HACETTEPE UNIVERSITY
INSTITUTE OF POPULATION STUDIES

CHANGES IN MORTALITY TRENDS AND PATTERNS IN THE PRE-COVID-19 PERIOD: CAUSE OF DEATH ANALYSIS OF LIFE EXPECTANCY AND LIFESPAN VARIATION IN TURKEY

Zehra YAYLA ENFİYECİ

Department of Demography

PhD Dissertation

Ankara
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Changes in Mortality Trends and Patterns in the Pre-Covid-19 Period: Cause of Death Analysis of Life Expectancy and Lifespan Variation in Turkey

Zehra YAYLA ENFIYECI

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ABSTRACT

This dissertation, which aims to analyse the recent pattern and trend in death rates in the pre-Covid period, consists of three articles. In the first essay quality of cause of death data for 2013-2019 were evaluated by focusing on garbage codes. By using WHO recommendations, garbage codes were determined and redistributed to target codes. Findings indicate that garbage cardiovascular diseases constituted the highest share of total garbage codes and increased between 2013 and 2019. The second essay evaluates the burden of communicable diseases by using Markov-chain modelling. Acccording to the results, communicable diseases increased significantly in 2013-2019 period and had higher impact on elderly. Findings also suggest that age at death distribution of males shifted towards older ages. Furthermore, the increase in communicable diseases points to a different pattern of mortality in Turkey's epidemiological transition.

The third essay aims to evaluate the age and cause contributions to life expectancy and lifespan variation between 2013 and 2019 by using decomposition method. Sex difference in life expectancy and lifespan variation were also analyzed with decomposition method. In this essay, the threshold age separating premature mortality from old-age mortality was also calculated. Findings of the essay disclose that males gained higher life expectancy than females due to the improvement in malignant neoplasms and unintentional injuries at ages below threshold age. Increasing life expectancy among females stem from the improvement in cardiovascular diseases at age above threshold ages. This study also indicates that increasing communicable diseases are an important barrier to the higher life expectancy. Sex gap in life expectancy shows the female advantage at all causes. Sex difference in lifespan variation indicates that females have higher variation at older ages and lower variation at young and adult ages than males.

To sum up, results of this dissertation highlight three important results. Firstly, garbage cardiovascular diseases is an important issue in cause of death data that needs to be addressed. Secondly, communicable diseases are becoming dangerous for elderly. Thirdly, females and males have different mortality patterns. While mortality improvement is seen at older ages among females (expansion of mortality), mortality improvement is seen at younger ages among males (compression of mortality).

Key words: Cause of death, garbage code**,** lifespan variation, communicable disease, threshold age.

ÖZET

Covid öncesi dönemde ölüm oranlarındaki güncel örüntüyü ve eğilimi incelemeyi amaçlayan bu tez, üç makaleden oluşmaktadır. İlk makalede, 2013-2019 yılları arasındaki ölüm nedeni verilerinin kalitesi, iyi tanımlanmamış kodlara odaklanılarak değerlendirilmiştir. DSÖ önerisi kullanılarak iyi tanımlanmamış kodlar belirlenmiş ve ve bu kodlar hedef kodlara yeniden dağıtılmıştır. Bulgular, iyi tanımlanmamış kardiyovasküler hastalıkların toplam iyi tanımlanmamış kodlar içinde en yüksek payı oluşturduğunu ve 2013-2019 yılları arasında artış gösterdiğini ortaya koymaktadır.

İkinci makale, Markov zinciri modellemesi kullanarak bulaşıcı hastalıkların yükünü değerlendirmektedir. Sonuçlara göre, bulaşıcı hastalıklar 2013-2019 döneminde önemli ölçüde artmış ve yaşlılar üzerinde daha yüksek etkiye sahip olmuştur. Bulgular erkeklerde ölüm yaşı dağılımının ileri yaşlara doğru kaydığını göstermektedir. Ayrıca, bulaşıcı hastalıklardaki artış, Türkiye'nin epidemiyolojik geçiş sürecinde farklı bir ölüm örüntüsüne işaret etmektedir.

Üçüncü makale, ayrıştırma yöntemini kullanarak 2013-2019 yılları arasında beklenen yaşam süresi ve yaşam süresi varyasyonuna yaş ve ölüm nedenlerinin katkılarını değerlendirmektedir. Ayrıştırma yöntemi ile yaşam beklentisi ve yaşam süresi varyasyonundaki cinsiyet farkı da analiz edilmiştir. Bu makalede ayrıca erken yaş ölümlülüğünü yaşlı ölümlülüğünden ayıran eşik yaş da hesaplanmıştır. Makalenin bulguları, eşik yaşın altındaki yaşlarda malign neoplazmlar ve kasıtsız yaralanmalardaki iyileşme nedeniyle erkeklerin kadınlardan daha yüksek yaşam beklentisi kazandığını ortaya koymaktadır. Kadınlarda beklenen yaşam süresinin artması ise eşik yaşın üzerindeki yaşlarda kardiyovasküler hastalıklardaki iyileşmeden kaynaklanmaktadır. Bu çalışma aynı zamanda artan bulaşıcı hastalıkların daha yüksek yaşam beklentisi önünde önemli bir engel olduğunu göstermektedir. Yaşam beklentisindeki cinsiyet farkı incelendiğinde, tüm nedenlerde kadınların avantajlı olduğu görülmektedir. Yaşam süresi varyasyonundaki cinsiyet farkı, kadınların erkeklere göre ileri yaşlarda daha yüksek, genç ve yetişkin yaşlarda ise daha düşük varyasyona sahip olduğunu göstermektedir.

Özetlemek gerekirse, bu tezin sonuçları üç önemli sonucu vurgulamaktadır. İlk olarak, iyi tanımlanmamış kardiyovasküler hastalıklar ölüm nedeni verileri içinde önlem alınması gereken önemli bir konudur. İkincisi, bulaşıcı hastalıklar yaşlılar için tehlikeli hale gelmektedir.

Üçüncüsü ise, kadınlar ve erkekler farklı ölüm örüntülerine sahiptir. Kadınlarda ileri yaşlardaki ölümlerde iyileşme görülürken (ölümlüğün genişlemesi), erkeklerde daha genç yaşlardaki ölümlerde iyileşme görülmektedir (ölümlülüğün sıkışması).

Anahtar kelimeler: Ölüm nedeni, iyi tanımlanmamış kodlar, yaşam süresi varyasyonu, bulaşıcı hastalıklar, eşik yaş.

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ABBREVIATIONS

ABPRS	Address Based Population Registration System
CDs	Communicable disease
CoD	Cause of death
DNS	Death Notification System
ICD	International Statistical Classification of Diseases and Related Health Problems
INJs	Injuries
NCDs	Noncommunicable disease
TurkStat	Turkish Statistical Institute
UCoD	Underlying cause of death
WHO	World Health Organization

I. GENERAL INTRODUCTION AND BACKGROUND

I.1. General Introduction

Mortality pattern of Turkey changed significantly in the 20th century. In the first part of the century, high rates of communicable diseases, infant and child mortality were main obstacles on the higher life expectancy. Additionally, during these years, Turkey participated in many wars, including the First World War. In this period, life expectancy was 44 years (Shorter & Macura, 1982). In the second part of the century, infant mortality and infectious diseases and other communicable diseases decreased significantly. During this period, life expectancy at birth exceeded 65 years for both sexes. Total fertility rate also decreased in this period. The distribution of mortality by age has changed from a U-shape with high infant, child, and old-age mortality to a J-shape with low infant and child mortality and high old-age mortality. The increase of life expectancy in this century was mainly driven by the improvement in infancy and childhood period.

Data based on the registration system was not sufficient in 20th century in Turkey. In particular, death registers did not cover the entire population and were not reliable. Main source of vital statistics was censuses. The first census in the Turkish Republic took place in 1927. The second census was conducted in 1935 and since then censuses were conducted every 5 years until 1990. The last census was conducted in 2000. With the 1970 census, some questions were included in the census to indirectly measure infant and child mortality. However, censuses were not sufficient to understand Turkey's mortality pattern. As a complementary source of vital statistics, demographic surveys were conducted. Coale-Demeny model life tables were among the widely used techniques for estimation of mortality pattern of Turkey (Ansley J. Coale & Demeny, 1966; UN, 1982). As can be seen, the main indicators of vital events in the 20th century are generally estimated indicators. Many reforms have been made to the registration system to understand the precise value of the measurement of vital events and to improve the efficiency of the registration system. The first and most

important step in these reforms is the digitalisation of the registration system. The digitalized registration system (MERNIS) was introduced in the early 2000s and since then, various changes have been made to the system to ensure the reliability of the data.

This study is composed of three main essays and uses the 2013-2019 death and population registration, namely causes of death dataset and Address Based Population Registration System (ABPRS) population data from Turkish Statistical Institute (TurkStat). TurkStat's cause of death dataset provides data on cause of death, sex, age and province (place of permanent residence) for each person who died in the period 2013-2019. The population dataset presents the total number of population by sex and single age for each year between 2013 and 2019.

In this dissertation, each essay consists of abstract, introduction, literature review, data and methods, findings, conclusion, and discussion sections. The introduction section explains general information on classification of cause of death, accuracy, and quality of death certificate, and then described the theoretical framework which were used in the second and third essays. In the theoretical framework part, epidemiological transition theory, mortality compression/expansion and shifting mortality hypothesis are handled. Then the mortality trends and patterns across countries were reviewed. At the end of introduction section common demographic concepts and definitions were presented (Table I.1.1).

The first essay includes the process of data preparation and distribution of garbage codes. In the first essay we tried to answer below research questions:

1. How does the percentage of garbage codes change by age and sex between 2013 and 2019?
2. Which types of garbage codes are most used?
3. How does the pattern of leading causes of death change before and after the distribution of garbage codes?
4. What policies should be implemented to reduce the use of garbage codes?

In the second essay, we aim to explore the causes and trends of death in Turkey for the 2013-2019 period, with special emphasis on the increase in communicable diseases. These analyses were conducted with discrete time, age-classified Markov-chain model. Related research questions of the second essay are:

1. How do recent gains in life expectancy and modal age at death vary by sex and age?
2. How do causes of death change recently?
3. Are there different trends in causes of death patterns in terms of sex and age?
4. Is Turkey still in the last phase of epidemiological transition?

The third essay aims to explain contribution of age and cause of death to the change in life expectancy and lifespan variation between 2013-2019 period. Related research questions of the third essay are:

1. How does the lifespan variation change by sex, age and causes of death between 2013 and 2019?
2. How do ages contribute lifespan inequality?
3. What causes of death contribute positively/negatively to lifespan variation?
4. Are there any differences in life disparity between the sexes?

The synthesis section aims to summarize key findings of the essays by addressing the research questions and discussing the contribution and limitations of the study.

I.2. Coding and Classifying Cause of Death

Understanding the causes of death is of vital importance in monitoring health problems in the population, evaluating health priorities, and planning public health programs and interventions. Effective public health policies need accurate, timely, complete, and comparable cause of death data (Anderson, 2011; Flagg & Anderson, 2021). Although almost every country has civil registration systems, many of these

systems are not well functioned and do not cover the entire population (Anderson, 2011; Mathers et al., 2005). Quality of civil registration systems varies across geographical regions and socio-economic status. Generally, vital statistics and registration systems have higher standards in developed countries (Teixeira et al., 2019). In the World, only sixty-eight percent of countries have at least 90% percent of coverage in death registration (UNSD, 2021). Today, most of Sub-Saharan African countries have less than 50% coverage or no data on death registration; some of Latin American and Northern Africa countries less than 75% coverage at death registration (UNSD, 2021). In such kind of conditions, countries developed some strategies to produce cause of death statistics. China and India use Sample Registration System (SRS) to overcome coverage problem. SRS provide estimates for mortality profile of these countries. In this system, birth and death events are monitored periodically by visiting households which are selected randomly (Anderson, 2011; Liu et al., 2016). Interviewing with household members, details of vital events are gathered. However, since the information on cause of death are gathered from household member who has generally no medical expertise, verbal autopsy collected by surveys is also used with for cause of death statistics in India and China (Liu et al., 2016; Setel et al., 2005). Although combination of SRS and verbal autopsy provide valid results for these countries, such kind of data does not enable international comparison. Therefore, cause of death data which are obtained from vital registration system and reported by a physician is the best way for the collection (Anderson, 2011).

Cause of death data taken from death registration system is generally collected by death certificates. To provide international comparability, WHO has established a standard medical death certificate (Figure II.2.1). This certificate enables to collect standardized cause of death information and makes easier for cross country comparison (Anderson, 2011). Death certificate is completed by physician because especially cause of death part of the form requires clinical expertise. If death occurs in suspicious conditions, then coroner certifies the cause of death.

Data quality has long been investigated by researchers. Some have defined data quality as “fit for use by data users”, while others have defined criteria including but

not limited to accuracy, relevance, comparability, timeliness, and accessibility (Mahapatra et al., 2007). Global Burden of Disease (GBD) studies have an important role in improvement of accuracy and quality of cause of death data. GBD produces health estimates and statistics for almost all countries based on the analytical methodology (Teixeira et al., 2019). GBD collaborators have also investigated some methods to improve the data quality. To assess the efficiency of death registration system Phillips and colleagues (2014) proposed a summary index including six criteria. Findings indicated that the proportion of garbage codes (GCs) has important impact on the index value of death registration system (Phillips et al., 2014). In simple terms, GCs are identified as causes that can't be categorised as the underlying cause of death (UCoD). GCs include not only the "Symptoms, signs and abnormal clinical and laboratory findings not elsewhere classified" which is known as R codes, but also include other codes which are considered as no value for public policies. Both GBD and WHO collaborators proposed some GC lists to evaluate the quality of cause-of-death statistics (Mathers et al., 2005; Naghavi et al., 2010, 2020; WHO, 2018). Furthermore, GCs should be redistributed to well defined causes to make global comparisons and accurate health policies (Naghavi et al., 2010, 2020).

Accurate cause of death data gives the true UCoD and the sequence of events that lead to death. However, cause of death data generally has some accuracy problems. The common three factors affecting the accuracy of cause of death information are: 1) unawareness about the importance of accurate certification of cause of death, 2) inadequate training on how to properly formulate and record the correct cause of death, 3) decreasing rate of clinical autopsy (Anderson, 2011; Blokker et al., 2017; Espinosa-Brito & Mendoza-Amat, 2017). In the first case, completion of death certificate is generally seen as boring administrative task. In this situation, less-experienced physician who has little or no idea about the clinical history of decedent may be given completion of the certificate (Anderson, 2011). When second factor occurs, the sequence of events that resulted in death does not make sense. In the third case, clinical autopsy plays important role in education of physicians and understanding the true UCoD. In this sense, second factor and third factor are associated with each other. Because if physicians have adequate training and

practicing, the quality of certificates will be more satisfactory. Apart from above factors, accuracy of some sensitive cause of death such as HIV/AIDS, suicide or abortion related causes etc. may be underreported for legal, cultural or social reasons in developed and developing countries alike (Carmo et al., 2021; Mathers et al., 2005; Say et al., 2014; Tøllefsen et al., 2012).

I.3. Theoretical Framework

There are some theories on longevity and quality of survival. While the first theoretical view is on the societal constraints on human mortality, other theoretical views are on the biological constraints on human mortality (Manton, 1982).

I.3.1. Societal Constraints on Human Mortality

I.3.1.1. Epidemiologic Transition Theory

According to the theory which evaluates societal constraints on human mortality, health and disease patterns are characterized by demographic and socioeconomic transitions (Omran, 1971). Omran (1971) describes 3 phases that change the life expectancy. The first stage is named as “the age of pestilence and famine”. In this phase death rates are high and fluctuating. Positive checks (war, epidemics, famine etc.) determine the pattern of mortality. The second stage is “the age of receding pandemic”. Characteristic of this phase is that epidemic decrease and mortality rates fall. Life expectancy at birth improves by about 20 years. The last phase is “the degenerative and man-made diseases”. Decline of mortality rates continues and life expectancy increases. In this phase main cause of deaths are degenerative diseases. Shift from phase 1 to phase 3 requires some changes in socio-demographic structure. According to this theory, epidemic transition is possible with modernization. Higher standard of livings, nutrition, improved sanitation and immunization etc. provides this transition (Omran, 1971). In the epidemiologic transition the most benefitted group in decreasing death rates are children and young women.

After epidemiologic transition theory proposed by Omran (1971), some modifications have been made in the theory. Some researchers viewed these modifications as a new transition in the theory (Armelagos, 2009; Barrett et al., 1998; Harper & Armelagos, 2010) while others viewed as a continuation of Omran's theory (Gaziano, 2010; Olshansky et al., 1998; Olshansky & Ault, 1986; R. G. Rogers & Hackenberg, 1987).

Barrett and colleagues (1998) have proposed an extended multi-epidemiological transitions framework that takes into account the pattern of disease emerging from the Palaeolithic period to the industrial revolution and beyond (Armelagos, 2009; Barrett et al., 1998; Harper & Armelagos, 2010). This framework covers the epidemiological history of human in a very comprehensive way and involves 3 transitions. The first transition begins with agricultural revolution, the second transition is associated with industrial revolution, and the third transition is accompanied by accelerated globalization of the world. Barrett and colleagues (1998) take the classic epidemiologic transition (Omran, 1971) in the second epidemiologic transition.

First, Barrett and colleagues (1998) and Harper and Armelagos (2010) constructed the Paleolithic baseline to understand the previous history of agricultural revolution which led to the first epidemiologic transition. In the Paleolithic age basic diet needs were met from the resources of the environment. In this age human were hunter-gatherer and they were mobile and live with small population size. So, transmission of many infectious diseases was very difficult. Moreover, foods in the Paleolithic age contain less pathogenic microorganisms than in the later ages. This means that diets in the Paleolithic age were more health promoting than later ages. This situation shows that emerging developments in the epidemiologic transition does not always brought benefits for human health but also brought damages (Barrett et al., 1998; Harper & Armelagos, 2010). On the other hand, it should be noted that in Paleolithic era birth and death rates were low and population size were kept stable due to the low birth rates (Angel, 1984; A. J. Coale, 1974). Disadvantages of agricultural revolution such as worse nutrition and increasing infectious diseases caused high death

and birth rates. After agricultural revolution, population size was kept stable due to high death rates (Armelagos et al., 1991).

The first epidemiologic transition defines shifting from hunter-gatherer society to settled society. Agricultural revolution changed the lifestyle of human. In the early cities of agrarian communities, human faced with crowded and unhygienic living conditions. Sedentary life provided to live with large population size. These crowded communities fostered the spread of infections and parasitic. The lack of sanitary sewerage system caused the spread of parasites and infections (Barrett et al., 1998). On the other hand, some new infections appeared through the products of domesticated animals such as cow, sheep, and cattle. The very familiar infections such as Tuberculosis, Q fever, brucellosis, smallpox, and measles were contracted from these domesticated animals. Furthermore, there were lack of dietary diversity and staple crops at the beginning of the agrarian period. Subsistence level food caused to nutrient deficiencies, famine, and drought periods in these communities. In addition to this, inadequate nutrition increased the epidemics and severity of diseases. At these stage cholera, plague, typhus, measles, and other viral infections were very common. Briefly, this transition is characterized by high birth and death rates and the leading causes of death are infectious diseases and nutrition deficiencies.

The second epidemiologic transition is described by transition from agriculture to industrial revolution. This transition covers Omran's epidemiologic transition (Barrett et al., 1998). Significant decline of infectious diseases and pandemics give place to degenerative and manmade diseases. Progress in the medical technology and sanitation were the main reasons for decline of infectious diseases. The second transition was faster for less developed countries than developed countries. After the Second World War biomedical innovations accelerated the decline of child mortality and improvement in life expectancy in these countries. On the other hand, at this stage extended life expectancy has brought disadvantage; human have begun to spend much more time with chronic diseases (Alter & Riley, 1989).

The third epidemiologic transition is typified by three trends. The first is the emergence of infectious diseases that had previously been controlled. The second one is that emergence of new infections which make a big contribution to adult mortality. The third is that many of re-emerging infections are antibiotic resistant pathogens. All these features are results of accelerating globalization. In addition to this, Barrett and colleagues (1998) and Harper & Barrett (2010) have drawn attention to the delayed chronic diseases to the older ages in high-income countries experiencing the third epidemiologic transition.

Apart from the epidemiological transition model described above, some researchers have described the new stages as a continuation of Omran's epidemiological transition theory. Olshansky & Ault (1986) have described a fourth stage, “The Age of Delayed Degenerative Diseases”. This stage is characterized by a rapid mortality decline at older ages. These deaths are postponed to later ages at which human become more fragile to dead from degenerative diseases. Postponement of mortality is due to the improved medical technology. Furthermore, deaths due to infectious and parasitic diseases had been prevented by modern medicine.

Rogers & Hackenberg (1987) also proposed a fourth stage extension. Original epidemiologic transition theory did not refer to deaths caused by injuries and human behaviors. According to this stage, man-made diseases, individual behaviors, and potentially destructive lifestyles such as physical inactivity and excessive drinking etc. affect morbidity and mortality. Furthermore, Rogers & Hackenberg (1987) emphasized that individual behaviors have an impact on infectious and parasitic diseases. For example, increasing rate of HIV/AIDS is due to the individual lifestyle and man-made causes.

Olshansky and colleagues (1998) defined the fifth phase of the epidemiological transition as the reappearance of infectious and parasitic diseases. Population ageing, urbanization and densely settled cities contributed the spread of the infectious and parasitic diseases. These diseases became very dangerous for people who have fragile or compromised immune system due to the age or medical treatments for degenerative

diseases. Especially older and advanced aged people are at the risk of infection and severe damages. On the other hand, genetic variants of bacteria made the treatment difficult because of antibiotic resistant (Olshansky et al., 1998).

Another fifth stage was proposed by Gaziano, "The Age of Obesity and Inactivity" (2010). Gaziano assumes the fourth stage of epidemiologic transition as the "The Age of Delayed Degenerative Diseases" proposed by Olshansky and Ault (1998). In the fifth stage, progress in the postponement of morbidity and mortality to older ages is threatened by individual bad habits and lifestyle that result obesity and inactivity. Furthermore, this adverse lifestyle is transferred to the later ages and obese children are generally becoming obese adults. This "obesity epidemic" is seen in both developed and developing countries and trigger the dramatic increase in cardiovascular disease (Gaziano, 2010).

I.3.2. Biological Constraints on Human Mortality

According to the theories on biological constraints on human mortality (Fries, 1980; Hayflick, 1974; Keyfitz, 1978) increasing life expectancy and decreasing mortality rates will reach their limits due to the physiological conditions in which senescence will show its characteristics on human life span. In this theory "rectangularization of survival curve" is explained. This curve describes the proportion of survivors of a cohort. It follows a path near 1.0 until arriving senescence age. In this age it suddenly drops 0.0. So, by eliminating premature deaths from some fatal diseases large portion of survivors will accumulate at the older ages and they will die due to biological incapability of organs, that is, "natural death". According to this model even if disease related mortality is eliminated, unless aging rate of organs are changed, significant improvement in life expectancy is not possible. There have emerged two different opinions in the biological constraints on human mortality. Gruenberg (1977) and Kramer (1983) propound theory of pandemic mortality and Fries (1980, 1983) propounds compression of morbidity theory.

According to theory of pandemic morbidity, although deaths from chronic diseases decline, incidence of these diseases does not decrease. So, an individual with this chronic disease lives in worse quality (Gruenberg, 1977; Kramer, 1983). Gruenberg (1977) defines the increasing prevalence and longer duration of chronic diseases as "failures of success". Although fatal complications of chronic diseases have been eliminated, nonfatal part of these diseases have worsened the population's health. In the compression of morbidity theory, due to the better lifestyles, healthier diet, awareness about risk factors such as smoking, alcohol, high calorie foods etc., health status remains in better quality until advanced ages and morbidity is postponed to later ages (Fries, 1980, 1983).

Fries'(1980) compression of morbidity theory also fits very well to mortality concept. Similar to the compression of morbidity, compression of mortality refers that as more people live longer and maximum lifespan increase slowly, then age at death distribution shifts to older ages, which is referred to as mortality compression (Fries, 1980). Trends in the age pattern of the death rate determine the strength of compression. If death rates at young and adult ages declines faster than at older ages, then deaths at young and adult ages will be postponed to later ages and then compression will occur. On the other side, if mortality rates at older ages declines faster than young and adult ages than old people will live longer and deaths at older ages will be postponed to later ages and thus will lead to expansion of mortality at older ages (Rothenberg et al., 1991; Zhang & Vaupel, 2008). The net compression in the population is determined by comparing the compression of mortality at young and adult ages and expansion of mortality at older ages.

Another concept which is generally taken together with mortality compression is "*mortality delay*" or "*shifting mortality*" (Canudas-Romo, 2008; Kannisto, 2001; Vaupel, 2010). Mortality delay occurs when mortality at all ages declines and the age distribution of mortality shifts to the right, preserving the shape of the distribution. As a result of the mortality delay, the age at death increases but lifespan variation remains unchanged. In other words, the rise of the modal age at death together with the

stagnation in lifespan variation is the evidence of mortality delay (Canudas-Romo, 2008).

In early studies, compression of mortality was tested by using truncated ages which is the conditional on surviving to a certain age (Myers & Manton, 1984; Rothenberg et al., 1991). However, this method was found a bit problematic because the result of compression was changing according to the selected age (Nusselder & Mackenbach, 1996; Robine, 2001). A common methodology used for determining compression is the investigation of standard deviation above and below the modal age at death (Cheung & Robine, 2007; Kannisto, 2001; Ouellette & Bourbeau, 2011). Regarding compression of mortality, some researchers introduced the concept of "threshold age" which separates the compression between younger and older ages (Zhang & Vaupel, 2008, 2009). Accordingly, while a reduction in premature deaths creates compression and decreasing lifespan variation, a reduction among elderly mortality creates expansion in old age mortality and increase of lifespan variation.

All these methods confirm that, according to the literature, the main indicator of mortality compression is decreasing lifespan variation. (Bergeron-Boucher et al., 2015).

I.3.3. Global Mortality Patterns and Trends

The twentieth century marked a great improvement in life expectancy of about 30 years in most of the countries. In the first part of 20th century, main contribution to life expectancy came from the improvement in infancy, childhood, and early adult periods (Bergeron-Boucher et al., 2015; Crimmins & Beltran-Sanchez, 2011). Better living conditions, improvement in medicine and nutrition provided great gains in life expectancy. Decline in infancy and early adult ages also contributed to decreasing lifespan variation, which led to mortality compression in many developed countries (Bergeron-Boucher et al., 2015; Edwards & Tuljapurkar, 2005). Hubert and colleagues (2002) conducted a longitudinal study on lifestyle, habits of aging cohort between the years 1986 and 1998. They found the supportive evidence for compression of

morbidity. Results showed that being in a healthier lifestyle postpone disability to the later ages and living with disability in a shorter period (Hubert et al., 2002).

In the second part of the 20th century, leading causes of death became cardiovascular diseases and malignant neoplasms. Mortality at adult and older ages began to decline and improvement in adult and older mortality accelerated the increasing life expectancy with stagnant lifespan variability (Cheung et al., 2009; Cheung & Robine, 2007; Christensen et al., 2009; Crimmins & Beltran-Sanchez, 2011). Canudas-Romo (2008) investigated the lifespan variation around the modal age for some selected developed countries for years between 1900 and 2005. Findings prove that although modal age increased over time, decline of variation around the modal age was slowdown. This study show that many developed countries experienced the shifting mortality pattern in the second half of the 20th centuries (Canudas-Romo, 2008). Recent improvements in life expectancy are substantially driven by improvements in adult and older ages.

In addition to the age at death, the pattern of cause of death has also changed in this century. In the early twentieth century, infectious diseases were among the leading causes of death, whereas today cardiovascular diseases and malignant neoplasms have become the leading causes, accounting for more than 50% of all deaths (Crimmins & Beltran-Sanchez, 2011). The change in cause of death pattern also brought the discussions of longer lives with worse health conditions (Christensen et al., 2009; Crimmins & Beltran-Sanchez, 2011). Today, older ages are more susceptible to chronic diseases and disabilities.

On the other hand, some countries could not enjoy with the high life expectancy. HIV/AIDS outbreak in Sub-Saharan Africa decreased life expectancy substantially in 1900s and 2000s (Bongaarts et al., 2011; van Raalte, 2021). Between 1960 and 1985 former Soviet Union countries experienced decreasing life expectancy due to the high cardiovascular diseases, fluctuated alcohol consumption and violence (Aburto & van Raalte, 2018; Luy et al., 2011). Former Soviet Union countries experienced increase in life expectancy between 1985 and 1987, due to the

Gorbachev's anti-alcohol campaign in 1985. However, with the collapse of campaign in 1987, life expectancy decreased significantly (Luy et al., 2011; Shkolnikov et al., 2001).

In the last century, the Eastern Mediterranean countries had a lack of quality data. Data for these countries, including Turkey, were very limited. According to mortality estimates, Eastern Mediterranean countries have three times the risk of dying from injuries compared to high-income countries (WHO, 2014). Moreover, these countries are currently among the countries with the highest risk of dying or becoming disabled due to injury or violence-related conditions, leading to a decline in life expectancy (Bairami et al., 2023; Mokdad et al., 2018). As expected, injury and violence-related deaths mostly affect the young and adult population and the male population. Although there has been an improvement in injury-related mortality, it is still higher in Eastern Mediterranean countries (Bairami et al., 2023).

In Latin America and Caribbean countries almost half of deaths from homicides occur among 15-29 age group and this excess mortality at young ages create a burden on mortality pattern in these countries (Institute of Health Metrics and Evaluation IHME, 2018). Research indicated that excess male deaths due to suicides, violence and injuries were increasing and at higher rates in Brazil, Colombia, Ecuador, Panama and Paraguay between 1950 and 2005 (Palloni & Pinto-Aguirre, 2011). Another study also proved that homicide deaths among males made the largest contribution to the life expectancy difference between Latin American and Caribbean countries and high-income countries in the period 2005-2014 (Canudas-Romo & Aburto, 2019).

Mortality data in most of Asian countries is of lower standard than in European, North American, and South American countries (Zhao, 2011). In most Asian countries under-registration and other reporting issues affected the reliability of mortality data severely. Based on the UN and WHO mortality data and estimations on mortality, between 1950 and 2010, life expectancy increased 28 years in Asian countries. This substantial increase stem from two improvements in mortality. The first is the

reduction in infant and child mortality and the second is the significant reduction in adult mortality. Adult mortality declined during 1950 and 1960s and adult and old age mortality continued to decline between 1970s and 1990. Life expectancy at birth was 57 and 61.9 years for males and females, respectively. Improvement in adult mortality was not homogeneous in Asia. Hong Kong, Singapore, Japan experienced great developments and life expectancy in these countries was high in 1990s. On the other hand, China, Lebanon, Syrian Arab Republic and Turkey experienced low socioeconomic status and moderate life expectancy level in 1990. Furthermore, Afghanistan, Iraq, Democratic People's Republic of Korea, Kazakhstan, Uzbekistan, and some other countries experienced increase in adult mortality due to wars, famines, collapse of the Soviet Union, increase in AIDS (Zhao, 2011).

America experienced increases in life expectancy since 1970. The gain in life expectancy was generally stem from the decline of mortality due to cardiovascular diseases (CVD) (Mehta et al., 2020). Thanks to the progress in pharmacology and medical surgery areas, CVDs decreased substantially until 2010. However, research evidence shows that since 2010 gain in life expectancy have stalled and begun to decline in recent years due to the rise in CVDs (Mehta et al., 2020). In another study, it was found that there has been high and increasing mortality at 45-54 age group among white non-Hispanic Americans due to increasing rate of suicides and drug poisonings since 1999 (Case & Deaton, 2015). Similarly, England and Wales also experienced a stagnation in life expectancy. Leon and colleagues (2019) investigated the life expectancy trends in England and Wales by comparing with 22 high-income countries. The notable finding of the study is that the stalling improvement in life expectancy in England and Wales since 2011. This deterioration was the result of the divergent mortality rates at 25-50 age group (Leon et al., 2019).

Today, at the time the thesis was written, the world was faced with the Covid-19 pandemic and the mortality due to Covid-19 have become the alarming event. At this time people were quarantined, education suspended, and international mobility and migration were taken under control. When we look at the epidemiology of Covid-19, people who are age 65 and over, people who have chronic diseases are among the

high-risk groups. In the beginning of the pandemic the Covid-19 was riskier for rich people due to the travel and vacation internationally. Then higher risk of pandemic shifted to poor people (Mamelund & Dimka, 2021). Research evidence indicated that socioeconomically disadvantaged people, immigrants, and Black and indigenous people were at the higher risk of Covid-19 hospitalizations and deaths in high-income countries (Dimka & Mamelund, 2020; Hatcher et al., 2020; Mamelund & Dimka, 2021; Torche & Nobles, 2022)

Table I.1.1. Common demographic concepts and definitions used in the dissertation

Completeness	Percentage of deaths with medically certified cause of death
Coverage	Percentage of population covered by medical certification of cause of death
Garbage code	Causes of death which are insufficient to explain the underlying causes of death. Garbage codes include ill-defined codes.
Ill-defined code	ICD codes under "symptoms, signs and ill-defined conditions (ICD-10 codes R00-R99) category
Immediate causes	Refers to the final cause resulting in death
Intermediate causes	Refers to events arising from the underlying cause
Underlying cause of death	Refers to disease or injury that triggers the events leading to death
Premature mortality/death	Refers to death occurring before life expectancy
Modal age at death	Refers to the age at which the higher proportion of deaths occurred
Years of life lost (YLL)	Refers to the average years an individual would have lived if they had not died prematurely. YLL is a summary measure of premature mortality.
Threshold age	Refers to the age below which a reduction in mortality reduces lifespan variation and above which a reduction in mortality increases lifespan variation
Life expectancy	Refers to the average number of years a human would live in a year if individuals would expose the age specific mortality rates of that year
Lifespan variation	Indicates the variability of the life expectancy
Life disparity	Refers to the average remaining life expectancy at the ages when death occurs; it is a measure of life years lost due to death

I.4. References

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II. ESSAY 1: QUALITY ASSESSMENT OF CAUSE OF DEATH DATA IN TURKEY BETWEEN 2013-2019: GARBAGE CODE ISSUE

Abstract

Background/Aim: Sufficient quality of cause of death data is a sine qua non for a well-performing death registration system. Garbage codes are a major barrier to the quality of cause of death data. This essay aims to 1) determine the types of garbage codes for 2013-2019 period, 2) assign each type of garbage codes to target causes, and 3) indicate the mortality pattern before and after distribution of garbage codes.

Method: Garbage codes were identified based on WHO cause of death classifications. In the redistribution process, we used fixed proportions determined by stochastic models for some garbage codes and proportional distribution for the remaining garbage codes.

Results: According to the findings, proportion of total garbage codes increased between 2013 and 2019. Garbage cardiovascular diseases consisted of most share of garbage codes and the proportion was high among older ages and among females. The percentage of ill-defined conditions (R codes) decreased in 2013-2019 period.

Conclusion: Although the rise in total garbage codes, Turkey has sufficient quality for cause of death data.

Key words: Garbage code, ill-defined causes, cause of death, Turkey

II.1. Introduction

Death registrations are in poor quality in many countries (Naghavi et al., 2020). Therefore, the produced statistics do not help to develop effective health policies. WHO and World Bank put some goals for the universal registration of birth, deaths and cause of death to achieve by 2020, 2025 and 2030 (World Bank & World Health Organization, 2014). Accordingly, registered, reported and certified deaths with main characteristics should be minimum 60%; proportion of reliable and certified cause of death occurred in hospitals should be minimum 80% by 2020. Despite these targets, only less than a third of deaths worldwide are attributed to a cause (de Savigny et al., 2017). Even in countries which have complete and high coverage vital registration system, the quality of cause of death data is inadequate (Ahern et al., 2011).

