in databases that generated mortality crossover (COALE & KISKER, 1986; COALE & LI, 1992, PRESTON et al. 1996; ELO & PRESTON, 1994).

The quality of age information is related to literacy levels and the quality of age information diminishes with age (PALLONI et al., 2014; AGOSTINHO, 2009; HORTA, 2012). In Brazil, there are signs of age reporting problems are more severe in less developed regions (AGOSTINHO, 2009; HORTA, 2012). Death registry system are also less consistent in these same regions (BORGES, 2017; QUEIROZ et al., 2017). In that context, it is likely that higher modal ages of death in less developed states are related to age reporting problems.

Another potential explanation relates to a probable difference in the death registry system coverage by age, not only differences in coverage by age, but a varying one among states. Death Distribution Methods (DDM) estimates, generally, are based in an age interval that is not (or is less) influenced by migration flows (QUEIROZ & SAWEYR, 2012; HILL et al., 2009; MURRAY et al., 2010). Usually, very young and old ages are more likely to be influenced by random fluctuation (MURRAY et al., 2010). In Brazil, Queiroz and Sawyer (2012) showed that death coverage level slightly decline with age. If the slope of the coverage decline varies between states, using the same correction factor (as we did) for each state would imply in varying underestimates of mortality at older ages between them. If coverage quality worsens faster with age for one state, or there is no age difference in registry system in other, the mortality level at older ages would be differently underestimated and, consequently, old age mortality measures would be influenced. Ahead in this Chapter (section 6.4), an example of mortality crossover between states will be presented and more broadly discussed.

6.3 Median age at death and lifespan variability

Figures 6.7 (males) and Figure 6.8 (females) presents the median age of death (y-axis), Q1 (starting point in plotted line), Q3 (ending point in plotted line) and IQR (line length) for each Federal Unit (FU) in every analyzed year. As in Figures 6.1 and 6.4, there are clear signs of convergence in those indicators with time. In 1980 (Figure 6.7A), among males, the gap between the highest and lowest median age of death was equal to 14.68 and diminished to 4.45 in 2010 (Figure 6.7D). As observed in the modal age of death, inter-state gap among females was lower than the observed for men. Between 1980 and 2010, the difference between the highest median age of death to the lowest (females) diminished from 9.06 to 3.56.

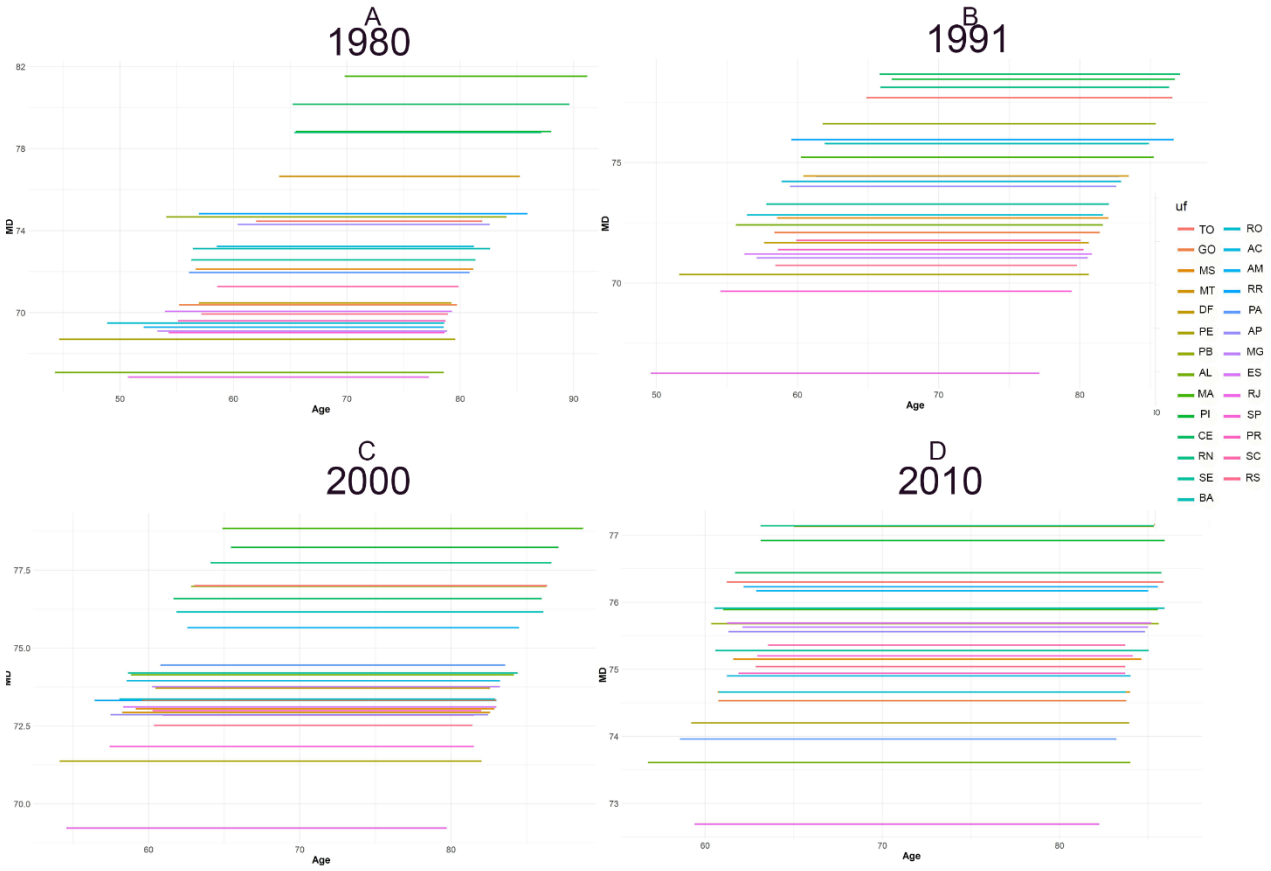
Between 1980 and 2010, for both males and females, SD (M+) also converged. Male gap between the highest and the lowest male standard deviation above the mode diminished from 5.02 (7.23-12.25) to 2.65 (7-99-10.64), while female gap diminished more modestly (from 4.12 to 3.8). Another sign of convergence, for both males (Figure 6.7) and females (Figure 6.8), the starting points (Q1) and ending points (Q3) are closer in 2010 (Panel D) than they were in the all previous years. Part of the observed convergence might be related to data quality improvement. With passing years, the highest MD diminished (for both males and females), probably indicating that the earlier estimates are less reliable.

In every analyzed year, states in green tones - from Northeast region - presented the highest MD. The only exception (also observed in the modal age analysis) among females, Distrito Federal in 2010 (Figure 6.8D). In both Figures 6.7 and 6.8, more clearly in 1980 (Panel A) and 1991 (Panel B), green tone lines (Northeast) and blue tone lines (North) are located at the top of the distribution. In 2010 (Panel D), the distribution is less clear about a regional pattern but it still presents green lines among those with highest MD. Inter-state gap in every indicator (Q1, Q3, IQR) for males and females diminished with time, indicating both data improvement and convergence in mortality indicators.

As observed in the modal age of death, highest median ages of death were also registered in states from Northeastern and Northern regions. Interestingly, at the

other hand, states from those same regions presented the highest IQR, shown by the line size in Figures 6.7 and 6.8. In 2010 (Males), for example, among the highest estimated IQR, four were observed in Northeastern states and one in a Northern state. A very similar picture was observed with female data. A regional pattern in SD(M+) was not clearly observed, especially for males. Considering females, in every year more developed states were among the best achievers, however, along with states from North and Northeast regions.

Figure 6.7 – Median age at death, Q1, Q3 and IQR, Males 1980-2010

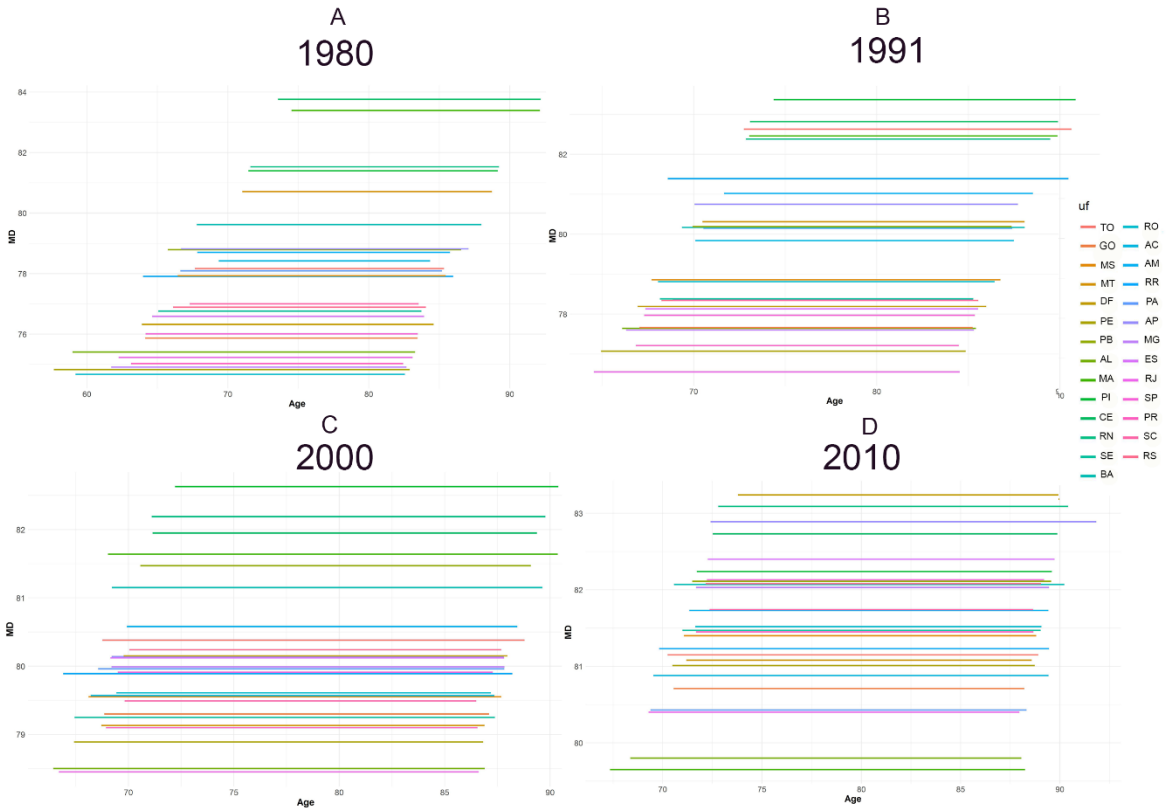


Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

Edwards and Tuljapurkar (2005) point out that higher socioeconomic inequality is associated with higher age at death variability. In terms of IQR, results presented in Figure 6.7 and 6.8 goes in the same direction authors highlight. At the other hand, higher median age in the less developed region is unexpected and require more caution in the analysis. The fact that Northeastern states and

Northern state presented higher MD gives strength to that view that with might be dealing with an age reporting problem, affecting more importantly older age estimates.

Figure 6.8 – Median age at death, Q1, Q3 and IQR, Females 1980-2010



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

The excess young adult mortality in Rio de Janeiro and São Paulo is reflected in male IQR observed in 1991 and 2000, as Panels B and C (Figure 6.7) shows. Gonzaga and Souza (2016) observe that Southeast region (where Rio de Janeiro and São Paulo are located) presented the highest IQR in 1991 and 2000. In 2010, same authors observed the highest IQR for females and the second highest for males in the Northeast region. In their work, highest male IQR was observed in Central-West region and the second highest in the Northeast. Gonzaga and Souza (2016) applied a different smoothing methods - MortalitySmooth[10]

[10] Carmarda, 2012

(CAMARDA, 2012) and they did not corrected the number of deaths by undercounting, part of the observed differences could be related to that methodological differences.

6.4 Mortality crossover

Throughout this Chapter, several signs of age reporting problems were observed. There was a high concentration of deaths at exact age 30. There also was an abrupt change in the age at death distribution at exact age 60 (more significant for males). In female analysis, more importantly, there was signs of digit preference at exact age 80. These indicators might be also related to the model estimation, more importantly in 1980 and 1991. The fact that both the highest modal and median ages of death were observed in Northeastern states, given their socioeconomic level and what most mortality and health studies indicate (BRANT et al., 2017; MORAIS-NETO et al., 2016; WAISENFIZ, 2013; FRANÇA et al., 2017b; MALTA et al., 2017; BORGES, 2017), can also be understood as another indicator of potential problems.

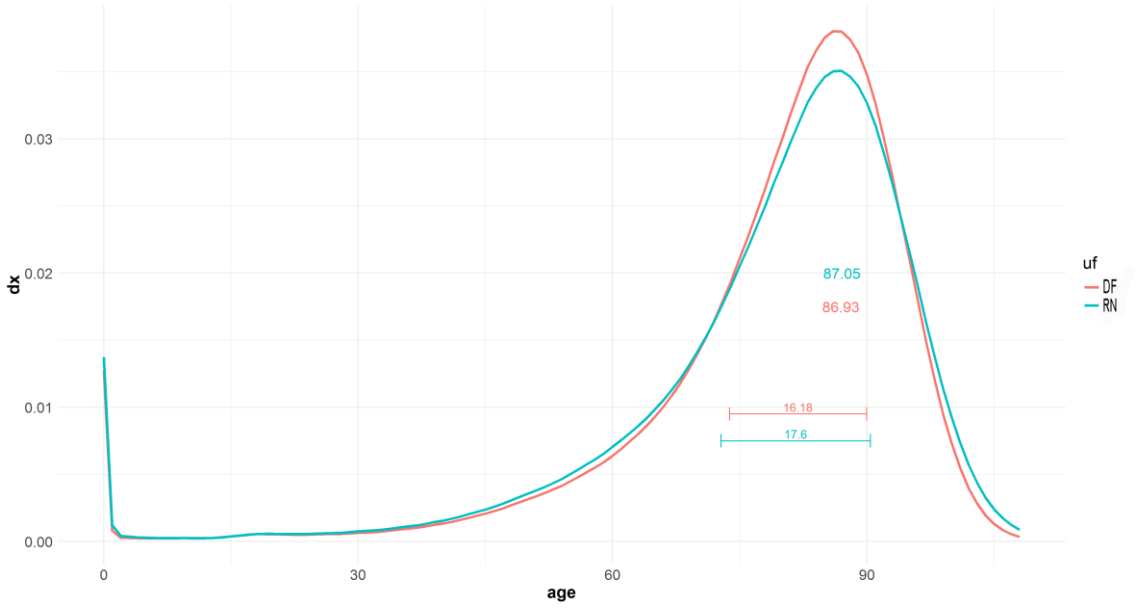
Age misreporting is hard to identify and to correct (PALLONI et al. 2014). For example, Preston et al. (1996) matched individuals in death registry in the 1980's with the same individuals in early 20th century censuses. In Brazil, given the availability of data, as well as the lack of the necessary information in the databases, it would be impossible to establish a similar exercise. However, several authors wrote about the possible impacts of age misreporting in mortality estimates and that literature may help understand if the observed mortality outcomes could be influenced by age reporting problems (COALE & KISKER, 1986; COALE & LI, 1991; ELO & PRESTON, 1994; PRESTON et al., 1996, PRESTON et al., 1999; PALLONI et al., 2014).

Based on the state level modal age estimates, presented in Figures 6.5 and 6.6, two FU (for each sex) were selected to take a closer look. We plotted mortality estimates from the highest achieving state along with a more socioeconomic developed one. Rio Grande do Norte (Northeast) presented the highest M for both males and females in 2010. Distrito Federal was the selected comparison

state because it presented the highest non-Northeastern female modal age at death and was among the highest non-Northeastern male modal age.

Figure 6.9 presents female age at death distribution from Rio Grande do Norte (RN) and Distrito Federal (DF). From the start of the distribution (0) until around age 70, the number of deaths registered by age is always higher in Rio Grande do Norte. From age zero to age 5, the difference in the number of deaths is very clear. From that point until around age 30, the dissimilarities are hard to visualize in Figure 6.9. A divergent path was registered in the 30 to 70 interval - with more deaths in Rio Grande do Norte. That difference in the proportion of deaths is also reflected in Q1, the first quarter of the hypothetical cohort dies, approximately, one year earlier in RN, compared to DF.

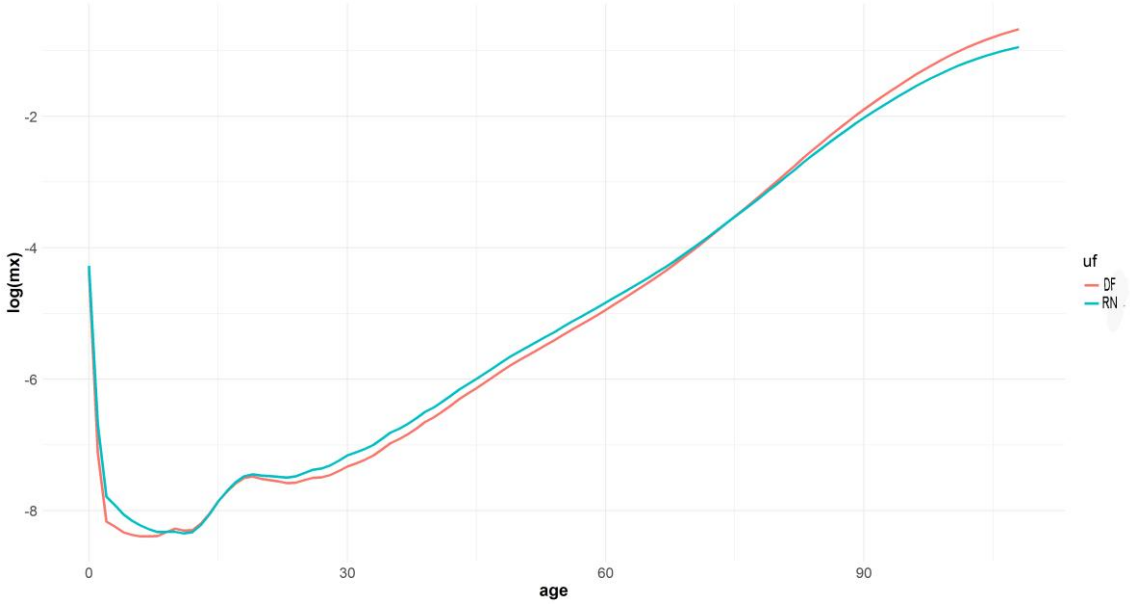
Figure 6.9 – Age at death distribution Rio Grande do Norte (RN) X Distrito Federal (DF), Female, 1980-2010, Brazil, TOPALS



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

Age of death distributions in Figure 6.9 crossover at age 72. It is interesting to observe that RN and DF curves rise at different paces. From age 70 on, Distrito Federal presents a steeper rise in the age at death distribution. That difference in the pace of the curves is also observed after the modal of death, as DF curve also declines at a steeper pace. That divergent dynamic in the number of deaths at older ages is also reflected in Q3. Different from Q1, the age that indicate that 25% is still alive is almost half a year (0.44) higher in RN, after presenting a one year lower Q1. Figure 6.10 shows that, except from a brief interval around age 10, Rio Grande do Norte presents higher mortality from ages 0 to around age 75. After that point, DF mx rises faster than RN.

Figure 6.10 – Age specific mortality rates Rio Grande do Norte (RN) X Distrito Federal (DF), Female, 1980-2010, Brazil, TOPALS



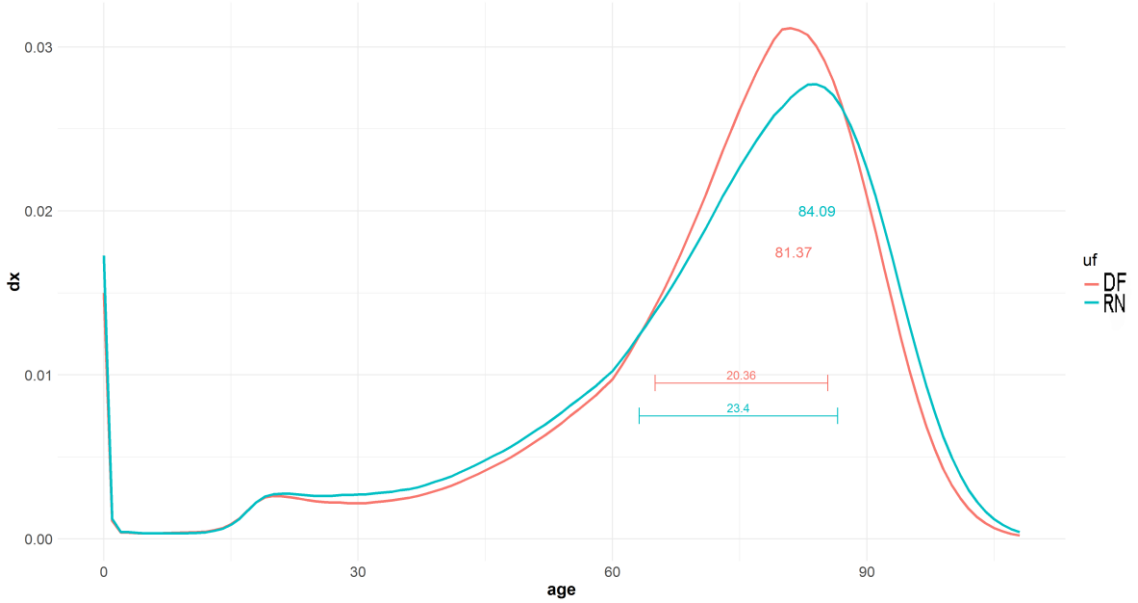
Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

The differences in trajectories are even more representative in Figure 6.11, considering male age at death distributions. After age 0, where the number of deaths is considerably higher in RN, the divergence in Figure 6.11 is only

perceivable after 23. The number of young adult deaths is very similar in both states at age 23 (RN local mode). RN is among the states with rising homicide rates between 2000 and 2010 (WAISENFIZ et al. 2013). DF had in 2001 high homicide rate, and varied little along the last analyzed decade (WAISENFIZ et al. 2013). RN has a higher number of deaths by age from age 23 to age 64, point where the curves crossover.

After age 60 (Figure 6.11), the number of deaths rises faster in DF. As observed for females, it also declines at a steeper pace. The differences in Q1 and Q3 are even more expressive for males. RN lost the first quarter of the hypothetical cohort almost two years earlier (1.88) and, however, reached the remaining 25% point 1.15 year after. That difference in old age mortality represented a modal age 2.72 years higher. Figure 6.12 reinforce what was shown in age at death distribution, except from a brief interval from ages 10 to 14, DF has lower mortality rates until age 66, where ASMR crossover.

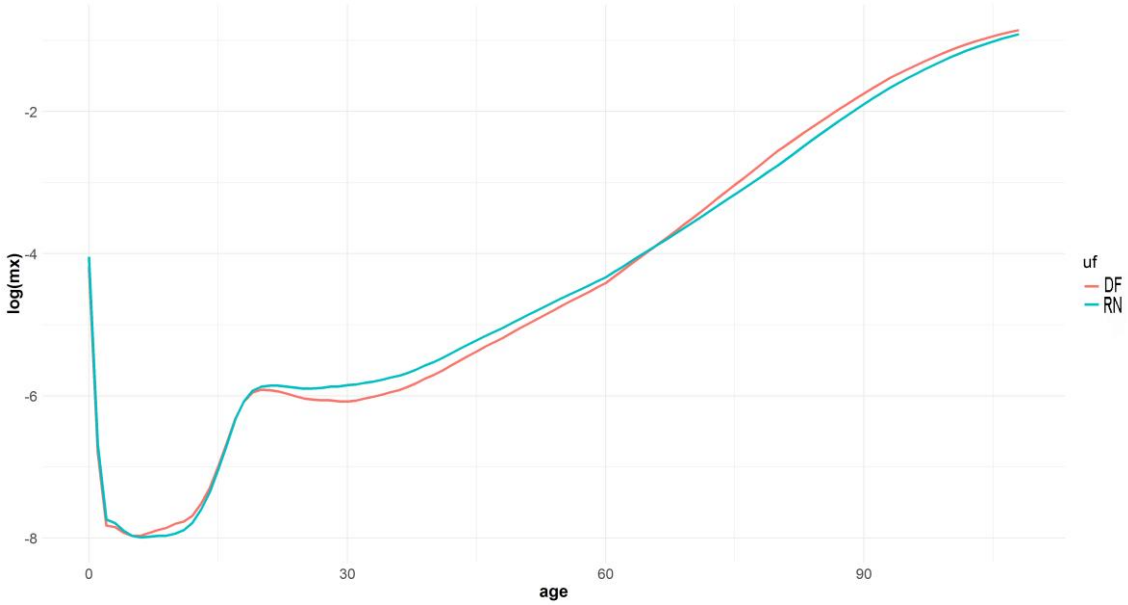
Figure 6.11 – Age specific mortality rates Rio Grande do Norte (RN) X Distrito Federal (DF), Male, 1980-2010, Brazil, TOPALS



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

It is unlike that old age mortality in Rio Grande do Norte, for both males and females, is lower than in Distrito Federal, besides what Figures 6.9-6.12 have shown. It would be necessary an important selection effect to explain such unexpected mortality path change. According to that view, as the more fragile dies, the more robust remaining individuals present lower mortality at older ages (COALE & KISKER, 1986; VAUPEL & YASHIN, 1985; VAUPEL et al., 1979; MANTON et al., 1987). Considering the elements available, it seems more plausible that the observed crossover is a consequence of age misreporting (AGOSTINHO, 2009; HORTA, 2012; PALLONI et al., 2014; GOMES & TURRA, 2009; PRESTON et al., 1996). As Coale and Kisker (1990) argue, harsh conditions at young ages are more likely to lead to higher mortality at older ages, not the opposite.