Cause of death quality is generally measured by the proportion of ill-defined or garbage codes in total deaths. Ill-defined and garbage codes are different concepts. The "garbage code" term was firstly used by Murray and Lopez (1996) to define the causes which are insufficient to explain the underlying causes of death in the assessment of Global Burden of Disease (Murray et al., 1996). On the other hand, ill-defined codes took its name from some specific ICD codes. Ill-defined codes refer ICD codes under "symptoms, signs and ill-defined conditions (ICD-10 codes R00-R99)" category (Mathers et al., 2005). Garbage codes cover all ICD codes which do not give a clear explanation for underlying cause of death and create contamination in causes of death data (Mathers et al., 2005). Briefly, ill-defined codes are subset of garbage codes.

In this essay we aimed to determine the types of garbage codes and evaluate the usability of cause of death data for 2013-2019 period. Under this aim, we answered the below research questions:

- How does the percentage of garbage codes change by age and sex between 2013 and 2019?
- Which types of garbage codes are most commonly used?

- How does the pattern of leading cause of death change before and after the distribution of garbage codes?
- What policies should be implemented to reduce the use of garbage codes?

The reasons for choosing the years 2013-2019 to answer these research questions are as follows: First, there are some issues with respect to generalizability (coverage and completeness) and quality of cause of death data for years prior to 2013 (Akgün et al., 2012; Yayla, 2016). By 2013, electronical "death notification system (DNS)" was put into practice. With this system death registration has become more complete and quality of ICD-codes become more valid for performing analysis with this data. So, the reason why we put 2013 as the starting year is to want to study with more reliable data. The claim that post-2013 data is of good quality is supported by a recent cross-country study in which Turkey was classified as having a well-performing vital registration system despite being among the low and medium socio-demographic index countries (Iburg, Mikkelsen, & Richards, 2020). Secondly, 2019 was selected for the end year due to the being the most recent data. On the other hand, working with the pre-Covid period prevented misinterpretation caused by mortality fluctuations during the Covid period.

II.2. Literature Review

II.2.1. Death Registration in Turkey

Death and burial procedures in Turkey are determined by several legal frameworks. These are "Public Health Law (1930), Municipal Law (1930), Metropolitan municipality law (2004), Regulations on family practice (2011), and Regulations on transport funeral and burial with the construction of cemetery (2013)". The last regulation, Regulations on transport funeral and burial with the construction of cemetery (2013), includes detailed identification for burial processes and certification of deaths. When we investigate the development of Turkish death registration system, we see two important years that can be considered as turning points of the registration system: 2009 and 2013.

Before 2009, there were two kinds of death certificates; one was for General Directorate of Civil Registration and Nationality through Central Civil Registration System (MERNIS) (for official record) and the other one was for TurkStat (for statistical and burial purposes). Death document collected by MERNIS was gathered for all deaths in Turkey. On the other hand, death certificates collected by TurkStat were only provided by health centers in provincial and district centers. So, the statistics produced did not accurately reflect the health profile of the country.

In 2009, death certificates collected by MERNIS were added to the TurkStat death data pool through matching process. In addition, the cause of death section of the TurkStat death certificate has been expanded and detailed. Death codes were made compatible with ICD codes. Before 2009, Turkey used the 8th version of ICD (ICD-8). By 2009, ICD-10 was put into practice. Another reform of the death registration system in Turkey was implemented in 2013. The electronic death notification system (DNS) was introduced, and the death certificate collected by MERNIS was abolished. This innovation has enabled all deaths to be collected in a single repository. With these reforms, there has been a significant improvement in the quality and completeness of

the data collected by the registry system (Özdemir et al., 2015; Teker et al., 2020; Yayla, 2016; Yayla & Çavlin, 2019).

II.2.2. Adoption of the International Statistical Classification of Diseases and Related Health Problems (ICD)

ICD enables universal comparison of cause of death data. The roots of efforts to uniformly name and classify diseases date back to 1851 (Gear et al., 1961). At the first statistical congress in 1853, uniform nomenclature and classification of diseases was accepted as a precondition for international comparison (Gear et al., 1961; WHO, 2007). At the second statistical congress in 1855, William Farr and Marc d'Espine suggested two different lists. Farr's classification consisted of five groups: "*epidemic diseases, constitutional diseases, local diseases arranged according to the anatomical site, developmental diseases and injury*" (Mahapatra et al., 2007; WHO, 2007). Cause list proposed by d'Espine was classified by the pathological and etiological characteristics of diseases. At the end of the Congress, a classification consisting of both lists was adopted. The first revision of ICD was prepared in 1891 by Dr. J. Bertillon, and adopted in 1893. The Bertillon classification consisted of three lists for its application: detailed list of 161 titles, intermediate list of 99 titles and abridged list of 44 titles. Following the recommendation of the American Public Health Association to review the classification every ten years, the first *international list of causes of death* conference was held in 1900 in Paris with the participation of 26 countries. In this conference a detailed list of causes of death was accepted. The next conferences for the revisions of the *international list of causes of death* were held in 1910, 1920, 1929 and 1938 (WHO, 2016). The Sixth Decennial Revision Conference was held in 1948 in France and the first revision of ICD (ICD-6) begun in this conference. This conference launched a new period for the vital and health statistics. In addition to the approving the international rules for the underlying cause of death, the conference suggested the international cooperation for vital statistics (WHO, 2016). From the past to the present, many changes have been made regarding causes of death with ICD

revisions. With the latest changes, the most recent version of the revisions, ICD-11, was launched on 1st January 2022[1].

Although the ICD provides a standardised list of causes of death and guidelines for identifying the Underlying Cause of Death (UCoD) with each revision, Naghavi and colleagues (2010) highlighted three problems that arise in comparisons of public health analysis. Firstly, with the revisions in the ICD codes, there have been changes in the causes of death and the codes assigned for these causes. Especially, for time series analysis, cause of death data needs to be compatible with ICD revisions. The second problem is that with the ICD-6 revision, some tabulation lists of causes of death were provided. Although these aggregated lists enabled the practical reporting of cause of death data for developing countries, they also caused the loss of details of UCoD. The last problem is that with the ICD-6 revision, other than ICD codes for UCoD, inclusion of some ICD codes for defining medical background of the deceased such as reasons for admission to hospital etc., have created complexity in the coding process. Although these codes were introduced as conditions that unlikely to cause death (WHO, 2016), they are used as UCoD (Naghavi et al., 2010).

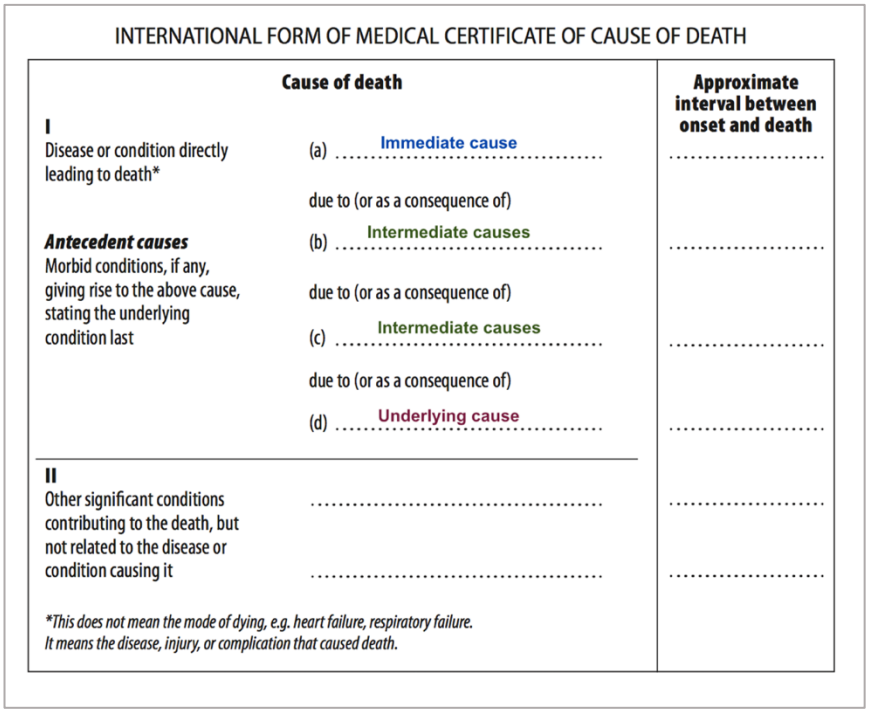
II.2.3. Frameworks for Classification and Assignment of Garbage Codes

Comparability of cause of death data is only possible using a standard death certificate and standard coding. Figure II.2.1 shows the international form of death certificate. This form gives information about the chain of events leading to death. Underlying cause refers to the disease or injury that triggers the events leading to death; intermediate causes refer to events arising from the underlying cause; and immediate cause refers to the final event resulting in death. Among these three types of causes, only underlying causes are reported for statistical purposes (WHO, 2016). According to definition of WHO, UCoD is defined as "the disease or injury which initiated the train of morbid events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury" (WHO, 2016). Incorrect

[1] https://www.who.int/standards/classifications/classification-of-diseases

certification of UCoD stem from either incorrect diagnosis or incomplete information of cause of death data. Garbage codes cannot denote the UCoD (senility, headache etc.) (Mikkelsen et al., 2018). They show a symptom or condition which takes part in the chain of causes leading to death and garbage codes are insufficient to define a cause of death (Mikkelsen et al., 2018). When we look at the literature, we see that there is not a standard classification for garbage codes. The best known and most widely used classifications belong to Global Burden of Disease studies, WHO and ICD.

Figure II.2.1. International medical certification of cause of death



Global Burden of Disease study: Naghavi and colleagues (2010) developed four types of garbage codes for 2010 GBD study. Type 1 includes causes which cannot be considered as UCoD such as essential primary hypertension, atherosclerosis etc. Type 2 codes refer intermediate causes for example heart failure, septicaemia etc. Type 3 are immediate causes (cardiac arrest, respiratory failure etc.). Type 4 defines causes which are specified insufficiently in ICD chapters (ill-defined cancer sites etc.). The detailed table of garbage codes proposed by (Naghavi et al., 2010) was presented in Appendix (Table A. 1). Apart from this classification, GBD proposed expanded lists of garbage codes in the next series.

World Health Organization (WHO): WHO proposed four types of garbage codes (WHO, 2018). In this dissertation, we have used these groupings for the garbage codes and details of these codes are provided in the methodology section. Classification and assignment of garbage codes were determined based on the studies (Ahern et al., 2011; Mathers et al., 2006; Stevens et al., 2010). This classification is also used by academic researchers (Cendales & Pardo, 2018; Mahapatra et al., 2007; Nalini et al., 2018; Plass et al., 2013; World Health Organization, 2010). WHO also proposed an expanded list as shown below (Table II.2.1):

Table II.2.1. Expanded list of garbage codes proposed by WHO (2018)

ICD-10 code(s)	**Description**
A40-A41	Streptococcal and other septicaemia
C76, C80, C97	Ill-defined cancer sites
D65	Disseminated intravascular coagulation [defibrination syndrome]
E86	Volume depletion
I10	Essential (primary) hypertension
I269	Pulmonary embolism without mention of acute cor pulmonale
I46	Cardiac arrest
I472	Ventricular tachycardia
I490	Ventricular fibrillation and flutter
I50	Heart failure
I514	Myocarditis, unspecified
I515	Myocardial degeneration
I516	Cardiovascular disease, unspecified
I519	Heart disease, unspecified
I709	Generalized and unspecified atherosclerosis
I99	Other and unspecified disorders of circulatory system
J81	Pulmonary oedema
J96	Respiratory failure, not elsewhere classified
K72	Hepatic failure, not elsewhere classified
N17	Acute renal failure
N18	Chronic renal failure
N19	Unspecified renal failure
P285	Respiratory failure of newborn
Y10-Y34, Y872	External cause of death not specified as accidentally or purposely inflicted

ICD-10th revision: The ICD-10 is a good guide for detail explanation for UCoD. The ICD manuals explain the rules and procedures for choosing immediate,

intermediate and underlying causes. On the other hand, it provides limited guidance for garbage codes except ill-defined codes (R codes) which are presented in detail in "Chapter XVIII Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified".

In addition to the above classifications, Naghavi and colleagues (2020) categorized the garbage codes according to the importance level for policy implications by answering these questions: Which codes should be prioritized in policy making and which are more problematic? Regarding these questions they classified garbage codes into 4 levels. Level 1 (very high) includes the garbage codes which require serious policy implications. In this level we cannot know the broad disease category (communicable, noncommunicable or injury) of the garbage code. In, level 2 (high) we know the garbage code that true UCoD is probably belong to one or two of the broad disease categories. Level 3 (medium) includes the garbage codes for which the true UCoD probably belongs to the same ICD chapter, such as "unspecified cancer". This level of garbage codes has some policy value. In the last group, level 4 (low), garbage codes most probably belong to a unique disease or injury category. In this category, garbage codes do not create a significant impact for public health policies (Naghavi et al., 2020). According to this classification level 1-3 are unhelpful codes for policy implications.

II.2.4. Assignment of Garbage Codes

To improve the validity of public health analysis, garbage codes should be redistributed to target causes. For this reason, some researchers, GBD collaborators and WHO have suggested several methods for reassigning procedure (Ahern et al., 2011; Johansson et al., 2006; Mathers et al., 2006; Naghavi et al., 2010; Stevens et al., 2010; WHO, 2008, 2018). In the literature, there are four main methods for redistribution of garbage codes: multiple cause analysis, negative correlation, impairment and proportional redistribution (Johnson et al., 2021).

Multiple cause analysis is performed based on the cause of death certificates (Figure II.2.1). As it is seen from the Figure II.2.1, multiple cause data includes the a

sequence of circumstances leading to death. By using the intermediate and UCoD information on the certificate, garbage codes can be redistributed. Multiple cause analysis was used in several studies. Some of these studies used stochastic models for assignment of garbage codes (Ahern et al., 2011; Fink et al., 2012; Foreman et al., 2016). Ahern and colleagues (2011) proposed a method for determine and redistribution of garbage codes. They used regression analysis grouped by age, sex and country development status. After analysis some predicted proportions for redistribution of garbage codes were obtained. As a case example they applied this method for heart failure. Findings showed that for all countries, ischemic heart disease had the highest proportion, and second highest proportion was hypertensive diseases for developing countries and cardiomyopathy and chronic obstructive pulmonary disease for developed countries (Ahern et al., 2011). Foreman and colleagues (2016) recommended a Bayesian mixed effects multinomial logistic model to redistribute garbage codes on more plausible causes. For this aim they used the death certificates of US for years between 1979 and 2011. Accordingly, they clarified that death certificates include too much evidence for real UCoD. Generally, a plausible underlying cause was among the causes that contribute to death. By using proposed method, 48% of heart failure, 25% of ischemic heart disease, 15% of chronic respiratory diseases were redistributed.

Other than stochastic models, some studies used the coarsened exact matching approach based on the medical death certificate (Agnieszka Fihel & Muszyńska-Spielauer, 2021; Stevens et al., 2010). Stevens and colleagues (2010) proposed the coarsened exact matching based on the multiple cause of death on certification of death. They used this method for redistribution of heart failure assigned as UCoD. They conducted the analysis for three countries: US, Brazil and Mexico and used demographic and autopsy matches for death which were autopsied. According to the findings, IHD had the highest percentage of heart failure deaths. Redistributed proportions of chronic chronic obstructive pulmonary disease, diabetes, and hypertensive heart disease were larger in Mexico and Brazil than in the US. Fihel and Spielauer (2021) also used the coarsened exact matching for elimination of cardiovascular garbage codes. Furthermore, some studies performed expert judgement

to determine the associations between causes of death (Naghavi et al., 2010). Based medical certification of cause of death, Naghavi and colleagues (2010) performed a combination of several models (proportionate redistribution, stochastic models, and expert judgement) to determine and assign garbage codes. As a limitation, although multiple cause analysis is very useful for analyzing causes of death, obtaining such kind of data is not always possible (Johnson et al., 2021).

According to the negative correlation method, there is a significant negative relationship between some garbage codes and some UCoD, i.e. some garbage codes increase as some underlying causes decrease. This method is generally applied for garbage codes undetermined diabetes, unspecified stroke, and undetermined malignant neoplasm (Johnson et al., 2021; Vos et al., 2020). Fihel and Mesle (2016) proposed a redistribution procedure for garbage cardiovascular diseases by using negative correlation method. They practiced the application for four countries: Czech Republic, Poland, Russia and the United Kingdom. In the study they applied a statistical analysis which include statistically significant and negative correlations between garbage cardiovascular diseases and other well-defined causes. However, this method became unsuccessful for Poland and Russia. In the next step, for these countries, they kept out the disruptions of well-defined causes in time series and then assigned garbage codes. This study indicated that there is no unique solution for all countries regarding distribution of garbage codes (A. Fihel & Mesle, 2016).

According to impairment method, the GBD defines some causes which can be the result of multiple causes. This method is used when it is not possible to determine the real underlying cause of death and the measure of years lived with disability (YLDs) is used for redistribution process (Johnson et al., 2021).

Lastly, proportional redistribution method reallocates garbage codes to related underlying causes proportionally by age, sex, province, year etc. Proportional redistribution is proposed for garbage codes which give no clue about underlying causes, for example R00-R99 codes (Johnson et al., 2021; Mathers et al., 2006; WHO, 2018).

II.2.5. Quality of death registration

Problems in health issues and health priorities in policy-making decisions would be better understood if there is a complete and comparable cause of death data by age and sex pattern in population (Lopez, 2013).

The basic product of death registration system is cause of death statistics. Rao and colleagues (2005) proposed 9 criteria under four categories to assess the quality of cause of death data. The first category is generalizability, that is representativeness of population. Coverage and completeness are two criteria for this category. In the reliability category, consistency of data is expected. "Consistency of the level of causes of death with general pattern of mortality" and "consistency of cause-specific mortality rates over time" are criteria for reliability of the cause of death data. Validity of the data is the most important category of the quality of the cause of death data (Rao et al., 2005). Use of ill-defined codes, consistency in age and sex specific causes of death are three criteria under content validity (information for the proportion of certificates certified by doctors; periodic comparisons for the accuracy of the causes of death). The last category for quality is policy relevance. Under this category timeliness and geographical disaggregation of cause of death data is determinant criteria. Franca and colleagues (2008) used these criteria for the assessment of cause of death data in Brazil for 2002-2004 period. They found that especially completeness and coding of garbage causes was very problematic. Total garbage codes were 18.3 percent and R00-R99 codes constituted the high share of garbage codes (13.1%). Garbage codes for cardiovascular diseases were 3.3% (França et al., 2008). Similar to the criteria proposed by Rao and colleagues (2005), Mahapatra and colleagues (2007) also evaluated the quality of death registration of almost all countries based on the pre-2005 cause of death data. Quality of death registration systems in some countries including, Turkey, India and China were found as limited use, which means "data before 1996 or completeness is less than 50% or data in non-standard format or data of partial coverage".

In GBD 2013, Mikkelsen and colleagues (2015) investigated the vital statistics performance index (VSPI) to measure the vital registration systems for 148 countries or territories for 1980-2012 period. VSPI includes 6 items: completeness of death registration, quality of death certification, cause specific mortaliy level, quality of age and sex registration, and timeliness of data (Philips et al., 2014). VSPI ranges between 0 and 1; as it closes to 1, it means a better vital registration performance and vice versa. Categorization is defined as "very low (<0.25)" , "low (0.25-0.49)", "medium (0.50-0.69)", "high (0.70-0.84)", "very high (0.85-1.00)". Findings showed that especially European countries have had the strongest vital system since 1980. Completeness, data quality and detail of cause of death data are the main reasons for poor performing system (VSPI <0.70) (Mikkelsen et al., 2015). According to the study, although Turkey had poor performing vital system between 1980 and 2012, findings showed that, Turkey got substantial progress in vital system performance especially between 2010 and 2012 (increased from 0.25 in 2005-2009 period to 0.41 in 2010-2012 period).

Iburg and colleagues (2020) found a negative association between socio-demographic development index (SDI) and the quality of mortality data based on the cause of death data from 20 countries, including Turkey. According to findings, in all 20 countries, heart failure, senility and other ill-defined causes were the most commonly coded garbage codes. Countries with lower socio-demographic development index (SDI) generally had higher proportion of more severe garbage codes than higher SDI countries. Main reason for the high share of garbage codes in low- and middle-income countries was due to lack of medical assistance in most of deaths. On the other hand, findings of the study also revealed that five countries including Turkey in low and middle SDI group had well performing death registration system (Iburg, Mikkelsen, & Richards, 2020). Iburg and colleagues (2020) also measured the VSPI quality of selected countries. According to the 2015 dataset from the WHO database, Turkey's VSPI quality was measured between 0.70-0.84, which means it has a well-functioning system.

In most countries, records of cause of death are reported by medical doctors or practitioners. Compare to developed countries, in developing countries, deaths are

more underreported and causes of death data lacks the medical attention (Mathers et al., 2005). Research findings show that two-thirds of deaths worldwide have poor data quality and that many deaths, particularly in low- and middle-income countries, occur where there are no health personnel available to document the cause of death (Adair et al., 2020; de Savigny et al., 2017). On the other hand, one study discloses that lack of physicians does not necessarily mean that the quality of death registration is poor. In Greenland, there is not any physician in some settlements. In these places deaths are certified by nurse or another official. Consequently, VSPI quality of Greenland was found higher than expected (Iburg, Mikkelsen, & Richards, 2020).

In GBD 2016, another system was developed for measurement of vital registration systems using a star rating index between 0 and 5 (Naghavi et al., 2017). This system uses the completeness level and proportion of age standardized major garbage causes. Then these two components are used for determination percent well certified (PWC) value. According to this PWC value star rating is defined. For example, if a vital registration system has a PWC between 0.1 and 0.35 ($0.1 \leq PWC < 0.35$), the vital registration system receives 2 stars. Johnson and colleagues (2021) used the star rating index for GBD countries to evaluate the quality of death data with 2015 or more recent data. The study also includes a detailed appendix presenting the star rating index and percentages of garbage codes by country. According to the findings, Turkey received 4 stars ($0.65 \leq PWC < 0.85$) for vital registration quality. The percentage of the first 5 garbage codes (1-heart failure unspecified right or left, 2-left heart failure, 3-sepsis (non-maternal and neonatal sepsis), 4-all ill-defined code for causes of death and 5-shock, cardiac arrest, coma) in total deaths is 10.09%.

França and colleagues (2020) assesses the change in the quality of cause of death by regional level for 1996-2016 period. Findings showed that total garbage code level decreased nearly 40%. This decline was stem from the decline of R-codes and related to the health policy which aimed to increase the investigation of deaths occurred at home and hospitals performed by Ministry of Health since 2005. Furthermore, decline in garbage codes occurred mostly in low-medium SDI regions. The highest proportion of garbage codes was among older ages especially age 80 and

over which was an expected result due to the high number of comorbidities at older ages (França et al., 2020).

Cendales and Pardo (2018) evaluated the quality of cause of death data in Colombia for 2007-2011 period for 5 cities. They used the four types of garbage code proposed by (WHO, 2017). Study disclosed that if certification was not certified by physician proportion of sign, symptom or ill-defined condition and injuries with intention is not determined were higher; if certification was issued by a physician, then cancer deaths for unspecified sites and garbage cardiovascular deaths were higher.

Murray and colleagues (2006) identified the effect of individual and community factors on the probability of being certified a death as one of the three cardiovascular garbage codes (general atherosclerosis and unspecified heart disease, heart failure, and cardiac arrest) by using multinomial logistic regression. According to findings, race and sex factors were strongly associated with being assigned as garbage cardiovascular diseases. Blacks and Native Americans were among the highest risk groups (Murray et al., 2006).

In another study, Murray and colleagues (2008) investigated the individual and community factors on being certified as diabetes by using multinomial logistic regression model. In this study they selected the deaths if diabetes was registered as a contributory cause of death and assigned as UCoD rather than cardiovascular, other noncommunicable and communicable diseases. Results disclosed that if death occurred in hospitals or if there was an autopsy or if decedents had higher BMI, higher education level, or insurance than deaths with diabetes which was one of the multiple contributing causes were most probably being assigned as cardiovascular diseases as UCoD (Murray et al., 2008).

II.3. Data and Method

II.3.1. Data Sources

The cause of death and population data for this dissertation were taken from TurkStat by sex and single age up to age 100 and over (100+). In the cause of death dataset, each row corresponded to one death, and age, sex, place of residence and ICD-10 code were available for each death.

II.3.1.1. Data Preparation

In the population dataset, we included only citizens of the Republic of Turkey. This is because while the majority of legally residing foreigners in Turkey are registered in the population registration system, only a negligible portion of this group is included in the death registration system (Yayla, 2016). To provide the correspondence between event (death) and exposure population, we excluded legally residing population from population dataset. Moreover, immigrants other than legally residing foreigners do not pose a problem for our data, as they are not registered in both the cause of death and civil registration systems. In the next step, we calculated the mid-year population for each year between 2013 and 2019. Mid-year population for each year between 2013 and 2019 is presented below (Table II.3.1).

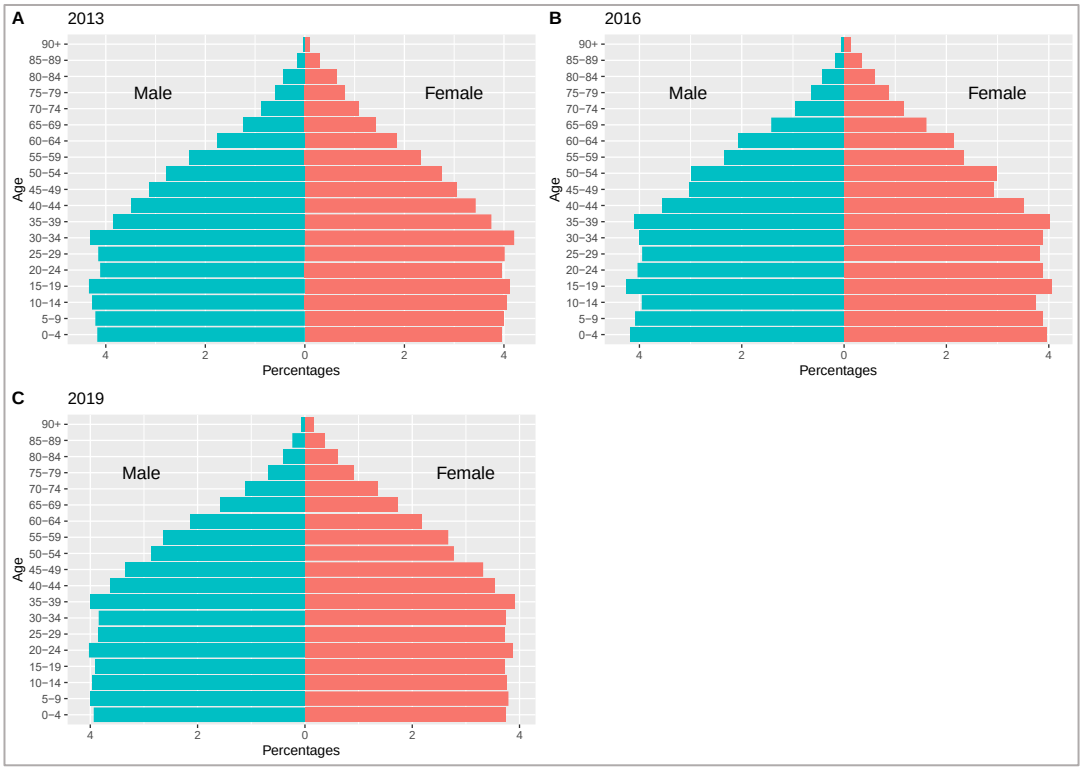
Table II.3.1. Mid-year population for the years between 2013-2019*

2013	**Total**	**75,166,434**
	Male	37,731,477
	Female	37,434,958
2014	**Total**	**76,054,464**
	Male	38,170,552
	Female	37,883,912
2015	**Total**	**76,997,548**
	Male	38,643,660
	Female	38,353,888
2016	**Total**	**77,917,435**
	Male	39,102,149
	Female	38,815,287
2017	**Total**	**78,827,508**
	Male	39,548,787
	Female	39,278,721
2018	**Total**	**79,739,250**
	Male	40,002,149
	Female	39,737,101
2019	**Total**	**80,637,010**
	Male	40,448,042
	Female	40,188,969

* Population data includes only citizens of the Republic of Turkey.

Furthermore, Figure II.3.1. shows the population distribution by sex and age group for some selected years (2013, 2016 and 2019). According to this figure, population distributions are almost identical in all pyramids, as there is not a large time gap between 2013 and 2019. Two patterns are evident in the pyramids: first, the population at child and adult ages is highly dense, and second, the female population becomes higher than the male population as age increases. Turkey's population structure is similar to that of developed countries.

Figure II.3.1. Population pyramids for 2013, 2016 and 2019.



II.3.1.2. Cause of death data

Prior to data analysis, three important data preparation steps have been applied to cause of death data. In the first step four-digit ICD-10 codes were converted into broad cause categories according to the cause categories listed in Annex Table A of the WHO technical paper for Global Health Estimates (GHE) (WHO, 2018). Table II.3.2 shows the broad cause categories used for this dissertation. In the second step, deaths of unknown sexes were redistributed proportionally within cause-age groups of known sexes. Then, deaths of unknown ages are redistributed proportionally within cause-sex groups of known ages. Total number of deaths of unknown sex and age is presented in Appendix Table A.2 and Table A.3. In the final step, for each broad cause categories, total number of deaths by cause, age and sex were calculated, and some ICD-10 codes were mapped as garbage codes and then garbage codes were redistributed.

Garbage codes are causes that do not provide information for UCoD. Before the analysis, these codes should be redistributed for a valid analysis result (Mathers et al., 2006; Naghavi et al., 2010).

According to the GHE cause list, there are four types of garbage codes. Garbage code-1 includes ICD-10 codes from R00 to R99 except R95. These codes are referred as the “Symptoms, signs and ill-defined conditions” (WHO, 2018). Garbage code-2 is ICD-10 codes Y1, Y2, Y30, Y31, Y32, Y33, Y34, Y872. These codes are injuries where the intent is not determined. Garbage code-3 are neoplasms of other and unspecified sites (C76, C80, and C97). Garbage code-4 contains heart failure, ventricular dysrhythmias, generalized atherosclerosis and ill-defined complications of heart diseases (I472, I490, I46, I50, I514, I515, I516, I519 and I709) (WHO, 2018). Table II.3.3. presents the total number of garbage codes by four types and total number of deaths.

After the identification of garbage codes, we redistributed them to target causes as recommended by WHO (2018) and Mathers and colleagues (2006). Garbage code-1 (R00-R94, R96-R99) was redistributed proportionally by age and sex to non-injury causes of death. Garbage code-2 (Y10-Y34, Y872) was redistributed proportionally by age and sex to all injury causes of death. Garbage code-3 (C76, C80, C97) was redistributed by age and sex to all sites excluding liver, pancreas, ovary and lung cancers. Garbage code-4, garbage cardiovascular diseases were redistributed by proportions to target causes presented below table (Table II.3.4.). Proportions and target causes in this table are suggested by WHO for Eastern European and central Asian countries (WHO, 2018). As described in the "Literature Review" section, there are other methodologies for the identification of GCs. Since we did not have medical documentation for any deaths, we could not use any multiple cause analysis. Furthermore, since the impairment assignment method requires morbidity data and the negative correlation method is only valid for some causes, we used proportional distribution methods for GC-1, GC-2 and GC-3 and WHO-recommended fixed proportions for GC-4.

Table II.3.2. Cause of death groups and corresponding ICD-10 codes

Table I.3.1.2.1. Cause of death groups and corresponding ICD-10 codes

Communicable, maternal, perinatal and nutritional conditions	**ICD-10 codes**	**Noncommunicable diseases**	**ICD-10 codes**	**Injuries**	**ICD-10 codes**
Infectious and parasitic diseases	A00-B99, G00-G04, G14, N70-N73, P37.3, P37.4	Malignant neoplasms	C00-C97	Unintentional injuries	V01-X40, X43, X46-59, Y40-Y86, Y88, Y89
Respiratory infectious	H65-H66, J00-J22, P23, U04	Other neoplasms	D00-D48	Intentional injuries	X60-Y09, Y35-Y36, Y870, Y871
Maternal conditions	O00-O99	Diabetes mellitus	E10-E14 (minus E10.2-E10.29, E11.2-E11.29, E12.2, E13.2-E13.29, E14.2)		
Nutritional deficiencies	D50-D53, D64.9, E00-E02, E40-E46, E50-E64	Endocrine/blood/ immune disorders	D55-D64 (minus D64.9), D65-D89, E03-E07, E15-E34, E65-E88		
Neonatal conditions	P00-P96 (minus P23, P37.3, P37.4)	Mental and substance use disorders	F04-F99, G72.1, Q86.0, X41-X42, X44, X45		
		Neurological conditions	F01-F03, G06-G98 (minus G14, G72.1)		
		Sense organ diseases	H00-H61, H68-H93		
		Cardiovascular diseases	I00-I99		
		Respiratory diseases	J30-J98		
		Digestive diseases	K20-K92		
		Genitourinary diseases	E10.2-E10.29,E11.2-E11.29,E12.2,E13.2-E13.29,E14.2, N00-N64, N75-N76, N80-N98		
		Skin diseases	L00-L98		
		Musculoskeletal diseases	M00-M99		
		Congenital anomalies	Q00-Q99 (minus Q86.0)		
		Oral conditions	K00-K14		
		Sudden infant death syndrome.	R95		

Table II.3.3. Total number of garbage codes and deaths

	Year	R-codes (GC-1)	Garbage injuries (GC-2)	Garbage cancer sites (GC-3)	Garbage CVD (GC-4)	Total # GCs	Total # deaths
Female	2013	3879	0	820	12389	17088	161964
	2014	4535	0	828	13708	19071	174779
	2015	3535	0	839	15112	19486	181022
	2016	4105	0	918	16836	21859	191199
	2017	3724	2	910	17560	22196	190680
	2018	4115	0	941	17677	22733	190397
	2019	3026	1	894	17639	21560	188334
Male	**Year**	**R-codes (GC-1)**	**Garbage injuries (GC-2)**	**Garbage cancer sites (GC-3)**	**Garbage CVD (GC-4)**	**Total # GCs**	**Total # deaths**
	2013	4499	0	1049	10096	15644	198909
	2014	4609	0	1242	11518	17369	208897
	2015	3993	0	1184	13177	18354	216015
	2016	4908	0	1242	14857	21007	228990
	2017	4611	7	1279	15714	21611	226201
	2018	6089	0	1346	15802	23237	226644
	2019	3430	3	1257	16185	20875	222066

Table II.3.4. Redistribution fractions and target causes for garbage cardiovascular causes of death (ICD10 4-digit codes I472, I490, I46, I50, I514, I515, I516, I519, and I709) (WHO, 2018)

Global Health Estimates (GHE) target causes								
	Redistribution fractions for females				**Redistribution fractions for males**			
	Cardiovascular diseases (I00-I99)	Cardiomyopathy,myocarditis,endocarditis (I30-I33,I38,I40,I42)	Chronic obstructive pulmonary disease (J40-J44)	Congenital heart anomalies (Q20-Q28)	Cardiovascular diseases (I00-I99)	Cardiomyopathy,myocarditis,endocarditis (I30-I33,I38,I40,I42)	Chronic obstructive pulmonary disease (J40-J44)	Congenital heart anomalies (Q20-Q28)
0	0.00	0.03	0.00	0.97	0.00	0.03	0.00	0.97
1-4	0.01	0.12	0.02	0.85	0.01	0.10	0.03	0.86
5-9	0.01	0.24	0.04	0.71	0.03	0.21	0.04	0.72
10-14	0.04	0.26	0.08	0.62	0.05	0.30	0.06	0.59
15-19	0.15	0.27	0.09	0.49	0.18	0.36	0.08	0.38
20-24	0.42	0.22	0.13	0.23	0.46	0.30	0.09	0.15
25-29	0.55	0.23	0.10	0.12	0.57	0.29	0.06	0.08
30-34	0.58	0.24	0.10	0.08	0.65	0.26	0.06	0.03
35-39	0.66	0.21	0.09	0.04	0.72	0.21	0.05	0.02
40-44	0.73	0.16	0.09	0.02	0.78	0.16	0.05	0.01
45-49	0.77	0.13	0.09	0.01	0.81	0.13	0.06	0.00
50-54	0.89		0.11		0.92		0.08	
55-59	0.89		0.11		0.9		0.1	
60-64	0.89		0.11		0.88		0.12	
65-69	0.90		0.10		0.86		0.14	
70-74	0.91		0.09		0.85		0.15	
75-79	0.92		0.08		0.85		0.15	
80-84	0.93		0.07		0.86		0.14	
85+	0.94		0.06		0.89		0.11	

II.4. Results

Figure II.4.1 presents the percentage distribution of garbage codes for the relevant year. According to this figure, garbage cardiovascular diseases (GC-4) constituted the major portion of the garbage codes. In 2019, this portion was around 7% for males and higher for females at 9%. Symptoms, signs and ill-defined conditions (GC-1) decreased to the level of 1.6% among females and 1.5% among males in 2019. GC-3, which defines garbage cancer sites, was stable over the years and remained at around 0.5% for both sexes. Between 2013-2019 period, total percentage of garbage codes increased from 7.9% to 9.4% among males and from 10.6% to 11.4% among females.