Figure 6.12 – Age specific mortality rates Rio Grande do Norte (RN) X Distrito Federal (DF), Male, 1980-2010, Brazil



Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

In Brazil, there are important signs of reporting problems and that it has a regional component (HORTA, 2012; AGOSTINHO, 2009; TURRA, 2012; GOMES &

TURRA, 2009). For example, Agostinho (2009) observed age misreporting signs were more severe in both censuses and death registry system in Northeast and North regions. As far as we know, no work have related birth registration problems with age misreporting. However, in Brazil civil registration is still a problem, and it is more severe in North and Northeast regions (IBGE, 2014). In past decades, it was even more expressive (IBGE, 2014). Lack of registration, or late registration could be related to unprecise date of birth information and, consequently, age misreporting.

Crossover could also be related to a varying difference in the death coverage system by age. If, for example, coverage decline faster with age in Rio Grande do Norte than in Distrito Federal, older age mortality would be more underestimated in the first. We applied a unique correction factor for all ages. If coverage varies with age at different levels in each state, it is possible that we are dealing with different levels of underestimation. As already mentioned, it could be one of the reason behind the higher modal age in unexpected states. Exploring the existence of this potential problem is beyond the scope of the dissertation. Although, it opens an interesting research venue to be explored in a near future.

In the present Chapter, we showed clear convergence in the studied mortality measures in the 1980-2010 period at Federal Units level. Modal age, IQR, and median age at death gaps between highest and lowest achieving states diminished very expressively. Considering males, gap in those measures dropped to at least half of what it was in the beginning of the series. Age at death distribution curves got closer to each other, indicating both convergence of mortality between states, as well as improvements in data quality. The timing differences in young mortality increase was demonstrated by the comparison of Southeastern and Northeastern/Northern states. The age at death distribution curves also represented an interesting analytical tool to capture the mortality dynamics in the local mode. The unexpected higher modal age at death in Northeastern states calls for attention when studying old age mortality in Brazil. There are several indication that elderly information in Brazil might present problems, and we showed that those issues might vary internally.

CHAPTER 7 - CONCLUSION

In this dissertation, we used the median age at death, the modal age at death, the interquartile range and the standard deviation above the mode to analyze mortality changes in Brazil between 1980 and 2010. Those measures highlighted important features of mortality dynamic registered in that period. The modal age of death and median age at death rose between 1980 and 2010, indicating mortality decline in younger and older ages. Both age at death variability measures demonstrated that lifespan in less uncertain for males and females. Although, measured by SD (M+), the lifespan variability diminished more modestly for males (1980-2010) and presented a more erratic path.

Changes in MD reassembled characteristics of mortality change registered in Brazil. Very clearly, it reflected the observed rise in young adult mortality between 1980 and 1991 (BORGES, 2017; SIMÕES, 2002; CAMARANO et al., 1997; WAISENFIZ, 2014; CERQUEIRA, 2014). Compared to female, between 1980 and 1991, MD rose very modestly in a period of important infant mortality decline (MERRICK, 1985; FRANÇA et al. 2017a; BORGES, 2017; SIMÕES, 2002). The age at death distribution curves captured male young adult mortality rise between 1980 and 1991. After the appearance of the local mode in those ages, male mortality did not diminish at that part of the curve, mainly due to differences in external cause mortality between states (WAISENFIZ, 2014; CERQUEIRA, 2014; IPEA, 2017; CAMPOS et al., 2015; MALTA et al., 2017). The comparison between São Paulo, Rio de Janeiro, Bahia and Pará helped visualizing how these internal differences behaved between 1980 and 2010 and how the timing of the rise in mortality differed.

The diminution of old age mortality in Brazil, implied by the rise of M, was identified elsewhere, as it was the fact that females mortality declined more expressively (BORGES, 2017; SIMOES, 2002; CORREA& MIRANDA-RIBEIRO, 2017; GONZAGA & SOUZA, 2016; KANSO, 2014). In more recent decades M rose faster, it is plausible to expect a further rise if we follow same path observed in developed countries (CANUDAS-ROMO, 2008; OUELLETE et al., 2012; HORIUCHI et al., 2013). Between 1980 and 2010, modal age increased for males (1.96) and females (5.31) at different levels, increasing sex gap in the

modal age at death. The median age at death also presented a higher increase for females in the same period. Male and female M and MD, however, moved in the same direction, despite the divergence (rising gap). Co-movement in mortality measures between sexes, even in periods with rising gap, was also observed in the countries from the Human Mortality Database (NOYMER & VAN, 2014).

We were also interested in using the set of measures to compare male and females mortality. Male mortality was always higher than females, in every year and age. In both variability measures, women were in better position. In 1980 and 1991, females SD (M+) was higher than males, however that result might be related to the model fit in those years. In that years, especially 1980, the fitted curves presented apparent problems at the end of the distribution. Female advantage in terms of the modal age at death and lifespan variability has been observed in several other countries (CHEUNG et al. 2009; CHEUNG & ROBINE, 2007; HORIUCHI et al. 2013; OUELLETE et al., 2012; GONZAGA et al., 2018; ALVAREZ et al., 2020).

During the first studied decade (1980-1991), period marked by male young adult mortality rise, MD gender gap increased. Male population lost the first quarter of its hypothetical cohort (Q1) more 10 years earlier than females in 1991. The most important causes of death in Brazil (LEITE et al., 2015; FRANÇA et al., 2017b; BRANT et al., 2017) indicate that behavior is the primary reason behind such expressive gap. For each death of a women related to external causes, eight men died (MOURA et al., 2015). Alvarez et al. (2020) argue that external causes are the main impediment for mortality convergence between LAC and developed countries among males. Gonzaga et al. (2018) found that male age at death variability rose in LAC countries (except for Brazil, Argentina, and Colombia), after a declining period, mainly due to violence and traffic accidents. In the countries were it did not increase, the declining path decelerated (GONZAGA et al., 2018).

Age at death variability measures (IQR and SD(+)) did not show signs that the compression of mortality process has been replaced by the shifting of survival curve. During that process, observed in many developed countries, the modal age at death rises at the same time as lifespan variability remains stable.

(CANUDAS-ROMO, 2008; CHEUNG et al., 2009; WILMOTH & HORIUCHI, 1999). In Brazil, both IQR and SD (M+), for both sexes, diminished in the period, indicating that lifespan distribution is compressed in a narrower age interval. That result goes in the same direction observed by other authors (GONZAGA et al., 2018; GONZAGA and SOUZA, 2016; GONZAGA et al., 2009. KANSO, 2014).

The overall decline in dispersion measures for females was more expressive in SD (M+). Between 1980 and 2010, male IQR diminished more than females, however, in 2010, males (22.62) presented higher IQR than females had in 1980 (19.46). Between 1980 and 1991, male IQR diminished from 24.75 to 24.19, very discrete result compared to female decline (1.16). In that same period, Q1 for females rose more than twice as registered in male population. In consequence, female IQR diminished more expressively, even after presenting a higher Q3 rise. Alvarez et al. (2020) argue that people in LAC live shorter lives and experience more uncertainty age at death, in comparison with developed countries, due to a more dispersed amenable disease mortality along the age span. In Denmark, the between 1975 and 1995, female lifespan inequality stagnated along with life expectancy at birth, mainly in consequence of mortality increase related to tobacco consumption (ABURTO et al. 2018).

Variability measures also indicated that states more socioeconomically unequal had higher variability in the age at death. The five highest IQR in 2010 were observed in Northeast (4) and North (1) states, the poorer and the more unequal regions (MEDEIROS et al., 2015; IBGE, 2018). Edwards and Tuljapurkar (2005) in the USA observed that those in worst socioeconomic condition presented higher lifespan variability and shorter lifespan. Firebaugh et al. (2014) found that 87% of the difference in the variance in the age at death among blacks and whites in the USA was explained by differences in the age at death for those dying for the same cause. According to van Haalte et al. (2018) inequality of the length of life is the most fundamental of all inequalities, since all other kinds of inequality are conditional on being alive. Variability in the moment of death influences important decisions in life cycle such as schooling, working, savings and retirement (LEE, 2003; OPPEN & VAUPEL, 2002; TULJAPURKAR, 2010).

We were also interested in analyzing mortality differentials between geographic units. At Federal Units level, results showed that mortality converged in the analyzed period. Gap between the lowest and highest states median age at death (males) diminished expressively between 1980 (14.68) and 2010 (4.45). Modal age at death gap between the highest and lowest achieving states declined from 10.43 to 4.83 in the same period (males). IQR gap also diminished, for both sexes. The convergence between age at death distribution curves was very impressive as well, the shape of the distributions became more similar overtime and they also became more close to each other. At state level, female at death distribution differences as are less remarkable, especially at young adult ages.

Mortality analysis based on the age at death distribution offered a rich analytical perspective at geographic units level, since it captures intrinsic features of mortality change. Overall, male dx curves got less heterogeneous from 1980 to 2010, except from young adult ages. It was also possible to observe that in some states young adult mortality rose the most between 1980 and 1991 and in others after 2000. In Northeastern and Northern states, young adult mortality rise was more concentrated in the 2000-2010 period. In that same decade, states from those regions registered expressive homicide and traffic accidents rates rise (MORAIS NETO, 2012; WAISENFIZ et al., 2013).

Another important aim in the present dissertation was the comparison and discussion about the resulting differences from the applied smoothing methods. We found that overtime estimates become more similar, except when using Log-Quad. For both males and females in 2010, it is hard to identify the differences between Beers, TOPALS and, in a lesser extent, the raw curve. From that last result, it is perceivable that data quality improved in the analyzed period. However, even in 2010, the difference between TOPALS and raw modal age was higher than one year, despite the close fit the model presented, highlighting the importance of the smoothed curve in modal age estimation. Beers presented a satisfactory fit; however, generally, the estimated curves (at the top of the distribution) were flatter and leaned to the right. It also had the same kind of difficulty capturing the pace of the rise in the local mode for young adult males. Probably, it could not capture that well fast rising risk of death. Additionally, more

generally at the end of the distribution, while interpolating binned data, some ages resulted in negative values and, consequently, in a disturbed curve.

For both sexes, in every year considered, estimated Log-Quad modal age was lower and results were considerably different from TOPALS and the others. LQ coefficients are based on life tables from countries that may present a different mortality profile. Probably, in most datasets, old age mortality was considerably higher than the observed in Brazil for the same level of 5q0 and 45q15 - input parameters in to model estimation. After the point where age at death distribution starts to increase, LQ curves rises earlier, faster, and declines at a steeper pace than the other estimates. Brazilian data has age reporting problems in both censuses and vital statistics systems that, probably, are related to the observed differences in the slopes and, in consequence, the modal age at death (AGOSTINHO, 2009; TURRA, 2012; HORTA, 2012; GOMES & TURRA, 2009). Age misreporting in higher ages usually underestimates mortality, independently of the direction of the bias (PRESTON et al., 1999) and affect mortality more severely at older ages (HORTA, 2012; COALE & KISKER, 1991; ORTEGA & GARCIA, 1986; PRESTON et al., 1996).

The magnitude of the impacts related to age misreporting is hard to identify and to correct (PRESTON et al., 1996; PRESTON et al., 1999; PALLONI et al., 2016). Another potential cause of the underestimation of mortality at older ages, that could explain the higher modal age in TOPALS, relates to the unique correction factor applied in the number of deaths. There are signs that the coverage of deaths is worst in younger and older ages, in part related to sampling problems (MURRAY et al., 2010). Queiroz and Sawyer (2012) indicate that death coverage in Brazil worsens after age 60. Therefore, if death coverage declines with age, using a unique factor might underestimate mortality at older ages. The last age from the original dataset considered in LQ is 60, which means that, potentially, it would be less affected by a possible old age mortality underestimation related to underreporting of deaths

Differences between LQ and TP were higher for males, especially at young adult ages. LQ was not able to capture the excessive mortality for males at that part of the distribution. Compared to all other estimates, between age 15 and 50,

approximately, LQ age at death distribution curve presented a flatter path at a lower level. LQ coefficients are based on few non-European countries, the model could not capture the excess mortality observed in young adult ages. Sharrow et al. (2014) observed a similar difficulty in Log-Quad to fit HIV mortality hump in Lesotho. Another problem with LQ estimates, in 1980, LQ dx curve presented higher level in most parts of the age distribution. In that case, high child mortality in 1980 influenced LQ estimates for all other ages. The probability of dying between birth and exact age 5 (5q0) is the most important parameter in LQ estimation, the model can be fitted only based on that parameter. Given the high infant mortality observed in Brazil in 1980 (FRANÇA et al. 2017a; MERRICK, 1985; BARRETO et al. 2011; SCHRAMM et al., 2004; SIMÕES, 2002), the model, probably, overestimated mortality in other ages.