Figure II.4.1. Percent distribution of garbage codes: 2013-2019

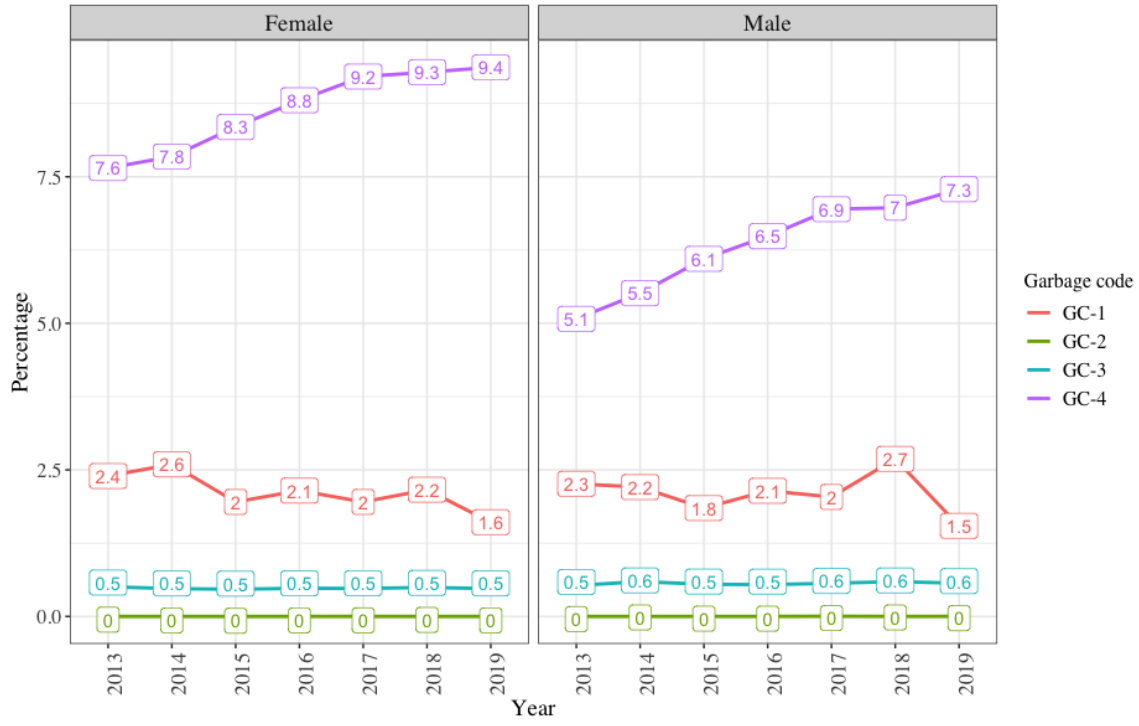


Table II.4.1 and Table II.4.2 show the percentage distribution of the four types of garbage codes in total garbage causes among females and males, respectively. According to Table II.4.1, garbage CVD has the highest share of total garbage, with R-codes ranking second. All kind of garbage codes decreased between 2013 and 2019 period. Also, the percentage of garbage codes increases with increasing age. The same

pattern applies to males (Table II.4.2). When females and males are compared, females have a higher percent of garbage CVD than males, while males have a higher percent of R-codes and garbage cancer sites than females.

Table II.4.1. Percentage distribution of garbage codes, female 2013 and female 2019

Female, 2013						Female, 2019					
Age group	**R-codes (GC-1)**	**Garbage injuries (GC-2)**	**Garbage cancer sites (GC-3)**	**Garbage CVD (GC-4)**	**Total**	**Age group**	**R-codes (GC-1)**	**Garbage injuries (GC-2)**	**Garbage cancer sites (GC-3)**	**Garbage CVD (GC-4)**	**Total**
0	1.16	0.00	0.01	0.34	1.51	**0**	0.07	0.00	0.00	0.14	0.21
1-4	0.45	0.00	0.01	0.17	0.63	**1-4**	0.15	0.00	0.03	0.14	0.32
5-9	0.19	0.00	0.01	0.08	0.29	**5-9**	0.03	0.00	0.02	0.04	0.09
10-14	0.21	0.00	0.01	0.08	0.29	**10-14**	0.03	0.00	0.02	0.05	0.10
15-19	0.17	0.00	0.00	0.08	0.25	**15-19**	0.09	0.00	0.01	0.03	0.14
20-24	0.18	0.00	0.02	0.08	0.28	**20-24**	0.07	0.00	0.01	0.07	0.16
25-29	0.19	0.00	0.04	0.06	0.29	**25-29**	0.07	0.00	0.02	0.08	0.18
30-34	0.31	0.00	0.08	0.09	0.48	**30-34**	0.12	0.00	0.02	0.12	0.26
35-39	0.25	0.00	0.06	0.19	0.50	**35-39**	0.23	0.00	0.06	0.22	0.51
40-44	0.39	0.00	0.16	0.27	0.82	**40-44**	0.24	0.00	0.12	0.35	0.71
45-49	0.33	0.00	0.20	0.58	1.11	**45-49**	0.30	0.00	0.18	0.62	1.10
50-54	0.42	0.00	0.38	0.87	1.67	**50-54**	0.36	0.00	0.26	0.93	1.55
55-59	0.54	0.00	0.44	1.47	2.45	**55-59**	0.43	0.00	0.40	1.90	2.73
60-64	0.64	0.00	0.51	2.70	3.86	**60-64**	0.65	0.00	0.40	3.15	4.21
65-69	0.74	0.00	0.59	3.80	5.13	**65-69**	0.83	0.00	0.53	4.78	6.14
70-74	1.49	0.00	0.53	7.28	9.29	**70-74**	1.12	0.00	0.49	7.40	9.02
75-79	2.20	0.00	0.63	10.70	13.53	**75-79**	1.52	0.00	0.53	11.86	13.92
80-84	4.16	0.00	0.63	17.26	22.05	**80-84**	2.13	0.00	0.51	15.47	18.12
85+	8.69	0.00	0.50	26.39	35.58	**85+**	5.57	0.00	0.52	34.44	40.53
Total %	**22.70**	**0.00**	**4.80**	**72.50**	100.00	**Total %**	**14.04**	**0.00**	**4.15**	**81.81**	100.00
Total #	**3879**	**0**	**820**	**12389**	**17088**	**Total #**	**3026**	**1**	**894**	**17639**	**21560**

Table II.4.2. Percent distribution of garbage codes, male 2013 and male 2019

Male, 2013						Male, 2019					
Age group	**R-codes (GC-1)**	**Garbage injuries (GC-2)**	**Garbage cancer sites (GC-3)**	**Garbage CVD (GC-4)**	**Total**	**Age group**	**R-codes (GC-1)**	**Garbage injuries (GC-2)**	**Garbage cancer sites (GC-3)**	**Garbage CVD (GC-4)**	**Total**
0	1.31	0.00	0.01	0.42	1.74	**0**	0.10	0.00	0.00	0.16	0.25
1-4	0.61	0.00	0.01	0.19	0.81	**1-4**	0.15	0.00	0.00	0.14	0.29
5-9	0.28	0.00	0.01	0.04	0.33	**5-9**	0.05	0.00	0.02	0.09	0.15
10-14	0.29	0.00	0.03	0.12	0.44	**10-14**	0.06	0.00	0.01	0.08	0.15
15-19	0.74	0.00	0.02	0.22	0.98	**15-19**	0.17	0.00	0.03	0.14	0.34
20-24	0.79	0.00	0.02	0.15	0.95	**20-24**	0.30	0.01	0.02	0.19	0.51
25-29	0.85	0.00	0.06	0.19	1.10	**25-29**	0.24	0.00	0.03	0.16	0.44
30-34	0.74	0.00	0.03	0.18	0.95	**30-34**	0.25	0.00	0.02	0.30	0.57
35-39	0.82	0.00	0.09	0.39	1.30	**35-39**	0.34	0.00	0.09	0.49	0.92
40-44	0.97	0.00	0.10	0.71	1.78	**40-44**	0.44	0.00	0.07	0.68	1.19
45-49	1.31	0.00	0.26	1.11	2.67	**45-49**	0.68	0.00	0.20	1.40	2.28
50-54	1.43	0.00	0.43	2.20	4.07	**50-54**	0.84	0.00	0.28	2.44	3.56
55-59	1.61	0.00	0.68	3.41	5.70	**55-59**	1.31	0.00	0.65	4.06	6.03
60-64	1.61	0.00	0.87	4.23	6.70	**60-64**	1.68	0.00	0.76	6.26	8.70
65-69	1.70	0.00	0.89	5.45	8.03	**65-69**	1.50	0.00	0.95	8.01	10.47
70-74	2.21	0.00	1.09	8.03	11.33	**70-74**	1.63	0.00	0.93	9.30	11.86
75-79	2.58	0.00	0.99	10.46	14.03	**75-79**	1.52	0.00	0.83	11.39	13.75
80-84	3.95	0.00	0.75	13.57	18.27	**80-84**	1.84	0.00	0.57	11.96	14.38
85+	4.97	0.00	0.36	13.48	18.82	**85+**	3.33	0.00	0.54	20.29	24.16
Total %	**28.76**	**0.00**	**6.71**	**64.53**	**100.00**	**Total %**	**16.43**	**0.01**	**6.02**	**77.53**	**100.00**
Total #	**4499**	**0**	**1049**	**10096**	**15644**	**Total #**	**3430**	**3**	**1257**	**16185**	**20875**

Tables II.4.3-II.4.6 show the distribution of the ten leading causes of death by age groups for 2013, before and after the distribution of garbage codes for females and males, respectively. According to Table II.4.3, garbage codes account for the third highest proportion after CVD and malignant neoplasms among females. The highest share of garbage codes was assigned to CVDs (Table II.4.4). A comparison of the distribution of deaths between males and females shows that males have higher percentages of malignant neoplasms, respiratory diseases and unintentional injuries than females in 2013 (Table II.4.3 and Table II.4.5). In addition to this, percentages of GCs are higher among females.

Tables II.4.7-II.4.10 present the distribution of the ten leading causes of death by age groups for 2019, before and after the distribution of garbage codes for females and males, respectively. Similar to 2013, the highest proportion of garbage codes was assigned to CVDs. A comparison of percentages of deaths between 2013 and 2019 indicates that percentages of CVDs and malignant neoplasms decreased for both sexes. On the other hand, percentages of neurological conditions, respiratory infectious and infectious and parasitic diseases and garbage codes increased for both sexes between 2013 and 2019 period.

Table II.4.3. Percent distribution of cause of death before redistribution of GCs, female 2013

Age group	Cardiovascular diseases	Malignant neoplasms	Respiratory diseases	Neurological conditions	Genitourinary diseases	Diabetes mellitus	Resp.inf & Inf.parasiticdis.	Neonatal cond.	Unintentional injuries	Other	GC	Total
0	0.02	0.01	0.03	0.07	0.01	0.00	0.19	1.79	0.04	1.37	0.16	3.69
1-4	0.01	0.06	0.03	0.11	0.01	0.00	0.09	0.01	0.16	0.27	0.07	0.82
5-9	0.01	0.06	0.01	0.05	0.01	0.00	0.04	0.00	0.09	0.08	0.03	0.38
10-14	0.02	0.05	0.00	0.05	0.01	0.00	0.02	0.00	0.07	0.08	0.03	0.34
15-19	0.03	0.06	0.01	0.04	0.01	0.00	0.03	0.00	0.10	0.15	0.03	0.45
20-24	0.03	0.07	0.01	0.04	0.01	0.00	0.03	0.00	0.10	0.12	0.03	0.43
25-29	0.04	0.12	0.02	0.04	0.01	0.00	0.03	0.00	0.09	0.15	0.03	0.54
30-34	0.09	0.21	0.01	0.03	0.02	0.01	0.02	0.00	0.09	0.18	0.05	0.72
35-39	0.14	0.37	0.02	0.04	0.02	0.01	0.04	0.00	0.08	0.17	0.05	0.95
40-44	0.27	0.57	0.04	0.04	0.03	0.03	0.05	0.00	0.10	0.15	0.09	1.38
45-49	0.48	0.84	0.07	0.06	0.07	0.05	0.07	0.00	0.10	0.16	0.12	2.01
50-54	0.71	1.20	0.13	0.06	0.12	0.11	0.10	0.00	0.12	0.23	0.18	2.95
55-59	1.11	1.41	0.18	0.08	0.20	0.23	0.16	0.00	0.12	0.29	0.26	4.04
60-64	1.70	1.58	0.33	0.12	0.27	0.37	0.18	0.00	0.12	0.40	0.41	5.47
65-69	2.58	1.75	0.50	0.16	0.39	0.56	0.28	0.00	0.15	0.48	0.54	7.37
70-74	4.14	1.99	0.77	0.33	0.55	0.81	0.41	0.00	0.20	0.67	0.98	10.85
75-79	5.94	1.94	1.09	0.67	0.63	0.95	0.55	0.00	0.23	0.78	1.43	14.22
80-84	8.64	1.85	1.59	1.19	0.78	1.08	0.76	0.00	0.35	1.07	2.33	19.63
85+	10.71	1.33	1.74	1.73	0.82	0.90	0.92	0.00	0.53	1.33	3.75	23.76
Total	**36.65**	**15.48**	**6.57**	**4.92**	**3.98**	**5.11**	**3.97**	**1.79**	**2.85**	**8.12**	**10.55**	**100.00**

Table II.4.4. Percent distribution of cause of death after redistribution of GCs, female 2013

Age group	Cardiovascular diseases	Malignant neoplasms	Respiratory diseases	Neurological conditions	Genitourinary diseases	Diabetes mellitus	Resp.inf & Infparasiticdis.	Neonatal cond.	Unintentional injuries	Other
0	0.02	0.02	0.03	0.07	0.01	0.00	0.20	1.85	0.04	1.46
1-4	0.02	0.07	0.04	0.12	0.01	0.00	0.10	0.01	0.16	0.30
5-9	0.02	0.07	0.01	0.06	0.01	0.00	0.05	0.00	0.09	0.09
10-14	0.02	0.06	0.01	0.06	0.01	0.00	0.03	0.00	0.07	0.09
15-19	0.03	0.07	0.02	0.04	0.01	0.00	0.03	0.00	0.10	0.16
20-24	0.04	0.07	0.01	0.04	0.01	0.00	0.03	0.00	0.10	0.13
25-29	0.04	0.14	0.02	0.04	0.01	0.00	0.03	0.00	0.09	0.16
30-34	0.10	0.23	0.01	0.04	0.02	0.01	0.02	0.00	0.09	0.19
35-39	0.16	0.38	0.02	0.04	0.03	0.01	0.04	0.00	0.08	0.18
40-44	0.31	0.61	0.04	0.04	0.03	0.03	0.06	0.00	0.10	0.16
45-49	0.54	0.87	0.07	0.06	0.07	0.05	0.07	0.00	0.10	0.16
50-54	0.81	1.26	0.14	0.06	0.12	0.11	0.11	0.00	0.12	0.23
55-59	1.27	1.48	0.20	0.09	0.21	0.23	0.16	0.00	0.12	0.30
60-64	1.97	1.65	0.36	0.12	0.28	0.37	0.19	0.00	0.12	0.41
65-69	2.97	1.83	0.54	0.16	0.39	0.56	0.28	0.00	0.15	0.48
70-74	4.91	2.08	0.85	0.33	0.56	0.82	0.42	0.00	0.20	0.68
75-79	7.09	2.04	1.20	0.69	0.64	0.97	0.56	0.00	0.23	0.80
80-84	10.55	1.96	1.76	1.22	0.80	1.11	0.78	0.00	0.35	1.09
85+	13.83	1.44	1.99	1.81	0.85	0.94	0.96	0.00	0.53	1.40
Total	**44.69**	**16.35**	**7.32**	**5.09**	**4.08**	**5.23**	**4.09**	**1.85**	**2.85**	**8.44**

Table II.4.5. Percent distribution of cause of death before redistribution of GCs, male 2013

Age group	Cardiovascular diseases	Malignant neoplasms	Respiratory diseases	Neurological conditions	Genitourinary diseases	Diabetes mellitus	Resp.inf & Infparasiticdis.	Neonatal cond.	Unintentional injuries	Other	GC	Total
0	0.02	0.01	0.04	0.06	0.01	0.00	0.19	1.90	0.05	1.21	0.14	3.62
1-4	0.01	0.05	0.02	0.11	0.01	0.00	0.08	0.00	0.20	0.26	0.06	0.80
5-9	0.01	0.06	0.01	0.04	0.01	0.00	0.02	0.00	0.13	0.09	0.03	0.40
10-14	0.02	0.06	0.00	0.06	0.00	0.00	0.05	0.00	0.16	0.11	0.03	0.49
15-19	0.03	0.09	0.01	0.07	0.01	0.00	0.04	0.00	0.42	0.24	0.08	0.98
20-24	0.04	0.08	0.01	0.06	0.01	0.01	0.03	0.00	0.40	0.32	0.07	1.03
25-29	0.07	0.12	0.02	0.03	0.01	0.00	0.03	0.00	0.41	0.29	0.09	1.07
30-34	0.14	0.16	0.02	0.04	0.02	0.01	0.05	0.00	0.37	0.28	0.07	1.16
35-39	0.23	0.27	0.03	0.04	0.03	0.02	0.06	0.00	0.36	0.26	0.10	1.40
40-44	0.53	0.50	0.05	0.04	0.05	0.03	0.08	0.00	0.35	0.29	0.14	2.05
45-49	0.96	1.06	0.12	0.06	0.07	0.08	0.11	0.00	0.37	0.32	0.21	3.35
50-54	1.50	1.88	0.22	0.08	0.12	0.15	0.14	0.00	0.34	0.40	0.32	5.15
55-59	2.22	2.82	0.45	0.10	0.22	0.25	0.21	0.00	0.31	0.47	0.45	7.49
60-64	2.81	3.42	0.70	0.12	0.29	0.32	0.26	0.00	0.29	0.52	0.53	9.26
65-69	3.25	3.46	0.97	0.17	0.33	0.38	0.33	0.00	0.25	0.52	0.63	10.29
70-74	4.04	3.38	1.38	0.29	0.48	0.48	0.41	0.00	0.28	0.59	0.89	12.21
75-79	4.80	2.99	1.58	0.54	0.52	0.50	0.49	0.00	0.31	0.63	1.10	13.47
80-84	5.44	2.46	1.83	0.77	0.62	0.47	0.61	0.00	0.30	0.70	1.44	14.64
85+	4.32	1.20	1.23	0.71	0.51	0.26	0.55	0.00	0.29	0.57	1.48	11.13
Total	**30.43**	**24.07**	**8.66**	**3.40**	**3.31**	**2.96**	**3.72**	**1.91**	**5.58**	**8.08**	**7.86**	**100.00**

Table II.4.6. Percent distribution of cause of death after redistribution of GCs, male 2013

Age group	Cardiovascular diseases	Malignant neoplasms	Respiratory diseases	Neurological conditions	Genitourinary diseases	Diabetes mellitus	Resp.inf & Infparasiticdis.	Neonatal cond.	Unintentional injuries	Other
0	0.02	0.01	0.04	0.07	0.01	0.00	0.19	1.96	0.05	1.28
1-4	0.01	0.06	0.02	0.11	0.01	0.00	0.09	0.00	0.20	0.29
5-9	0.01	0.07	0.01	0.04	0.01	0.00	0.02	0.00	0.13	0.10
10-14	0.03	0.07	0.00	0.06	0.00	0.00	0.05	0.00	0.16	0.12
15-19	0.05	0.11	0.01	0.08	0.01	0.00	0.04	0.00	0.42	0.26
20-24	0.05	0.10	0.01	0.07	0.01	0.01	0.03	0.00	0.40	0.34
25-29	0.10	0.15	0.02	0.04	0.02	0.00	0.04	0.00	0.41	0.30
30-34	0.16	0.18	0.02	0.05	0.02	0.01	0.05	0.00	0.37	0.29
35-39	0.28	0.30	0.03	0.04	0.03	0.02	0.07	0.00	0.36	0.27
40-44	0.61	0.53	0.05	0.05	0.05	0.03	0.08	0.00	0.35	0.30
45-49	1.08	1.12	0.13	0.06	0.07	0.08	0.12	0.00	0.37	0.33
50-54	1.70	1.96	0.24	0.08	0.12	0.15	0.15	0.00	0.34	0.41
55-59	2.51	2.93	0.48	0.10	0.22	0.26	0.22	0.00	0.31	0.47
60-64	3.14	3.54	0.75	0.13	0.29	0.33	0.27	0.00	0.29	0.53
65-69	3.66	3.58	1.04	0.18	0.33	0.39	0.33	0.00	0.25	0.53
70-74	4.64	3.52	1.49	0.30	0.48	0.49	0.41	0.00	0.28	0.60
75-79	5.58	3.12	1.73	0.55	0.53	0.50	0.49	0.00	0.31	0.64
80-84	6.49	2.58	2.02	0.79	0.64	0.48	0.62	0.00	0.30	0.72
85+	5.45	1.28	1.40	0.74	0.53	0.27	0.57	0.00	0.29	0.60
Total	**35.56**	**25.20**	**9.51**	**3.53**	**3.40**	**3.03**	**3.85**	**1.96**	**5.58**	**8.37**

Table II.4.7. Percent distribution of cause of death before redistribution of GCs, female 2019

Age group	Cardiovascular diseases	Malignant neoplasms	Respiratory diseases	Neurological conditions	Genitourinary diseases	Diabetes mellitus	Resp.inf & Infparasiticdis.	Neonatal cond.	Unintentional injuries	Other	GC	Total
0	0.01	0.01	0.02	0.03	0.01	0.00	0.10	1.25	0.02	0.87	0.02	2.33
1-4	0.01	0.03	0.02	0.09	0.01	0.00	0.06	0.00	0.07	0.17	0.04	0.48
5-9	0.01	0.04	0.00	0.04	0.01	0.00	0.02	0.00	0.04	0.05	0.01	0.23
10-14	0.01	0.04	0.01	0.04	0.00	0.00	0.01	0.00	0.04	0.05	0.01	0.22
15-19	0.02	0.05	0.00	0.05	0.00	0.00	0.03	0.00	0.05	0.12	0.02	0.34
20-24	0.03	0.06	0.01	0.03	0.01	0.00	0.03	0.00	0.06	0.12	0.02	0.35
25-29	0.03	0.08	0.01	0.03	0.01	0.00	0.03	0.00	0.05	0.10	0.02	0.34
30-34	0.06	0.16	0.01	0.03	0.01	0.01	0.04	0.00	0.05	0.12	0.03	0.52
35-39	0.12	0.31	0.02	0.03	0.02	0.01	0.05	0.00	0.05	0.12	0.06	0.80
40-44	0.20	0.45	0.03	0.03	0.03	0.02	0.08	0.00	0.05	0.11	0.08	1.07
45-49	0.38	0.71	0.06	0.05	0.06	0.04	0.12	0.00	0.08	0.16	0.13	1.80
50-54	0.54	0.98	0.11	0.06	0.11	0.07	0.18	0.00	0.07	0.17	0.18	2.47
55-59	0.94	1.35	0.17	0.08	0.18	0.17	0.27	0.00	0.08	0.27	0.31	3.83
60-64	1.50	1.62	0.28	0.12	0.30	0.27	0.45	0.00	0.09	0.32	0.48	5.43
65-69	2.22	1.73	0.43	0.19	0.45	0.39	0.61	0.00	0.10	0.43	0.70	7.26
70-74	3.17	1.79	0.65	0.34	0.59	0.59	0.94	0.00	0.12	0.58	1.03	9.80
75-79	4.87	1.72	1.01	0.67	0.80	0.68	1.42	0.00	0.17	0.72	1.59	13.67
80-84	6.20	1.45	1.25	1.13	0.91	0.68	1.83	0.00	0.22	0.87	2.07	16.61
85+	12.53	1.55	2.24	2.99	1.61	1.00	3.73	0.00	0.47	1.69	4.64	32.45
Total	**32.86**	**14.15**	**6.32**	**6.02**	**5.12**	**3.93**	**9.99**	**1.25**	**1.87**	**7.05**	**11.45**	**100.00**

Table II.4.8. Percent distribution of cause of death after redistribution of GCs, female 2019

Age group	Cardiovascular diseases	Malignant neoplasms	Respiratory diseases	Neurological conditions	Genitourinary diseases	Diabetes mellitus	Resp.inf & Infparasiticdis.	Neonatal cond.	Unintentional injuries	Other
0	0.01	0.01	0.02	0.03	0.01	0.00	0.10	1.25	0.02	0.89
1-4	0.01	0.04	0.02	0.09	0.01	0.00	0.06	0.00	0.07	0.19
5-9	0.01	0.04	0.00	0.04	0.01	0.00	0.02	0.00	0.04	0.06
10-14	0.01	0.04	0.01	0.04	0.00	0.00	0.01	0.00	0.04	0.05
15-19	0.02	0.05	0.00	0.05	0.00	0.00	0.03	0.00	0.05	0.12
20-24	0.03	0.06	0.01	0.03	0.01	0.00	0.03	0.00	0.06	0.12
25-29	0.04	0.08	0.01	0.03	0.01	0.00	0.03	0.00	0.05	0.10
30-34	0.07	0.17	0.01	0.03	0.02	0.01	0.04	0.00	0.05	0.12
35-39	0.15	0.33	0.02	0.03	0.03	0.01	0.05	0.00	0.05	0.13
40-44	0.24	0.48	0.03	0.03	0.03	0.02	0.08	0.00	0.05	0.11
45-49	0.46	0.75	0.07	0.05	0.06	0.04	0.12	0.00	0.08	0.16
50-54	0.65	1.03	0.12	0.06	0.11	0.08	0.18	0.00	0.07	0.17
55-59	1.15	1.42	0.20	0.08	0.18	0.17	0.27	0.00	0.08	0.27
60-64	1.84	1.70	0.32	0.12	0.31	0.27	0.46	0.00	0.09	0.33
65-69	2.74	1.82	0.49	0.19	0.45	0.40	0.62	0.00	0.10	0.44
70-74	3.99	1.87	0.73	0.35	0.59	0.60	0.96	0.00	0.12	0.59
75-79	6.19	1.81	1.13	0.68	0.82	0.69	1.44	0.00	0.17	0.73
80-84	7.95	1.54	1.40	1.15	0.93	0.69	1.86	0.00	0.22	0.88
85+	16.53	1.65	2.53	3.06	1.64	1.02	3.81	0.00	0.47	1.73
Total	**42.11**	**14.89**	**7.13**	**6.15**	**5.22**	**4.00**	**10.18**	**1.25**	**1.88**	**7.21**

Table II.4.9. Percentage distribution of leading cause of death before redistribution of GCs, male 2019

Age group	Cardiovascular diseases	Malignant neoplasms	Respiratory diseases	Neurological conditions	Genitourinary diseases	Diabetes mellitus	Resp.inf & Infparasiticdis.	Neonatal cond.	Unintentional injuries	Other	GC	Total
0	0.01	0.01	0.01	0.03	0.00	0.00	0.12	1.37	0.02	0.85	0.02	2.45
1-4	0.01	0.04	0.02	0.08	0.01	0.00	0.05	0.00	0.10	0.17	0.03	0.50
5-9	0.01	0.05	0.00	0.04	0.00	0.00	0.02	0.00	0.05	0.05	0.01	0.24
10-14	0.01	0.05	0.01	0.04	0.00	0.00	0.02	0.00	0.07	0.06	0.01	0.27
15-19	0.03	0.06	0.01	0.08	0.00	0.00	0.04	0.00	0.19	0.16	0.03	0.60
20-24	0.04	0.08	0.01	0.05	0.01	0.00	0.04	0.00	0.20	0.22	0.05	0.71
25-29	0.05	0.08	0.01	0.04	0.01	0.00	0.04	0.00	0.22	0.22	0.04	0.70
30-34	0.09	0.13	0.01	0.03	0.01	0.00	0.04	0.00	0.18	0.21	0.05	0.76
35-39	0.22	0.23	0.02	0.03	0.03	0.01	0.05	0.00	0.22	0.23	0.09	1.12
40-44	0.42	0.38	0.04	0.04	0.04	0.02	0.08	0.00	0.20	0.23	0.11	1.56
45-49	0.78	0.70	0.09	0.05	0.09	0.05	0.15	0.00	0.22	0.28	0.21	2.62
50-54	1.35	1.32	0.15	0.08	0.12	0.10	0.23	0.00	0.24	0.35	0.33	4.29
55-59	2.03	2.32	0.37	0.10	0.21	0.18	0.42	0.00	0.26	0.46	0.57	6.92
60-64	2.73	3.27	0.72	0.14	0.33	0.29	0.64	0.00	0.24	0.54	0.82	9.72
65-69	3.28	3.68	0.99	0.21	0.44	0.35	0.87	0.00	0.23	0.58	0.98	11.62
70-74	3.61	3.30	1.24	0.30	0.54	0.39	1.05	0.00	0.21	0.56	1.11	12.32
75-79	4.11	2.79	1.49	0.53	0.62	0.39	1.28	0.00	0.20	0.63	1.29	13.31
80-84	3.91	1.94	1.38	0.67	0.64	0.32	1.41	0.00	0.19	0.57	1.35	12.37
85+	6.13	1.64	1.83	1.29	0.97	0.38	2.19	0.00	0.30	0.88	2.27	17.90
Total	**28.81**	**22.04**	**8.39**	**3.86**	**4.08**	**2.48**	**8.74**	**1.37**	**3.56**	**7.27**	**9.40**	**100.00**

Table II.4.10. Percent distribution of cause of death after redistribution of GCs, male 2019

Age group	Cardiovascular diseases	Malignant neoplasms	Respiratory diseases	Neurological conditions	Genitourinary diseases	Diabetes mellitus	Resp.inf & Infparasiticdis.	Neonatal cond.	Unintentional injuries	Other
0	0.01	0.01	0.01	0.03	0.00	0.00	0.12	1.38	0.02	0.87
1-4	0.01	0.04	0.02	0.09	0.01	0.00	0.06	0.00	0.10	0.19
5-9	0.01	0.06	0.00	0.05	0.00	0.00	0.02	0.00	0.05	0.06
10-14	0.01	0.05	0.01	0.04	0.00	0.00	0.02	0.00	0.07	0.06
15-19	0.04	0.06	0.01	0.08	0.00	0.00	0.04	0.00	0.19	0.17
20-24	0.06	0.08	0.02	0.06	0.01	0.00	0.04	0.00	0.21	0.23
25-29	0.06	0.09	0.01	0.04	0.01	0.00	0.04	0.00	0.22	0.23
30-34	0.12	0.14	0.01	0.03	0.01	0.00	0.04	0.00	0.18	0.22
35-39	0.27	0.24	0.03	0.04	0.03	0.01	0.05	0.00	0.22	0.24
40-44	0.49	0.40	0.04	0.04	0.04	0.02	0.09	0.00	0.20	0.23
45-49	0.93	0.74	0.10	0.06	0.09	0.05	0.16	0.00	0.22	0.28
50-54	1.59	1.38	0.17	0.08	0.13	0.10	0.24	0.00	0.24	0.36
55-59	2.42	2.43	0.42	0.10	0.21	0.19	0.42	0.00	0.26	0.47
60-64	3.30	3.40	0.80	0.14	0.33	0.29	0.65	0.00	0.24	0.55
65-69	3.97	3.82	1.11	0.22	0.45	0.35	0.89	0.00	0.23	0.59
70-74	4.40	3.44	1.38	0.31	0.55	0.39	1.06	0.00	0.21	0.57
75-79	5.07	2.90	1.66	0.53	0.62	0.39	1.29	0.00	0.20	0.64
80-84	4.94	2.02	1.56	0.68	0.65	0.32	1.43	0.00	0.19	0.58
85+	7.96	1.72	2.08	1.32	0.99	0.39	2.24	0.00	0.30	0.90
Total	**35.66**	**23.02**	**9.44**	**3.94**	**4.16**	**2.52**	**8.90**	**1.38**	**3.56**	**7.43**

II.5. Conclusion and Discussion

This paper aims to assess the quality of cause of death data for the period 2013-2019 by addressing the issue of garbage codes. We used WHO (2018) recommendations for determination and redistribution process of garbage codes. According to the findings, garbage codes constituted the nearly 11% of total deaths among females and 9% of total deaths among males for 2013-2019 period. A study that applied the same methodology as our paper in determining garbage codes for the period 2009-2017 found similar percentages for total garbage codes (Teker et al., 2020). Furthermore, Özdemir and colleagues (2015) evaluated the death registration system in Turkey by comparing 2001-2008 and 2009-2013 periods by handling completeness of death registration and proportions of garbage causes. For garbage causes they calculated the R-codes and garbage cardiovascular diseases for the evaluation. The findings show that garbage causes declined from about 45% in 2000 to about 15% in 2009 and to about 10% in 2013 (Özdemir et al., 2015). In another study Özdemir and colleagues (2017) evaluated the Turkey's epidemiological process between 1980 and 2013. They measured two types of garbage codes: ill-defined signs and symptoms (R-codes) and garbage cardiovascular diseases. According to the findings, the percentage of garbage codes calculated for 2013 is consistent with the findings of our study for 2013 (Özdemir et al., 2017).

This study also indicated that garbage cardiovascular diseases (GC-4) constituted the highest share of garbage codes (in 2013, 73% for females and 65% for males) and continued to increase between 2013 and 2019 period. As a result, the highest proportion of garbage codes was assigned to the CVDs. In a study conducted for 6 developed countries also showed that heart failure (I50.9) was among the commonly used garbage code in these countries (Mikkelsen et al., 2020). Among these, Germany stands out as a country with the highest use of code I50.9 (Heart failure, unspecified). Johnson and colleagues (2021) investigated in detail the global use of garbage codes for 2015. Accordingly, globally, unspecified lower respiratory infectious had the highest percentage (nearly 29% for both sexes) of garbage coded deaths for under 1 year of age. For age 50 and over, unspecified type of stroke and

unspecified heart failure were the most prominent garbage codes. Johnson and colleagues (2021) also presented the country specific garbage codes including Turkey. Based on 2015 Turkey cause of death data, the top five garbage codes were heart failure unspecified left or right, left heart failure, sepsis (non-maternal and neonatal sepsis), R-codes, shock, cardiac arrest, and coma (Johnson et al., 2021). In another study (Iburg, Mikkelsen, Adair, et al., 2020) some countries including Turkey were selected according to the socio-demographic index (SDI) to evaluate the relationship between usage of garbage code and SDI. In the study, analysis for Turkey was performed with 2015 cause of death dataset taken from WHO. Results showed that among the top ten leading causes of death, heart failure unspecified (I50.9) was ranked third for males and second for females. In addition, while there are 5 garbage coded causes among the top ten leading causes of death for females, there are 3 garbage coded causes for males (Iburg, Mikkelsen, Adair, et al., 2020).

Findings of this essay also indicated that ill-defined signs and symptoms (R-codes) were at the second rank and although decline between 2013 and 2019 for both sexes, they still constituted around 15% of total garbage codes. In literature, R-codes are defined as the level-1 (very high) garbage codes; that means this type of garbage codes have no policy value for public health and should be prioritized in health policies (Naghavi et al., 2020). Furthermore, some codes in garbage cardiovascular diseases and garbage cancer sites are in level-2 (high) and some codes are in level-3 (medium) garbage codes. Although these codes have some policy values compared to the garbage codes in level-1, garbage codes in level 1-3 are high impact garbage codes which are obstacle for efficient health policies. Denmark and Japan are among the countries that use R-codes extensively (Mikkelsen et al., 2020). In depth analysis indicated that senility (R54) in Japan and R99 in Denmark are among the commonly used GCs. Some research findings suggest that R codes are more likely to be certified as the underlying cause of death in individuals with lower levels of education and less access to health services (França et al., 2020).

Results of our study also showed that proportion of garbage codes is higher among older ages. Some age groups tend to have less qualified cause of death data.

The higher prevalence of comorbidities such as diabetes mellitus, malignant neoplasms and hypertension among the elderly makes it difficult to determine UCoD (Alpérovitch et al., 2009; França et al., 2020). Similar to Turkey, in most of developed countries about 90% of all GCs occur among elderly (Mikkelsen et al., 2020). According to research assessing the unusable and insufficiently specified garbage codes in 6 high income countries (Australia, Canada, Denmark, Germany, Japan and Switzerland) (Mikkelsen et al., 2020), 18% of cause of death was unusable. Japan had the highest share in unusable (25%) and insufficiently specified causes (11%). Other ill-defined and unspecified deaths (R99), heart failure (I50.9) and senility (R54) were among the commonly used garbage codes. Mikkelsen and colleagues showed that garbage coding are significant problems among countries experiencing population ageing. Adair and colleagues (2019) disclosed that proportion of dementia was higher in Australia than in Japan. The significant disparity of registered dementia proportions was associated with the different certification practices of two countries. In another study (Iburg, Mikkelsen, Adair, et al., 2020) it was clarified that the cause "senility" (R54) is a frequently used garbage code in Japan. These two studies suggest that the higher rates of dementia in Australia may be a result of the higher use of the garbage code instead of dementia in Japan. Although GBD proposed methods for this kind of problems, quality of cause of death at older ages remain problematic (Adair et al., 2019; Alpérovitch et al., 2009).

According to the WHO assessment framework (WHO, 2010) there are some criteria for sufficient quality of cause of death data. According to these criteria, percentage of ill-defined causes (R00-R99) should be less than 10% for ages 65 and over and less than 5% for ages below 65. Furthermore, total percentage of garbage codes (garbage heart disease and heart failure; cancers with ill-defined site; injuries that intention is undetermined) should be less than 10-15% (WHO, 2010). Our results indicated that cause of death data for 2013-2019 period has sufficient quality of cause of death. The most qualitative data comes from injury-related deaths, as there are zero garbage injuries for the period 2013-2019. However, there are still some considerations regarding data quality. The increase in garbage cardiovascular diseases (GC-4) is an urgent issue that needs to be addressed with caution.

The cause of death is often positioned somewhere between “the medical profession’s understandable bias towards the living patient, and the public-health profession’s lack of interest in individual outcomes”, and this results with the loss of the most important part of the public health information (Lopez et al., 2007). Health policy value of civil registration system will increase significantly with correct certification and coding of cause of death (Lopez et al., 2007). The low quality is partly due to inadequate training of physicians in completing the ICD coding process or a lack of awareness of the public health importance of correct coding (Naghavi et al., 2020).

ICD emphasis that if another reasonable cause of death is available on the certificate, mode of death could not be used as UCoD (WHO, 2016). There are several reasons for misrecorded or misdiagnosed cause of death. First, physicians could not be accessed the detailed and accurate records to understand the real UCoD. Secondly, physicians may have inadequate training for completion of death certificate. Lastly, physicians may deliberately record incorrectly for some political or financial reasons (Ahern et al., 2011). Moreover, even if physicians may know the use of the ICD, they should be trained in certifying the chain of events leading to death to provide that coder can correctly determine the underlying cause of death (Mikkelsen et al., 2020).

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APPENDIX A. SUPPLEMENTARY TABLES FOR ESSAY 1

Table A.1. Garbage codes proposed by Naghavi and colleagues (2010)

GC Type	ICD-10 codes
Type 1	A31.1, A59, A60.0, A71-A74, A63.0, B00.0, B07, B08.1, B08.8, B30, B35-B36, F32-F33.9, F40-F42.9, F45-F48.9, F51-F53.9, F60-F98.9, G43-G45.9, G47-G52.9, G54-G54.9, G56-G58.9, H00-H04.9, H05.2-H69.9, H71-H80.9, H83-H93, J30, J33, J34.2, J35, K00-K11.9, K14, L04-L08.9, L20-L25.9, L28-L87.9, L90-L92, L94, L98.0-L98.3, L98.5-L98.9, M03, M07, M09-M12, M14-M25, M35.3, M40, M43.6-M43.9, M45.9, M47-M60, M63-M71, M73-M79, M95-M99, N39.3, N40, N46, N60, N84-N93, N97, Q10-Q18, Q36, Q38.1, Q54, Q65-Q74, Q82-Q84, R00-R99, B94.8, B949.9, G80-G83, Y86, Y87.2, Y89, I10, I15, I70
Type 2	A40-A41, A48.0, A48.3, E85.3-E85.9, E86-E87, G91.1, G91.3-G91.8, G92, G93.1-G93.6, I26, I27.1, I44-I45, I49-I50, I74, I81, J69, J80-J81, J86, J90, J93, J93.8-J93.9, J94, J98.1-J98.3, K65-K66, K71-K72 (except K71.7), K75, K76.0-K76.4, K92.0-K92.2, M86, N14, N17-N19
Type 3	D65, I45-I46, J96
Type 4	C80, C26, C39, C57.9, C64.9, C76, D00-D13, D16-D18, D20-D24, D28-D48, A49.9, B83.9, B99, E88.9 I51, I99, X59, Y10-Y34

Table A.2. Number of deaths of unknown sex

	Age	Cause of death	Number of death
2013	0	Congenital anomalies	24
	0	GC-1	2
	0	GC-4	1
	0	Neonatal conditions	18
	0	Sudden infant death syndrome	1
	Unknown	GC-1	2
	Unknown	Unintentional injuries	1
	Total		49
2014	0	Congenital anomalies	32
	0	GC-1	3
	0	Neonatal conditions	30
	0	Respiratory infectious	1
	0	Sudden infant death syndrome	1
	Unknown	Cardiovascular diseases	1
	Unknown	GC-1	2
	Unknown	Unintentional injuries	3
	Total		73
2015	0	Congenital anomalies	30
	0	Neonatal conditions	23
	0	Sudden infant death syndrome	2
	18	Unintentional injuries	1
	27	Intentional injuries	1
	45	Respiratory diseases	1
	50	Malignant neoplasms	1
	55	Unintentional injuries	1
	58	Cardiovascular diseases	1
	68	GC-4	1
	72	Cardiovascular diseases	1
	Unknown	GC-1	3
	Total		66
2016	0	Congenital anomalies	24
	0	Digestive diseases	1
	0	Endocrine disorders	1
	0	Neonatal conditions	27
	0	Respiratory infectious	1
	0	Sudden infant death syndrome	3
	36	GC-1	2
	63	Unintentional injuries	1
	80	Cardiovascular diseases	1
	Unknown	GC-1	3
	Unknown	Unintentional injuries	1
	Total		65
2017	0	Congenital anomalies	21
	0	Neonatal conditions	24
	0	Sudden infant death syndrome	2
	20	Infectious and parasitic diseases	1
	20	Unintentional injuries	1
	45	Unintentional injuries	1
	51	Respiratory infectious	1
	84	GC-1	1
	Unknown	GC-1	5
	Unknown	Unintentional injuries	1
	Total		58

Table A.3. Number of deaths of unknown age

	Sex	Cause of death	Number of death
2013	Female	GC-1	4
	Female	Unintentional injuries	3.2
	Male	Cardiovascular diseases	1
	Male	GC-1	4
	Male	Intentional injuries	4
	Male	Unintentional injuries	17.8
	Total		34
2014	Female	Cardiovascular diseases	2.2
	Female	GC-1	4.4
	Female	Intentional injuries	2
	Female	Respiratory diseases	1
	Female	Unintentional injuries	7
	Male	Cardiovascular diseases	4.8
	Male	Digestive diseases	1.0
	Male	GC-1	18.6
	Male	Intentional injuries	4.0
	Male	Malignant neoplasms	1.0
	Male	Respiratory diseases	1.0
	Male	Unintentional injuries	22.0
	Total		69.02
2015	Female	GC-1	4.6
	Female	Intentional injuries	1.0
	Female	Unintentional injuries	12.0
	Male	GC-1	18.4
	Male	Unintentional injuries	44.0
	Total		80.0
2016	Female	GC-1	6.6
	Female	Sudden infant death syndrome	1.0
	Female	Unintentional injuries	8.5
	Male	Cardiovascular diseases	4
	Male	Congenital anomalies	1
	Male	Digestive diseases	2
	Male	GC-1	24.4
	Male	Intentional injuries	5
	Male	Unintentional injuries	30.5
	Total		83.0
2017	Female	Cardiovascular diseases	1.0
	Female	GC-1	3.8
	Female	Intentional injuries	1.0
	Female	Respiratory diseases	1.0
	Female	Respiratory infectious	2.0
	Female	Unintentional injuries	1.0
	Male	Cardiovascular diseases	3.0
	Male	Digestive diseases	1.0
	Male	GC-1	20.2
	Male	Infectious and parasitic diseases	1.0
	Male	Intentional injuries	2.0
	Male	Unintentional injuries	19.0
	Total		56.0

III. ESSAY 2: CAUSES OF DEATH IN TURKEY: HOW THE INCREASE IN THE BURDEN OF COMMUNICABLE DISEASES VARY BY SEX AND AGE IN TURKEY?*

Abstract

Background/Aim: Causes of death statistics are essential tools for public health, but Turkey lags in the number of studies on causes and trends of death. This study measures causes and trends of death in Turkey for the 2013-2019 period, with special emphasis on the increase in communicable diseases (CDs).

Method: This study has a representative research design based on the national population and cause of death registration systems. Causes of death with International Classification of Diseases, Tenth Revision (ICD-10) codes were grouped and garbage codes were determined and redistributed. To understand how the increase in the burden of CDs vary by sex and age, modal age at death, age-specific death rates, probability of eventual death, years of life lost (YLL) due to three main causes of death were calculated by using discrete absorbing Markov chain model.

Results: According to results, modal age at death among male population shifted to older ages, the share of respiratory infectious diseases and other infectious and parasitic diseases increased rapidly between 2013 and 2019, just before the onset of COVID-19 pandemic.

Conclusion: Overall, our results suggest that burden of CDs increased for both sexes, and elderly male population was among the most effected group. Since non-communicable diseases were still the leading causes of death, increasing rate of CDs may create an extra burden on health system.

Key words: Causes of death, communicable diseases, Markov chain, modal age at death, Turkey.

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III.1. Introduction

Coale (1974) express that demographic transition does not involve only one revolution, it involves two revolutions which led to two transitions. The first revolution is "Neolithic revolution" which is often "overlooked" and the second is industrial revolution (Coale, 1974; Aykut Toros, 2016). Birth and death rates have shifted from low to high level during the Neolithic revolution. Farming technologies in Neolithic period accelerated the close settlement of hunter gatherers and crowded areas. These changes increased spread of the infectious diseases and the contamination of food and water and these caused higher death rates. On the other hand, higher death rates were compensated by higher birth rates (Coale, 1974; Aykut Toros, 2016). In the industrial revolution period, birth and death rates have shifted from high to low rates. Improved medical technology, better sanitation and nutrition have declined death rates. Furthermore, the spread of modern contraceptive methods and longer life expectancy have decreased the fertility rates.

Similar to the demographic transition, Omran (1971) have developed a theory of epidemiologic transition which is based on the general pattern of mortality and causes of death for some developed countries. Omran (1971) describes 3 phases that change the life expectancy. In the first phase ("the age of pestilence and famine") death rates are high and fluctuating. Epidemics, famine and war etc. determine the pattern of mortality. Characteristic of the second phase ("the age of receding pandemic") is that epidemic begins to decrease and mortality rates fall. Life expectancy at birth increases about 20 years. In this phase the most benefitted group are children and young women (Olshansky et al., 1998; Olshansky & Ault, 1986; Omran, 1971). The last phase is "the degenerative and man-made diseases". Decline of mortality rates continues and life expectancy increases. In this phase main cause of deaths are degenerative diseases such as cancer, heart disease, accidents etc. Shifting from phase 1 to phase 3 requires some changes in socio-demographic structure. According to this theory, epidemic transition is possible with modernization. Higher standard of livings, nutrition, improved sanitation and immunization etc. enables this transition (Olshansky & Ault, 1986; Omran, 1971).

Turkey has followed an epidemiologic transition parallel with its demographic transition process. The background section of this essay summarizes Turkey's epidemiological history up to 2013, including the pre-transition period. Epidemiologic stage of post 2013 period will be discussed in “Conclusion” part of the essay. This essay examines the causes and trends of death in Turkey for the study period 2013-2019, highlights the increasing share of communicable diseases, and examines whether there are any sex and age differences in causes and trends of death. The 2013-2019 period is significant because Turkey implemented an electronic death notification system in 2013, so mortality data is more complete compared to the registration practices of previous year (Özdemir, 2012; Özdemir et al., 2015; Yayla, 2016). Since the most recent available data on causes of death is for 2019, we selected the period as 2013-2019. Furthermore, focusing on pre-2020 data has also helped to avoid any fluctuation that may arise due to the COVID-19 pandemic. We revealed these trends in more detail using high quality death registration data. We addressed the following inter-related research questions: (1) How do recent gains in life expectancy and modal age at death vary by sex and age? (2) How do causes of death change recently? (3) Are there different trends in the pattern of causes of death in terms of sex and age? (4) Is Turkey still in the last phase of epidemiological transition? Regarding these questions, we first measured life expectancy and modal age at death to reveal the age pattern at death, and then disclosed trends in causes of death by sex.

There are lots of studies for Turkey handling the non-communicable diseases (NCDs) or chronic diseases (Balbay et al., 2018; Breda et al., 2021; Sozmen & Unal, 2014). Since communicable diseases have a small share among the causes of death, studies on communicable diseases are very few. In order to implement sustainable, accurate and timely health policies, first of all, it is necessary to understand the trends and patterns of diseases, to identify vulnerable groups and to define the risk factors of diseases correctly. This study shows that communicable diseases were already on the rise before the time of COVID-19 pandemic, and reveals the most affected groups on the basis of age and sex. Considering the current burden of non-communicable diseases and the burden created by COVID-19 pandemic, it is of great importance to include measures related to communicable diseases in health policies.

III.2. Literature Review

III.2.1. Background

Along with the socio-economic transformations Turkey has undergone, its demographic structure is also changing rapidly. Turkey has followed an epidemiologic transition parallel with its demographic transition process. Shorter & Macura (1982) divided the demographic transformation of Turkey into 3 periods as 1923-1955, 1955-1985 and 1985 and beyond, and the following researchers (Koç et al., 2010; Toros, 1985) referred this classification in their studies.

During the pre-transition period before 1923, like many countries, Turkey struggled with some diseases at certain periods in its history. Before the foundation of the Turkish Republic, settlement in the rural areas were common, high mortality and infectious diseases and epidemics were major health problems. People were living in poverty and the male population were dying due to the wars. According to the 1927 census which is the first census of Turkish Republic, sex ratio was 93 males per 100 females (Shorter, 1985; TurkStat, 2021d). Life expectancy is estimated as 30 years and crude death rate is estimated as 36 per thousand for 1923 (Shorter, 1985). Furthermore, the First World War played a major role in spreading of epidemics. Struggle with malaria, tuberculosis, trachoma, smallpox and syphilis began in this period (Tekir, 2019; Temel, 2008). In addition to these diseases, tuberculosis, diarrhea, typhus, cholera and measles were also among common infectious diseases (Bakar et al., 2017; Karabulut, 2007; Panzac, 1997; Tekeli & İlkin, 2004).

In the first stage of transition (1923-1955), life expectancy at birth increased to 44 years and infant mortality rate decreased from 306 to 233 per thousand (Shorter & Macura, 1982). Furthermore, because of the dangerous extent of infectious diseases, in 1930 Ministry of Health was established and General Hygiene law was put into effect to make provision for infectious diseases. In the meantime, Public Health Law number 1593 was put into effect (Umumi Hıfzısıhha Kanunu , 1930). By this law, struggle against the infectious and other diseases was taken under the responsibility of

the State specifically through some precautions against infectious diseases (Umumi Hıfzısıhha Kanunu , 1930). Furthermore, physical examination before marriage and notification and follow-up of the tuberculosis and other epidemics became compulsory.

In 1960s vaccinations programs against tuberculosis, smallpox, and polio came into effect in throughout the country (Bakar et al., 2017). On the other hand, cardiovascular diseases became common disease. In 1965, distribution and sale of contraceptive methods were permitted and ban on abortion was removed (Nüfus Planlaması Hakkında Kanun, 1965). Abortion was allowed if there is a life threat for mother or if it is known that child will be born disabled. This implementation provided the decline of total fertility rate and better reproductive health. In this period family planning programs, child and maternal health services were expanded (Bakar et al., 2017; Koç et al., 2010). Moreover, life expectancy reached to 59 years in this period (Shorter & Macura, 1982). Improvements in education and health sector accelerated the urbanization process.

During the third stage of transition, by the 1980s, thanks to the immunization programs smallpox and polio substantially declined. In 1983, induced abortion has been allowed until the 10^{th} week of pregnancy and sterilization for both sexes has been legalized (Nüfus Planlaması Hakkında Kanun, 1983). Urbanization and education level have increased. Infant mortality rate declined to 11 per thousand (TurkStat, 2021a) and maternal mortality decreased to 15.7 per hundred thousand in 2013 (TurkStat, 2021e). Life expectancy at birth reached to 78 years in 2013 (TurkStat, 2021c). Total fertility rate has been declined to replacement level which is 2.1 child per woman in 2013 (TurkStat, 2021a). Furthermore, communicable diseases left their place to noncommunicable diseases.

III.2.2. Literature on Mortality Trends in Turkey

In literature, there are studies which provide useful insights on the mortality trend and patterns in Turkey. Bakar and colleagues (2017) present an archive study to

discuss the demographic and epidemiological transition of Turkey between 1931 and 2015. They have described four time periods: pre-1923, 1923-1960, 1960-1980 and 1980-2015. In this long interval, life expectancy has increased, total fertility rate has declined to 2.1, and urbanization and level of education have increased. Longer life expectancy and lower fertility rate have created a rise in elderly population. The main causes of death have become chronic diseases and cancers. They have emphasized the necessity of policies with regard to management of aging and chronic diseases (Bakar et al., 2017). Özdemir and colleagues (2017), analyzed the epidemiologic transition of Turkey between 1980 and 2013. In their analysis they followed standart classification of the causes of death into 3 main groups: communicable diseases, noncommunicable diseases and injuries.This study has shown that causes of death pattern has shifted from communicable diseases to noncommunicable or chronic and degenerative diseases between 1980 and 2013. They conclude that Turkey is in the last stage of the epidemiologic transition. However, they noted that communicable diseases remain a public health challenge due to the insufficient basic health services and inequality in access to health services (Özdemir et al., 2017).

National Burden of Disease and Cost Effectiveness Study (NBDCE) is a part of global burden of disease (GBD) project conducted by WHO. NBDCE study was conducted in 2002-2004 period (Refik Saydam Hygiene Center Presidency, 2004). This study is the one that predicts the burden of disease for Turkey and 5 regions level for the year of 2000. Akgün, Çolak and Bakar (2012) evaluated the cross-sectional verbal autopsy survey as part of the National Burden of Disease and Cost Effectiveness Project to understand the causes of death profile and quality of cause of death registration in Turkey (Akgün et al., 2012). In the master thesis Torun (2014) evaluates the potential years of life lost and potential gains in life expectancy by major causes of death for 2000 - 2008 period with decrement life table methods (Torun, 2014).

III.2.3. Burden of Communicable Diseases in the World

Infectious diseases have started to increase in the last of years 20th century in many parts of the world (McMichael, 2001; Weiss & McMichael, 2004). Poor

countries have also faced with increasing trend of infectious diseases. Spread of malaria, cholera and HIV/AIDS have increased the burden of diseases of poor countries (McMichael, 2001). In 1996, WHO warned against the dangers of infectious diseases:

> *"Until relatively recently, the long struggle for control over infectious diseases seemed almost over…..Far from being over, the struggle to control infectious diseases has become increasingly difficult. Diseases that seemed to be subdued, such as tuberculosis and malaria, are fighting back with renewed ferocity. Some, such as cholera and yellow fever, are striking in regions once thought safe from them. Other infections are now so resistant to drugs that they are virtually untreatable. In addition, deadly new diseases such as Ebola haemorrhagic fever, for which there is no cure or vaccine, are emerging in many parts of the world. At the same time, the sinister role of hepatitis viruses and other infectious agents in the development of many types of cancer is becoming increasingly evident. The result amounts to a global crisis: no country is safe from infectious diseases. The socioeconomic development of many countries is being crippled by the burden of these diseases. Much of the progress achieved in recent decades towards improving human health is now at risk."* (WHO, 1997)

Morens and colleagues (2004) emphasize the challenge by re/emergence of infectious diseases in all over the world. Northeastern part of United States, western Europe, Japan and southeastern Australia have been identified as "hotspots" for emerging diseases due to the population density, antibiotic drug resistance, and environmental factors (Jones et al., 2008; Morens et al., 2004). Semenza and colleagues (2016) conducted a study in Europe during 2008-2013 period to determine the key drivers of infectious diseases. According to their study travel and tourism is very important factor for infectious diseases. Food and water quality, natural environment, global trade, and climate are found as other drivers of infectious diseases (Semenza et al., 2016). Choe and colleagues (2018) examined the trends in infectious diseases in South Korea in 1983-2015. They found that age -standardized mortality at infectious disease decreased in 1983-1996 period and then increased in 1996-2015. They also showed that although there is an improvement in terms of infancy period in

Korea, infectious diseases have become problematic issue for elderly people (65+) (Choe et al., 2018).

III.3. Data and Method

III.3.1. Data sources

The primary data sources of this study are TurkStat registration statistics for causes of death, and population by sex and age for the 2013-2019 period. In the causes of death datasets, each row corresponded to one death and provided data on age, sex, place of residence, and ICD-10 coded cause of death for each death.

III.3.1.1. Data preparation

To begin the analysis, four-digit ICD-10 codes were converted to the broad cause categories. This process is applied according to the cause categories listed in Annex Table A of the WHO technical paper for Global Health Estimates (GHE) (WHO, 2018). Total number of deaths by cause, age and sex were extracted according to the GHE cause list, and some ICD-10 codes were mapped as garbage codes. Garbage codes are causes that do not provide information for underlying cause of death (Mathers et al., 2006; Naghavi et al., 2010). Before causes of death analysis is done, these codes should be redistributed for a valid analysis result (Naghavi et al., 2010).

In the population dataset, we included only citizens of the Republic of Turkey. Because while population registration system covers large proportion of legally residing foreigners, death registration system covers only a negligible part of this group in Turkey (Yayla, 2016). To provide the correspondence between event (death) and exposure population, we excluded legally residing population from population dataset. On the other hand, immigrants other than legal residing foreigners are not registered in both the cause of death and population registration systems anyway. Therefore, other immigrants did not pose a problem for our analysis. In the next step, we calculated the mid-year population for each year between 2013 and 2019. Total number of deaths and mid-year population were presented in the first essay.

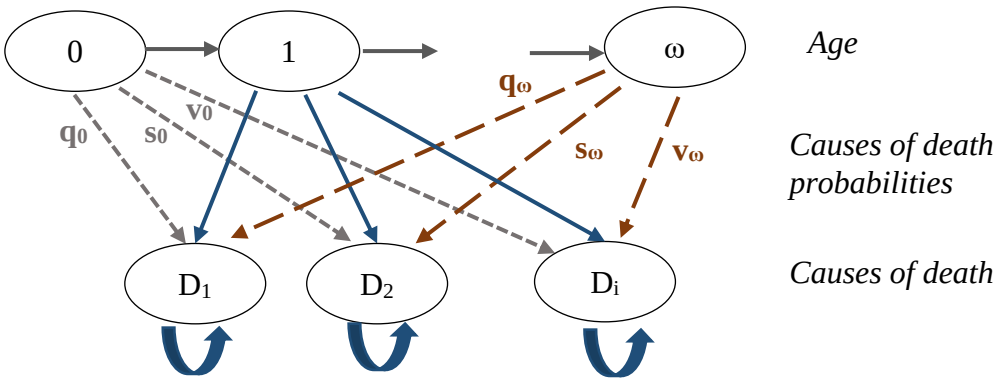
III.3.2. Data analysis

In this study, we applied Markov chain matrix approach to analyse the trend and pattern of causes of death. Markov chain is a stochastic process, which provides the probabilities of transition among states. Markov chain also satisfies the property of memorylessness, that is, probability of next state depends on the current states, not past states (Kemeny & Snell, 1976). Markov chain is very suitable for human life cycle, because, as Caswell (2009) states:

> "*The movement of an individual through its life cycle is a random process, and although the eventual destination (death) is certain, the pathways taken to that destination are stochastic and will differ even between identical individuals; this is individual stochasticity" and "…, because it accounts for all the possible pathways, and their probabilities, that an individual can follow through its life*" (Caswell, 2009 pp. 1763, 1764).

In this paper, we used the discrete-time absorbing Markov chain model. In this model, causes of death are included as absorbing states. Figure III.3.1 shows the figure of an age-classified life cycle with (0,1,…, ω) age classes and (D_1, D_2,…D_i) causes of death. In this figure, q_k, s_k and v_k where k = 0,1,…, ω define the death probabilities of causes D_1, D_2, and D_i, respectively.

Figure III.3.1. Markov chain model used in this study



Then we get the transition matrix corresponding to Figure III.3.1.:

$$\mathbf{P} = \begin{pmatrix} \mathbf{U} & \mathbf{0} \\ \mathbf{M} & \mathbf{I_i} \end{pmatrix}$$

Here, all capital letters are matrix. Therefore, P matrix consists of 4 matrices; U, M, 0 and Ii. U shows the transitions among the transient states. Since transient states refer to ages, U includes the survival probabilities on the sub-diagonal and zeros elsewhere (ω×ω). Matrix M refers to absorbing states with the dimension of i×ω. This matrix shows the death rates for each cause of death. I_i corresponds to the identity matrix with the dimension of i×i. I_i provides the remaining number of dead individuals in their absorbing states (Engelman et al., 2014). All matrices in the P matrix are constructed by calculating the age and cause-specific hazards. Formulas (1) and (2) defines the sex-specific hazards of causes of death:

$$h_{female} = \frac{\text{Age and cause specific female deaths in year t}}{\text{Age specific mid-year female population in year t}} \quad (1)$$

$$h_{male} = \frac{\text{Age and cause specific male deaths in year t}}{\text{Age specific mid-year male population in year t}} \quad (2)$$

After this calculation, hazard matrices took the form of

$$\mathbf{H} = \begin{pmatrix} h_{11} & h_{12} & . & . & h_{1i} \\ h_{21} & . & . & . & . \\ . & . & . & . & . \\ . & . & . & . & . \\ h_{\omega 1} & h_{\omega 2} & . & . & h_{\omega i} \end{pmatrix}$$

Cause of death →

Starting age ↓

where h_{ij} is the hazard due to cause j at age i. In the next steps, we calculated the longevity statistics (life expectancy, variance, and standard deviation), rates and probability of dying from each cause, years of life lost by causes.

III.3.2.1. Statistics of Longevity

If we define the longevity of an individual in age class W as the remaining time

until an absorption by a state, then we constitute the fundamental matrix, **N**:

$$\mathbf{N} = (\mathbf{I}_{\omega} - \mathbf{U})^{-1} \tag{1}$$

Matrix **N** provides the statistics of longevity, where the (i, j)th entry of matrix **N** is the mean time spent in state i, conditional on survival to state j (Caswell, 2006, 2009; Caswell & Ouellette, 2016; Caswell & Zarulli, 2018). The first two moments are:

$$\eta_1^{\mathrm{T}} = \mathbf{1}_{\omega}^{\mathrm{T}}\mathbf{N} \tag{2}$$

$$\eta_2^{\mathrm{T}} = \eta_1^{\mathrm{T}}(2\mathbf{N} - \mathbf{I}_{\omega}) \tag{3}$$

where $\mathbf{1}_{w}^{\mathrm{T}}$ is the transpose of the column vector of ones with $w' \; 1$ dimension and $\mathbf{I}_{w}$ is the identity matrix with $w' \; w$ dimension. Calculated statistics of longevity from these moments are:

$$\mathrm{E}(\eta) = \eta_1 \tag{4}$$

$$\mathrm{V}(\eta) = \eta_2 - \eta_1 \circ \eta_1 \tag{5}$$

$$\mathrm{SD}(\eta) = \sqrt{\mathrm{V}(\eta)} \tag{6}$$

$$\mathrm{CV}(\eta) = \mathrm{diag}(\eta_1)^{-1}\mathrm{SD}(\eta) \tag{7}$$

where $\circ$ denotes element by element multiplication, $\mathrm{E}(\eta)$ gives the life expectancy at each age; $\mathrm{V}(\eta)$, $\mathrm{SD}(\eta)$ and $\mathrm{CV}(\eta)$ are variance, standard deviation and coefficient of variation of longevity, respectively.

III.3.2.2. Probability Distribution of Eventual Death Due to Each Cause

Assuming that b_{ij} is the probability of dying from cause i at the current age j, then

$$\mathbf{B} = \mathbf{MN} \tag{8}$$

columns of **B** give the probability distribution of eventual cause of death for each age. Rows of **B** give the probability of dying from a cause for each age (Caswell, 2006, 2009)

III.3.2.3. Life Lost Due to Causes of Death

If we define $\mathbf{Z}_1$ as the matrix of mean life lost due to causes; that is,

$$\mathbf{Z}_1 = (E(\text{life lost} \mid \text{cause} = 1, \text{starting age} = j))$$

$$\mathbf{Z}_1 = (h_1^T \text{Ä} \mathbf{I}_a)\hat{\mathbf{B}} \tag{9}$$

and column sums of $\mathbf{Z}_1$ give the total life lost for each cause.

III.3.2.4. Modal age at death

The modal age at death indicates the age at which the higher proportion of deaths occurred. To understand trends in modal age, two aspects should be considered (Canudas-Romo, 2010): (1) Mortality changes at ages younger than modal age, (2) Mortality changes at ages older than modal age. If the improvement in mortality occurs at ages below the modal age, the modal age will not change because the increase in the number of survivors reaching the modal age leads to more deaths at the same modal age but does not move the modal age forward. However, when the improvement occurs at ages above the modal age, this improvement will shift the modal age to older ages (Canudas-Romo, 2010; Horiuchi et al., 2013). In this study, modal age at death was calculated using the P-spline smoothing procedure. P-spline uses a flexible

nonparametric approach, and it is assumed as highly efficient for fitting mortality rates to obtain smoothed mortality rates (Camarda, 2008; Currie et al., 2004). Another strength of the P-spline approach is that it also provides good estimates for data with some quality or completeness issues (Horiuchi et al., 2013). Since we wanted to show the modal age among adults, infant and child mortality data excluded and so, P-spline smoothing method was applied to mortality data for age 10 and over. This procedure was performed by using "MortalitySmooth" package in R programming version 4.1.0.

Furthermore standard deviation above the modal age (SD(M+)) was calculated for each year to monitore the changes in variation. Let μ(x) be force of mortality at age x. Then corresponding survival function is,

$$S(x) = \exp(-\int_0^x \mu(t)\, dt) \tag{10}$$

Density function of age at death distribution is calculated by

$$f(x) = \mu(x)S(x) \tag{11}$$

Let M denote modal age, standard deviation above modal age is calculated by the following formula:

$$SD(M+) = \sqrt{\frac{\int_M^\omega (x-M)^2 f(x)\, dx}{\int_M^\omega f(x)\, dx}} \tag{12}$$

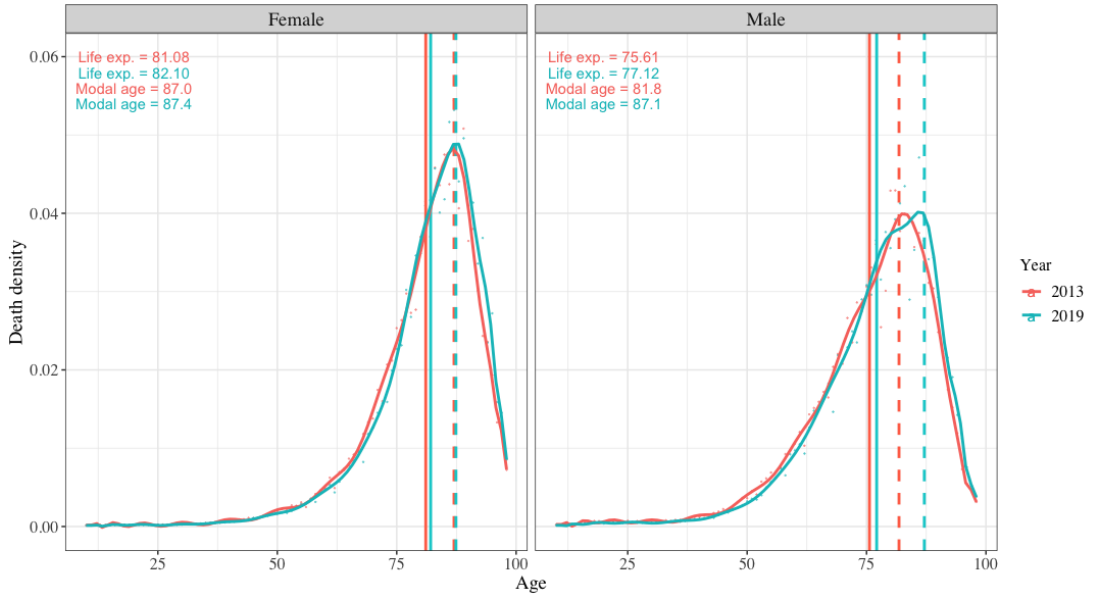
where ω is the maximum age reached in the population.

III.4. Results

In this part, we first evaluated the longevity statistics for both sexes and then further analyzes on causes of death.

Life expectancy at birth for female population increased 1.02 years between 2013 and 2019 (Appendix Table B.1 and Table B.2). Variance and standard deviation in age at death provide information about inequality in death and progression in mortality. Higher variation or standard deviation indicates higher uncertainty about age at death. Results show that variation decreases with increasing age, as expected. Between 2013 and 2019, variation has decreased at all ages among women. Male life expectancy increased 1.5 years in seven years. As in females, the variation in life expectancy has decreased at all ages among males in 2013-2019 period (Appendix Table B.3 and Table B.4). When we compare by sex, we see a similar pattern in 2013 and 2019: male variances in life expectancy are higher up to age of 60, after which female variances become higher.

Figure III.4.1. Distribution of death density in 2013 and 2019 by sex (solid vertical lines show life expectancy and dashed lines show modal age at death)



Note: Figure shows density curves for age 10 and over

Figure III.4.1 shows the death density curves, life expectancy and modal age at death in 2013 and 2019 for each sex. According to figure, death density rised sharply after age 50 and reached peaks at ages 80s for both sexes. The life expectancy of females increased by almost a year, while that of males increased by 1.5 years. Modal ages increased 0.4 and 5.3 years among females and males, respectively.