State level modal age at death presented an unexpected result as the highest achieving M were more frequently observed at the Northeast region. We would expect the highest modal age at death in more develop states (MONTEIRO NETO, 2014; PAMPEL et al., 2005; CUTLER et al. 2006; LANTZ et al., 1998; EDWARDS & TULJAPURKAR, 2005). There were signs of reporting problems in the concentration of death at age 30, abrupt slope changing at age 60 and concentration of deaths at age 80 (females). They were more frequently observed in Northeast and North states in 1980 and 1991. States from those regions present more reporting problems and poorer death system coverage (HORTA, 2012; AGOSTINHO, 2009; QUEIROZ et al., 2017). It is possible, however, that that these problems are also related to the ages where the knots were positioned in TOPALS estimation.

Comparatively, states that had higher mortality all along the age distribution presented lower old age mortality and, consequently, a higher modal age at death. Preston et al. (1996) demonstrated that mortality crossover between blacks and whites in the USA was consequence of age misreporting in the first group. Rio Grande do Norte and Distrito Federal mortality crossover could be a consequence of some sort of selection effect, we have no means to exclude completely that possibility. However, given regional differences in reporting quality identified in the literature (AGOSTINHO, 2009; HORTA, 2012; TURRA, 2012), it is more likely that we are dealing with that same kind of problem.

Another approach that could, at least partially, explain the observed crossover relates to the decline of death registry system coverage with age (QUEIROZ & SAWYER, 2012). If in one state quality does not vary with age or vary little, comparatively that state would be less affected to potential mortality underestimation at older ages. In that sense, partially, mortality crossover would be a consequence of varying levels of undercounting of death in old age between states.

7.1 Research agenda

Our results shed lights on some important questions, but also point towards some future research venues. At geographic unit level, mortality presented interesting dynamics and it would be very enriching to understand how sex differentials evolved with time at state level. There were timing differences in young adult mortality rise between states from the Southeast and Northeast/North regions (BORGES, 2017; SIMÕES; IPEA, 2017; WAISENFILZ, 2013), the highest ASMR sex gap was observed at those ages in studied years. Other causes of death presented different rhythm of decline at state level as well, and it also varied between males and females (SASTRY, 1997; DUARTE et al., 2002; FRANÇA et al., 2017a; (MARTINS-MELO et al., 2018; BRANT et al., 2017). . It is likely that regional pattern of convergence and divergence in sex mortality differ, among other things, with the moment of the onset of mortality transition and with socioeconomic indicators. A similar approach as the adopted in the present dissertation could help understand the sex gap at geographic level.

An additional potential venue to be explored after this dissertation is to use the age at death distribution framework, and the set of mortality measures, to look at specific causes of death. The pattern of changes in age at death distribution is influenced by specifics causes of death, it would be interesting to explore to what extent different causes of the influence the timing of death (ALVAREZ et al., 2020; FIREBAUGH et al., 2018). In that case, it would be interesting the select more recent years as data problems and differences in quality are even more important when causes of death are considered.

Another promising research venue would be to investigated further death coverage differences at older ages. Exploring that issue in deep would substantially contribute to old mortality analysis, as mortality has been declining at older ages at a great extent. However, old age mortality estimates is heavily influenced by reporting and coverage problems. One initial exploration into that problem would be to compare if death coverage varies with age at different levels between geographic units. If confirmed, that could, at least partially, explain mortality crossover between states and the unexpected higher modal age at death registered in Northeastern states. At the same time, indirect techniques would help identify the extent of age misreporting at different geographic units level, as other authors have applied (AGOSTINHO, 2009; GOMES & TURRA, 2009; COALE & KISKER, 1990; ELO & PRESTON, 1994). Trying to differentiate, if possible, the size and impact of those two potential problems would broadly contribute of the old age mortality literature.

CHAPTER 8 - BIBLIOGRAPHICAL REFERENCES

Abreu, D.M.X.; César, C.C.; França, E.B. (2009). Gender differences in avoidable mortality in Brazil (1983-2005). Cad. Saúde Pública, 25(12):2672-2682.

Aburto, J.M.; van Raalte, A.A. (2018). Lifespan dispersion in times of life expectancy fluctuation: the case of Central and Eastern Europe. Demography, 55:2071–2096. https://doi.org/10.1007/s13524-018-0729-9.

Agostinho, C. S. Estudo sobre a mortalidade adulta, para Brasil entre 1980 e 2000 e Unidades da Federação em 2000: uma aplicação dos métodos de distribuição de mortes. Tese (Doutorado). – Universidade Federal de Minas Gerais. Belo Horizonte.

Albuquerque, M.V; Viana, A.L.A.; Lima, L.D.; Ferreira, M.P.; Fusaro, E.R.; Iozzi, F.L. (2017). Regional health inequalities: changes observed in Brazil from 2000-2016. Ciência & Saúde Coletiva, 22(4):1055-1064. DOI: 10.1590/1413-81232017224.26862016.

Alvarez, J.A; Aburto, J.M.; Canunda-Romo, V. (2020). Latin American convergence and divergence towards the mortality profiles of developed countries, Population Studies, 74:1, 75-92, DOI: 10.1080/00324728.2019.1614651

Arriaga, E. E. & Davis, K. (1969). The pattern of mortality change in Latin America. Demography, 6 (3): 223-242.

Barbi, E.; Lagona, F.; Marsili, M.; Vaupel, J.W.; Wachter, K.W.(2018). The plateau of human mortality: Demography of longevity pioneers. Science 360, 1459–1461.

Barreto ML, Teixeira MG, Bastos FI, Ximenes RAA, Barata RB, Rodrigues LC (2011). Successes and failures in the control of infectious diseases in Brazil: social and environmental context, policies, interventions, and research needs. The Lancet, 377, pp. 1877-1889.

Barreto, S. M.; Miranda, J. J.; Figueroa, J.P.; Schmidt, M.I.; Munoz, S.; Kuri-Morales, P.P; Silva-Jr, J.B. (2012). Epidemiology in Latin America and the Caribbean: current situation and challenges, International Journal of Epidemiology, Volume 41, Issue 2, 1 April 2012, Pages 557–571, https://doi.org/10.1093/ije/dys017.

Baudish, A. (2011). The pace and shape of ageing. Methods in Ecology and Evolution, 2, 375–382. doi: 10.1111/j.2041-210X.2010.00087.x

Baudish, A.; Vaupel, J.W. (2012). Getting to the Root of Aging. Science 338 (6107), 618-619. DOI: 10.1126/science.1226467.

Barufi et al. (2012). Infant mortality in Brazil, 1980-2000: A spatial panel data analysis BMC Public Health, 12:181.

Béltran-Sanches, H.; Razak, F.; Subramanian, S.V. (2014). Going beyond the disability-based morbidity definition in the compression of morbidity framework. Glob Health Action, 7: 24766. http://dx.doi.org/10.3402/gha.v7.24766.

Béltran-Sanches, H.; Andrade, F.C.D. (2013). Educational and Sex Differentials in Life Expectancies and Disability-Free Life Expectancies in São Paulo, Brazil, and Urban Areas in Mexico. J Aging Health, 25(5). doi:10.1177/0898264313491425.

Bergeron-Boucher, M.P; Ebeling, M.; Canudas-Romo,V. (2015). Decomposing changes in life expectancy. Demographic Research: Volume 33, Article 14

Bongaarts, J. (2005). Long-range trends in adult mortality: Models and projection methods. Demography, 42(1):23–49.

Bongaarts, J. (2009). Trends in senescent life expectancy. Population Studies, 63 (3), pp. 203-213. DOI: 10.1080/00324720903165456.

Booth, H.; Gerland, P.(2015). Demographic Techniques: Data Adjustment and Correction. International Encyclopedia of the Social & Behavioral Sciences, 2nd edition, Volume 6. http://dx.doi.org/10.1016/B978-0-08-097086-8.31011-X.

Borges, G. M. (2017). Health transition in Brazil: regional variations and divergence/convergence in mortality. Cadernos de Saúde Pública, 33(8), 2017.

Borges, G. M. (2018). Consistent population estimates: an application to Brazil. (2018). Dissertation. University of California, Berkeley.

Brant, L.C.C; Nascimento, B.R.; Passos, V.M.A.; Duncan, B.B.; Bensenhõr, I.J.M.; Malta, D.C.; Souza, M.F.M; Ishtani, L.H.; França, E.; Oliveira, M.S.; Mooney, M.; Naghavi, M.; Roth, G.; Ribeiro, A.L.P. (2017). Variations and particularities in cardiovascular disease mortality in Brazil and Brazilian states in 1990 and 2015: estimates from the Global Burden of Disease. Rev Bras Epidemiol, 20 (SUPPL 1), pp. 116-128. DOI: 10.1590/1980-5497201700050010.

Brown, N. J. L.; Albers, C. J.; Ritchie, S.J. (2017). Contesting the evidence for limited human lifespan. Nature 546. http://dx.doi.org/10.1038/nature22784.

Burguer, O.; Baudisch, A.; Vaupel, J.W. (2012). Human mortality improvement in evolutionary context. PNAS, 109 (44) 18210-18214.

Cai, L.; Lubitz, J. (2007). Was There Compression of Disability for Older Americans From 1992 to 2003?. Demography, 44 (3), pp. 479-495. https://doi.org/10.1353/dem.2007.0022.

Camargos, M.C.S.; Gonzaga, M.R. (2015). Viver mais e melhor? Estimativas de expectative de vida saudável para a população brasileira. Cad. Saúde Pública, 31(7):1460-1472. http://dx.doi.org/10.1590/0102-311X00128914.

Canudas-Romo, V. and Wilmoth, J. R. (2007). Record measures of longevity. Presentation at the Population Association of America 2007 meeting held in New York.

Canudas-Romo, V. (2008). The modal age at death and the shifting mortality hypothesis. Demographic Research 19(30): 1179 1204.doi:10.4054/DemRes.2008.19.30.

Canudas-Romo, V. (2010). Three measures of longevity: Time trends and record values. Demography 47(2): 299–312. doi:10.1353/dem.0.0098.

Canudas-Romo, V.; Saikia, N.; Diamond-Smith, N. (2015). The contribution of age-specific mortality towards male and female life expectancy differentials in India and selected States, 1970-2013. Asia-Pacific Population Journal, 30(2), pp.1-20.

Camarano, A.A.; Beltrão, K.I.; Araújo, H.E.; Pinto, M.S. (1997). Transformações no Padrão Etário da Mortalidade Brasileira em 1979—1994 e o seu Impacto na Força de Trabalho. IPEA. Texto Para Discussão, N° 512.

Campos, M.R.; von Doellinger, V.R.; Mendes, L.V.P.; Costa, M.F.S.; Pimentel, T.G.; Schramm, J.M.A. (2015). Morbidity and mortality associated with injuries: results of the Global Burden of Disease study in Brazil, 2008. Cad. Saúde Pública, Rio de Janeiro, 31(1):1-17. http://dx.doi.org/10.1590/0102-311X00191113.

Carey (2003). Life Span: A Conceptual Overview. Population and Development Review, Vol. 29, Supplement: Life Span: Evolutionary, Ecological, and Demographic Perspectives, pp.1-18.

Carvalho, J.A.M. (1974). Regional Trends in Fertility and Mortality in Brazil. Population Studies, 28 (3), pp. 401-421.

Carvalho, J.A.M.; Wood, C.H. (1978). Mortality, Income Distribution, and Rural-Urban Residence in Brazil. Population and Development Review, 4 (3), pp. 405-420.

Cerqueira, D.R.C. (2014). Causas e consequências do crime no Brasil. Tese (Doutorado). PUC-Rio. Rio de Janeiro.

Chandran,A.; Sousa , T.R.V.; Guo, Y; Bishai, D. ; Pechansky. F. 2012). Road Traffic Deaths in Brazil: Rising Trends in Pedestrian and Motorcycle Occupant Deaths, Traffic Injury Prevention, 13(sup1), pp.11-16, DOI: 10.1080/15389588.2011.633289.

Cheung, S. L. K.; Robine, J. M. (2007). Increase in common longevity and the compression of mortality: The case of Japan. Population Studies, v. 61, p. 85-97.

Cheung SLK, Robine JM, Paccaud F, Marazzi A. (2009). Dissecting the compression of mortality in Switzerland, 1876–2005. DemographicResearch. 21(19):569–598.

Crimmins, E.M.; Béltran-Sanches, H. (2010). Mortality and morbidity trends: is there compression of morbidity? Journal of Gerontology: Social Sciences, 66B(1), 75–86, doi:10.1093/geronb/gbq088.

Crimmins, E.M.; Kim, J.K.; Solé-Auró, A. (2010) Gender differences in health: results from Share, Elsa and HRS. European Journal of Public Health, Vol. 21, No. 1, 81–91.

Coale, A.J.; Kisker, E. (1990). Decfects in Data on old-Age Mortality in the United States: New Procedures for Calculating Mortality Schedules and Life Tables at highest Ages. Asia and Pacific Population Forum, 4(1).

Coale, A.J.; Li, S. (1991). The Effect of Age Misreporting in China on the Calculation of Mortality Rates at Very High Ages. Demography, 28(2), pp. 294-301.

Corrêa, E.R.P; Miranda-Ribeiro, A. (2017). Ganhos em expectativa de vida aonascer no Brasil nos anos 2000: impacto das variações da mortalidade por idade e causas de morte. Ciência & Saúde Coletiva, 22(3):1007-1017.