Figure III.4.2. Modal age at death: 2013-2019

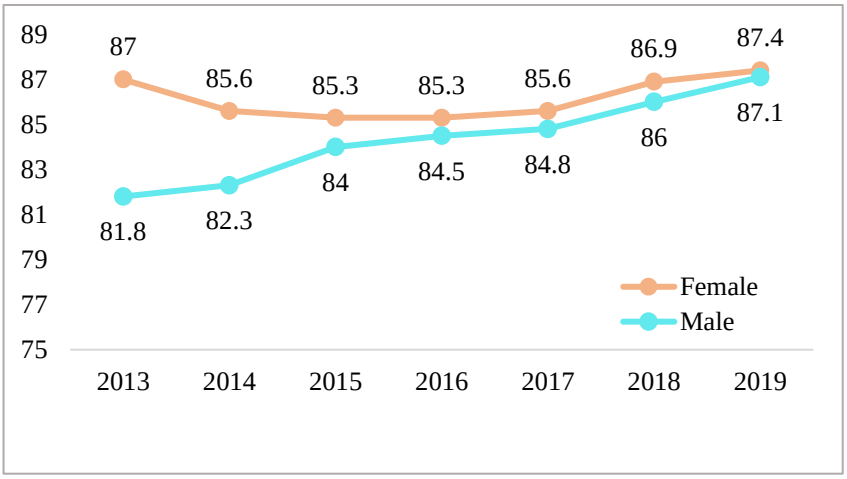
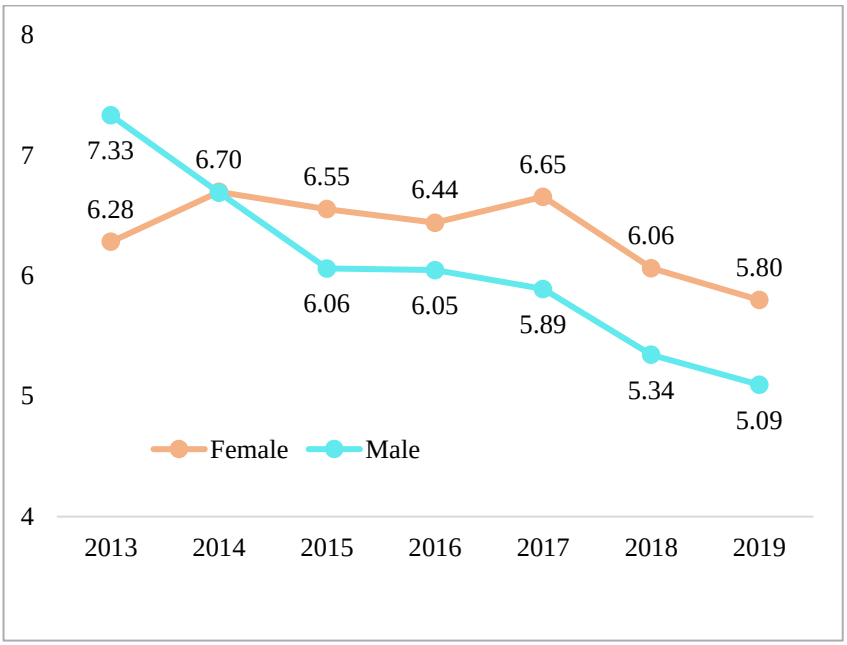


Figure III.4.2 indicates the modal age at death between 2013 and 2019. The most notable trend in this figure is the decrease in modal age at death difference between females and males. Modal age for males has increased each year and arrived 87.1 in 2019. On the other hand, the modal age for females has remained relatively stable, with modal ages ranging between 86 and 87 during this period.

Figure III.4.3. Standard deviation above the modal age (SD(M+)): 2013-2019



According to Figure III.4.3, SD(M+) among males decreased from 7.3 to 5.1 years between 2013 and 2019. The rate of decline of SD(M+) among males slowed down between 2015 and 2017, but across all years, the rate of decline of SD(M+) is faster for males than for females. Among females, SD(M+) is fluctuating between 2013 and 2017 period and after 2017 it is decreasing to 5.8 years.

Figure III.4.4. Percent distribution of cause of death: 2013-2019

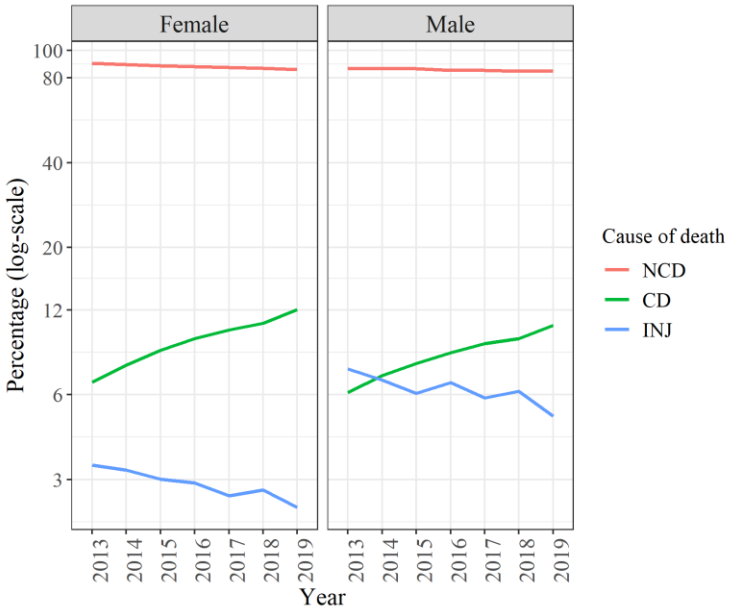


Figure III.4.4 shows the percent distribution of three main causes of death. According to this figure, the share of non-communicable diseases (NCDs) and injuries are decreasing over the years, but percent decline of NCDs is very slow. NCDs still constitute the major causes of death, which stands at 86% and 84% in 2019 among females and males, respectively. Injuries (INJs) have a decreasing trend for both sexes but are higher among males than females.

By contrast, percentages of communicable, maternal, perinatal and nutritional conditions (CDs) are rising rapidly. CDs percentages almost double for both sexes, rising from 6.7% to 12% among females and from 6.1% to 11% among males between 2013 and 2019.

Figure III.4.5. Age-specific death rates for children under 5

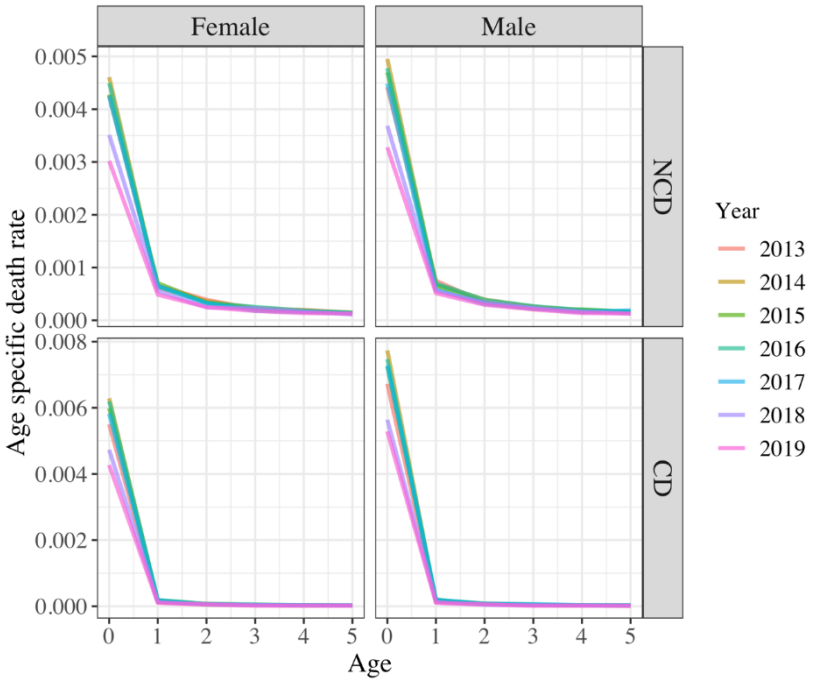


Figure III.4.6. Age-specific death rates among individuals aged 50 and older

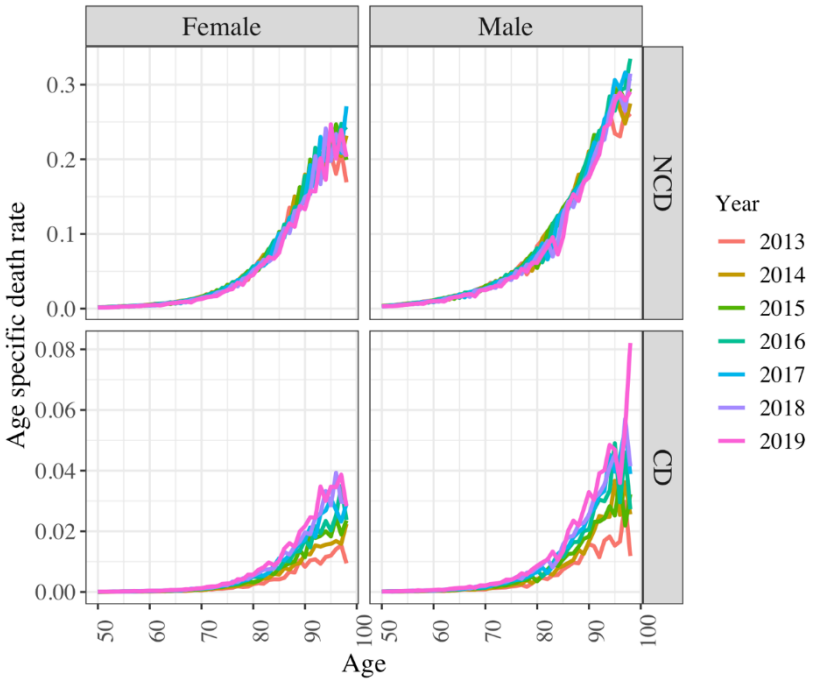


Figure III.4.5 and Figure III.4.6 shows the age specific death rates (ASDRs) of NCDs and CDs for under age 5 and age 50 and over, respectively. For the under 5, rates of NCDs and CDs are highest at age 0 and there is rapid decline until age 1. Infancy period ASDRs are decreasing for NCDs and CDs over the years.

For the age 50 and over group, while NCDs death rates are decreasing over the years, rates are higher at older ages and among males. Figure III.4.6 also shows that CDs' death rates have an increasing trend from 2013 to 2019. and rates are higher among males.

Figure III.4.7. Probability of dying from NCDs, CDs and INJs by age and sex

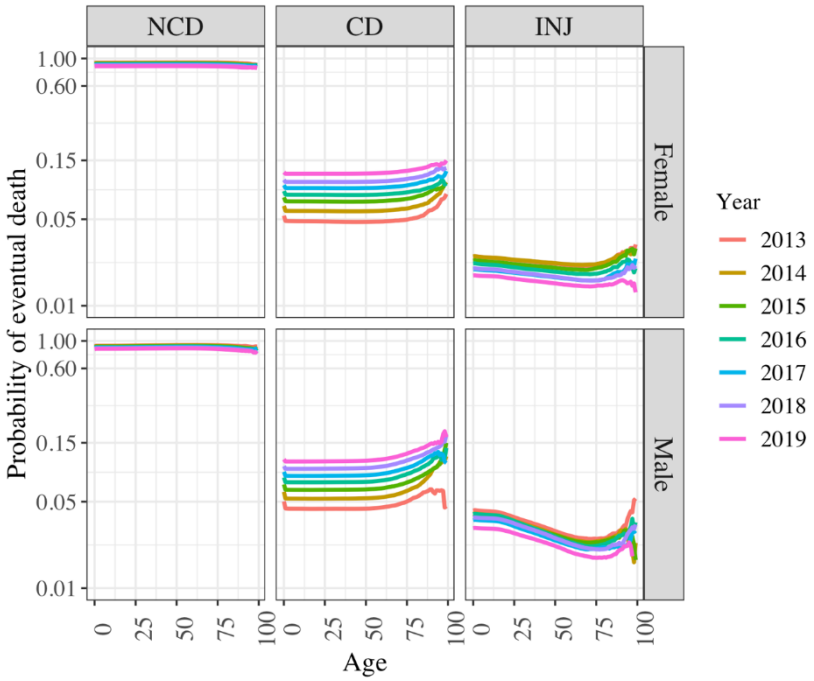


Figure III.4.7 presents the change of death probabilities (in logarithmic scale) among three main causes of death by age and sex over the years. Although the probability of dying from NCDs has the highest category for all ages, average NCDs probabilities have declined slowly between 2013 and 2019, falling from 0.92 to 0.87 among males and from 0.93 to 0.87 among females. There are, however, rapid rises for CDs probabilities, which increased from an average of 0.05 to 0.12 for both sexes. Up to the age of 75, females are more likely to die from CD, while after this age it is

higher for males. Meanwhile, probabilities of INJs have decreased from 0.03 to 0.02 for males, and from 0.02 to 0.015 among females.

To understand the contribution of different causes of death related to the increasing trend in death probabilities of CDs, we examined the sub-categories of CDs and split them into five parts (WHO, 2013, 2018): infectious and parasitic diseases, maternal conditions, neonatal conditions, nutritional deficiencies and respiratory tract infectious diseases.

Figure III.4.8. Probability of dying from communicable, maternal, perinatal and nutritional conditions by age and sex

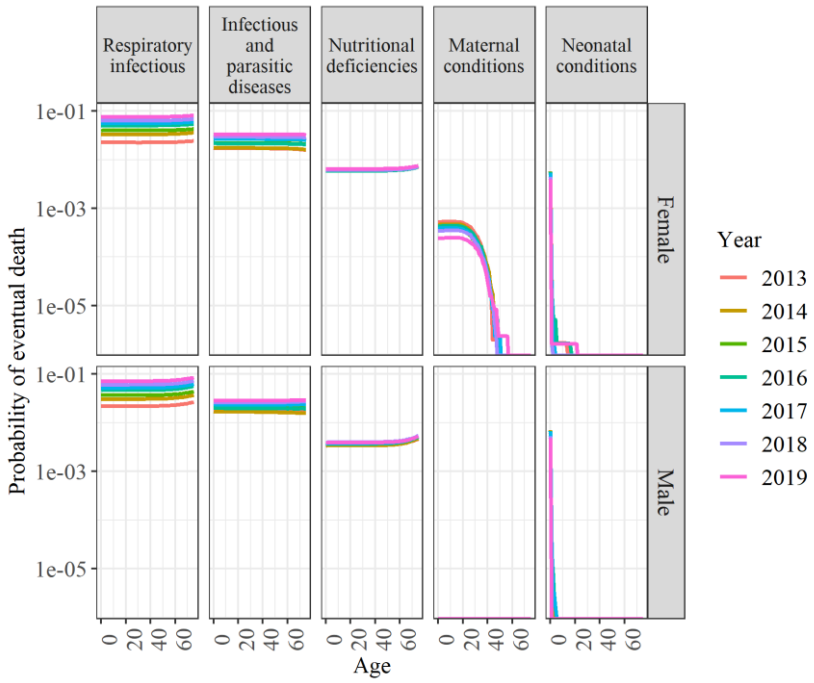


Figure III.4.8 shows the change of probabilities in logarithmic scale. According to the findings, this rise is due to the increase in respiratory tract infectious diseases, as well as infectious and parasitic diseases. Respiratory tract infectious diseases constitute the highest share of CDs' death probabilities, and they show an increase over the years. Infectious and parasitic diseases are also increasing over the years. Probability of nutritional deficiencies is almost at the same level from 2013 to 2019

and that probability increases with age. Dying from maternal conditions decreases after women exit their 20s and there is declining trend between 2013 and 2019.

Figure III.4.9. Distribution of years of life lost (YLL) by 3 main cause of death groups (NCD, CD, INJ)

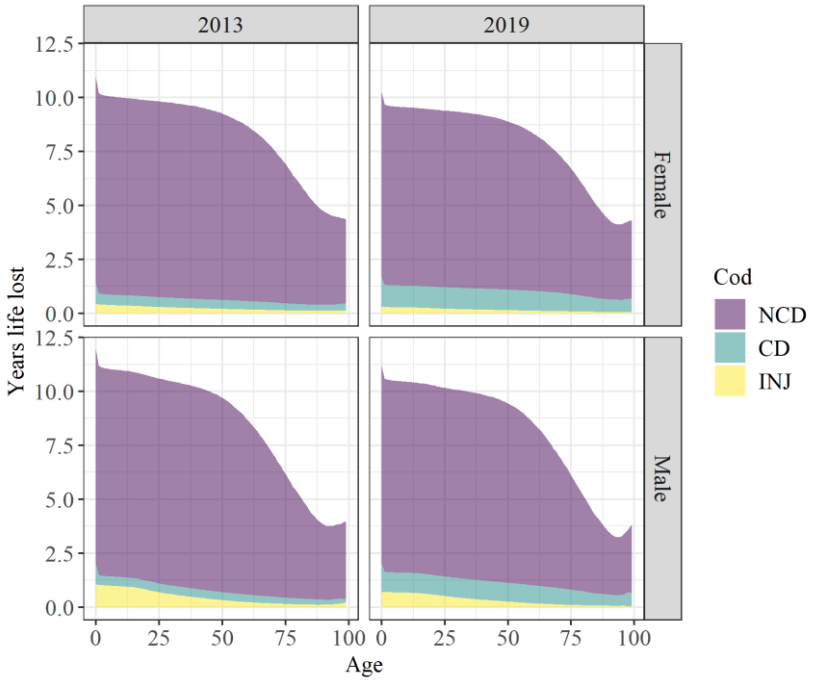


Figure III.4.9 presents the years of life lost (YLL) by 3 main causes of death. Between 2013 and 2019, total YLL at birth decreased from 10.9 to 10.2 years and from 11.9 to 11.2 among females and males, respectively. On the other hand, YLL at birth from CDs went up from 0.95 to 1.37 and 0.96 to 1.34 among females and males, respectively. Age specific YLLs due to CDs increased more than doubled at all ages between 2013 and 2019, peaking in age seventies.

III.5. Conclusison and Discussion

Since the establishment of the Turkish Republic has made progress in its healthcare system. Life expectancy at birth reached a higher level, infant and child mortality rates declined considerably (Buzcu et al., 2017). This study examines the age and cause patterns of mortality by sexes in Turkey for 2013-2019 period. There have been improvements in the death notification system over the years, especially for coverage of causes of death. However, reporting of causes needs further improvement particularly for ill-defined causes. In the data preparation stage of this study, we assessed the initial data from the registration system and applied necessary modifications by redistributing garbage codes.

During the study period, life expectancy at birth rised nearly 1 year among females and 1.5 years among males. Moreover, as a novel finding of our study, modal age at death increased 0.4 years among females and 5.3 years among males in six years. This finding indicates that, distribution of the bulk of the bell-shaped deaths around the modal age shifted toward older ages for males and increased only slightly for females. This study also indicates that modal age at death converged in males and females. Although life expectancy is affected by changes in mortality at any age, modal age is only affected by changes in old age mortality (Horiuchi et al., 2013). This means that in 2013-2019 period, the improvement in mortality in older ages was higher for males than for females. Similar to this result, Horiuchi and collagues (2013) also found that in France, female-male gap in modal age got closer between 1972 and 2009. Furthermore, it may be concluded that different gains in life expectancy and modal age death between sexes is due to the fact that males just experienced the improvement females had previously experienced in adult mortality (Mathers et al., 2015). In many developed countries gains in life expectancy among males were later than among females. For example, in accordance with our findings, Mesle and Vallin (Meslé & Vallin, 2006) revealed that Sweden, England, Denmark and Japan were also experienced the sex difference in gains in life expectancy between 1980-2000 period (Meslé & Vallin, 2006). Standard deviation above the mode (SD(M+)) indicates the inequalities in older ages. Acoording to finding of this study, SD(M+) in 2019 was

lower than in 2013 for both sexes, indicating that variation at older ages decreased over time. However, rate of decrease differed between males and females. SD(M+) for females remained more or less at the same level until 2017. On the other hand, SD(M+) among males decreased continuously between 2013 and 2019. The different trend between women and men suggests that the compression of mortality has most likely ended for females and is still continuing for males. Similar pattern in SD(M+) was also observed for females in Japan, Canada, France and USA in 2000s (Ouellette and Bourbeau, 2011).

According to our results, NCDs have decreased from 87% to 84% among males and from 90% to 86% among females between 2013 and 2019, yet NCDs were still the leading causes of death. This finding shows that NCDs maintained the first rank among the causes of death, as was the case before 2013 (Özdemir et al., 2017), but their share decreased. Similar to this finding, some studies have shown a reduction in cardiovascular diseases over a period before 2013, thanks to medical and surgical advances (Dinç et al., 2013; Unal et al., 2013). We also found that both ASDRs, death probabilities and YYL due to NCDs decreased slightly over the years. Considering the importance of relation between chronical diseases and aging and according to elderly focused results, NCDs death rates are higher in elderly population for both sexes.

Death probability of INJs decreased over the years, but while deaths from INJs show a declining trend, they are still high for the male population. Probabilities here are also higher at younger ages and among males, therefore there is need for detailed analysis to enhance subsequent studies.

Undoubtedly, the most important result of this study is the increasing share of communicable diseases. CDs reached 11% among males and 12% among females in 2019. We found that CDs death rates were higher but declined over the years among children under five. Notably, CDs death rates increased at older ages and were higher among male population in the 2013-2019 period. Death probabilities of CDs also increased almost 2.5 times from 2013 to 2019 for both sexes. The main reason for this

increasing trend stemmed from the rising probability of respiratory infectious and infectious and parasitic diseases. In accordance with our results, GBD study also show that lower respiratory infectious increased 73% from 2009 to 2019 and become among the top 10 causes of death in Turkey (IHME Institute for Health Metrics and Evaluation, 2020)(Institute for Health Metrics and Evaluation, 2020). Furthermore, YLL due to CDs doubled at all ages between the analysis period and reached its maximum level in the age 70s. Furthermore, increase of CDs is an unexpected trend for the last stage of epidemiological transition. The fact that deaths from CDs particularly affect the elderly points to a different pattern of mortality. However, since the time interval of this paper (7 years) is too short to get a clear understanding of Turkey's epidemiologic stage, we can say that this trend points to a different pattern of mortality in the epidemiologic transition for Turkey.

Overall, our findings suggested three important results. Firstly, the age pattern of mortality has shifted significantly towards older age groups among males as a result of compression of the mortality to older ages. Secondly, both ASDRs and death probabilities of CDs increased between 2013 and 2019 for both sexes; however, these indicators were higher among elderly male population. Finally, YLL due to CDs increased at all ages for both sexes.

Proportion of elderly population (65+) in total population in Turkey increased from 7.7 to 9.1 percent between 2013 and 2019, respectively (TurkStat, 2021b). Increasing elderly population also effected the patterns of causes of death. According to our results, the shift in deaths, especially in males, to older ages and the addition of CDs burden to the existing NCDs in the elderly indicate the necessity of taking health measures for the elderly population. A similar result was obtained by a study (Désesquelles et al., 2015) arguing that aging is a risk factor for infectious diseases and treatment is very difficult at advanced ages. Furthermore, Choe and colleagues (2018) found that infectious diseases have become an issue for age 65+ in South Korea (Choe et al., 2018). Trends and patterns of communicable diseases also differ among developed and developing countries. In a study it was revealed that contrary to the low-middle income countries such as Vietnam, Mongolia and Indonesia, deaths due to

lower respiratory tract infectious diseases (LRTI) were increased in upper-middle income and high-income countries (such as Japan, Singapore and Taiwan) between 2000 and 2017 (Feddema et al., 2021). The different trends in LRTIs have been ascribed to different risk factors in developed and developing countries. Main risk factors of LRTI are aging population in high income countries, while in developing countries malnutrition, smoke pollution and lack of effective preventive measures (Feddema et al., 2021). McDonald and colleagues (2018) estimated the mortality burden of influenza between 2000 and 2013 among 60 years and older people in Netherlands. According to the findings, burden of influenza was highest among age group 80-84 years (McDonald et al., 2018). Similar to these studies, in a study performed for Hong Kong, it was found that CDs showed the greatest increases between 2001 and 2010 for both sexes and mostly effected the elderly population (Plass et al., 2013). The most plausible explanation for why CDs affect older people more now than in the past is increasing life expectancy and changing mortality and morbidity pattern of elderly. In the past, most of the elderly lived with chronic diseases for a shorter period of time, whereas today the elderly can live with chronic diseases for a longer period of time (Diaconu et al., 2022). Therefore, living longer with chronic diseases makes older people more vulnerable to CDs.

As in the rest of the world, the fight against COVID-19 pandemic has been on Turkey's agenda for the last few years. COVID-19 left an extra burden on the health system especially concerning elderly population. To avoid post-pandemic health crisis, existing health problems should be handled urgently (Chan & Horne, 2021). Therefore, new strategies in health services to improve prevention, diagnosis and treatment are essential. Increasing vaccine coverage, improvement in early diagnosis and antibiotic treatment among elderly population may be an efficient preventive measures (Feddema et al., 2021; Fullman et al., 2018).

One of the strengths of this study is that it presents not only the recent age specific death rates but also longevity statistics, probability distribution of causes of death and YLL by cause, age and sex. Another strength of this study is that due to the

improvement in death registration system as of 2013, results were obtained from more reliable cause of death data.

When the findings of our study and international studies are evaluated together, the need for detailed studies on CDs among the elderly male population, where the burden of NCDs was already high, is clearly seen. Further analyses are also needed on the impact of causes of death on life expectancy or modal age at death to understand which diseases are improving or worsening.

III.6. References

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APPENDIX B. SUPPLEMENTARY TABLES FOR ESSAY 2

Table B.1. Age distribution of longevity statistics, female 2013

	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14
l_x	1.000	0.990	0.989	0.989	0.988	0.988	0.988	0.987	0.987	0.987	0.987	0.987	0.987	0.986	0.986
LE	81.08	80.88	79.95	79.00	78.03	77.05	76.07	75.09	74.10	73.12	72.13	71.14	70.15	69.16	68.18
Variance	223.76	161.39	155.48	151.93	149.85	147.98	146.59	145.43	144.25	143.25	142.40	141.62	140.86	140.10	139.25
SD	14.96	12.70	12.47	12.33	12.24	12.16	12.11	12.06	12.01	11.97	11.93	11.90	11.87	11.84	11.80
CV	0.18	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.17	0.17	0.17	0.17	0.17
	15	**16**	**17**	**18**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**	**27**	**28**	**29**
l_x	0.986	0.986	0.986	0.985	0.985	0.985	0.985	0.984	0.984	0.984	0.984	0.983	0.983	0.983	0.983
LE	67.19	66.21	65.22	64.24	63.25	62.27	61.28	60.29	59.31	58.32	57.34	56.35	55.36	54.38	53.39
Variance	138.33	137.39	136.27	135.35	134.49	133.65	132.85	131.98	131.09	130.26	129.69	128.93	128.18	127.42	126.57
SD	11.76	11.72	11.67	11.63	11.60	11.56	11.53	11.49	11.45	11.41	11.39	11.35	11.32	11.29	11.25
CV	0.18	0.18	0.18	0.18	0.18	0.19	0.19	0.19	0.19	0.20	0.20	0.20	0.20	0.21	0.21
	30	**31**	**32**	**33**	**34**	**35**	**36**	**37**	**38**	**39**	**40**	**41**	**42**	**43**	**44**
l_x	0.982	0.982	0.982	0.981	0.981	0.981	0.980	0.980	0.979	0.979	0.978	0.977	0.976	0.975	0.975
LE	52.41	51.43	50.45	49.46	48.48	47.50	46.52	45.55	44.57	43.59	42.62	41.65	40.69	39.72	38.76
Variance	125.69	124.85	124.08	123.17	122.35	121.48	120.55	119.53	118.44	117.55	116.44	115.22	114.01	112.65	111.32
SD	11.21	11.17	11.14	11.10	11.06	11.02	10.98	10.93	10.88	10.84	10.79	10.73	10.68	10.61	10.55
CV	0.21	0.22	0.22	0.22	0.23	0.23	0.24	0.24	0.24	0.25	0.25	0.26	0.26	0.27	0.27
	45	**46**	**47**	**48**	**49**	**50**	**51**	**52**	**53**	**54**	**55**	**56**	**57**	**58**	**59**
l_x	0.973	0.972	0.971	0.970	0.969	0.967	0.965	0.962	0.960	0.958	0.955	0.952	0.950	0.946	0.942
LE	37.80	36.84	35.88	34.93	33.98	33.04	32.11	31.18	30.25	29.33	28.41	27.50	26.57	25.66	24.77
Variance	109.84	108.47	107.09	105.65	104.07	102.20	100.33	98.29	96.53	94.39	92.43	90.29	88.68	86.58	84.27
SD	10.48	10.41	10.35	10.28	10.20	10.11	10.02	9.91	9.83	9.72	9.61	9.50	9.42	9.30	9.18
CV	0.28	0.28	0.29	0.29	0.30	0.31	0.31	0.32	0.32	0.33	0.34	0.35	0.35	0.36	0.37
	60	**61**	**62**	**63**	**64**	**65**	**66**	**67**	**68**	**69**	**70**	**71**	**72**	**73**	**74**
l_x	0.937	0.932	0.926	0.921	0.914	0.907	0.899	0.889	0.880	0.868	0.855	0.841	0.824	0.808	0.787
LE	23.88	23.00	22.15	21.26	20.42	19.57	18.75	17.93	17.12	16.34	15.58	14.81	14.10	13.36	12.68
Variance	81.90	79.58	76.84	74.86	72.29	69.86	67.19	64.61	62.11	59.39	56.61	54.02	51.08	48.64	45.84
SD	9.05	8.92	8.77	8.65	8.50	8.36	8.20	8.04	7.88	7.71	7.52	7.35	7.15	6.97	6.77
CV	0.38	0.39	0.40	0.41	0.42	0.43	0.44	0.45	0.46	0.47	0.48	0.50	0.51	0.52	0.53
	75	**76**	**77**	**78**	**79**	**80**	**81**	**82**	**83**	**84**	**85**	**86**	**87**	**88**	**89**
l_x	0.767	0.742	0.716	0.687	0.660	0.633	0.597	0.557	0.521	0.477	0.435	0.390	0.349	0.299	0.261
LE	12.00	11.37	10.74	10.16	9.53	8.89	8.37	7.90	7.37	6.96	6.53	6.17	5.78	5.58	5.25
Variance	43.27	40.52	37.96	35.37	33.27	31.44	29.36	27.28	25.66	23.94	22.51	21.18	20.15	19.04	18.31
SD	6.58	6.37	6.16	5.95	5.77	5.61	5.42	5.22	5.07	4.89	4.74	4.60	4.49	4.36	4.28
CV	0.55	0.56	0.57	0.59	0.61	0.63	0.65	0.66	0.69	0.70	0.73	0.75	0.78	0.78	0.81

	90	91	92	93	94	95	96	97	98	99+
l_x	0.214	0.181	0.147	0.119	0.097	0.076	0.059	0.047	0.036	0.030
LE	5.17	4.94	4.86	4.74	4.59	4.57	4.60	4.50	4.57	4.36
Variance	17.52	16.95	16.43	16.00	15.74	15.56	15.31	15.09	14.81	14.64
SD	4.19	4.12	4.05	4.00	3.97	3.94	3.91	3.88	3.85	3.83
CV	0.81	0.83	0.83	0.84	0.86	0.86	0.85	0.86	0.84	0.88

Note: l_x : Survivorship, LE: mean life expectancy, SD: standard deviation, CV: coefficient of variation

Table B.2. Age distribution of longevity statistics, female 2019

	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14
l_x	1.000	0.993	0.992	0.992	0.991	0.991	0.991	0.991	0.991	0.991	0.991	0.990	0.990	0.990	0.990
LE	82.10	81.70	80.75	79.78	78.80	77.81	76.83	75.84	74.85	73.86	72.86	71.87	70.88	69.89	68.90
Variance	192.56	144.90	140.58	138.36	136.92	135.86	134.84	133.92	133.33	132.65	132.00	131.32	130.67	130.02	129.46
SD	13.88	12.04	11.86	11.76	11.70	11.66	11.61	11.57	11.55	11.52	11.49	11.46	11.43	11.40	11.38
CV	0.17	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.16	0.16	0.16	0.16	0.16	0.17
	15	**16**	**17**	**18**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**	**27**	**28**	**29**
l_x	0.990	0.990	0.989	0.989	0.989	0.989	0.989	0.988	0.988	0.988	0.988	0.988	0.987	0.987	0.987
LE	67.91	66.92	65.94	64.95	63.96	62.98	61.99	61.01	60.02	59.03	58.04	57.06	56.07	55.08	54.09
Variance	128.80	128.06	127.22	126.43	125.50	124.55	123.79	123.04	122.35	121.55	120.87	120.16	119.59	119.03	118.37
SD	11.35	11.32	11.28	11.24	11.20	11.16	11.13	11.09	11.06	11.03	10.99	10.96	10.94	10.91	10.88
CV	0.17	0.17	0.17	0.17	0.18	0.18	0.18	0.18	0.18	0.19	0.19	0.19	0.20	0.20	0.20
	30	**31**	**32**	**33**	**34**	**35**	**36**	**37**	**38**	**39**	**40**	**41**	**42**	**43**	**44**
l_x	0.987	0.986	0.986	0.986	0.986	0.985	0.985	0.984	0.984	0.983	0.983	0.982	0.982	0.981	0.980
LE	53.10	52.11	51.13	50.14	49.16	48.18	47.20	46.22	45.24	44.26	43.29	42.31	41.34	40.37	39.40
Variance	117.76	117.14	116.45	115.68	114.84	113.98	113.11	112.20	111.34	110.39	109.47	108.48	107.48	106.27	105.16
SD	10.85	10.82	10.79	10.76	10.72	10.68	10.64	10.59	10.55	10.51	10.46	10.42	10.37	10.31	10.25
CV	0.20	0.21	0.21	0.21	0.22	0.22	0.23	0.23	0.23	0.24	0.24	0.25	0.25	0.26	0.26
	45	**46**	**47**	**48**	**49**	**50**	**51**	**52**	**53**	**54**	**55**	**56**	**57**	**58**	**59**
l_x	0.979	0.978	0.977	0.976	0.974	0.973	0.971	0.970	0.968	0.966	0.963	0.960	0.957	0.954	0.951
LE	38.43	37.47	36.51	35.56	34.61	33.66	32.72	31.78	30.83	29.89	28.97	28.05	27.13	26.23	25.31
Variance	104.08	102.64	101.29	99.71	98.17	96.53	94.91	93.22	91.69	90.05	88.06	85.98	84.01	81.78	79.94
SD	10.20	10.13	10.06	9.99	9.91	9.82	9.74	9.65	9.58	9.49	9.38	9.27	9.17	9.04	8.94
CV	0.27	0.27	0.28	0.28	0.29	0.29	0.30	0.30	0.31	0.32	0.32	0.33	0.34	0.34	0.35
	60	**61**	**62**	**63**	**64**	**65**	**66**	**67**	**68**	**69**	**70**	**71**	**72**	**73**	**74**
l_x	0.946	0.942	0.936	0.932	0.926	0.919	0.911	0.903	0.893	0.885	0.874	0.861	0.847	0.831	0.815
LE	24.42	23.54	22.67	21.77	20.90	20.06	19.22	18.39	17.57	16.73	15.94	15.16	14.40	13.65	12.89
Variance	77.60	75.24	72.76	70.90	68.67	66.02	63.46	60.97	58.36	56.33	53.71	51.21	48.61	46.09	43.83
SD	8.81	8.67	8.53	8.42	8.29	8.13	7.97	7.81	7.64	7.51	7.33	7.16	6.97	6.79	6.62
CV	0.36	0.37	0.38	0.39	0.40	0.41	0.41	0.42	0.43	0.45	0.46	0.47	0.48	0.50	0.51
	75	**76**	**77**	**78**	**79**	**80**	**81**	**82**	**83**	**84**	**85**	**86**	**87**	**88**	**89**
l_x	0.796	0.773	0.750	0.721	0.695	0.661	0.627	0.587	0.547	0.503	0.464	0.425	0.376	0.326	0.285
LE	12.19	11.51	10.83	10.23	9.58	9.02	8.45	7.96	7.47	7.04	6.54	6.06	5.71	5.43	5.07
Variance	41.33	38.81	36.53	33.89	31.83	29.50	27.43	25.27	23.30	21.36	19.86	18.58	17.24	15.98	15.04
SD	6.43	6.23	6.04	5.82	5.64	5.43	5.24	5.03	4.83	4.62	4.46	4.31	4.15	4.00	3.88
CV	0.53	0.54	0.56	0.57	0.59	0.60	0.62	0.63	0.65	0.66	0.68	0.71	0.73	0.74	0.77