Couzin-Frankel, J. (2011). A Pitched Battle Over Life Span. Science 333 (6042), 549-550. DOI: 10.1126/science.333.6042.549.

Christensen, K.; Vaupel, J.W. (1996). Determinants of longevity: genetic, environmental and medical factors. Journal of Internal Medicine, 240: 333-341.

Cullen, M.R.; Baiocchi, M.; Eggleston, M.; Loftus, P; Fuchs, V. (2015). The Weaker Sex? Vulnerable Men, Resilient Women, and Variations in Sex Differences In Mortality Since 1900. Nber Working Paper Series. Working Paper 21114, http://www.nber.org/papers/w21114.

Cutler, D. Deaton, A., Lleras-Muney, A., (2006). The Determinants of Mortality. Journal of Economic Perspectives, American Economic Association, vol.20(3), pages 97-120, Summer.

Daniel, E.; Germiniani, H.; Nazareno, E.R.; Braga, S.V.; Winker, A.M.; Cunha, C.L.P. (2005). Mortality Trend due to Ischemic Heart Diseases in the City of Curitiba – Brazil, from 1980 to 1998. Arquivos Brasileiros de Cardiologia, 85(2), pp.100-104.

de Beer, J.; Bardoutsos, A.; Janssen, F. (2017). Maximum human lifespan may increase to 125 years. Nature 546, http://dx.doi.org/10.1038/nature22792.

Deeg, D.J.H.; Van Vliet, M.J.G.; Kardaun, J.W.P.F; Huisman, M. (2013). Understanding the mortality decline at older ages. Improved life course or improved present period? Annual Review of Gerontology and Geriatrics, v.33, n.1, p.261-291.

Del Popolo, F. (2000). Los problemas en la declaración de la edad de la población adulta mayor en los censos. Población y desarrollo, 8, Cepal.

Dong, X., Milholland, B. &Vijg, J. (2016). Evidence for a limit to human lifespan. Nature 538, 257–259.

Drummond Jr, M.; Barros, M.B.A. (1999). Desigualdades socioespaciais na mortalidade do adulto no Município de São Paulo. Rev. Bras. Epidemiol. 2(1/2), pp. 34-49.

Duarte, Elisabeth Carmen et al. Expectativa de vida ao nascer e mortalidade no Brasil em 1999: análise exploratória dos diferenciais regionais (2002). RevPanamSaludPublica, v. 12, n. 6, p. 436-44.

Dyrting, S. (2017). Smoothing Migration Intensities With Penalised TOPALS. Working paper. DOI: 10.13140/RG.2.2.18841.11363.

Edwards, R. D.; Tuljapurkar, S. (2005). Inequality in life spans and a new perspective on mortality convergence across industrialized coutries. Populationand Development Review, New York, v. 31, n. 4, p. 645-674.

Elo, I.T.; Preston, S.H. (1994). Estimating African-American Mortality from Inaccurate Data. Demography, 31(3), pp.427-458.

Ezzati M, Friedman AB, Kulkarni SC, Murray CJL (2008) Correction: The Reversal of Fortunes: Trends in County Mortality and Cross-County Mortality Disparities in the United States. PLOS Medicine 5(5): e119.

França, E.B.; Lansky, S. (2008). Mortalidade Infantil Neonatal no Brasil: Situação, Tendências e Perspectivas. Informe de Situação e Tendências: Demografia e Saúde, RIPSA (Textos de Apoio, Texto 3).

França, E.B; Lansky, S.; Rego, MAS; Malta, DC; França, JS; Teixeira, R; Porto, D; Almeida, MF; Souza, MFM; Szwarcwald, CL; Mooney, M; Naghavi, M; Vasconcelos, AMN (2017a). Leading causes of child mortality in Brazil, in 1990 and 2015: estimates from the Global Burden of Disease study. Rev Bras Epidemiol, 20 (SUPPL 1), pp. 46-60.

França, Elisabeth B., Valéria Maria de Azeredo Passos, Deborah Carvalho Malta, Bruce B. Duncan, Antonio Luiz P. Ribeiro, Mark DC Guimarães, Daisy MX Abreu et al. (2017b). "Cause-specific mortality for 249 causes in Brazil and states during 1990–2015: a systematic analysis for the global burden of disease study 2015." Population health metrics 15, no. 1: 39.

Fries, J.F. (1980). Aging, natural death, and the compression of morbidity. New England Journal of Medicine 303:130–35.

Fenelon, Andrew. Geographic divergence in mortality in the United States. Population and development review, v. 39, n. 4, p. 611-634, 2013.

Frenk, J.; Bobadilla, J.L.; Sepúlveda, J.; Cervantes, M.L. (1989). Health transition in middle-income countries: new challenges for health care. Health Policy and Planning; 4(1): 29-39.

Frenk, J.; Frejka, T.; Bobadilla, J.L.; Stern, C.; Lozano, R.; Sepúlveda, J.; José, M. (1991). La Transición Epidemiológica en América Latina. Boletín de la Oficina Sanitaria Panamericana. 111(6).

Glei, D.; Horiuchi, S. (2007). The narrowing sex differential in life expectancy in high-income populations: effects of differences in the age pattern of mortality. Population Studies. 61(2): 141-159

Gomes, M.M.F.; Turra, C.M. (2009). The number of centenarians in Brazil: Indirect estimates based on death certificates. Demographic Research, 20(20), pp. 495-502. DOI: 10.4054/DemRes.2009.20.20.

Gonzaga, M. R. (2008). Compressão da mortalidade: entendendo a variabilidade da idade à mortenapopulação do Estado de São Paulo, 1980-2005. Dissertação (Mestrado) – Universidade Federal de Minas Gerais. Belo Horizonte.

Gonzaga, M. R.; Queiroz, B.L.; Machado, C.L. (2009). Compression of mortality: a study on the variability of age at death in the State of São Paulo, Brazil, 1980-2005. Cadernos de saúde publica, 25, no. 7 (2009): 1475-1485.

Gonzaga, M. R.;,Schmertmann, C.P. (2016). Estimating age-and sex-specific mortality rates for small areas with TOPALS regression: an application to Brazil in 2010." Revista Brasileira de Estudos de População 33, no. 3 (2016): 629-652.

Gonzaga, M.R. ; Costa, J. V. (2016). Compressão da Mortalidade tendências e diferenciais regionais na variabilidade da idade à morte no Brasil 1980-2010. In: VII Congreso de la Asociación Latinoamericana de Población e XX Encontro Nacional de Estudos Populacionais, 2016, Foz do Iguaçu-PR. Anais do VII Congreso de la Asociación Latinoamericana de Población e XX Encontro Nacional de Estudos Populacionais, 2016.

Gonzaga, M. R.; Queiroz, B.L.; Lima, E. (2018). Compression of Mortality: The Evolution in the Variability in the Age of Death in Latin America. Relap, 23, pp. 9-35 http://dx.doi.org/10.31406/n23a2.

Grey, A.D.N.J.; Ames, B.N.; Andersen, J.K.; Bartke, A.; Campisi, J.; Heward, C.B.; McCarter, R.J.M., Stock, G. (2002). Time to Talk SENS: Critiquing the Immutability of Human Aging. Ann. N.Y. Acad. Sci. 959: 452–462.

Gruenberg, E.M. (1977). The Failures of Success. The Milbank Memorial Fund Quarterly, 55(1), pp. 3–24.

Hill, M.E; Preston, S.H.; Rosenwaike, I. (2000). Age Reporting Among White Americans Aged 85+: Results Of A Record Linkage Study. Demography, 37 (2), pp. 175–186.

Hill, K. (2017). Analytical Methods to Evaluate the Completeness and Quality of Death Registration: Current State of Knowledge. Tech. rep. Technical Paper No. 2017/2. New York: United Nations Department of Economic and Social Affairs Population Division.

Hill, K.; You, D.; Choi, Y. (2009). Death Distribution Methods for Estimating Adult Mortality: sensitivity analysis with simulated data errors. Demographic Research, vol. 21 (9), pps 235-254 .

Horiuchi, S., Ouellette, N., Cheung, S.L.K., and Robine, J.M. (2013). Modal age at death: lifespan indicator in the era of longevity extension. Vienna Yearbook of Population Research 11: 37–69.

Horta, C.J.G. (2012). Idade Declarada nos Censos Demográficos e a Qualidade da Informação. Trabalho apresentado no XVIII Encontro Nacional de Estudos Populacionais, ABEP, Águas de Lindóia/SP – Brasil.

Hu, S.; Yu, C. (2014). The Application of Model Life Table Systems in China: Assessment of System Bias and Error. Int. J. Environ. Res. Public Health, 11, pp. 12514-12531. doi:10.3390/ijerph111212514.

Hughes, B.G.; Hekimi, S. (2017). Many possible maximum lifespan trajectories. Nature, 546, http://dx.doi.org/10.1038/nature22786.

IBGE (2000). Estatísticas do Registro Civil. Volume 27. Instituto Brasileiro de Geografia e Estatística.

IBGE (2010). Observações sobre a evolução da mortalidade no Brasil: o passado, o presente e perspectivas. Rio de Janeiro, Instituto Brasileiro de Geografia e Estatística.

IBGE (2014). Estatísticas do Registro Civil. Volume 41. Instituto Brasileiro de Geografia e Estatística.

IBGE (2017). Tábua completa de mortalidade para o Brasil – 2016: Breve análise da evolução da mortalidade no Brasil – Instituto Brasileiro de Geografia e Estatística.

IBGE (2018). Síntese de Indicadores Sociais: Uma análise das condições de vida da população brasileira 2018. Estudos e Pesquisa: Informação Demográfica, N° 39.

Jubithana, A.I.F. (2018). Mortality due to external causes of death in Suriname, 2004-2012. Trabalho apresentado no XXI Encontro Nacional de Estudos Populacionais, ABEP, Poços de Caldas/MG – Brasil.

IPEA (2017). Atlas da Violência 2017. Instituto de Pesquisa Econômica Aplicada Disponível em: http://ipea.gov.br/portal/images/170609_atlas_da_violencia_2017.pdf

Kannisto, V., J. Lauritsen, A. R. Thatcher, and J. W. Vaupel (1994). Reductions in mortality at advanced ages: Several decades of evidence from 27 countries. Population and Development Review 20, 793–810.

Kannisto, V. (2000). Measuring the compression of mortality. Demographic research, v. 3, n. 6.

Kannisto, V. (2001). Mode and Dispersion of the Length of Life. Population: An English Selection 13:159-71.

Keilman, N; Pham, DQ; Syse, A. (2017). Period and cohort life expectancy, mortality compression, and age at death distribution. Paper prepared for presentation at the 4th Human Mortality Database Symposium, Berlin, 22-23 May 2017.

Kramer, The rising pandemic of mental disorders and associated chronic diseases and disabilities. Acta Psychiatrica Scandinavica, 62(S285), pp.382-397. https://doi.org/10.1111/j.1600-0447.1980.tb07714.x.

Lantz, P.M; House, J.S; Lepkowsi, J.M; Williams, D.R.; Mero, R.P; Chen, J. (1998). Socioeconomic factors, health behavior, and mortality: Results from a nationally representative prospective study of US adults. JAMA, 279 (21): 1703-08.

Laurenti, R.; Mello-Jorge, M.H.P.. Gotlieb, S.L.D. (2004). A mortalidade materna nas capitais brasileiras: algumas características e estimativa de um fator de ajuste. Rev. Bras. Epidemiol.;7(4):449-60.

Leite, I.C; Valente, J.G; Schramm, J.M.A; Daumas, R.P.; Rodrigues, R.N.; Santos, M.F.; Oliveira, A.F.; Silva, R.S.; Campos, M.R.; Mota, J.C. (2015). Carga de doença no Brasil e suas regiões, 2008. Cad. Saúde Pública, Rio de Janeiro, 31(7):1551-1564. http://dx.doi.org/10.1590/0102-311X00111614.

Lee, R. (2003). LEE, R. 2003. The Demographic Transition: Three Centuries of Fundamental Change. Journal of Economic Perspectives 17.

Lee, R.; Goldstein, J.R. (2003). Rescaling the Life Cycle: Longevity and Proportionality. Population and Development Review, 29, Supplement: Life Span: Evolutionary,Ecological, and Demographic Perspectives, pp. 183-207.

Lenart, A.; Vaupel, J.W. (2017). Questionable evidence for a limit to human lifespan. Nature, 546, http://dx.doi.org/10.1038/nature22790.

Lima, E.E.C.; Queiroz, B.L. (2014). Evolution of the deaths registry system in Brazil: associations with changes in the mortality profile, under-registration of death counts, and ill-defined causes of death. Cad. Saúde Pública, 30(8):1721-1730. http://dx.doi.org/10.1590/0102-311X00131113.

Luy, M. (2003). Causes of Male Excess Mortality: Insights from Cloistered Populations. Population and Development Review, 29 (4), pp. 647-676.