	90	91	92	93	94	95	96	97	98	99+
l_x	0.240	0.202	0.165	0.134	0.102	0.081	0.058	0.044	0.032	0.024
LE	4.84	4.56	4.37	4.13	4.12	3.92	4.09	4.07	4.27	4.29
Variance	14.16	13.54	13.09	12.90	12.91	13.09	13.53	13.83	14.11	14.12
SD	3.76	3.68	3.62	3.59	3.59	3.62	3.68	3.72	3.76	3.76
CV	0.78	0.81	0.83	0.87	0.87	0.92	0.90	0.91	0.88	0.88

Note: l_x : Survivorship, LE: mean life expectancy, SD: standard deviation, CV: coefficient of variation

Table B.3. Age distribution of longevity statistics, male 2013

	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14
l_x	1.000	0.989	0.988	0.987	0.987	0.986	0.986	0.986	0.985	0.985	0.985	0.985	0.985	0.984	0.984
LE	75.61	75.46	74.54	73.59	72.62	71.64	70.66	69.68	68.70	67.71	66.73	65.75	64.76	63.78	62.80
Variance	256.55	195.18	189.31	186.11	183.76	182.13	180.74	179.55	178.46	177.45	176.41	175.37	174.43	173.37	172.03
SD	16.02	13.97	13.76	13.64	13.56	13.50	13.44	13.40	13.36	13.32	13.28	13.24	13.21	13.17	13.12
CV	0.21	0.19	0.18	0.19	0.19	0.19	0.19	0.19	0.19	0.20	0.20	0.20	0.20	0.21	0.21
	15	**16**	**17**	**18**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**	**27**	**28**	**29**
l_x	0.984	0.983	0.983	0.982	0.981	0.981	0.980	0.979	0.979	0.978	0.977	0.977	0.976	0.975	0.975
LE	61.82	60.85	59.88	58.92	57.96	57.00	56.04	55.07	54.11	53.14	52.18	51.21	50.24	49.28	48.31
Variance	170.72	169.19	167.39	165.33	163.06	160.90	158.96	157.09	155.27	153.52	151.83	149.99	148.56	146.95	145.51
SD	13.07	13.01	12.94	12.86	12.77	12.68	12.61	12.53	12.46	12.39	12.32	12.25	12.19	12.12	12.06
CV	0.21	0.21	0.22	0.22	0.22	0.22	0.22	0.23	0.23	0.23	0.24	0.24	0.24	0.25	0.25
	30	**31**	**32**	**33**	**34**	**35**	**36**	**37**	**38**	**39**	**40**	**41**	**42**	**43**	**44**
l_x	0.974	0.973	0.973	0.972	0.971	0.971	0.970	0.969	0.968	0.967	0.966	0.965	0.963	0.962	0.960
LE	47.34	46.37	45.40	44.43	43.47	42.50	41.53	40.57	39.61	38.64	37.69	36.74	35.79	34.84	33.90
Variance	144.17	142.79	141.62	140.36	138.95	137.74	136.35	135.01	133.69	132.42	130.84	129.29	127.64	125.92	124.10
SD	12.01	11.95	11.90	11.85	11.79	11.74	11.68	11.62	11.56	11.51	11.44	11.37	11.30	11.22	11.14
CV	0.25	0.26	0.26	0.27	0.27	0.28	0.28	0.29	0.29	0.30	0.30	0.31	0.32	0.32	0.33
	45	**46**	**47**	**48**	**49**	**50**	**51**	**52**	**53**	**54**	**55**	**56**	**57**	**58**	**59**
l_x	0.958	0.956	0.954	0.952	0.948	0.945	0.941	0.937	0.933	0.927	0.921	0.914	0.908	0.900	0.891
LE	32.97	32.04	31.11	30.18	29.28	28.39	27.49	26.62	25.73	24.89	24.05	23.22	22.37	21.55	20.76
Variance	122.19	120.09	118.19	116.30	113.87	111.21	108.75	105.87	103.45	100.30	97.14	93.85	91.09	88.00	84.50
SD	11.05	10.96	10.87	10.78	10.67	10.55	10.43	10.29	10.17	10.01	9.86	9.69	9.54	9.38	9.19
CV	0.34	0.34	0.35	0.36	0.36	0.37	0.38	0.39	0.40	0.40	0.41	0.42	0.43	0.44	0.44
	60	**61**	**62**	**63**	**64**	**65**	**66**	**67**	**68**	**69**	**70**	**71**	**72**	**73**	**74**
l_x	0.881	0.871	0.859	0.849	0.834	0.819	0.804	0.787	0.770	0.750	0.728	0.708	0.682	0.659	0.631
LE	19.977	19.206	18.461	17.671	16.955	16.247	15.546	14.861	14.154	13.507	12.880	12.224	11.654	11.028	10.473
Variance	81.117	77.694	74.086	71.255	67.658	64.152	60.761	57.398	54.460	51.155	47.877	45.058	41.773	39.116	36.191
SD	9.007	8.814	8.607	8.441	8.225	8.009	7.795	7.576	7.380	7.152	6.919	6.713	6.463	6.254	6.016
CV	0.45	0.46	0.47	0.48	0.49	0.49	0.50	0.51	0.52	0.53	0.54	0.55	0.55	0.57	0.57
	75	**76**	**77**	**78**	**79**	**80**	**81**	**82**	**83**	**84**	**85**	**86**	**87**	**88**	**89**
l_x	0.604	0.574	0.542	0.507	0.482	0.453	0.412	0.371	0.335	0.298	0.261	0.228	0.193	0.160	0.131
LE	9.89	9.36	8.85	8.39	7.77	7.21	6.83	6.47	6.05	5.69	5.35	4.99	4.71	4.48	4.23
Variance	33.63	31.05	28.50	25.93	24.30	22.72	20.77	18.91	17.39	15.95	14.66	13.62	12.70	11.89	11.28
SD	5.80	5.57	5.34	5.09	4.93	4.77	4.56	4.35	4.17	3.99	3.83	3.69	3.56	3.45	3.36
CV	0.59	0.60	0.60	0.61	0.63	0.66	0.67	0.67	0.69	0.70	0.72	0.74	0.76	0.77	0.79

	90	91	92	93	94	95	96	97	98	99+
l_x	0.104	0.082	0.063	0.048	0.035	0.025	0.018	0.013	0.009	0.007
LE	4.07	3.89	3.78	3.67	3.68	3.78	3.74	3.70	3.86	3.98
Variance	10.80	10.50	10.37	10.42	10.64	10.86	11.02	11.33	11.72	11.86
SD	3.29	3.24	3.22	3.23	3.26	3.30	3.32	3.37	3.42	3.44
CV	0.81	0.83	0.85	0.88	0.89	0.87	0.89	0.91	0.89	0.87

Note: l_x : Survivorship, LE: mean life expectancy, SD: standard deviation, CV: coefficient of variation

Table B.4. Age distribution of longevity statistics, male 2019

	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14
l_x	1.000	0.991	0.991	0.990	0.990	0.990	0.989	0.989	0.989	0.989	0.989	0.989	0.989	0.988	0.988
LE	77.12	76.78	75.84	74.87	73.89	72.91	71.92	70.94	69.95	68.96	67.97	66.98	65.99	65.00	64.01
Variance	220.72	171.64	167.59	165.24	163.64	162.48	161.57	160.62	159.88	159.17	158.50	157.78	157.13	156.37	155.59
SD	14.86	13.10	12.95	12.85	12.79	12.75	12.71	12.67	12.64	12.62	12.59	12.56	12.53	12.50	12.47
CV	0.19	0.17	0.17	0.17	0.17	0.17	0.18	0.18	0.18	0.18	0.19	0.19	0.19	0.19	0.19
	15	**16**	**17**	**18**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**	**27**	**28**	**29**
l_x	0.988	0.988	0.987	0.987	0.986	0.986	0.986	0.985	0.985	0.984	0.984	0.983	0.983	0.982	0.982
LE	63.03	62.05	61.07	60.10	59.12	58.15	57.17	56.20	55.23	54.26	53.29	52.31	51.34	50.36	49.39
Variance	154.67	153.28	152.09	150.74	149.23	147.61	146.43	144.90	143.46	141.87	140.63	139.36	138.11	136.90	135.76
SD	12.44	12.38	12.33	12.28	12.22	12.15	12.10	12.04	11.98	11.91	11.86	11.81	11.75	11.70	11.65
CV	0.20	0.20	0.20	0.20	0.21	0.21	0.21	0.21	0.22	0.22	0.22	0.23	0.23	0.23	0.24
	30	**31**	**32**	**33**	**34**	**35**	**36**	**37**	**38**	**39**	**40**	**41**	**42**	**43**	**44**
l_x	0.981	0.981	0.980	0.980	0.979	0.978	0.978	0.977	0.976	0.976	0.975	0.974	0.973	0.971	0.970
LE	48.41	47.44	46.46	45.48	44.51	43.53	42.56	41.59	40.62	39.65	38.69	37.73	36.77	35.81	34.85
Variance	134.62	133.59	132.53	131.49	130.39	129.42	128.33	127.33	126.25	124.97	123.67	122.37	121.04	119.64	118.38
SD	11.60	11.56	11.51	11.47	11.42	11.38	11.33	11.28	11.24	11.18	11.12	11.06	11.00	10.94	10.88
CV	0.24	0.24	0.25	0.25	0.26	0.26	0.27	0.27	0.28	0.28	0.29	0.29	0.30	0.31	0.31
	45	**46**	**47**	**48**	**49**	**50**	**51**	**52**	**53**	**54**	**55**	**56**	**57**	**58**	**59**
l_x	0.969	0.967	0.965	0.963	0.961	0.958	0.955	0.951	0.948	0.944	0.939	0.933	0.927	0.920	0.913
LE	33.90	32.96	32.02	31.09	30.16	29.24	28.34	27.44	26.54	25.64	24.77	23.92	23.07	22.25	21.40
Variance	116.82	115.12	113.43	111.52	109.56	107.45	105.12	102.72	100.56	98.22	95.51	92.64	89.76	86.54	83.92
SD	10.81	10.73	10.65	10.56	10.47	10.37	10.25	10.14	10.03	9.91	9.77	9.63	9.47	9.30	9.16
CV	0.32	0.33	0.33	0.34	0.35	0.35	0.36	0.37	0.38	0.39	0.39	0.40	0.41	0.42	0.43
	60	**61**	**62**	**63**	**64**	**65**	**66**	**67**	**68**	**69**	**70**	**71**	**72**	**73**	**74**
l_x	0.904	0.895	0.884	0.875	0.862	0.848	0.833	0.816	0.798	0.784	0.762	0.741	0.718	0.694	0.671
LE	20.61	19.82	19.04	18.23	17.48	16.77	16.05	15.36	14.68	13.93	13.30	12.65	12.01	11.40	10.76
Variance	80.50	77.17	73.85	71.15	67.80	64.19	60.79	57.35	53.88	51.35	47.94	44.89	41.91	38.96	36.44
SD	8.97	8.78	8.59	8.43	8.23	8.01	7.80	7.57	7.34	7.17	6.92	6.70	6.47	6.24	6.04
CV	0.44	0.44	0.45	0.46	0.47	0.48	0.49	0.49	0.50	0.51	0.52	0.53	0.54	0.55	0.56
	75	**76**	**77**	**78**	**79**	**80**	**81**	**82**	**83**	**84**	**85**	**86**	**87**	**88**	**89**
l_x	0.642	0.612	0.583	0.548	0.517	0.481	0.447	0.409	0.370	0.329	0.301	0.268	0.225	0.184	0.153
LE	10.19	9.64	9.07	8.59	8.04	7.57	7.08	6.63	6.23	5.88	5.33	4.87	4.62	4.41	4.10
Variance	33.66	30.96	28.61	25.98	23.90	21.68	19.78	17.89	16.06	14.23	13.14	12.15	11.05	9.98	9.17
SD	5.80	5.56	5.35	5.10	4.89	4.66	4.45	4.23	4.01	3.77	3.63	3.49	3.32	3.16	3.03
CV	0.57	0.58	0.59	0.59	0.61	0.61	0.63	0.64	0.64	0.64	0.68	0.72	0.72	0.72	0.74

	90	91	92	93	94	95	96	97	98	99+
l_x	0.123	0.096	0.075	0.056	0.040	0.028	0.019	0.012	0.008	0.005
LE	3.88	3.66	3.43	3.25	3.16	3.10	3.14	3.22	3.39	3.83
Variance	8.45	7.89	7.52	7.40	7.51	7.86	8.49	9.29	10.22	10.86
SD	2.91	2.81	2.74	2.72	2.74	2.80	2.91	3.05	3.20	3.30
CV	0.75	0.77	0.80	0.84	0.87	0.91	0.93	0.95	0.94	0.86

Note: l_x : Survivorship, LE: mean life expectancy, SD: standard deviation, CV: coefficient of variation

IV. ESSAY 3: TRENDS IN LIFE EXPECTANCY AND LIFESPAN VARIATION IN TURKEY

Abstract

Background/aim: We explored the changes in life expectancy and lifespan variation in the period 2013-2019, focusing on sex differences and contributions of causes of death and age.

Method: Population and cause of death data were gathered from TurkStat. We obtained life expectancy, lifespan variation and threshold ages for Turkey for each year between 2013 and 2019. Life disparity was used as a measure of lifespan variation. The life expectancy and lifespan variation for each sex were then decomposed into causes of death and age components by using the line-integral model.

Results: Between 2013 and 2019, male's life expectancy increased much more than female's life expectancy mainly due to the reductions in infant mortality, improvement in unintentional injuries and malignant neoplasms at adult age and decline of malignant neoplasms and cardiovascular diseases at old ages. As for lifespan variation, up to threshold age, all causes except respiratory infections and infectious and parasitic diseases reduced variation. For males, the decline in lifespan variation was larger than for females, mainly due to the decline in unintentional injuries and malignant neoplasms at adult ages. When it comes to female-male gap in life expectancy and lifespan variation, we found that narrowing gap in life expectancy was mainly due to the reduction of male mortality due to malignant neoplasms and unintentional injuries.

Conclusion: To improve life expectancy and decrease variation, respiratory infectious diseases and infectious and parasitic diseases should be reduced, followed by a focus on deaths from cardiovascular diseases and malignant neoplasms. In addition, reducing unintentional injuries, which are still high in males, will also help to reduce the male-female life expectancy gap.

Key words: Lifespan variation, life expectancy, causes of death, decomposition techniques

IV.1. Introduction

Demographers use the term lifespan to refer the oldest age to which individuals can survive (Weeks, 2007). On the other hand, longevity means the ability to survive from one year to the next (Weeks, 2007). Longevity is calculated by life expectancy that is the average year a human would live in a year if individuals would expose the age specific mortality rates of that year (Preston et al., 2001; Smits & Monden, 2009; Weeks, 2007). Because life expectancy tells the average length of life based on the current mortality rates, it does not exactly give the actual length of human life. At this point lifespan variation, which indicates the variability of the length of life, plays an important role. Larger lifespan variation means higher uncertainty in the age at death (Aburto & van Raalte, 2018; Kannisto, 2001; van Raalte et al., 2011). Lifespan variation also discloses the heterogeneity in the health of population and countries with higher life expectancy have lower lifespan variation (Aburto et al., 2020; Aburto & van Raalte, 2018; Colchero et al., 2016; Vigezzi et al., 2022). Although lifespan variation and life expectancy are inversely related measures, two population with the same life expectancy may have different lifespan variations around the life expectancy (Aburto et al., 2020; Smits & Monden, 2009). While in some countries death rates may improve at all ages, in other countries this improvement may occur at older or younger ages.

At individual level, both life expectancy and lifespan variation have inferences on the individual's life course. From the societal level, effective policies such as investment in education, retirement policies require the life expectancy and lifespan variation measures (Aburto & van Raalte, 2018; van Raalte et al., 2011).

In this study, we focused on the changes in life expectancy and lifespan variation in Turkey between 2013 and 2019 and performed age and cause decomposition analysis to simultaneously understand the ages and causes of death that contributed to the changes in the life expectancy and lifespan variation. Furthermore, we calculated the threshold age at which improvement in mortality above and below this age had different impact on lifespan variation. Finally, we also analyzed the causes

and ages that contributed to the female-male gap in life expectancy and lifespan variation in 2013 and 2019. To the best of our knowledge, this is the first study that evaluate the life expectancy and lifespan variation patterns by age and cause in Turkey. It is unknown how Turkish lifespan variation changed and how causes of death contributed to changes in variation, and what kind of policies should be implemented for future. This study goes beyond the discussion about differences in mortality patterns between females and males in life expectancy and modal age at death. There are several reasons for our motivation to include lifespan variation. Firstly, it provides a more accurate comprehension of inequality in age at death and discloses the underlying causes of health inequalities. Second, understanding the contributions of age and cause to life expectancy and lifespan variations gives a clearer opinion about the future mortality scenarios.

This study has four aims:

- To calculate the change in life expectancy and lifespan variation between 2013 and 2019 by sex.
- To examine the age and cause contribution to changes in life expectancy and lifespan variation.
- To analyze the sex gap in life expectancy and lifespan variation
- To make policy inferences based on the findings.

IV.2. Literature Review

IV.2.1. Why Does Lifespan Variation Matter?

Lifespan variation indicates the certainty in the time of death. Higher lifespan variation indicates lower certainty in the anticipated death time and more heterogeneity in health status of population (Edwards, 2013; Edwards & Tuljapurkar, 2005; van Raalte et al., 2015). Increasing lifespan variation shows that the share of premature and preventable deaths are increasing (van Raalte et al., 2011). Due to the its important role, lifespan variation should be an area of interest to both academic researchers and policy makers.

Lifespan variation also enables to understand the health inequalities across subgroups such as among educational level (Edwards & Tuljapurkar, 2005; Permanyer et al., 2018; Sasson, 2016; van Raalte et al., 2011), income or occupational classes (Brønnum-Hansen, 2017; Edwards & Tuljapurkar, 2005; van Raalte et al., 2014, 2015), ethnic groups (Firebaugh et al., 2014) in a population.

At macro level, lifespan variation is the indicator of heterogeneity (Edwards & Tuljapurkar, 2005). At micro level, increase of lifespan inequality influences the individual's decisions on their life courses such as time of having child, saving money for the retirement, because such decisions depend not only the life expectancy but also on uncertainty about age at death (Aburto et al., 2020; Edwards & Tuljapurkar, 2005). Relationship of life expectancy and lifespan variation is also great importance in the field of economy. The theory of life cycle saving (Browning & Crossley, 2001) maintains that length of life very important factor on the saving and consumption decisions of people. For example, if parents think that children's lives are in danger, they may not want to invest in children's education due to the costly and non-tradable investment. In a high-risk environment in terms of length of life, people may choose the certainty of consumption over ambiguity of human capital investment (Edwards & Tuljapurkar, 2005). Another economical approach relates the fertility behavior and risk of child surviving (Kalemli-Ozcan, 2002). When the risk of surviving decreases,

individuals are more likely to have fewer children and to make more investments in their children's education. Edwards (2013) showed that reducing lifespan variation costs more than increasing life expectancy. According to the study, decline of 1 year in adult standard deviation corresponds to decline of half a year in life expectancy. If the variation is high in infancy period then the reduction in variation will be more costly (Edwards, 2013).

Particular characteristic of lifespan variation measures is that they give a threshold age which separates the "young-age component" from "old age component" (Zhang & Vaupel, 2009). Decline of mortality at any age leads to a higher life expectancy (James W. Vaupel & Romo, 2003), however this relation is not valid for lifespan variation. Lifespan variation decreases only when mortality decreases at ages below young-old threshold age (J. W. Vaupel et al., 2011; Zhang & Vaupel, 2009). Threshold age gives important information about the life expectancy-lifespan variation relationship: if improvement below threshold age outpaces the improvement above threshold age, life expectancy and lifespan variation will act inversely (Aburto et al., 2020; Aburto & van Raalte, 2018; Gillespie et al., 2014). The higher the life expectancy, the higher the threshold age is. This means that below threshold age, there is more age to save early lives and sustain the negative relationship between life expectancy and lifespan variation (Aburto et al., 2020; Shkolnikov et al., 2011; J. W. Vaupel et al., 2011). Improving life expectancy with lower lifespan variation has been expressed as "*compression of mortality"* or the "*rectangularization of survivorship"* (Aburto et al., 2020; Keyfitz; Hayflick; Fries). *Rectangularization of survivorship* describes the proportion of survivors of a cohort. It follows a path near 1.0 until arriving senescence age. In this age it suddenly drops 0.0. So, by eliminating premature deaths from some fatal diseases large portion of survivors will accumulate at the older ages and they will die due to biological incapability of organs, that is, "natural death".

IV.2.2. Differences in Life Expectancy and Lifespan Variation

If we look at the historical trend in life expectancy and lifespan variation in the world, between 18th century and middle of the 20th century, we see that the largest

gains in life expectancy and lifespan equality came from infancy period (J. W. Vaupel et al., 2011). Industrialization is a turning point in human history in terms of increasing life expectancy. Colchero and colleagues (2016) found that lifespan equality and life expectancy has increased greatly among industrialized human. Before industrialization, non-industrial humans and other primates were very similar in terms of life expectancy and lifespan equality measures. With industrialization, humans have begun differing from other primates in these measures (Colchero et al., 2016). With the progress in the medicine and better health conditions, infant mortality has substantially decreased, and life expectancy increased greatly. With these improvements, by the 21st century the effect of infancy period on life expectancy and lifespan variation has decreased significantly and effect of older ages has become important over time (Aburto et al., 2020). Aburto and colleagues (2020) showed that the effect of reduced mortality in infancy on lifespan variation was almost equal to the impact of saving lives between the ages of 76 and 80 years. Today, life expectancy has increased to a certain level in almost all countries and has approached each other. However, due to the differences in adult age variances, there are heterogenous trends among countries. Smits and Monden (2009) revealed the reasons for the different trends in lifespan variation in cross-country comparisons. For this aim, authors focused on the lifespan variations when the same levels of life expectancies were achieved by the countries. This approach provided to understand the differences in lifespan variation at similar level of life expectancy. Regarding the differences they proposed two hypotheses: "*The forerunner hypothesis*" and "*the diffusion hypothesis*". In the forerunner hypothesis, countries that achieve a certain life expectancy level first have lesser lifespan variation than countries that achieve the same life expectancy level afterwards. According to diffusion hypothesis, countries that achieve the same life expectancy level afterwards have lower levels of lifespan variation than forerunners of life expectancy, as they can benefit from the improvements in medicine and progress in the health innovations and take a lesson from experiences of forerunner countries. Findings of this study indicated that countries that got higher level of life expectancy sooner than others, was exposed to higher levels of lifespan inequality. Because while forerunner countries invested much in health developments, countries that fell behind reached these health innovations and developments faster and at lower

costs. Furthermore, decreasing lifespan inequality are easier for countries that fell behind due to the fact that decreasing adult mortality in these countries- where infectious diseases, injuries and accidental deaths are higher-had lower costs than the costs of decline of adult mortality in forerunner countries-where chronic diseases are the leading causes of death at adult ages (Smits & Monden, 2009). Apart from forerunner and diffusion hypothesis, there are some country examples that do not fit the above two hypotheses: Scenario of laggard countries. In this scenario, some developed countries which reaching the high level of life expectancy later in time have higher levels of variation (Seaman et al., 2016). Studies of Seaman and colleagues (2016) and Shkolnikov and colleagues (2011) can be given for the examples of laggard countries. Seaman and colleagues (2016) indicated the life expectancy levels Scotland and English and Wales for the years 1950 to 2012. They also compared the differences in lifespan variations by measuring the lifespan variations for the same levels of life expectancy. Scotland's higher lifespan variation was due to the lower old age and higher adult age mortality rate. At the same level of life expectancy, Scotland takes the advantage of old age mortality however, it is a temporal advantage due to the expansion of mortality and raised uncertainty at the age of death. The similar mortality pattern, expansion of mortality due to the high mortality rates at adult ages, have been observed in the USA (Shkolnikov et al., 2011).

Shkolnikov and colleagues (2011) found that below the threshold age coronary and heart diseases, causes of infant mortality had the higher contribution to decreases lifespan variability in USA and England and Wales between 1980 and 2002. Furthermore, decreasing level of lung cancers, traffic accidents and violence among males and decline of breast and cancers among females produced a significant contribution to lifespan variation. Although USA and England and Wales both decreased the variation, due to the excess mortality at adult ages, USA had higher variation than England and Wales.

To sum up, the life expectancy improvement generally continues with the decline of lifespan variation (Colchero et al., 2016). Since mortality improvement at young and adult ages has outstripped the improvement at older ages and there has been a compression of mortality, that is, most of the deaths have shifted towards older ages (Aburto & van Raalte, 2018; J. W. Vaupel et al., 2011; Wilmoth & Horiuchi, 1999). However, some studies indicated that in subpopulation level or in some periods increase in lifespan variation and life expectancy have occurred simultaneously (Aburto et al., 2018; Aburto & van Raalte, 2018; Brønnum-Hansen, 2017; Sasson, 2016; Seaman et al., 2016; van Raalte et al., 2014) due to the retardation in the decrease of young and adult age mortality with continued improvement in mortality of the elderly (Aburto & van Raalte, 2018; Engelman et al., 2014).

IV.2.3. Sex Difference in Life Expectancy and Lifespan Variation

Sex is an important determinant of life expectancy and lifespan variation. It is known that females tend to have longer life expectancy and lower lifespan variation compared to males (Aburto et al., 2020; Colchero et al., 2016; Edwards & Tuljapurkar, 2005). On the other hand, females' capacity to live with disabilities is higher than males. This means that although females live longer than males, they spend these extra years of life in poor health (Austad, 2006; Nusselder et al., 2010; Wingard, 1984). Although sex difference in life expectancy has been accepted as normal, it has been found that gap in life expectancy between females and males emerged with the increase of cardiovascular diseases and malignant neoplasms among adult mortality. The sex gap in life expectancy widened as deaths from infectious and parasitic diseases and maternal and infancy conditions declined and heart disease and neoplasms became the leading causes of death (Beltrán-Sánchez et al., 2015). There are two important factors that affect the excess male mortality: biological and behavioral factors. Among biological factors, hormonal and chromosomal factors are the main factors influencing the gap in life expectancy between sexes. X chromosome carries genes which affect the immune system. Having two X chromosome provides an advantage for females against most of the infectious and parasitic diseases (Giefing-Kröll et al., 2015; Oksuzyan et al., 2008; Waldron, 1985). So, infectious and parasitic diseases are more

fatal for males especially for ages 30 and above (Waldron, 1985). In addition to the biological factors, researches showed that gap in female-male life expectancy does not always stem from biological factors (McCartney et al., 2011; Oksuzyan et al., 2008; Thorslund et al., 2013). Historical trends of female-male gap in the life expectancy shows that gap changed in some periods (Thorslund et al., 2013). Gap in life expectancy at age 65 was around 1.1 years between 1750 and 1960; it reached 3.8 years in 1960 and 1990 and decreased to 3.1 years after 1990s. This finding implies that apart from biological factors, there are other factors that affect the female and male survivorship. According to a research (Glei & Horiuchi, 2007), narrowing gap in life expectancy was due to the sex differences in the age pattern of mortality, that is, variation of males' mortality decreased and contributed to narrowing sex gap in life expectancy.

The second important factor which causes the excess male mortality is behavioral factor. Main contributors of behavioral factors are smoking, alcohol abuse and risk-taking behaviors (McCartney et al., 2011; Oksuzyan et al., 2008). Deaths from injuries, accidents and violent are higher among males due to the risk-taking behavior of males. Garcia and Aburto (2019) showed that Venezuelan males have higher variation and lower life expectancy associated with higher rates of violence at adult ages (García & Aburto, 2019).

Deaths caused by ischaemic heart disease and malignant neoplasms also are strongly associated with smoking and alcohol abuse (Austad, 2006; Beltrán-Sánchez et al., 2015; Giefing-Kröll et al., 2015; Waldron, 1985). In another study (Lindahl-Jacobsen et al., 2013), it was found that behavioral factors do not explain the sex gap in life expectancy alone. They carried out a research in Utah where risk-taking behaviors among male population was low and they compared the female-male differentials in life expectancy with Sweden and Denmark where risk-taking behaviors among males relatively higher than males in Utah. Findings showed that sex gap in life expectancy in Utah did not differ from the Denmark and Sweden.

Several studies indicated that early deaths with higher lifespan variation were more common among underprivileged groups (Aburto & van Raalte, 2018; Brønnum-Hansen, 2017; Firebaugh et al., 2014; van Raalte et al., 2011). Edwards and Tuljapurkar (2003) evaluated the lifespan inequality and life expectancy trends by socio-economic groups in advanced countries since 1960. One of the reasons for high variances among males was the external causes of deaths at young and adult ages. Furthermore, some groups in population were more fragile in life expectancy and variation measures. For example, African-Americans, people with low education, and people in the poor quintile have higher variance and lower life expectancy. Similar to Edwards and Tuljapurkar's (2003) findings, some studies revealed that lifespan variation is lower and life expectancy is higher among individuals with higher levels of education, individuals with higher socio-economic status, or those belonging to certain races and ethnicities (Meara et al., 2008; Montez et al., 2011; Olshansky et al., 2012; Sasson, 2016; Firebaugh et al., 2014). Firebaugh and colleagues (2014) investigated the reasons of lifespan variability among black and whites in the United States. The study indicated that cause and sex specific intervention is crucial for decreasing racial disparities. van Raalte and colleagues (2011) evaluated the lifespan variation in low-educated groups for ten European countries between 1990 and 2000. In this group, greater lifespan variation was mainly due to higher deaths from injuries and neoplasms at younger ages. In another study, van Raalte and colleagues (2014) investigated the lifespan variation by age and causes of death among Finnish occupational class for the years between 1971 and 2010. According to results, while life expectancy improved among all classes, lifespan variation decreased in only nonmanual class. Reason for this difference was stem from early-adult age mortality. Although variation among elderly decreased in all classes, early-adult age variation decreased only in the upper nonmanual class. Since decline in early-adult variation due to circulatory diseases was offset by increase in lifespan variation due to circulatory diseases at older ages and high external mortality at adult ages, lifespan variation remained at stagnant level. In another study, life disparity and life expectancy were investigated among socioeconomic groups based on the register data on income and mortality between the years 1986 and 2014 (Brønnum-Hansen, 2017). According to the study, there are increasing gap between lowest and highest income quartile.

Saving early deaths is higher among highest quartile and there was no compression of mortality among lowest quartiles.

IV.3. Data and Method

IV.3.1. Data

In this study, population and cause of death datasets were taken from TurkStat by sex, year and single ages. In cause of death data, all causes were in ICD-10 coding. The data on population were obtained from Address-Based Population Registration System (ABPRS) of TurkStat for 2013-2019. In the data preparation process, cause of deaths with ICD-10 codes were grouped according to Annex Table A of WHO technical paper for Global Health Estimates. After the distribution of garbage codes, we selected top ten causes of death for analysis.

IV.3.2. Method

In this study, we investigated the indicators of life expectancy and lifespan variation since birth. We did not exclude the infancy and childhood periods as it is done in some studies (Edwards & Tuljapurkar, 2005; Gillespie et al., 2014; Smits & Monden, 2009; Tuljapurkar, 2010) due to the fact that infancy and childhood periods are important contributors of life expectancy and lifespan variation in Turkey.

In the literature, various dispersion indicators have been suggested to measure the variation in lifespan (Kannisto, 2001; Shkolnikov et al., 2003; Tuljapurkar, 2010; van Raalte & Caswell, 2013; Wilmoth & Horiuchi, 1999). These studies showed that since there are strong correlations between variation measures, used variation measure does not matter for results of the study (Colchero et al., 2016; van Raalte & Caswell, 2013; J. W. Vaupel et al., 2011).

Absolute and relative indicators measure the lifespan differences but, while absolute measures give the lifespan variability in number of years, relative measures are obtained by dividing absolute indicators to life expectancy (Appendix Table C.1). Relative variation measures such as Keyfitz's entropy, coefficient of variation etc.

gives information about the spread of death distribution compared with its average value (life expectancy) (Colchero et al., 2016; Shkolnikov et al., 2003). Absolute indicators can remain constant if all lifespans increase or decrease in an equal number of years (Vigezzi et al., 2022).

Relative measures allow to compare populations which have different level of life expectancy and different species (Aburto et al., 2019; Baudisch, 2011; Wrycza et al., 2015; Colchero et al., 2016; Smits & Monden, 2009). Absolute measures are, on the other hand, appropriate to understand lifespan variation in different countries and subpopulations (Aburto et al., 2019; Brønnum-Hansen, 2017; van Raalte et al., 2014). Calculations of absolute measures and their relative counterparts are presented in Appendix Table C.1.

Although there are strong correlations between variation measures, some studies indicated that relative and absolute lifespan measures may not give the similar trends (Mackenbach et al., 2016; Vigezzi et al., 2022). Vigezzi and colleagues (2022) showed that choice of lifespan indicator (relative or absolute) can be very important in the periods of mortality increases /shocks (such as famines and epidemics). According to the study, during the mortality crises relative and absolute lifespan variation behave differently. While relative lifespan variations increase, absolute lifespan variations decrease during mortality crises (Vigezzi et al., 2022).

In this study, we showed the contributions to life expectancy and lifespan variation by using relative and absolute measures. As an absolute measure, we selected the life disparity ($e^{\dagger}$) measure, average years of life lost, because its interpretation is simple in public health context. Total change in disparity equals to the sum of every age-cause specific contribution to disparity (Aburto & van Raalte, 2018). To alleviate any concern, we performed decomposition analysis also with a relative measure, CoV, following the researchers (Aburto et al., 2018; Wrycza et al., 2015).

We could not find substantial differences with the results presented in this essay (see Appendix Figure C.1).

IV.3.2.1. Decomposition technique

Before decomposition analysis we first calculated the age and cause specific death rates. Let m_a^c be the age and cause specific death rate of cause c at age a. Then,

$$m_a^c = m_a \times prop_a^c \quad (1)$$

where m_a is age-specific death rate at age a

$$m_a = \frac{Total\ number\ of\ death\ at\ age\ \boldsymbol{a}}{Mid-year\ population\ at\ age\ \boldsymbol{a}} \quad (2)$$

and $prop_a^c$ denotes the proportion of cause c at age a in total death counts at age a

$$prop_a^c = \frac{Number\ of\ death\ at\ age\ \boldsymbol{a}\ from\ cause\ \boldsymbol{c}}{Total\ number\ of\ death\ at\ age\ \boldsymbol{a}} \quad (3)$$

After calculation of age-cause specific death rates we performed the decomposition process. In this study we used the line integral model of decomposition proposed by Horiuchi et al. (2008). Decomposition methods proposed by researchers (Andreev et al., 2002; Arriaga, 1984; Gupta, 1999; Kitagawa, 1955; Pollard, 1988; Pullum et al., 1989) focus on discrete changes in life expectancy, while line integral method (Horiuchi et al., 2008) handles the continuous change. In decomposition analysis if sum of the main effects (direct and indirect effects) does not match the overall change of dependent variable, there will be a discrepancy which is called interaction effect or residual term. Interaction effect and residual terms make the interpretation a bit difficult (Horiuchi et al., 2008). Kitagawa's decomposition method gives an interaction term and Delta method (Pullum et al., 1989) gives a residual term. Line integral method does not have an interaction or residual terms and allows many covariates in continuous change.

Let y be the differentiable function of n covariates represented by $x = [x_1, x_2, \dots, x_n]$ Then y can be expressed as the function of x. We can express y as $y = f(x_1, x_2 \dots, x_n)$. Any impact of x on y produces a change in y. Let both y and x depend on time t and let x be a differentiable vector function of t between t_1 and t_2. Then, we define the function as $y(t) = f\big(x(t)\big) = f(x_1(t), x_2(t), \dots, x_n(t))$ and change in y between t_1 and t_2 can be expressed as,

$$y(t_2) - y(t_1) = \int_{t_1}^{t_2} \frac{d}{dt} y(t) dt = \int_{t_1}^{t_2} \left\{ \sum_{i=1}^{n} \frac{\partial}{\partial x_i(t)} y(t) \frac{d}{dt} x_i(t) \right\} dt \qquad (4)$$

For simplicity, if we drop the t from equation (4) , then we get,

$y_2 - y_1 = \sum_{i=1}^{n} c_i$ where $c_i = \int_{x_{i1}}^{x_{i2}} \frac{\partial y}{\partial x_{i1}} dx_i$. According to this equation, c_i is the total change in y produced by x_i. In other words, c_i is the effect of x_i on y.