Luy, M.; Gast, K. (2014). Do women live longer or do men die earlier? Reflections on the causes of sex differences in life expectancy. Gerontology, v.60, p.143–153.

Manton, K.G. (1982). Changing Concepts of Morbidity and Mortality in the Elderly Population. The Milbank Memorial Fund Quarterly. Health and Society, 60 (2), pp. 183-244.

Manton, K.G.; Patrick, C.H.; Johnson, K.J. (1987). Health Differentials between Blacks and Whites: Recent Trends in Mortality and Morbidity. The Milbank

Quarterly, Vol. 65, Supplement 1 (Part 1). Currents of Health Policy: Impacts on Black Americans (1987), pp. 129-199.

Manton, K.G.; Vaupel, J.W. (1995). Survival After The Age of 80 in The United States, Sweden, France, England, And Japan. The New England Journal of Medicine, 333(18), pp. 1232-1235.

Malta, D.C.; Moura, L; Prado, R.R.; Escalante, J.C.; Schmidt, M.I.; Duncan, B.B. (2014). Mortalidade por doenças crônicas não transmissíveis no Brasil e suas regiões, 2000 a 2011. Epidemiol. Serv. Saúde, 23(4):599-608. doi: 10.5123/S1679-49742014000400002.

Malta, D.C.; Minayo, M.C.S.; Filho, A.M.S.; Silva, M.M.A.; Montenegro, M.M.S.; Ladeira, R.M.; Morais-Neto, O.L.; Melo, A.P; Mooney, M.; Naghavi, M. (2017). Mortality and years of life lost by interpersonal violence and self-harm: in Brazil and Brazilian states: analysis of the estimates of the Global Burden of Disease Study, 1990 and 2015. Rev Bras Epidemiol; 20 SUPPL 1: 142-156.

Martins-Melo, F.R; Carneiro, M.; Ramos-Júnior, A.N.; Heukelbach, J.; Ribeiro, A.L.P.; Werneck, G.L. (2018). The burden of Neglected Tropical Diseases in Brazil, 1990-2016: A subnational analysis from the Global Burden of Disease Study 2016. PLoS Negl Trop Dis 12(6): e0006559. https://doi.org/10.1371/journal.pntd.0006559.

Medeiros M, Souza PHGF, Castro FA (2015). The stability of income inequality in Brazil, 2006-2012: an estimate using income tax data and household surveys. Ciência & Saúde Coletiva, 20(4):971-986.

Mello-Jorge, M.H.P.; Gotlieb, S.L.D.; Sobol, M.L.M.; Almeida, M.F.; Latorre, M.R.D.O. (1993). Avaliação do Sistema de Informação Sobre Nascidos Vivos e o Uso de Seus Dados em Epidemiologia e Estatísticas de Saúde. Rev. Saúde Pública, 27 (6 Supl.).

Merrick, T.W. (1985). The Effect of Piped Water on Early Childhood Mortality in Urban Brazil, 1970 To 1976. Demography, 22 (1).

Meslé, F.; Vallin, J. . (2011). Historical Trends in Mortality. In: ROGERS, R.; CRIMMS, E. (eds). International Handbook of Adult Mortality, Springer Science+Business Media B.V.

Minnesota Population Center. Integrated Public Use Microdata Series, International: Version 6.5 [dataset]. Minneapolis: University of Minnesota, 2018. http://doi.org/10.18128/D020.V6.5.

Ministério da Saúde. (2019). Sistema de Informação de Mortalidade (SIM). Datasus. - www.datasus.gov.br

Ministério da Saúde. (2015). Saúde Brasil 2014: Uma análise da situação de saúde. Brasília-DF.

Morais-Neto, O.L.; Montenegro, M.M.S.; Monteiro, R.A.; Siqueira-Júnior, J.B; Silva, M.M.A.; Lima, C.M.; Miranda, L.O.M.; Malta, D.C.; Silva-Júnior, J.B. (2012).

Mortality due to Road Traffic Accidents in Brazil in the last decade: trends and risk clusters. Ciência & Saúde Coletiva, 17(9):2223-2236.

Morais-Neto, O.L; Andrade, A.L; Guimarães, R.A.; Mandacarú, P.M.P.; Tobias, G.C. (2016). Regional disparities in road traffic injuries and their determinants in Brazil, 2013. International Journal for Equity in Health, 15(142). DOI 10.1186/s12939-016-0433-6.

Morteiro-Neto, A. (2014). Desigualdades Regionais no Brasil: Características e Tendências Recentes. IPEA. Boletim Regional, Urbano e Ambiental, 9, pp.67-81.

Moura, Erly Catarina de, Gomes, Romeu, Falcão, Marcia Thereza Couto, Schwarz, Eduardo, Neves, Alice Cristina Medeiros das, & Santos, Wallace. (2015). Desigualdades de gênero na mortalidade por causas externas no Brasil, 2010. Ciência & Saúde Coletiva, 20(3), 779-788.

Murray, C.J.L.; Rajaratnam, J.K.; Marcus, J.; Laakso, T.; Lopez, A.D. (2010). What Can We Conclude from Death Registration? Improved Methods for Evaluating Completeness. PLoS Med 7(4): e1000262. doi:10.1371/journal.pmed.1000262

Nepomuceno, M.R.; Turra, C.M. (2015). Trends in healthy life expectancy among older Brazilian women between 1998 and 2008. Rev Saúde Pública;49:1. DOI:10.1590/S0034-8910.2015049005472.

Nepomuceno, M.R.; Turra, C.M. (2019). The population of centenarians in Brazil: historical estimates from 1900 to 2000. MPIDR Working Paper WP 2019-015, August 2019.

Nikiforov, S.V.; Mamaev, V.B. (1998). The Development of Sex Differences in Cardiovascular Disease Mortality: A Historical Perspective. American Journal of Public Health, 88(9), pp. 1348-1353.

Oeppen, J.; Vaupel, J. W. (2002). Broken limits to life expectancy. Science, v. 296, n. 570, 1029-1031.

Olshansky, S.J. and Ault, A.B. (1986) The Fourth Stage of the Epidemiologic Transition: The Age of Delayed Degenerative Diseases. The Milbank Quarterly, 64, 355-391. http://dx.doi.org/10.2307/3350025.

Olshansky, S.J.; Carnes, B.A.; Cassel, C. (1990). In Search of Methuselah: Estimating the Upper Limits to Human Longevity. Science 250 (4981), 634-640. DOI: 10.1126/science.2237414.

Olshansky, S.J.; Rudberg, M.A.; Carnes, B.A.; Cassel, C.; Brody, J.A. (1991). Trading off Longer Life for Worsening Health: The Expansion of Morbidity Hypothesis. Journal of Aging and Health, 3(2), pp.194-216.

Olshansky, S.J.; Passaro, D.J.; Hershow, R.C.; Layden, J.; Carnes, B.A.; Brody, J.; Hayflick, L. Butler, R.N.; Alisson, D.B.; Ludwig, D.S. (2005). A Potential

Decline in Life Expectancy in the United States in the 21st Century. The New England Journal of Medicine, 352 (11), 1138-1145.

Omram, A. R. (1971). The epidemiologic transition: a theory of the epidemiology of population change. Milbank Memorial Fund Quarterly, 49 (Part 1): 509-538.

Omram, A.R. (1998). The epidemiologic transition theory revisited thirty years later, World Health Statistics Quarterly/Rapport trimestriel de statistiques sanitaires, vol. 51, n° 2-3-4, p. 99-119.

Ortega, A.; Garcia, V. (1985). Estudio Sobre la Mortalidad y Algunas Caracteristicas Socioeconomicas de la Poblacion de la Tercera Edad. Informe Sobre Resultados y Conclusiones De La Prueba Piloto (8 Al 12 De Abril De 1985). San José.

Ouellette, N. and Bourbeau, R. (2011). Changes in the age-at-death distribution in four low mortality countries: A nonparametric approach. Demographic Research 25(19): 595–628. doi:10.4054/DemRes.2011.25.19.

Ouellette, N., C. Camarda, and R. Bourbeau (2012). Regional disparities in Canadian adult and old-age mortality: A comparative study based on smoothed mortality ratio surfaces and age at death distributions. Canadian Studies in Population 39 (3-4), 79-106.

Palloni, A.; Pinto-Aguirre, G.; Pelaez, M. (2002). Demographic and health conditions of aging in Latin America and the Caribbean. International Journal of Epidemiology, 31:762-771.

Palloni, A.; Pinto-Aguirre, G. (2011). Adult Mortality in Latin America and the Carribbean. In: ROGERS, R.; CRIMMINS, E. (eds). International Handbook ofAdult Mortality, Springer Science+Business Media B.V. 2011

Palloni, A., Pinto, G. and Beltrán-Sánchez, H. (2014). Latin American Mortality Database (LAMBdA). [Machine-readable database], Madison: University of Wisconsin, 2014.

Palloni, A., Pinto, G. and Beltrán-Sánchez, H. (2016). Estimation of Life Tables in the Latin American Data Base (LAMBdA): Adjustments for Relative Completeness and Age Misreporting.https://www.un.org/en/development/desa/population/events/pdf/expert/26/notes/Palloni_2016_Est-of-life-in-LAMBdA.pdf .

Pampel, F.C; Krueger, P.M; Denney, J.T. (2010). Socioeconomic Disparities in Health Behaviors. Annu. Rev. Sociol. 36:349–70. 10.1146/annurev.soc.012809.102529.

Parahyba, M.I. (2006). Desigualdades de gênero em saúde entre os idosos no Brasil. Trabalho apresentado no XV Encontro Nacional de Estudos Populacionais, ABEP, realizado em Caxambú- MG – Brasil, de 18 - 22 de Setembro de 2006.

Portela, M.H.R.B. (1989). Sub-registro de nascimentos vivos em localidade do Estado de Piauí, Brasil. Rev. Saúde públ., S. Paulo, 23: 493-501.

Prata, P.R. (1992). A Transição Epidemiológica no Brasil. Cad. Saúde Públ.,8 (2):168-175.

Preston, S.H. Elo. I., Rosenwaike, I., Hill, M. (1996). African-American mortality at older ages: results of a matching study. Demography, v.22, n.2, pp 192-209.

Preston, S.H.; Elo. I.T., Stewart, Q. (1999). Effects of age misreporting on mortality estimates at older ages. Population Studies, Vol. 53, No. 2, pp. 165-177.

Preston, S.H.; Heuveline, P.; Guillot, M. (2001). Demography:Measuring and Modeling Population Processes. Blackwell Publishing.

Preston, SH; Wang, H. (2006). Sex mortality differences in the United States: the role of cohort smoking patterns. Demography, 43(4): 631-646.

Queiroz, B.L.; Sawyer, D.O.T. (2012). O que os dados de mortalidade do Censo de 2010 podem nos dizer? R. bras. Est. Pop., Rio de Janeiro, v. 29, n. 2, p. 225-238.

Queiroz, B.L.; Lima, E.E.C. (2016). National and subnational experience with estimating the extent and trend in completeness of registration of deaths in Brazil. United Nations Expert Group Meeting : "Methodology and lessons learned to evaluate the completeness and quality of vital statistics data from civil registration".

Queiroz, B.L.; Freire, F.H.M.A; Gonzaga, M.R; Lima, E.E.C (2017). Completeness of death-count coverage and adult mortality (45q15) for Brazilian states from 1980 to 2010. Rev Bras Epidemiol. May 2017; 20 SUPPL 1: 21-33.

Rau, R; Soroko, E. ;Jasilionis, D. ; Vaupel, J.W. (2008). Continued Reductions in Mortality at Advanced Ages. Population and Development Review; 34 (4):747–768.

Reis, C.S; Turra, C.M. (2016). Distribuição espacial dos centenários no Brasil: uma análise exploratória da qualidade dos dados dos censos de 2000 e 2010. Revista Espinhaço, 2016, 5 (1): 52-61.

Robine, J.M.; Saito, Y.(2003). Survival beyond Age 100: The Case of Japan. Population and Development Review, Vol. 29, Supplement: Life Span: Evolutionary, Ecological, and Demographic Perspectives, pp. 208-228.

Robine, J.M.; Saito, Y.; Jagger, C. (2009). The relationship between longevity and healthy life expectancy. Quality in Ageing and Older Adults, 10 (2), pp. 5 – 14. http://dx.doi.org/10.1108/14717794200900012.

Rogers, R.G.; Hackenberg, R. (1987). Extending epidemiologic transition theory: A new stage. Social Biology, v.34, n.3-4, p.234-243.

Rootzén, H; Zholud, D (2017). Human life is unlimited – but short. Extremes (2017) 20:713–728. https://doi.org/10.1007/s10687-017-0305-5.

Rosenwaike, I; Preston, S.W. (1984). Age overstatement and Puerto Rican longevity. Human Biology 56 (3): 503-525.

Rozing, M.P.; Kirkwood,T.B.L.;Westendorp, G.J. (2017). Is there evidence for a limit to human lifespan?. Nature, 546, p. E11-E12. doi:10.1038/nature22788.

Sastry, N. (1997). What explains rural-urban differentials in child mortality in brazil? Soc. Sci. Med. Vol. 44, No. 7. pp. 989-1002.