According to this model life disparity ($e^{\dagger}$) and life expectancy (e_0) correspond to dependent variable *y*. Covariates are age and cause specific mortality rates (Horiuchi et al., 2008). This model justifies that decomposition of a change in dependent variable is stated as the sum of the covariates' effects. Furthermore, since this model assumes the continuous/gradual change of covariates along the time, it also enables the elimination of interaction effects. In this study, total changes in y were computed by using the algorithm implemented by (Riffe, 2018).

We selected top ten causes of death and then decomposed total change in life expectancy and life disparity by age and cause. As proposed by Horiuchi and colleagues (2008), we decomposed the change for each year between 2013 and 2019 and then combined the six sets of decomposition results. All calculations were executed using R programming.

IV.3.2.2. Calculation of threshold age for life disparity

Zhang and Vaupel (2009) showed that in some conditions there is an age that separates early deaths from late deaths. According to their proposition:

Life lost due to death (or lost life expectancy) is

$$e^{\dagger} = \int_0^{\infty} d(a)e(a)da \tag{5}$$

where $e(a) = \frac{\int_a^{\infty} l(x)dx}{l(a)}$ (remaining life expectancy at age a) and $d(a) = l(a) \times \mu(a)$ (distribution of deaths in the life table). It should be noted that $l(a)$ is the probability of survival to age a with $l(a) = \exp(-\int_0^a \mu(x)dx)$ and $l(0) = 1$. Cumulative hazard function is calculated as $H(a) = \int_0^a \mu(x)dx$ and $(0) = 0$.

The age specific impact of reduction in death rates on lifespan disparity is derivative of $e^{\dagger}$ with respect to the change of mortality:

$$\begin{aligned} g(a) = \frac{de^{\dagger}}{-dln\mu(a)} &= d(a)\left[e^{\dagger}(a) - e(a)\left(1 + \ln l(a)\right)\right] \\ &= d(a)[e^{\dagger}(a) - e(a)\left(1 - \mathrm{H}(a)\right)] \end{aligned} \tag{6}$$

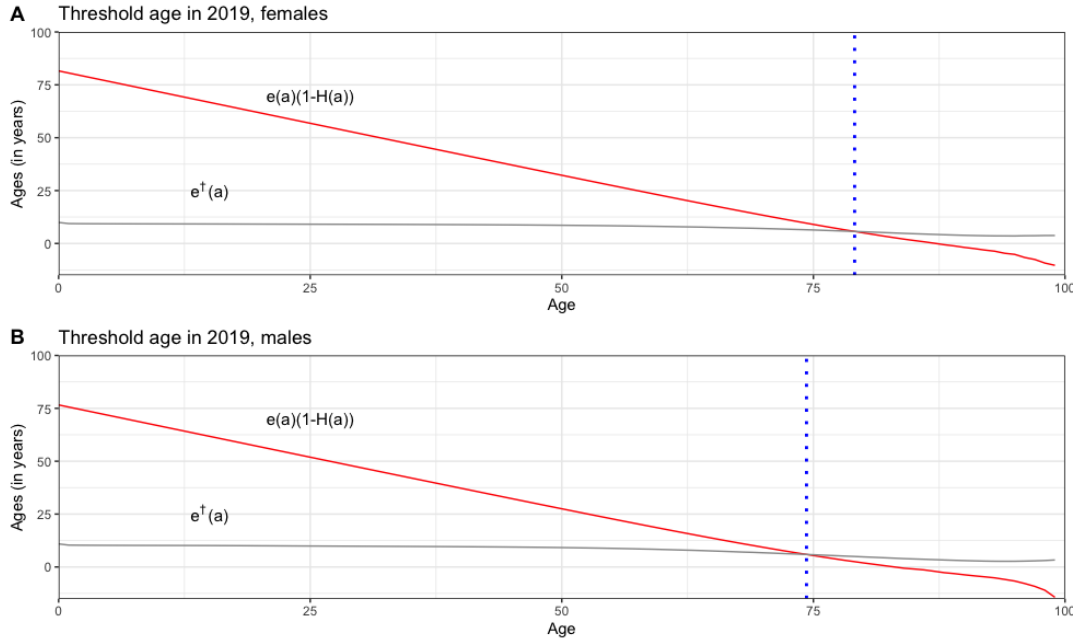
Zhang and Vaupel (2009) showed that there exists a unique threshold age ($a^{\dagger}$) greater than zero for life disparity ($e^{\dagger}$) when $\frac{e^{\dagger}}{e(0)} < 1$. If $\frac{e^{\dagger}}{e(0)} = 1$ then preventing deaths at age zero has not any effect on life disparity but preventing deaths above age zero increases the lifespan disparity. If $\frac{e^{\dagger}}{e(0)} > 1$ then there is not any threshold age, that is, preventing deaths at any age increases lifespan inequality.

In the light of these formulations, Zhang and Vaupel (2009) proved that the age at which

$$e^{\dagger}(a) = e(a)(1 - H(a)) \tag{7}$$

gives the threshold age $a^{\dagger}$ (Zhang & Vaupel, 2009). For better understanding, graphical depiction of this equality presented in Figure IV.3.1.

Figure IV.3.1. Graphical demonstration of the threshold age calculation for Turkey, 2019



IV.4. Results

Table IV. 4.1 shows the percent distribution of causes of deaths between 2013 and 2019. In this table except neurological conditions, genitourinary diseases and respiratory infections and infectious and parasitic diseases percentage of all causes decreased from 2013 to 2019 for both sexes. Cardiovascular and respiratory diseases remained almost at the same level among males.

Table IV.4.1. Percent distribution of causes of death by sex and years.

	Causes of death	**2013**	**2019**
Female	Cardiovascular diseases	44.7	42.1
	Malignant neoplasms	16.3	14.9
	Respiratory diseases	7.3	7.1
	Neurological conditions	5.1	6.1
	Genitourinary diseases	4.1	5.2
	Diabetes mellitus	5.2	4.0
	Resp.inf. & Inf.and parasitic dis.	4.1	10.2
	Neonatal conditions	1.9	1.3
	Unintentional injuries	2.8	1.9
	Other	8.4	7.2
	Total	**100.0**	**100.0**
Male	Cardiovascular diseases	35.6	35.7
	Malignant neoplasms	25.2	23.0
	Respiratory diseases	9.5	9.4
	Neurological conditions	3.5	3.9
	Genitourinary diseases	3.4	4.2
	Diabetes mellitus	3.0	2.5
	Resp.inf. & Inf.and parasitic dis.	3.9	8.9
	Neonatal conditions	2.0	1.4
	Unintentional injuries	5.6	3.6
	Other	8.4	7.4
	Total	**100.0**	**100.0**

Age and causes specific death rates for females and males are indicated in log-scale in Figure IV.4.1 and Figure IV.4.2, respectively. Cardiovascular and respiratory diseases a bit decreased in infancy and young ages for both sexes and they remained almost in the same level at adult and older ages. Malignant neoplasms decreased in almost every age, but decline is higher among males. Neurological conditions and genitourinary diseases decreased at infancy and young ages for both sexes; increased at adult and older ages among females and remained almost at the same level at adult and older ages among males. Respiratory infections and infectious and parasitic

diseases are important because death rates increased significantly at adult and older ages for both sexes. Unintentional injuries decreased significantly at young and adult ages, but decline is more among males. Other diseases in which congenital anomalies constituted the major parts decreased more among males at infancy and young ages.

Figure IV.4.1. Age-causes specific death rates, females

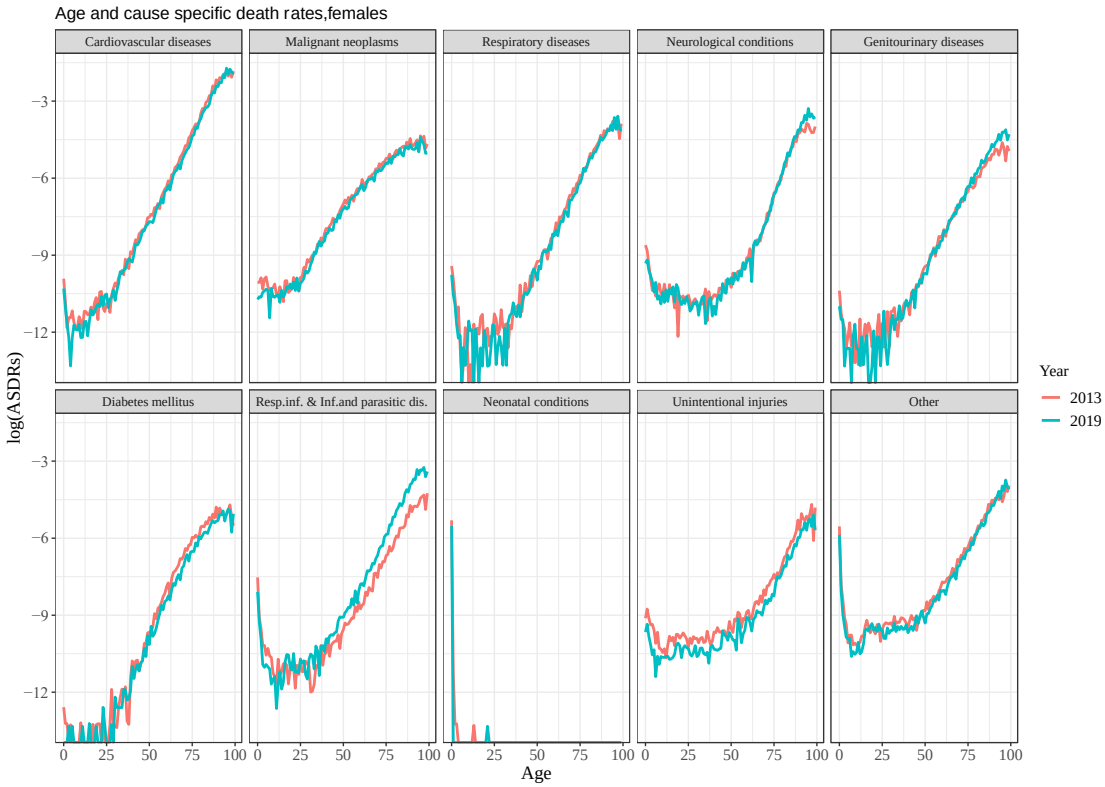
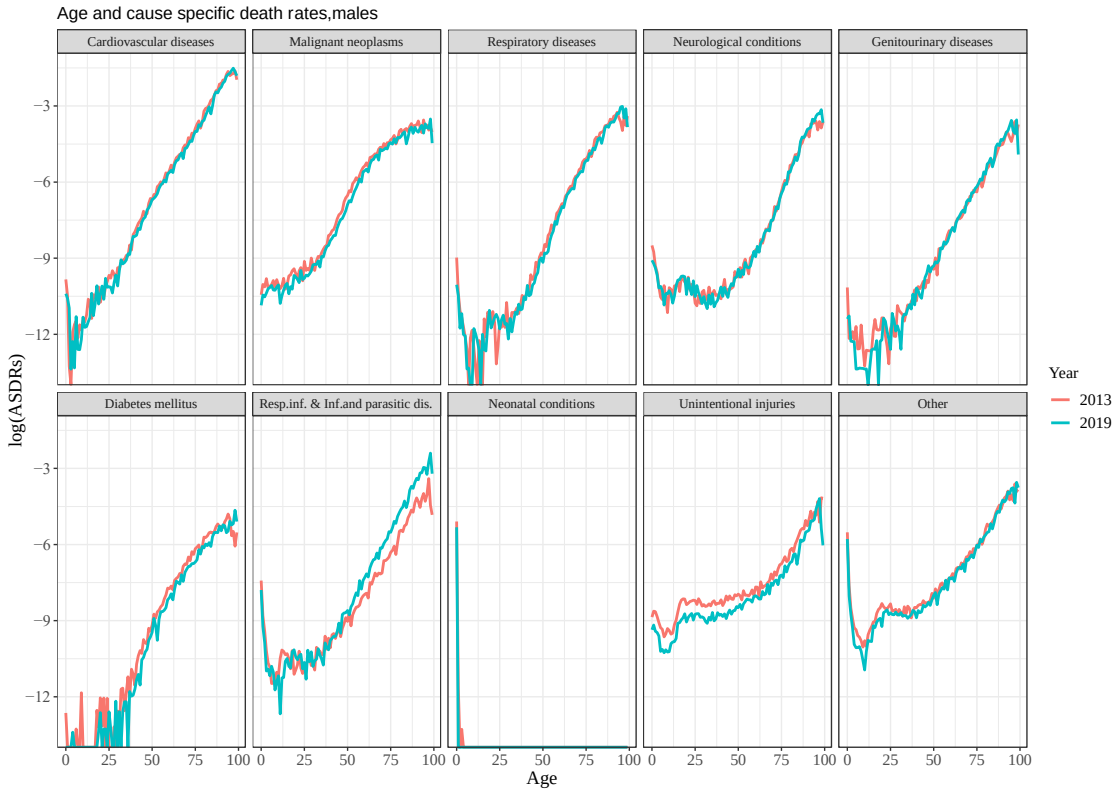


Figure IV.4.2. Age-causes specific death rates, males



According to Table IV.4.2, life expectancy of both sexes increased between 2013 and 2019 and it reached to 81.6 and 76.6 for females and males, respectively. Life disparity decreased from 11.4 to 10.7 among males and 10.4 to 9.7 among females. In parallel with life expectancy, threshold age also increased for both sexes.

Table IV.4.2. Distribution of life expectancy, life disparity, coefficient of variation and threshold ages by sex and years

	Year	Life expectancy (e(0))	Life disparity ($e^{\dagger}$)	Coefficient of variation (CoV)	Threshold age
Female	2013	80.6	10.4	0.1855	78.0
	2014	80.2	10.3	0.1872	77.8
	2015	80.3	10.2	0.1848	77.9
	2016	80.1	10.2	0.1872	77.9
	2017	80.6	10.0	0.1823	78.4
	2018	81.1	9.9	0.1756	78.7
	2019	81.6	9.7	0.1700	79.1
Male	2013	75.1	11.4	0.2133	72.8
	2014	75.0	11.3	0.2149	73.0
	2015	75.1	11.1	0.2104	73.1
	2016	74.8	11.3	0.2156	72.9
	2017	75.4	11.1	0.2098	73.4
	2018	75.9	11.0	0.2027	73.7
	2019	76.6	10.7	0.1940	74.3

IV.4.1. Age and cause specific contribution

Figure IV.4.3-A and IV.4.3-B show the age contributions to life expectancy for all years between 2013 and 2019 for females and males, respectively. All ages except the oldest ages have positive contributions on life expectancy. Age contribution to life expectancy is higher between age group 5-9 and 60-64 among males than among females. The greatest gain in life expectancy among females came from infancy period and ages between 65 and 75. For males, on the other hand, the largest contribution came from infancy period and ages between 50 and 75. When we come to figures IV.4.3-C and IV.4.3-D, parallel with the increasing life expectancy trend, life disparity at all ages up to age 75 decreased for both sexes. Life disparity among males, especially ages between 5 and 65, decreased more than the life disparity among females.

Figure IV.4.3. Age contributions to changes in life expectancy and life disparity between 2013 and 2019

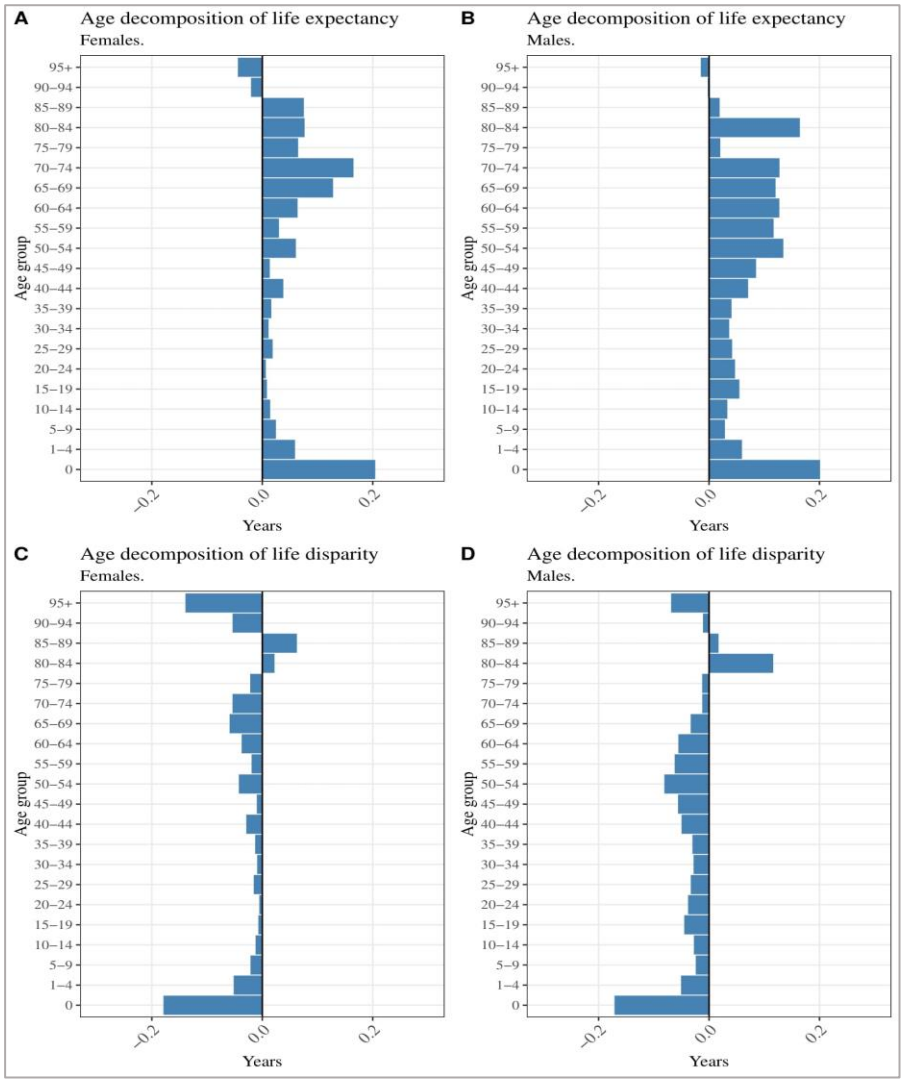


Figure IV.4.4 and Table IV.4.3 indicate the age-cause contribution to life expectancy (A-B) and life disparity (C-D) for each year interval between 2013 and 2019. Furthermore, table versions of Figure IV.4.4 were presented in Appendix Tables C.2-C.5. According to Figure IV.4.4-A and IV.4.4-B, improvements in neonatal conditions and other causes in infancy period had positive impact on life expectancy for both sexes. Decline in cardiovascular diseases at older ages among females and improvements of malignant neoplasms at older ages among males had the highest contribution to life expectancy. Furthermore, important contribution to life expectancy among males also comes from decline in unintentional injuries at young and adult

ages. On the other hand, increase in respiratory tract infections and infectious and parasitic diseases at ages 45 and above caused a reduction in life expectancy for each sex.

According to Figure IV.4.4-C and IV.4.4-D, below the threshold age, decline of inequality are higher among males than females. Decline in infant mortality among males and females is almost equal and has the highest impact on the decline of lifespan inequality. Furthermore, for males, unintentional injuries and malignant neoplasms below threshold age decreased variation significantly. For females, decline of cardiovascular diseases and diabetes mellitus below threshold age decreased the lifespan inequality. Increase of mortality due to neurological conditions at older ages among females decreased the lifespan inequality.

Furthermore, respiratory tract infections and infectious and parasitic diseases created a significant impact on life expectancy and lifespan inequality. Mortality from infectious diseases increased above age 45 for both sexes, it caused the highest decline of life expectancy (0.43 years among females and 0.35 years among males) (Table IV.4.3 and Appendix Table C.2 and Appendix Table C.3). If we look from the viewpoint of lifespan inequality, increase of infectious diseases increased the lifespan inequality below threshold age and decreased the lifespan inequality above threshold age. When we look at the net impact, due to the higher increase in mortality above threshold age, infectious diseases created a reduction in lifespan inequality.

In summary, decline of infancy related diseases and increase of respiratory tract infections and infectious and parasitic diseases at older ages for both sexes and decline of unintentional injuries and malignant neoplasms among males made major contribution to decreasing lifespan inequality. Improvement in the infancy related diseases, cardiovascular diseases, malignant neoplasms, diabetes mellitus (females), respiratory diseases (males) and unintentional injuries (males) created significant contribution to the increasing life expectancy.

Figure IV.4.4. Age and cause decomposition of life expectancy and life disparity between 2013 and 2019

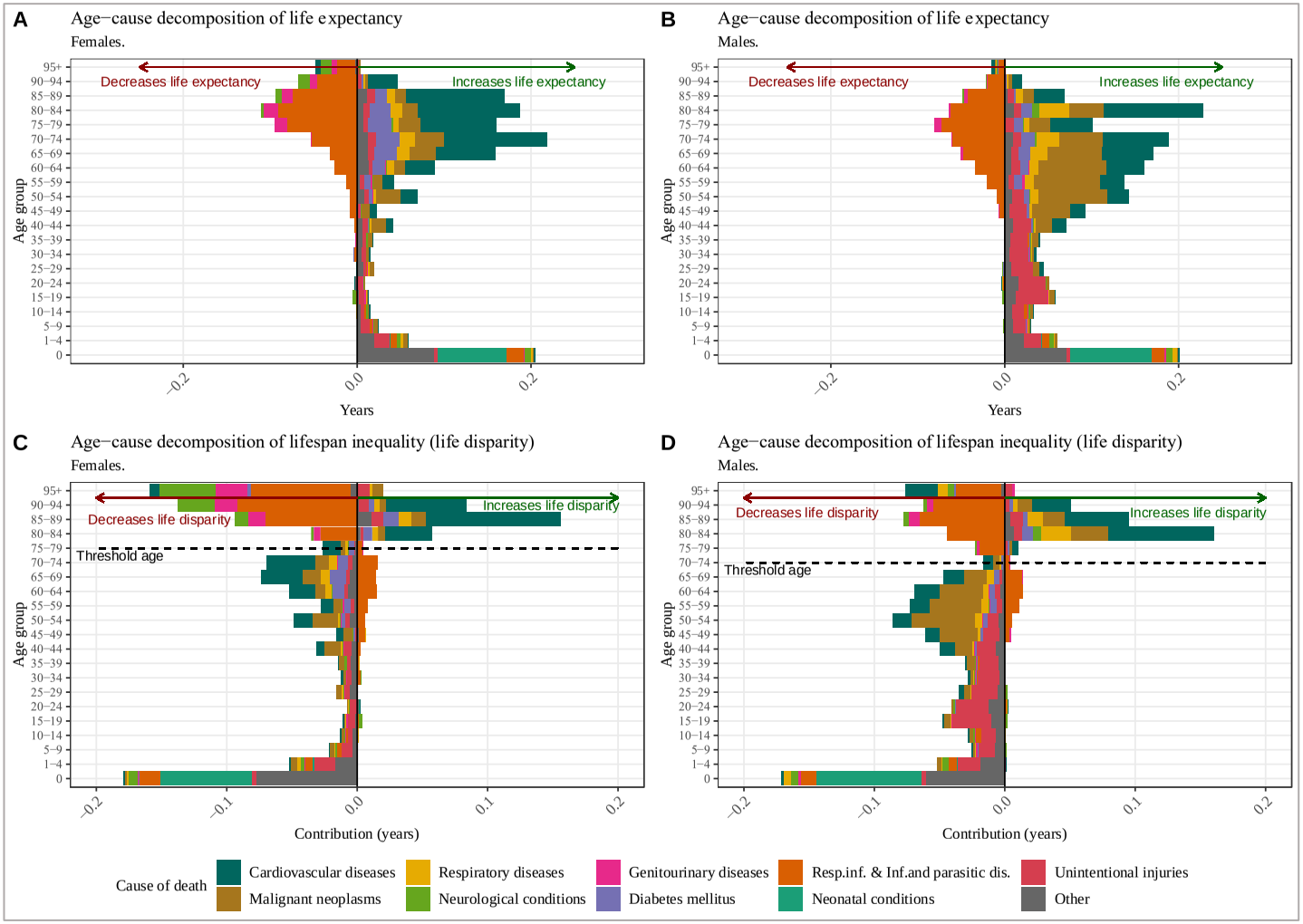


Table IV.4.3. Cause-specific contributions to the change in e(0), $e^{\dagger}$ and CoV among females and males, between 2013 and 2019

Sex		Causes of death	Contribution to e(0) (years)	Contribution to $e^{\dagger}$ (years)	Contribution to CoV (units)
Female	1	Cardiovascular diseases	0.63	0.05	-0.00023
	2	Malignant neoplasms	0.23	-0.08	-0.00173
	3	Respiratory diseases	0.08	-0.01	-0.00033
	4	Neurological conditions	-0.02	-0.10	-0.00092
	5	Genitourinary diseases	-0.05	-0.07	-0.00058
	6	Diabetes mellitus	0.15	-0.02	-0.00026
	7	Respiratory inf. & Inf.and parasitic dis.	-0.43	-0.22	-0.00184
	8	Neonatal conditions	0.08	-0.07	-0.00297
	9	Unintentional injuries	0.13	-0.05	-0.00197
	10	Other	0.21	-0.13	-0.00473
	Total contribution		**1.01**	**-0.70**	**-0.0156**
Male	1	Cardiovascular diseases	0.48	0.04	-0.00015
	2	Malignant neoplasms	0.53	-0.16	-0.00289
	3	Respiratory diseases	0.14	0.00	-0.00023
	4	Neurological conditions	0.02	-0.02	-0.00043
	5	Genitourinary diseases	-0.01	-0.02	-0.00029
	6	Diabetes mellitus	0.09	-0.01	-0.00017
	7	Resp.inf. & Inf.and parasitic dis.	-0.35	-0.19	-0.00192
	8	Neonatal conditions	0.10	-0.08	-0.00324
	9	Unintentional injuries	0.30	-0.17	-0.00520
	10	Other	0.22	-0.14	-0.00483
	Total contribution		**1.51**	**-0.76**	**-0.0194**

IV.4.2. Age and cause contribution to sex gap in life expectancy and life disparity

Figure IV.4.5, and Figure IV.4.6 indicate the age-cause contribution to the female-male difference in life expectancy and lifespan inequality, respectively. The gap in life expectancy decreased from 5.5 years in 2013 to 5 years in 2019. On the other hand, the gap in lifespan inequality stayed almost in the same level (0.99 years in 2013 and 0.93 years in 2019). Figure IV.4.5 present the female-male gap in 2013 (figure IV.4.5 above) and in 2016 (figure IV.4.5 below). Females had advantage in life expectancy across all ages and all causes. It is clear that between 2013 and 2019, the gap decreased mainly due to the decline of male mortality from malignant neoplasms and unintentional injuries up to age 65. In total, decreasing rate of malignant neoplasms and unintentional injuries contributed to the narrowing of the gap in life expectancy by 0.33 and 0.21 years, respectively (Table IV.4.4). On the other hand, respiratory infections and infectious and parasitic diseases contributed to the opposite side and widened the gap in life expectancy. When we look at the female-male gap in lifespan variation (Figure IV.4.6), we see that improvement in malignant neoplasms and unintentional injuries among males was not reflected in the gap in lifespan inequality as much as the gap in life expectancy. The notable decrease in the gap occurred between ages 15 and 45 (Figure IV.4.6-A and IV.4.6-B). Furthermore, it is seen that lifespan variation is higher among males below age 75 and lifespan variation is higher among females for ages above 75. In total, the major contribution to reducing the gap in the lifespan inequality comes from unintentional injuries (0.11 years), and the contribution of all other diseases remains small (Table IV.4.4). Since improvement in malignant neoplasms occurring in females aged 75 years and older was offset by improvement in males aged 74 years and younger, the net effect on the gap in lifespan variation did not change significantly. As a leading cause of death, cardiovascular diseases did not contribute to the difference in life expectancy and life disparity significantly.

Figure IV.4.5. Age and cause contributions to the gap between sexes in life expectancy

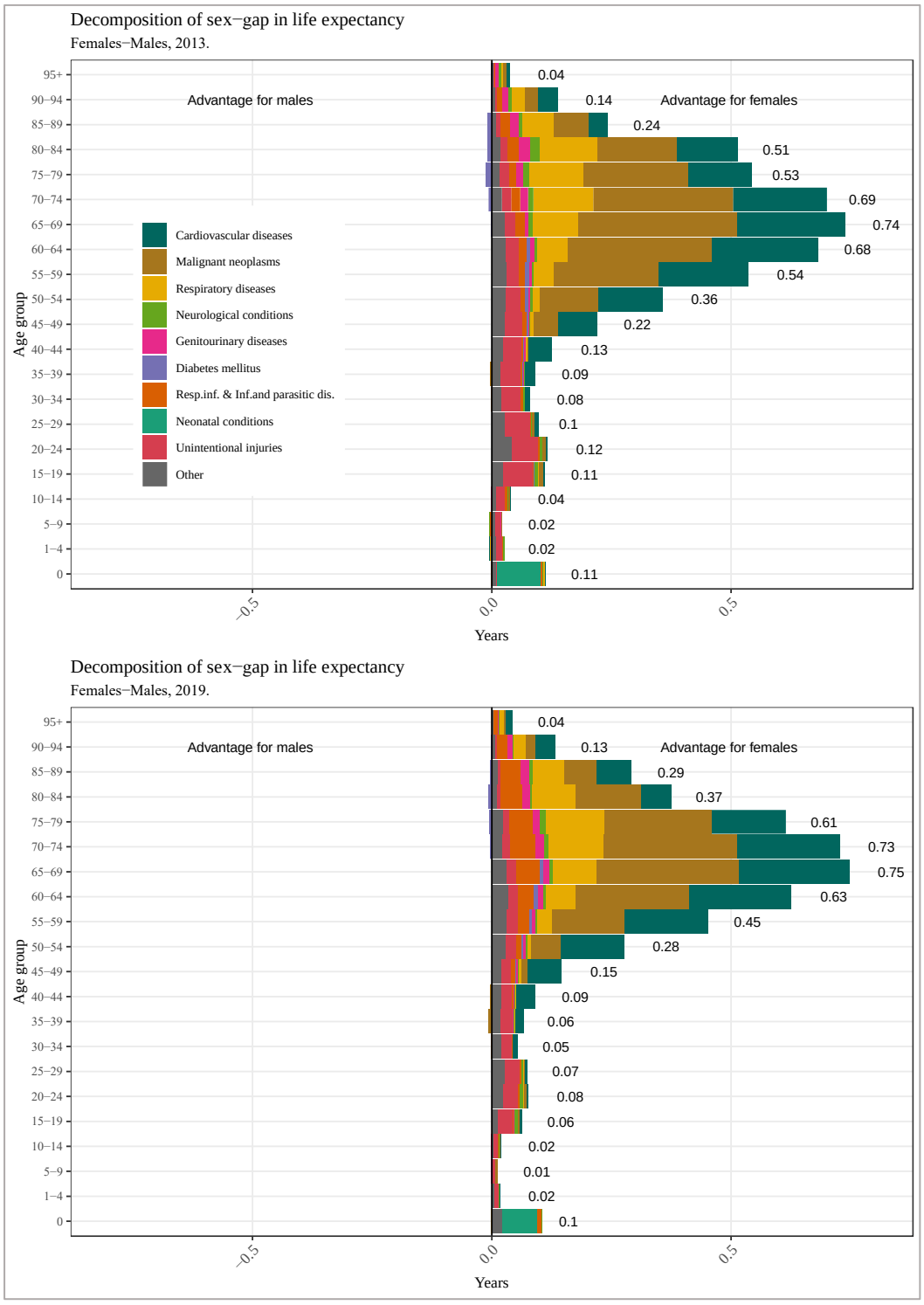


Figure IV.4.6. Age and cause contributions to the gap between sexes in life disparity

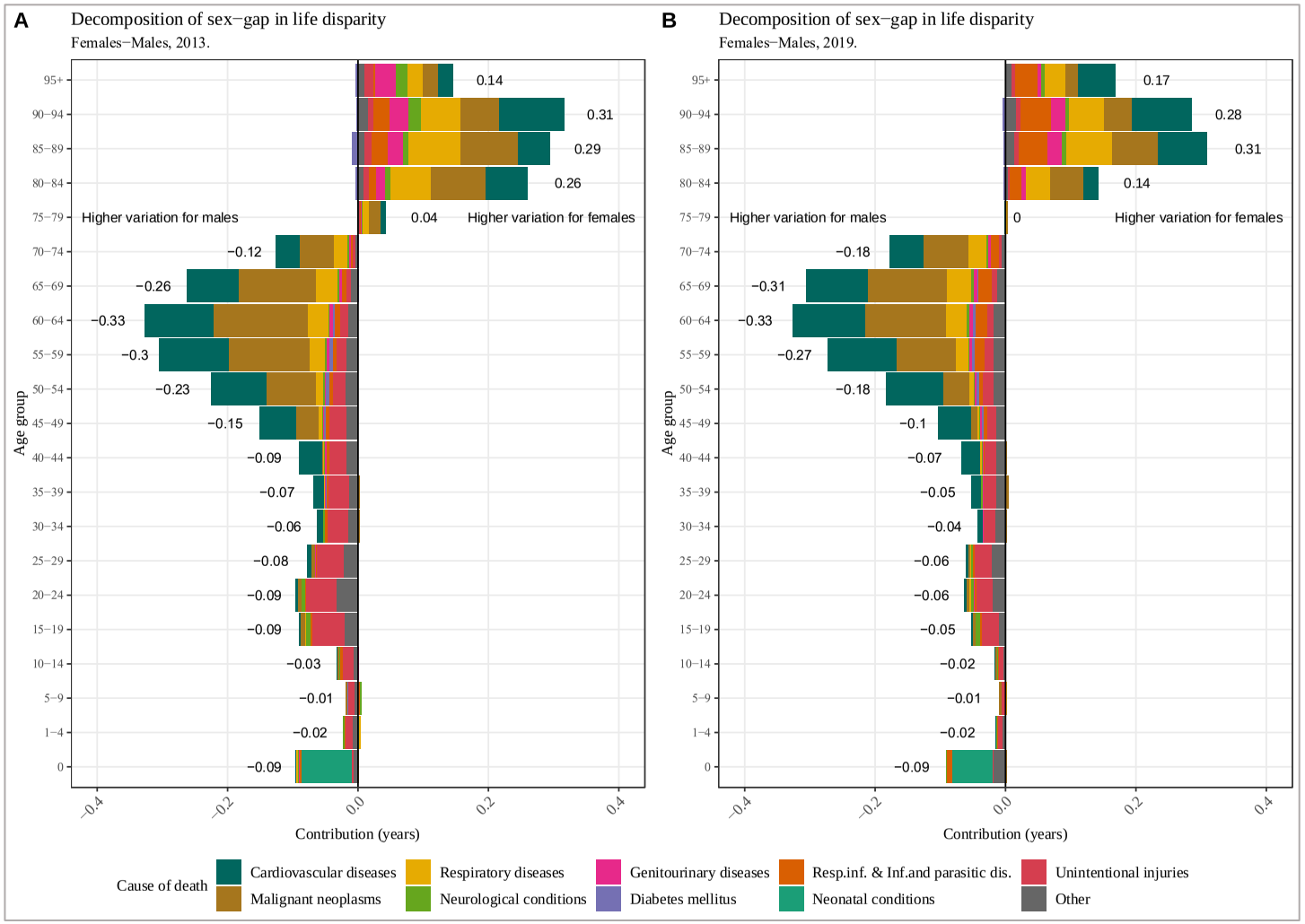


Table IV.4.4. Cause-specific contributions to the gap between females and males in e(0) and $e^{\dagger}$. (Females- Males)

	Causes of death	**2013**	**2019**
Life expectancy (e(0))	Cardiovascular diseases	1.50	1.47
	Malignant neoplasms	1.83	1.50
	Respiratory diseases	0.69	0.63
	Neurological conditions	0.11	0.09
	Genitourinary diseases	0.13	0.12
	Diabetes mellitus	-0.01	0.01
	Respiratory inf. & Inf.and parasitic dis.	0.19	0.37
	Neonatal conditions	0.09	0.07
	Unintentional injuries	0.55	0.34
	Other	0.39	0.36
	Total gap	**5.47**	**4.98**
	Causes of death	**2013**	**2019**
Life disparity ($e^{\dagger}$)	Cardiovascular diseases	-0.29	-0.32
	Malignant neoplasms	-0.30	-0.28
	Respiratory diseases	0.10	0.06
	Neurological conditions	0.02	-0.02
	Genitourinary diseases	0.08	0.03
	Diabetes mellitus	-0.04	-0.03
	Respiratory inf. & Inf.and parasitic dis.	0.01	0.05
	Neonatal conditions	-0.08	-0.06
	Unintentional injuries	-0.31	-0.20
	Other	-0.18	-0.16
	Total gap	**-0.99**	**-0.93**

IV.5. Conclusion and Discussion

In this essay we indicated the trend in death rates on life expectancy and lifespan variation. Although there is a strong correlation between lifespan variation measures, we performed the decomposition analysis with absolute (lifespan disparity) and relative (coefficient of variation) measures. Findings showed that there were not major differences in results. According to the results, life expectancy increased, and life disparity decreased between 2013 and 2019. We also analyzed the female-male gap in life expectancy and lifespan variation in 2013 and 2019. The female-male difference in life expectancy and lifespan variation decreased 0.5 years and 0.06 years, respectively.