Schimidt, M.I.; Duncan, B.B., Silva, G.A.; Menezes, A.M.; Monteiro, C.A.; Barreto, S.M.; Chor, D.; Menezes, P.R. (2011). Chronic non-communicable diseases in Brazil: burden and current challenges. Lancet. DOI:10.1016/S0140-6736(11)60135-9.

Schmertmann, C.P.; Gonzaga, M.R. (2018). Bayesian Estimation of Age-Specific Mortality and Life Expectancy for Small AreasWith Defective Vital Records. Demography, 55(4), pp.1363-1388. https://doi.org/10.1007/s13524-018-0695-2.

Schramm, J.M.A.; Oliveira, A.F.; Leite, I.C.; Valente, J.G.; Gadelha, A.M.J.; Portela, M.C.; Campos, M.R. (2004). Transição epidemiológica e o estudo de carga de doença no Brasil. Ciência & Saúde Coletiva, 9(4):897-908.

Schünemann, J.; Strulik, H.; Trimborn, T. (2017). The gender gap in mortality: How much is explained by behavior? Journal of Health Economics, 54, pp. 79–90.

Sharrow, D.J.; Clark, S.J.; Raftery, A.E. (2014). Modeling Age-Specific Mortality for Countries with Generalized HIV Epidemics. PLoS ONE 9(5): e96447. doi:10.1371/journal.pone.0096447.

Shkolnikov, V.M.; Andreev, E.E.; Begun, A.Z. (2003). Gini coefficient as a life table function: computation from discrete data, decomposition of differences and empirical examples. Demographic Research , 8(11), pp.305-358.

Siegel, J.C.; Swanson, D.A. (2004). The Methods and Materials of Demography. Second Edition. Elsevier Academic Press.

Signor, D.; Moura, G.V. Mosaico da distribuição de renda no Brasil: uma análise com base em 40 anos de dados da PNAD. XXI Encontro de Economia da Região Sul. Anpec-Sul, Curitiba, 2018.

Silva, B.G.C.; Lima, N.P.; Silva, G.S; Antúnez, S.F.; Seerig, L.M.; Restrepo-Méndez, M.C.; Wehrmeister, F.C. (2016). Maternal mortality in Brazil from 2001 to 2012: time trends and regional differences. Rev Bras Epidemiol; 19(3): 484-493.

Simões CCS (2002). Perfis de saúde e de mortalidade no Brasil: uma análise de seus condicionantes em grupos populacionais específicos. Brasília: Organização Pan-Americana da Saúde/Organização Mundial da Saúde.

Thatcher, A.R; Kannisto, V.; Vaupel, J.W. (1998). The Force of Mortality at Ages 80 to 120. Monographs On Population Aging, Vol.5. Odense University Press.

Tuljapurkar, S.; Li, N.; Boe, C. (2000). A universal pattern of mortality decline in the G7 countries. Nature., 405, p.789-792.

Turra, C.M. (2012). Os limites do corpo: A longevidade em uma perspectiva demográfica. Revista UFMG 19(1): 156–181.

United Nations (2016) – Human Development Report 2016 - United Nations Development Programme (UNDP).

Vallin, J.; Meslé, F. (2016). Highest life expectancies: Which leader after Japan? In Annual Meeting of the Population Association of America, Washington, DC.

van Raalte, A.A.; Caswell, H. (2013). Perturbation Analysis of Indices of Lifespan Variability. Demography 50:1615–1640. DOI 10.1007/s13524-013-0223-3.

Vaupel, J.W.; Manton, K.G.; Stallard, E. (1979). The Impact of Heterogeneity in Individual Frailty on the Dynamics of Mortality. Demography, 16(3), pp. 439-454.

Vaupel, J.W.; Yashin, A.I. (1985). Heterogeneity's Ruses: Some Surprising Effects of Selection on Population Dynamics. The American Statistician, 39(3), pp. 176-185.

Vaupel,J.W. (1997). The remarkable improvements in survival at older ages. Philosophical Transactions of the Royal Society of London: Biological Sciences 352: 1799–1804.

Vaupel,J.W.; Carey, J.R; Christensen, K; Johnson, T.E; Yashin, A.I; Holm, N.V.; Iachine, I.A; Kannisto, V.; Khazaeli, A.A.; Liedo, P.; Longo, V.D.; Zeng, Y.; Manton, K.G; Curtsinger, J.W. (1998). Biodemographic Trajectories of Longevity. Science 280 (5365), 855-860. DOI: 10.1126/science.280.5365.855.

Vaupel, J.W. (2010). Biodemography of human ageing. Nature 464(7288): 536–542. doi:10.1038/nature08984.

Vaupel, J.W.; Zhang, Z.; van Raalte, A. A. (2011). Life expectancy and disparity: an international comparison of life table data. BMJ Open; 1:e000128. doi:10.1136/bmjopen-2011-000128.

Victora, C.G.; Aquino, E.M.L.; Leal, M.C.; Monteiro, C.A.; Barros, F.C; Szwarcwald. (2011). Maternal and child health in Brazil: progress and challenges. Lancet, 377: 1863–76.

Waiselfisz J.J. (2013). Mapa da violência 2013: homicídios e juventude no Brasil. Brasília: Secretaria- Geral da Presidência da República.

WHO (2013). Global status report on road safety 2013: supporting a decade of action. World Health Organization 2013. ISBN 978 92 4 156456 4.

WHO (2015). Trends in maternal mortality 1990-2015. World Health Organization 2015.

Wilmoth, J. R.; Horiuchi S. E.(1999). Rectangularizationrevisited: variability of age at death within human populations. Demography, Chicago, v. 36, n. 4, p. 475- 495, nov.

Wilmoth, J.R. (2000). Demography of longevity: past, present, and future trends. Experimental Gerontology 35 (2000) 1111±1129

Wilmoth, J. R. (2002). Human longevity in historical perspective. In: Paola S. Timiras (ed.), Physiological Basis of Aging and Geriatrics, 3rded., Boca Raton, FL: CRC Press, pp. 11-24.

Wilmoth, JR, Boe C, and Barbieri, M. (2011). In: Eileen M. Crimmins, Samuel H. Preston, and Barney Cohen, Editors; Panel on Understanding Divergent Trends in Longevity in High-Income Countries; National Research Council

Wilmoth, J., Zureick, S., Canudas-Romo, V., Inoue, M., & Sawyer, C. (2012). A flexible two-dimensional mortality model for use in indirect estimation. Population Studies, 66(1), 1-28.

Wisser, O.; Vaupel, J.W. (2014). The sex differential in mortality: A historical comparison of the adult-age pattern of the ratio and the difference. MPIDR Working Paper WP 2014-005.

Wood, C.H.; Carvalho, J.A.M. (1994). Demografia de Desigualdade no Brasil. Rio de Janeiro: IPEA.

Wong., L. R.; Turra, C.M. (2007). O Sub-Registro de Nascimentos no Brasil. Banco Interamericano de Desenvolvimento, ATN/NS-8915-RS.

Wong, R.; Peláez, M.; Palloni, A.; Markides, K. (2006). Survey Data for the Study of Aging in Latin America and the Caribbean. Journal of Aging and Health, 18 (2), pp. 157-179. DOI: 10.1177/0898264305285655.

Wong, R.; Gertz, K.; Michaels-Obregon, A.; Palloni, A.(2011). Burden of Aging in Developing Countries: Disability Transitions in Mexico Compared to the United States. Working Paper.

CHAPTER 9 - ANNEXES

Table 9.1- Mode, median, Interquartile range, Q1, Q3 and standard deviation above the mode, Federal Units, Brazil, females, 1980

UF	Q1	MD	Q3	IQR	SD(M+)	M
Rondônia	59.19	74.67	82.56	23.37	8.13	80.27
Acre	69.36	78.42	84.35	14.99	7.67	80.51
Amazonas	67.84	78.70	85.77	17.94	8.93	80.72
Roraima	63.98	77.91	85.99	22.01	9.80	80.72
Pará	66.63	78.09	85.20	18.56	8.92	80.60
Amapá	66.68	78.82	87.08	20.40	10.45	80.72
Tocantins	67.67	78.17	85.34	17.67	9.82	80.40
Maranhão	74.52	83.39	92.14	17.62	11.79	80.93
Piauí	71.46	81.40	89.15	17.69	10.39	81.65
Ceará	73.56	83.76	92.20	18.64	10.13	85.27
R.G.Norte	71.61	81.53	89.24	17.63	10.47	81.56
Paraíba	65.74	78.79	86.55	20.81	10.01	80.69
Pernambuco	57.66	74.83	82.90	25.24	9.01	80.28
Alagoas	58.99	75.41	83.29	24.30	9.32	80.29
Sergipe	65.07	76.76	83.74	18.67	8.73	80.33
Bahia	67.80	79.62	87.98	20.18	10.86	80.72
Minas Gerais	61.73	74.91	82.67	20.94	9.09	80.14
Espírito Santo	64.63	76.59	83.92	19.29	9.21	80.31
Rio de Janeiro	62.25	75.23	83.11	20.87	8.72	80.24
São Paulo	64.17	76.01	83.48	19.31	8.69	80.28
Paraná	63.14	75.03	82.44	19.30	8.76	80.09
Santa Catarina	67.30	77.00	83.54	16.24	8.41	80.25
R.G.Sul	66.12	76.89	84.05	17.93	8.67	80.36
Mato Grosso do Sul	66.45	77.93	85.46	19.01	9.52	80.55
Mato Grosso	71.03	80.71	88.74	17.71	10.85	80.76
Goiás	64.14	75.87	83.46	19.32	9.51	80.18
Distrito Federal	63.90	76.32	84.60	20.70	9.32	80.50

Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

Table 9.2- Mode, median, Interquartile range, Q1, Q3 and standard deviation above the mode, Federal Units, Brazil, females, 1991

UF	Q1	MD	Q3	IQR	SD(M+)	M
Rondônia	68.06	78.81	86.43	18.37	9.79	80.68
Acre	70.09	79.84	87.48	17.39	10.47	80.60
Amazonas	71.66	81.02	88.52	16.86	10.23	80.96
Roraima	68.59	81.39	90.46	21.87	10.17	84.48
Pará	70.53	80.15	87.39	16.86	9.57	80.94
Amapá	70.05	80.75	87.70	17.65	6.58	85.44
Tocantins	72.74	82.63	90.62	17.88	9.41	84.85
Maranhão	73.04	82.46	89.87	16.83	8.75	84.83
Piauí	74.37	83.37	90.85	16.49	9.20	85.29
Ceará	73.08	82.82	89.89	16.81	7.77	86.30
R.G.Norte	72.86	82.38	89.47	16.61	7.89	85.61
Paraíba	69.95	80.19	87.36	17.42	8.64	83.02
Pernambuco	64.95	77.07	84.85	19.90	8.92	80.62
Alagoas	66.10	77.64	85.41	19.31	9.88	80.46
Sergipe	68.16	78.38	85.27	17.12	8.38	80.72
Bahia	69.36	80.17	88.08	18.72	9.95	81.76
Minas Gerais	66.31	77.60	85.30	18.99	8.95	80.72
Espírito Santo	67.37	78.13	85.52	18.16	9.25	80.60
Rio de Janeiro	64.56	76.55	84.52	19.97	8.47	80.74
São Paulo	67.30	77.97	85.35	18.05	8.67	80.76
Paraná	66.85	77.21	84.48	17.63	9.11	80.36
Santa Catarina	68.86	78.35	85.25	16.39	8.61	80.61
R.G.Sul	68.24	78.34	85.54	17.30	8.72	80.74
Mato Grosso do Sul	67.71	78.86	86.75	19.04	9.64	80.95
Mato Grosso	70.48	80.31	88.07	17.59	10.36	80.86
Goiás	67.04	77.66	85.23	18.19	8.91	80.66
Distrito Federal	66.94	78.19	85.97	19.03	7.80	83.23

Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

Table 9.3 - Mode, median, Interquartile range, Q1, Q3 and standard deviation above the mode, Federal Units, Brazil, females, 2000, TOPALS

UF	Q1	MD	Q3	IQR	SD(M+)	M
Rondônia	69.43	79.61	87.19	17.76	9.82	80.86
Acre	68.21	79.57	87.36	19.15	8.31	83.66
Amazonas	69.93	80.58	88.44	18.51	9.08	83.64
Roraima	66.91	79.89	88.21	21.30	7.65	85.73
Pará	68.57	79.96	87.83	19.26	8.41	84.23
Amapá	69.21	80.14	87.80	18.58	8.21	83.93
Tocantins	68.77	80.38	88.79	20.02	9.22	84.00
Maranhão	69.03	81.64	90.36	21.32	8.86	86.46
Piauí	72.21	82.63	90.39	18.19	8.74	86.09
Ceará	71.15	81.95	89.37	18.23	7.64	86.30
R.G.Norte	71.11	82.19	89.78	18.67	7.61	86.69
Paraíba	70.57	81.47	89.08	18.51	7.74	85.82
Pernambuco	67.42	78.89	86.83	19.41	8.34	83.42
Alagoas	66.44	78.50	86.90	20.46	9.03	83.17
Sergipe	67.45	79.25	87.38	19.93	8.85	83.44
Bahia	69.22	81.15	89.64	20.42	9.03	85.45
Minas Gerais	69.20	79.99	87.84	18.64	9.03	83.37
Espírito Santo	69.15	80.12	87.82	18.67	8.24	84.26
Rio de Janeiro	66.70	78.45	86.62	19.92	8.28	83.47
São Paulo	69.50	79.91	87.28	17.77	7.67	83.88
Paraná	68.94	79.10	86.56	17.62	9.16	81.35
Santa Catarina	69.82	79.49	86.50	16.68	7.77	82.78
R.G.Sul	70.05	80.24	87.68	17.63	8.12	83.81
Mato Grosso do Sul	68.11	79.55	87.68	19.56	8.99	83.48
Mato Grosso	68.72	79.13	86.89	18.17	10.03	80.73
Goiás	68.86	79.30	87.11	18.24	9.77	80.96
Distrito Federal	69.77	80.15	87.98	18.21	9.21	83.27

Source: Brazilian Censuses, 1980, 1991, 2000, 2010; Mortality Information System, Datasus (SIM)

Table 9.4 - Mode, median, Interquartile range, Q1, Q3 and standard deviation above the mode, Federal Units, Brazil, females, 2010, TOPALS

UF	Q1	MD	Q3	IQR	SD(M+)	M
Rondônia	71.64	81.52	89.07	17.43	9.10	84.10
Acre	69.53	80.88	89.43	19.90	9.83	83.76
Amazonas	71.34	81.73	89.42	18.08	8.67	85.21
Roraima	69.82	81.23	89.45	19.62	8.79	85.42
Pará	69.39	80.43	88.32	18.93	8.89	84.09
Amapá	72.41	82.89	91.83	19.42	10.69	83.83
Tocantins	70.25	81.15	88.91	18.66	8.53	85.09
Maranhão	67.35	79.65	88.24	20.89	9.25	84.13
Piauí	71.72	82.24	89.59	17.87	7.65	86.39
Ceará	72.52	82.73	89.87	17.36	7.22	86.82
R.G.Norte	72.80	83.09	90.41	17.60	7.82	87.05
Paraíba	71.49	82.11	89.56	18.06	7.74	86.34
Pernambuco	70.49	81.01	88.73	18.24	8.58	84.56
Alagoas	68.37	79.80	88.06	19.68	8.90	83.83
Sergipe	71.00	81.47	89.03	18.03	8.17	85.37
Bahia	70.57	82.07	90.22	19.65	8.62	86.33
Minas Gerais	71.68	82.03	89.45	17.77	7.75	85.95
Espírito Santo	72.27	82.40	89.73	17.46	8.00	86.15
Rio de Janeiro	69.29	80.40	87.96	18.67	7.22	85.49
São Paulo	72.23	82.13	89.20	16.97	7.48	86.11
Paraná	71.68	81.45	88.66	16.98	8.02	85.05
Santa Catarina	72.36	81.74	88.65	16.29	7.65	85.18
R.G.Sul	72.17	82.08	89.05	16.88	7.22	86.17
Mato Grosso do Sul	71.07	81.40	88.81	17.74	8.02	85.28
Mato Grosso	71.19	81.08	88.58	17.39	8.82	83.72
Goiás	70.55	80.71	88.20	17.65	8.30	83.98
Distrito Federal	73.79	83.24	89.97	16.18	6.89	86.93

Source: Brazilian Censuses. 1980. 1991. 2000. 2010; Mortality Information System. Datasus (SIM)

Table 9.5 - Mode. median. Interquartile range. Q1. Q3 and standard deviation above the mode. Federal Units. Brazil. males. 1980. TOPALS

UF	Q1	MD	Q3	IQR	SD(M+)	M
Rondônia	48.88	69.49	78.59	29.70	7.23	78.00
Acre	58.53	73.23	81.20	22.67	8.15	79.64
Amazonas	52.08	69.29	78.54	26.46	7.94	77.16
Roraima	56.96	74.82	85.92	28.96	11.42	80.56
Pará	56.08	71.96	80.82	24.75	8.47	79.31
Amapá	60.38	74.30	82.59	22.20	8.01	80.16
Tocantins	62.01	74.46	81.94	19.93	8.08	79.98
Maranhão	69.80	81.53	91.20	21.40	12.25	81.22
Piauí	65.51	78.83	88.03	22.52	10.52	81.83
Ceará	65.23	80.16	89.62	24.40	9.65	85.26
R.G.Norte	65.38	78.79	87.14	21.76	9.42	82.34
Paraíba	54.07	74.67	84.09	30.02	9.50	80.55
Pernambuco	44.64	68.70	79.56	34.91	8.70	79.13
Alagoas	44.27	67.08	78.54	34.27	8.85	77.68
Sergipe	56.28	72.57	81.34	25.07	8.31	79.73
Bahia	56.44	73.12	82.64	26.20	9.73	80.09
Minas Gerais	53.30	69.10	78.85	25.55	9.12	76.71
Espírito Santo	53.95	70.06	79.27	25.32	8.46	77.63
Rio de Janeiro	50.69	66.85	77.23	26.53	9.08	74.83
São Paulo	54.28	69.02	78.63	24.35	9.35	76.04
Paraná	55.12	69.60	78.69	23.57	8.88	76.48
Santa Catarina	58.56	71.27	79.85	21.29	8.65	77.33
R.G.Sul	57.19	69.93	78.90	21.70	9.00	75.88
Mato Grosso do Sul	56.68	72.12	81.15	24.46	9.36	79.38
Mato Grosso	64.03	76.64	85.25	21.22	10.26	80.39
Goiás	55.21	70.38	79.72	24.51	9.21	77.69
Distrito Federal	56.95	70.47	79.21	22.26	7.87	76.90

Source: Brazilian Censuses. 1980. 1991. 2000. 2010; Mortality Information System. Datasus (SIM)

Table 9.6 - Mode. median. Interquartile range. Q1. Q3 and standard deviation above the mode. Federal Units. Brazil. males. 1991. TOPALS

UF	Q1	MD	Q3	IQR	SD(M+)	M
Rondônia	56.42	72.82	81.65	25.23	8.75	79.92
Acre	58.87	74.21	82.92	24.05	8.66	80.24
Amazonas	62.03	75.78	84.75	22.72	9.87	80.44
Roraima	59.56	75.95	86.66	27.10	11.70	80.49
Pará	61.32	74.43	82.80	21.48	9.39	80.06
Amapá	59.46	74.01	82.57	23.11	7.64	80.30
Tocantins	64.88	77.69	86.56	21.68	10.14	80.87
Maranhão	60.24	75.22	85.25	25.01	9.32	82.27
Piauí	66.67	78.46	86.74	20.07	7.90	83.80
Ceará	65.82	78.67	87.11	21.29	7.97	84.39
R.G.Norte	65.87	78.12	86.33	20.45	7.87	83.52
Paraíba	61.79	76.61	85.38	23.59	7.61	83.41
Pernambuco	51.61	70.35	80.63	29.01	8.43	79.30
Alagoas	55.63	72.41	81.64	26.01	8.53	79.84
Sergipe	57.78	73.27	82.06	24.28	8.16	80.02
Bahia	61.92	75.79	84.91	22.99	9.67	80.60
Minas Gerais	57.10	71.04	80.56	23.46	8.98	77.85
Espírito Santo	56.23	71.20	80.87	24.64	7.80	78.78
Rio de Janeiro	49.60	66.25	77.12	27.51	9.41	74.72
São Paulo	54.54	69.66	79.42	24.89	9.02	77.04
Paraná	58.61	71.38	80.26	21.65	9.07	77.49
Santa Catarina	59.90	71.77	80.05	20.15	8.33	77.39
R.G.Sul	58.43	70.73	79.80	21.37	9.02	76.40
Mato Grosso do Sul	58.55	72.70	82.02	23.47	9.38	79.51
Mato Grosso	60.41	74.44	83.47	23.06	9.29	80.22
Goiás	58.36	72.09	81.41	23.06	8.76	79.06
Distrito Federal	57.63	71.67	80.63	23.00	8.22	78.58

Source: Brazilian Censuses. 1980. 1991. 2000. 2010; Mortality Information System. Datasus (SIM)

Table 9.7 - Mode. median. Interquartile range. Q1. Q3 and standard deviation above the mode. Federal Units. Brazil. males. 2000. TOPALS

UF	Q1	MD	Q3	IQR	SD(M+)	M
Rondônia	58.07	73.36	82.90	24.84	8.17	80.63
Acre	58.55	73.95	83.24	24.70	9.48	80.20
Amazonas	62.57	75.65	84.50	21.93	9.27	80.53
Roraima	56.44	73.32	83.00	26.56	9.70	80.17
Pará	60.79	74.45	83.58	22.79	9.62	80.16
Amapá	57.48	72.86	82.45	24.97	9.28	79.92
Tocantins	63.04	77.00	86.34	23.31	8.65	83.55
Maranhão	64.89	78.84	88.73	23.84	9.95	84.32
Piauí	65.45	78.23	87.10	21.65	9.35	83.16
Ceará	61.65	76.59	86.00	24.36	7.76	84.33
R.G.Norte	64.10	77.74	86.64	22.54	7.23	84.96
Paraíba	62.81	76.98	86.29	23.47	8.05	84.25
Pernambuco	54.12	71.37	82.02	27.90	9.08	79.68
Alagoas	58.85	74.14	84.16	25.31	9.43	80.74
Sergipe	58.64	74.20	84.41	25.77	8.41	83.05
Bahia	61.84	76.16	86.10	24.27	9.84	81.90
Minas Gerais	60.24	73.76	83.23	22.99	9.14	79.93
Espírito Santo	58.33	73.11	82.99	24.65	9.01	79.94
Rio de Janeiro	54.57	69.22	79.71	25.15	9.82	76.19
São Paulo	57.42	71.84	81.50	24.08	8.70	79.14
Paraná	60.26	72.99	81.99	21.73	8.84	79.26
Santa Catarina	60.95	72.85	81.52	20.57	8.32	78.74
R.G.Sul	60.36	72.52	81.41	21.06	8.93	78.48
Mato Grosso do Sul	59.16	73.05	82.87	23.71	9.71	79.65
Mato Grosso	58.27	72.93	82.58	24.31	9.24	79.80
Goiás	59.57	73.33	82.97	23.39	9.32	79.77
Distrito Federal	60.46	73.71	82.57	22.11	7.75	79.84

Source: Brazilian Censuses. 1980. 1991. 2000. 2010; Mortality Information System. Datasus (SIM)

Table 9.8 - Mode. median. Interquartile range. Q1. Q3 and standard deviation above the mode. Federal Units. Brazil. males. 2010. TOPALS

UF	Q1	MD	Q3	IQR	SD(M+)	M
Rondônia	60.79	74.66	83.74	22.96	9.70	80.21
Acre	61.23	74.90	84.01	22.79	8.72	80.72
Amazonas	62.89	76.17	85.01	22.12	9.68	80.56
Roraima	62.18	76.23	85.55	23.37	9.71	80.93
Pará	58.59	73.96	83.21	24.61	9.32	80.21
Amapá	61.32	75.56	84.82	23.50	10.64	80.30
Tocantins	61.22	76.30	85.88	24.66	8.27	83.67
Maranhão	61.01	75.89	85.55	24.55	10.27	80.70
Piauí	63.15	76.92	85.94	22.79	8.68	82.77
Ceará	61.69	76.44	85.75	24.06	7.99	83.67
R.G.Norte	63.14	77.14	86.54	23.40	8.51	84.09
Paraíba	60.35	75.68	85.60	25.25	8.03	84.13
Pernambuco	59.22	74.20	83.94	24.72	8.97	81.15
Alagoas	56.78	73.61	84.00	27.22	9.81	80.56
Sergipe	60.58	75.28	85.03	24.45	8.06	83.42
Bahia	60.52	75.91	85.93	25.41	9.32	82.79
Minas Gerais	62.11	75.63	85.00	22.90	8.32	83.04
Espírito Santo	61.25	75.69	85.18	23.93	9.06	81.75
Rio de Janeiro	59.41	72.69	82.25	22.85	8.86	79.27
São Paulo	62.95	75.20	84.14	21.18	8.05	81.66
Paraná	61.89	74.94	83.71	21.82	8.43	80.57
Santa Catarina	63.56	75.36	83.71	20.15	8.70	80.26
R.G.Sul	62.86	75.04	83.71	20.85	8.37	80.53
Mato Grosso do Sul	61.59	75.15	84.62	23.03	9.55	80.64
Mato Grosso	60.71	74.66	83.99	23.28	9.79	80.27
Goiás	60.75	74.53	83.77	23.01	9.16	80.36
Distrito Federal	65.02	77.13	85.39	20.36	8.75	81.37

Source: Brazilian Censuses. 1980. 1991. 2000. 2010; Mortality Information System. Datasus (SIM)