IV.5.1. Age and cause of death contributions to changes in e(0) and $e^{\dagger}$ between 2013 and 2019

A decline in mortality increases life expectancy, regardless of age. On the other hand, impact of mortality decline on lifespan variation is higher and negative below threshold age, the effect decreases as age increases and goes to the positive values after the threshold age. This means that causes which increase life expectancy and causes that decrease lifespan variation may not be the same (Aburto et al., 2018). Our findings also disclosed that causes that have created higher lifespan inequality are different from causes that have created higher life expectancy. While improvement in cardiovascular diseases and malignant neoplasms increased life expectancy significantly, improvement in infant mortality created a greater equality. It is very important to point out that causes of death at younger ages carry importance not only for higher life expectancy but also for low disparity. Improvement in infancy period led to highest reduction in the lifespan variation. Especially improvement in neonatal conditions and other diseases (mainly congenital anomalies) created the highest contribution to both life expectancy and lifespan variation.

According to the results, it was observed that diseases affecting the change in life expectancy and variation differ in age groups.

The mortality reduction in infancy period provided higher life expectancy and lower inequality. Especially improvement in neonatal conditions and other diseases (mainly congenital anomalies) created the highest contribution to both life expectancy and lifespan variation.

For ages from 1 to 14, improvement in unintentional injuries and other diseases (mainly congenital anomalies) made important contribution to rising life expectancy and declining variation. In this age group contribution is higher for male population than females.

For 15-34 age group, contribution remined very little among females, but thanks to the improvement in unintentional injuries, males got great advantage in life expectancy and lifespan variation.

Between age 35 and 64 decrease in malignant neoplasms and cardiovascular diseases among males provided great contribution to both measures. Compared to males, contribution among females remined very small. Furthermore, the increasing mortality due to infectious and parasitic diseases had the opposite effect for both sexes, i.e. it contributed to a decrease in life expectancy and an increase in lifespan variation.

From age 65 to threshold age of females (nearly age 78), improvement in cardiovascular diseases, malignant neoplasms and diabetes made great contribution to increasing life expectancy. However, increasing mortality from infectious and parasitic diseases offset this gain and net gain decreased significantly. For males, between age 65 and threshold age (nearly 74), improvement of malignant neoplasms and cardiovascular diseases contributed significantly. However, similar to females, increases of infectious diseases decreased the net gain life expectancy and lifespan variation.

Above threshold age, cardiovascular diseases and malignant neoplasms continued to improve, while mortality from infectious and parasitic diseases increased. So, total gain in life expectancy above threshold ages decreased. On the other hand, increase of infectious and parasitic diseases decreased the lifespan variation significantly.

Notable finding of this study is that dramatic increase in respiratory infections and infectious and parasitic diseases at ages above 45 for both sexes. It caused the highest loss in life expectancy for both sexes (0.43 years for females and 0.35 years for males). On the other hand, increase in mortality from respiratory infections and infectious and parasitic diseases at ages above threshold ages also led to highest net reductions in variation. As can be understood from this finding, lifespan variation can be reduced in two ways: firstly, by reducing the mortality rate below the threshold age and secondly, by increasing the mortality rate above the threshold age. In the first scenario, there is an increase in life expectancy and a decrease in health inequality. In the second scenario, there is a loss in life expectancy and a decrease in health inequality. It should be noted that any health policy cannot be based on an increase in mortality at any age. Therefore, policy makers should consider the first scenario to decrease the lifespan variation. To accelerate the health equity, it is essential to focus on causes of death at young and adult ages below threshold age rather than only focusing on the leading causes of death (Seligman et al., 2016).

Our findings showed that improving health in infancy and up to the threshold age is very important and that controlling cardiovascular disease and cancer at these ages can have important effects on lifespan variation.

There are important sex differences with regard to gain in life expectancy. Firstly, males contributed more than twice to the change in life expectancy due to decline of unintentional injuries than females. This finding is not unexpected, because males are more prone to unintentional injuries than females (Seligman et al., 2016).

Secondly, mortality from malignant neoplasms declined significantly above age 40 for both sexes, but especially for males. Hashim and colleagues (2016) pointed out a global decline in cancer rates except liver cancer for each sex and lung cancer for females (Hashim et al., 2016). Decline in malignant neoplasms led to greater gains in life expectancy and net reductions in lifespan variations among males. It should be noted that cancers that contributed to life expectancy positively may be different from the cancers that contributed lifespan equality positively (Seligman et al., 2016). So, there is a need for further analysis on cancer types.

Overall, the results can be summarized as follows: with continued progress against infant mortality, malignant neoplasms and unintentional injuries below the threshold age, males were able to benefit from improvement in life expectancy. Life expectancy gains among male population came from the improvement in young and adult mortality (compression of mortality). On the other hand, life expectancy gains among females came from improvement in older ages (expansion of mortality). Below the threshold age, only infant mortality improved significantly in females; other causes of death, such as malignant neoplasms, diabetes mellitus and unintentional injuries, improved relatively less. Cardiovascular diseases led to important gains in female's life expectancy, however improvement in cardiovascular diseases above threshold age caused rise of lifespan variation. In addition, the increase in mortality rates from neurological diseases and genitourinary diseases above threshold age allowed for lower variation but caused a decrease in life expectancy. Therefore, females could not benefit from improvements in life expectancy as much as males due to 1) low reductions in premature deaths and 2) increase in respiratory infections and infectious and parasitic diseases, genitourinary diseases and neurological conditions at older ages. It should be noted that different gains in life expectancy and lifespan variation between sexes is due to the fact that males just experienced the improvement females had previously experienced in adult mortality (Mathers et al., 2015). So, it does not make sense to expect equal gains in life expectancy and life expectancy variation for males and females above and below the threshold age. Thus, the reason why females do not achieve as high a life expectancy as males is due to increased mortality from

infectious diseases, genitourinary diseases and neurological conditions at older ages. Gain in female life expectancy stem from the improvement in cardiovascular diseases at old ages and this pattern shows the expansion of mortality among female population. In the literature, research evidences show that some countries also experienced the high life expectancy thanks to the advantage in old mortality (Seaman et al., 2016; Shkolnikov et al., 2011; Smits & Monden, 2009).

Results of this study indicated that without higher improvement in young and adult mortality, decline in old-age mortality will create a lifespan inequality. Age and cause specific death rates show that, cardiovascular diseases, malignant neoplasms and unintentional injuries are still high between age 35 and 75. Therefore, with the effective health policy by targeting the infant mortality, malignant neoplasms, cardiovascular diseases and unintentional injuries between ages 35 and 75, Turkey may increase its life expectancy and lifespan equality much more. Moreover, eliminating the respiratory infections and infectious and parasitic diseases should also be a priority health issue. As we mention above, lifespan variation is very costly and needs to be given priority in health policies, as the level of lifespan variation also reduces the value of the life expectancy gains (Edwards, 2013; Shkolnikov et al., 2011; van Raalte et al., 2011). The countries which reached the highest life expectancy and low lifespan variation achieved this balance by reducing the young and adult age mortality (J. W. Vaupel et al., 2011). Experiences of these countries show that policymakers should prioritize causes of infant mortality, young-age mortality in healthcare spending. Studies also show that role of genetic variation in lifespan variation is modest and does not explain the whole story about life disparity-life expectancy relationship (J. W. Vaupel et al., 2011).

IV.5.2. Age and cause of death contributions to male-female differences in e(0) and $e^{\dagger}$

According to the results of female-male gap in life expectancy, females hold the survival advantage for all ages and causes. Similar to our findings, some researches also showed the female advantage in life expectancy at all ages and all causes (Austad,

2006; Beltrán-Sánchez et al., 2015; Wingard, 1984). Thanks to the decline of male mortality due to malignant neoplasms and unintentional injuries at young and adult ages, the gap in life expectancy decreased half a year between 2013 and 2019. This finding is consistent with the result of Glei and Horiuchi's (2007) study, which emphasized that the reduction in lifespan inequality in male mortality rates contributes to the narrowing of the male-female gap in life expectancy. On the other hand, increase of respiratory infections and infectious and parasitic diseases mortality increased the gap in life expectancy. Although mortality from infectious and parasitic diseases increased among both sexes, it is seen that infections are more fatal diseases for males. The fatality of infections is higher for oldest ages. Some research evidences showed that females have biological advantage in immune protection due to genetic and hormonal factors (Bouman et al., 2005; Giefing-Kröll et al., 2015). Although biological factors play an important role to determine the causes of death for sexes, the gap between male-female life expectancy can not be explain with biological factors alone. Behavioral factors are also important determinants of the gap. For example, consumption of tobacco, alcohol and substance abuse (drugs) and dangerous behaviours in traffic among males increase the risks of dying from cardiovascular diseases, lung cancer and unintentional injuries (Giefing-Kröll et al., 2015; McCartney et al., 2011; Oksuzyan et al., 2008; Waldron, 1985). Therefore, determining the biological and social, behavioral and environmental factors in causes of death is very crucial. Although the gap in male-female life expectancy is normal to some extent, preventing the deaths other than biological causes will close the gap as much as possible. Our results suggest that reducing unintentional injuries, malignant neoplasms and cardiovascular disease among the male population below the threshold age would narrow the male-female gap in life expectancy and life expectancy variation.

For further studies, we suggest that studies on the effects of mortality change within social groups such as education level, income status or place of region or country comparisons will provide a clear information on the health inequality in population (Firebaugh et al., 2014; Gillespie et al., 2014; van Raalte et al., 2011, 2014, 2015).

IV.6. References

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APPENDIX C. SUPPLEMENTARY TABLES FOR ESSAY 3

Table C.1. Absolute and relative measures of lifespan variation

Absolute measure	**Relative measure**
Life disparity: $e^{\dagger} = \int_0^{\omega} d(x)e(x)dx$	Lifetable entropy (Keyfitz's entropy): $\overline{H} = \frac{\int_0^{\omega} d(x)e(x)dx}{e_0} = \frac{e^{\dagger}}{e_0}$
Standard deviation: $S_0 = \sqrt{\int_0^{\omega} (x - e_0)^2 d(x)dx}$	Coefficient of variation (CoV): $CoV = \frac{\sqrt{\int_0^{\omega} (x - e_0)^2 d(x)dx}}{e_0} = \frac{S_0}{e_0}$
Absolute Gini coefficient: $G_0^{abs} = e_0 - \frac{1}{l(0)^2} \int_0^{\omega} l(x)^2 \, dx$	Gini coefficient: $G_0 = \frac{e_0 - \frac{1}{l(0)^2} \int_0^{\omega} l(x)^2 \, dx}{e_0} = \frac{G_0^{abs}}{e_0}$
Interquartile range (IQR): $IQR = x_{25} - x_{75}$ where $l(x_{25}) = 0.25$, $l(x_{75}) = 0.75$ Interdecile range (IDR): $IDR = x_{10} - x_{90}$ where $l(x_{10}) = 0.10$, $l(x_{90}) = 0.90$ Intercentile range (ICR): $ICR = x_1 - x_{99}$ where $l(x_1) = 0.01$, $l(x_{99}) = 0.99$	
	Theil's index: $T = \int_0^{\omega} d(x)(\frac{x}{e_0} \log \frac{x}{e_0}) \, dx$
	Mean logarithmic deviation: $MLD = \int_0^{\omega} d(x)(log \frac{e_0}{x})$

Figure C.1. Age and cause decomposition of coefficient of variation (CoV)

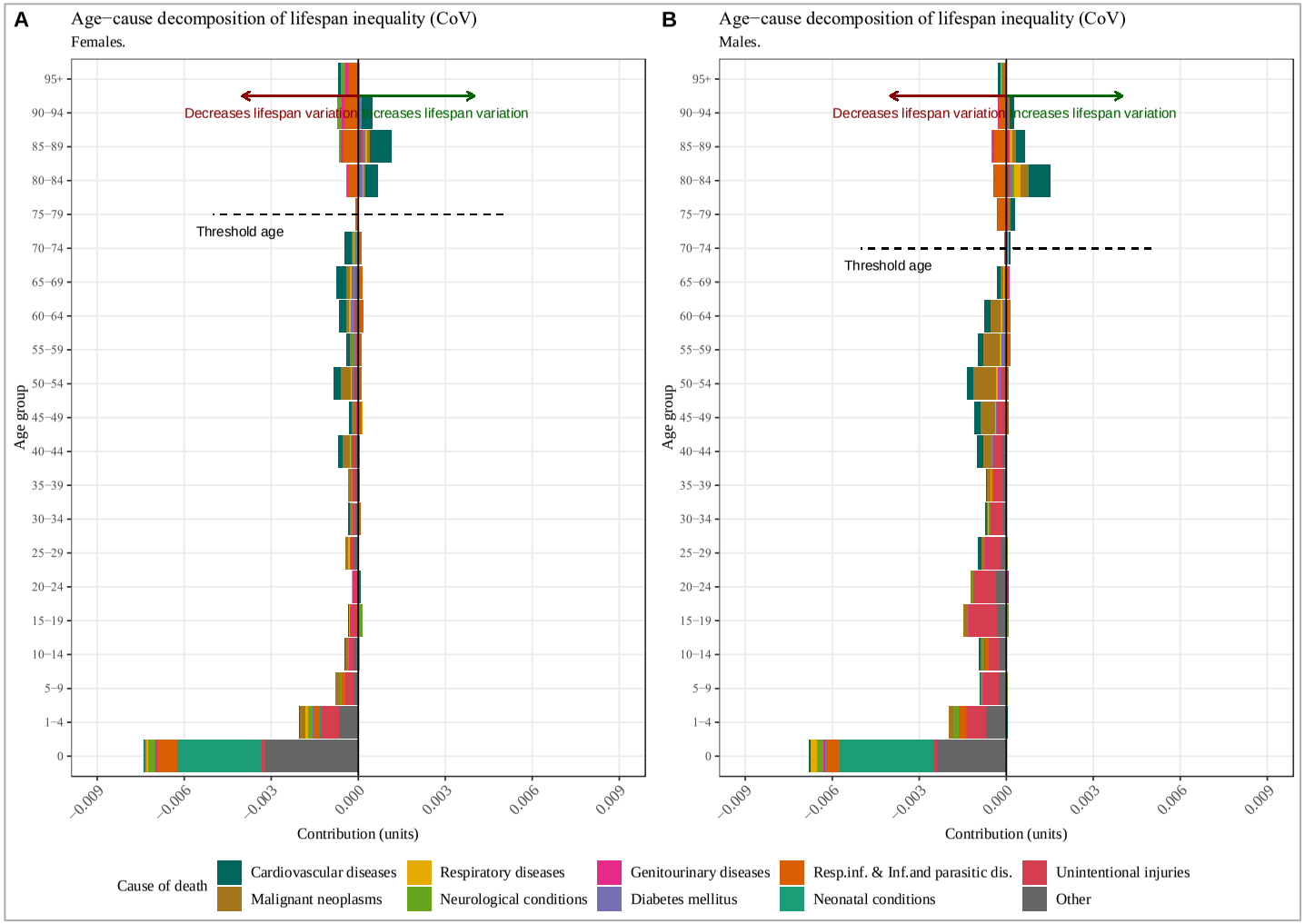

Table C.2. Age-cause contribution to life expectancy, females 2013-2019

Age	Cardiovascular diseases	Malignant neoplasms	Respiratory diseases	Neurological conditions	Genitourinary diseases	Diabetes mellitus	Resp.inf. & Inf.and parasitic dis.	Neonatal conditions	Unintentional injuries	Other
0	0.001	0.002	0.002	0.007	0.001	0.000	0.019	0.080	0.004	0.088
1-4	0.001	0.005	0.003	0.003	0.001	0.000	0.006	0.001	0.018	0.019
5-9	0.001	0.004	0.000	0.001	0.000	0.000	0.004	0.000	0.010	0.004
10-14	0.002	0.002	-0.001	0.001	0.001	0.000	0.002	0.000	0.004	0.004
15-19	0.001	0.001	0.002	-0.003	0.001	0.000	-0.001	0.000	0.006	0.002
20-24	0.000	0.001	0.000	0.001	0.001	0.000	-0.001	0.000	0.006	-0.002
25-29	0.000	0.005	0.002	0.001	0.000	0.000	0.000	0.000	0.004	0.006
30-34	0.002	0.003	0.000	0.000	0.000	0.000	-0.003	0.000	0.004	0.005
35-39	0.001	0.006	0.000	0.002	0.000	0.000	-0.001	0.000	0.004	0.006
40-44	0.008	0.016	0.001	0.002	0.000	0.001	-0.003	0.000	0.007	0.006
45-49	0.008	0.010	0.000	0.000	0.001	0.001	-0.007	0.000	0.002	-0.001
50-54	0.020	0.028	0.003	0.000	0.001	0.004	-0.009	0.000	0.006	0.008
55-59	0.014	0.010	0.000	0.000	0.003	0.007	-0.012	0.000	0.005	0.003
60-64	0.035	0.012	0.008	0.001	0.000	0.015	-0.026	0.000	0.005	0.013
65-69	0.070	0.030	0.015	0.000	0.000	0.026	-0.031	0.000	0.007	0.012
70-74	0.118	0.034	0.017	0.001	-0.001	0.027	-0.052	0.000	0.009	0.012
75-79	0.088	0.024	0.007	0.002	-0.016	0.027	-0.080	0.000	0.006	0.007
80-84	0.118	0.017	0.014	-0.004	-0.016	0.024	-0.090	0.000	0.007	0.007
85-89	0.113	0.012	0.010	-0.008	-0.013	0.013	-0.073	0.000	0.009	0.012
90-94	0.035	0.003	0.002	-0.013	-0.008	0.002	-0.046	0.000	0.004	0.001
95+	-0.006	0.002	0.000	-0.012	-0.006	-0.001	-0.020	0.000	0.002	-0.002
Total	**0.628**	**0.228**	**0.085**	**-0.016**	**-0.050**	**0.147**	**-0.425**	**0.081**	**0.128**	**0.208**

Table C.3. Age-cause contribution to life expectancy, males 2013-2019

Age	Cardiovascular diseases	Malignant neoplasms	Respiratory diseases	Neurological conditions	Genitourinary diseases	Diabetes mellitus	Resp.inf. & Inf.and parasitic dis.	Neonatal conditions	Unintentional injuries	Other
0	0.002	0.001	0.006	0.007	0.002	0.000	0.014	0.095	0.004	0.070
1-4	-0.001	0.004	0.001	0.005	0.000	0.000	0.008	0.001	0.021	0.021
5-9	0.001	0.002	0.001	-0.001	0.001	0.001	0.000	0.000	0.015	0.009
10-14	0.002	0.003	-0.001	0.002	0.000	0.000	0.005	0.000	0.013	0.008
15-19	0.001	0.006	0.000	-0.002	0.001	0.000	0.000	0.000	0.036	0.013
20-24	-0.001	0.002	-0.001	0.001	0.000	0.001	-0.001	0.000	0.031	0.015
25-29	0.004	0.007	0.000	-0.001	0.000	0.000	-0.001	0.000	0.025	0.007
30-34	0.003	0.002	0.001	0.002	0.001	0.001	0.001	0.000	0.022	0.005
35-39	0.002	0.008	0.000	0.001	0.001	0.001	0.002	0.000	0.020	0.006
40-44	0.017	0.019	0.002	0.001	0.001	0.002	0.000	0.000	0.020	0.009
45-49	0.017	0.044	0.003	0.000	-0.002	0.003	-0.005	0.000	0.019	0.005
50-54	0.025	0.080	0.009	0.001	0.001	0.006	-0.009	0.000	0.014	0.008
55-59	0.028	0.076	0.011	0.000	0.002	0.009	-0.020	0.000	0.007	0.004
60-64	0.044	0.079	0.009	0.001	0.001	0.010	-0.033	0.000	0.010	0.007
65-69	0.059	0.063	0.020	0.000	-0.003	0.013	-0.047	0.000	0.009	0.007
70-74	0.076	0.050	0.026	0.003	-0.001	0.015	-0.060	0.000	0.010	0.009
75-79	0.049	0.024	0.006	0.001	-0.008	0.011	-0.072	0.000	0.010	0.000
80-84	0.115	0.039	0.034	0.009	-0.001	0.012	-0.062	0.000	0.009	0.010
85-89	0.035	0.012	0.009	-0.002	-0.005	0.002	-0.042	0.000	0.007	0.003
90-94	0.012	0.004	0.002	-0.001	-0.002	0.001	-0.019	0.000	0.001	0.000
95+	-0.005	0.000	-0.002	-0.001	0.000	0.000	-0.007	0.000	0.001	-0.001
Total	**0.484**	**0.525**	**0.137**	**0.025**	**-0.011**	**0.087**	**-0.351**	**0.095**	**0.303**	**0.216**

Table C.4. Age-cause contribution to life disparity, female 2013-2019

Age	Cardiovascular diseases	Malignant neoplasms	Respiratory diseases	Neurological conditions	Genitourinary diseases	Diabetes mellitus	Resp.inf. & Inf.and parasitic dis.	Neonatal conditions	Unintentional injuries	Other
0	-0.001	-0.001	-0.002	-0.006	-0.001	0.000	-0.016	-0.070	-0.003	-0.077
1-4	-0.001	-0.005	-0.003	-0.003	-0.001	0.000	-0.006	-0.001	-0.016	-0.017
5-9	-0.001	-0.004	0.000	-0.001	0.000	0.000	-0.003	0.000	-0.008	-0.003
10-14	-0.001	-0.002	0.001	-0.001	0.000	0.000	-0.001	0.000	-0.003	-0.004
15-19	-0.001	-0.001	-0.001	0.003	-0.001	0.000	0.001	0.000	-0.005	-0.001
20-24	0.000	-0.001	0.000	-0.001	-0.001	0.000	0.000	0.000	-0.005	0.001
25-29	0.000	-0.004	-0.001	-0.001	0.000	0.000	0.000	0.000	-0.003	-0.005
30-34	-0.002	-0.003	0.000	0.000	0.000	0.000	0.003	0.000	-0.004	-0.004
35-39	-0.001	-0.004	0.000	-0.001	0.000	0.000	0.001	0.000	-0.003	-0.004
40-44	-0.006	-0.012	-0.001	-0.001	0.000	0.000	0.002	0.000	-0.005	-0.004
45-49	-0.006	-0.007	0.000	0.000	-0.001	-0.001	0.005	0.000	-0.002	0.001
50-54	-0.014	-0.019	-0.002	0.000	-0.001	-0.003	0.006	0.000	-0.004	-0.005
55-59	-0.009	-0.007	0.000	0.000	-0.002	-0.005	0.008	0.000	-0.003	-0.002
60-64	-0.020	-0.007	-0.005	-0.001	0.000	-0.009	0.015	0.000	-0.003	-0.007
65-69	-0.032	-0.014	-0.007	0.000	0.000	-0.012	0.014	0.000	-0.003	-0.005
70-74	-0.037	-0.011	-0.006	-0.001	0.000	-0.008	0.016	0.000	-0.003	-0.004
75-79	-0.014	-0.004	-0.002	-0.001	0.000	-0.004	0.004	0.000	-0.001	-0.001
80-84	0.036	0.005	0.005	-0.002	-0.005	0.007	-0.028	0.000	0.002	0.003
85-89	0.104	0.011	0.010	-0.010	-0.013	0.012	-0.070	0.000	0.009	0.011
90-94	0.062	0.005	0.005	-0.029	-0.017	0.004	-0.091	0.000	0.008	0.001
95+	-0.008	0.008	0.003	-0.044	-0.024	-0.003	-0.076	0.000	0.009	-0.005
Total	**0.048**	**-0.076**	**-0.008**	**-0.101**	**-0.067**	**-0.022**	**-0.217**	**-0.071**	**-0.046**	**-0.133**

Table C.5. Age-cause contribution to life disparity, male 2013-2019

Age	Cardiovascular diseases	Malignant neoplasms	Respiratory diseases	Neurological conditions	Genitourinary diseases	Diabetes mellitus	Resp.inf. & Inf.and parasitic dis.	Neonatal conditions	Unintentional injuries	Other
0	-0.001	-0.001	-0.005	-0.006	-0.002	0.000	-0.012	-0.081	-0.004	-0.060
1-4	0.001	-0.003	-0.001	-0.005	0.000	0.000	-0.007	-0.001	-0.018	-0.018
5-9	-0.001	-0.002	-0.001	0.001	-0.001	0.000	0.000	0.000	-0.013	-0.007
10-14	-0.001	-0.002	0.000	-0.002	0.000	0.000	-0.004	0.000	-0.011	-0.007
15-19	-0.001	-0.005	0.000	0.002	-0.001	0.000	0.000	0.000	-0.029	-0.010
20-24	0.001	-0.002	0.001	-0.001	0.000	-0.001	0.001	0.000	-0.025	-0.012
25-29	-0.004	-0.006	0.000	0.001	0.000	0.000	0.001	0.000	-0.020	-0.006
30-34	-0.002	-0.002	0.000	-0.001	-0.001	0.000	0.000	0.000	-0.017	-0.004
35-39	-0.002	-0.006	0.000	-0.001	0.000	-0.001	-0.002	0.000	-0.015	-0.004
40-44	-0.012	-0.013	-0.001	0.000	-0.001	-0.001	0.000	0.000	-0.014	-0.007
45-49	-0.011	-0.029	-0.002	0.000	0.001	-0.002	0.003	0.000	-0.013	-0.004
50-54	-0.015	-0.048	-0.005	0.000	0.000	-0.004	0.005	0.000	-0.008	-0.004
55-59	-0.015	-0.040	-0.006	0.000	-0.001	-0.005	0.011	0.000	-0.004	-0.002
60-64	-0.019	-0.034	-0.004	0.000	0.000	-0.004	0.014	0.000	-0.004	-0.003
65-69	-0.016	-0.018	-0.006	0.000	0.001	-0.004	0.013	0.000	-0.002	-0.002
70-74	-0.007	-0.005	-0.001	0.000	0.000	-0.001	0.004	0.000	-0.001	-0.001
75-79	0.005	0.002	-0.001	-0.001	-0.003	0.001	-0.019	0.000	0.002	0.000
80-84	0.082	0.028	0.024	0.006	-0.001	0.009	-0.044	0.000	0.006	0.007
85-89	0.049	0.017	0.012	-0.005	-0.008	0.003	-0.065	0.000	0.010	0.004
90-94	0.030	0.010	0.004	-0.003	-0.005	0.003	-0.054	0.000	0.003	0.000
95+	-0.025	0.000	-0.007	-0.005	0.003	-0.002	-0.035	0.000	0.005	-0.002
Total	**0.035**	**-0.159**	**-0.001**	**-0.021**	**-0.019**	**-0.009**	**-0.189**	**-0.081**	**-0.171**	**-0.142**

V. SYNTHESIS

This section synthesizes the essays in this dissertation by considering the research objectives, summarises the main findings of the study and presents the contributions, limitations, and recommendations for further analysis.

Throughout the World history there has been great changes in mortality patterns and trends. The main aims of human beings were to extend their lives and save premature mortality. For these aims infant and child mortalities, mortality at adult ages were lowered to some extent, however, there occurs lots of preventable death before "time to die" (Sen, 1998). Death registries and statistics are important resource for public health policies. Death statistics give information about the health and morbidity about population. According to Sen (1998), death data gives idea about more than health. Death data can adapt fast to changing economic and social circumstances and any change in socio-economic situation reflects to death data (Sen, 1998).

Death registration in Turkey has been improved significantly with reforms in 2009 and 2013. Before 2009, mortality data were collected separately for statistical and administrative purposes. Mortality data collected for administrative purposes covered all population however, cause of death part was not detailed. On the other hand, death data collected for statistical purposes covered only provincial and district centers and cause of death part was not detailed. Therefore, there were completeness and coverage issue in the registration system before 2009. In 2009, TurkStat began to produce death statistics for whole population by accessing the death records collected in registration system. Furthermore, cause of death part of the certificate was detailed and made in accordance with ICD codes. Although deficiencies in the death statistics was lowered substantially, coverage and completeness issues continued. Due to the dual recording system, there were sometimes inconsistencies between two death records. In 2013, it was decided to collect death records in one pool. Electronic death notification system (DNS) was implemented. Administrative and statistical tasks have

been carried out through DNS. Therefore, coverage and completeness issues were eliminated with this system.

Turkey's epidemiological transition process began with the foundation of Turkish Republic in 1923. Before this time, Turkey struggled in many wars, including the First World War. High infant and child mortality rates, the spread of infectious diseases throughout the country and high and fluctuating death rates are the notable characteristics of this pre-transition period. After the foundation of the Turkish Republic, struggle against infectious disease was accelerated and death rates began to decline. In the second part of the 20th century, cardiovascular diseases came to the fore, as well as the fight against infectious diseases. By the end of twentieth century, communicable diseases declined significantly and noncommunicable diseases became the main causes of death.

There have also been significant changes in the age and sex pattern of mortality from pre-transition period to 2019. Until the end of 20th century, improvement in infant and child mortality contributed greatly to the gain in life expectancy for both sexes. Although contribution of infancy period continues, this contribution decreased from past to present. With the increasing life expectancy, more people have reached to older ages and the contribution of adult and older ages to the increase in life expectancy has become equal to that of infancy. Until the end of of 20th century, elimination of infectious diseases at infant and young ages had increasing impact on life expectancy, while with the beginning of 21st century chronic diseases at adult and older ages have contributed increasing life expectancy. Furthermore, sex pattern of mortality has also changed. Due to the improvement in maternal and infant mortality, female's disadvantage in life expectancy in the past has turned into an advantageous situation today. On the other hand, although improvement in infant and child mortality also increased male life expectancy, male life expectancy lags female life expectancy. The main reasons for this sex pattern are usually male's high-risk environments such as working conditions or health-risk behaviors such as smoking, driving fast in traffic.

Emergence of aging population is important demographic change in post-transitional countries. Turkey has also been facing with aging population. Main characteristic of aging population is that the elderly population is increasing while the young and working population is decreasing due to the decreasing fertility rates. An ageing population also creates an imbalance in the economy as there will be fewer working age groups supporting a larger share of older people in retirement. Furthermore, aging population will require healthcare facilities and so policymakers should take precautions for necessary infrastructure.

The second decade of the 21st century has witnessed a pandemic caused millions of deaths in all over the world. Since its onset in China, the Covid-19 pandemic has expanded around the world in 2020. According to WHO database, from 2020 to March 2023, there have been 6.9 million deaths from Covid around the world (WHO, 2023). A high number of Covid-19 deaths occurred between 2020-2021. Covid-19, one of the world's biggest pandemics (Goldstein & Lee, 2020), has significantly affected the lives of people around the world. When we look at the demographic results of Covid-19, mortality rates have been directly affected by pandemic. The pandemic's impact on mortality has been exacerbated by the triggering effect of Covid-19 on the health system and the country's economic situation (Klancher Merchant, 2021; Sasson, 2021). Individuals aged 65 and over and individuals with chronic diseases are the most affected group. Increasing rate of mortality among elderly is the obvious effect of the Covid-19. Short-term effect of Covid-19 will be to increase healthy life expectancy, but within a short period of time this trend will reverse, leading to an increase in expected years of disability (Palloni, 2021) and increased demand for health services due to Covid-19 will further increase the current burden on the healthcare system. The long-term effects of the pandemic will be seen in the "sandwich generation" who were studying in high school, university, starting their professional life or getting married during the pandemic period (Palloni, 2021). Although short-term burden of Covid-19 in this population is quite mild, health problems that are likely to be seen in the years to come may leave important footprint on these cohorts. Research evidence show that organ damage, memory loss, cognitive

impairment, renal dysfunction, and heart irregularities are among some health problems that may be observed in this generation (Palloni, 2021).

The pandemic also effected the accessibility of mortality data. In developing countries, pre-existing inefficiencies in the vital registration system unable to meet timely and accurate information for assessing the dimensions of the pandemic. Some developed countries have also had difficulties in providing data especially in subgroup and area level data (Ho, 2021). Another issue with the data is that underreporting of Covid-19 related deaths for deaths occurred outside the hospital (Aburto et al., 2022). Furthermore, it is quite possible that some Covid-19 deaths were attributed to cardiovascular diseases. Such misclassifications can be misleading for health policies, especially when assessing the pattern of mortality in subgroup populations, as some groups may be more exposed to misclassifications (Aburto et al., 2022).

There are four main aims in this study. The first one was to measure the data quality of mortality data in 2013-2019 period. The data quality was evaluated by investigating the proportion of garbage code by age, sex, and garbage types. The selected period is important because the beginning period is the enforcement of the electronic DNS, and the importance of the ending period is that it is the recent year before Covid-19 pandemic. Therefore, selection of 2013-2019 period provide opportunity to evaluate functionality of DNS. The second aim of this study was to investigate the age, sex, and cause of death pattern mortality and to identify the drivers of the change in life expectancy and lifespan variation. The third aim was to explain the change in age, sex, and cause of death pattern with the known theories of longevity and to determine some policies for further improvement in mortality.

This study investigated the quality of cause of death data regarding garbage code. Results proved that ill-defined cardiovascular diseases are big issue for both sexes because the proportion increased between 2013 and 2019 and constituted the most share of garbage burden. Ill defined-signs and symptoms decreased for the same period. Furthermore, it can be said that quality of death data is sufficient for cause of death analysis. On the other hand, it is known that ICD code errors on death certificates

decreases the policy value of cause of death data (Mikkelsen et al., 2018). For this reason, it is necessary to provide trainings for death certification process to medical students and doctors. It is also important to raise awareness of the significance of correct death certification for public health (Mikkelsen et al., 2018).

This study also evaluated the epidemiological transition of Turkey pass through. According to results, burden of communicable diseases increased greatly between 2013-2019 period. This rise mostly effected the older aged people and males were affected much more than females. Although 2013-2019 period is too short to evaluate epidemiological transition, as far as we know, this kind of mortality trend did not occur in the history of Turkey and this kind of trend which was seen in seven consecutive years can not be coincidental. It can be said that Turkey experiences a new pattern in mortality in which NCDs are leading causes of death and increasing CDs created an extra burden on mortality. In a study evaluating mortality rates in Turkey between 2009 and 2017, the increase in mortality rates due to CDs among the elderly was emphasized (Teker et al., 2020).

In this dissertation we evaluated the recent age-sex and cause patterns of mortality. First of all, we disclosed that there are notable differences in the sex pattern of mortality. Modal age at death among males increased significantly between 2013 and 2019 (shown in the second essay) while it was increased so small for the same period. Further analysis by cause of death showed that while males caught a good improvement at young and adult ages by decreasing the deaths due to unintentional injuries, malignant neoplasms, and congenital anomalies; females caught this improvement mostly at older ages by decreasing deaths due to cardiovascular diseases. These findings indicates that males and females experienced different mortality patterns for 2013-2019 period. It is seen that mortality compression- due to decline in lifespan variation below threshold age- and mortality delay – due to rise at modal age at death- are dominant patterns for males. On the other hand, expansion of mortality is the most notable characteristic of female mortality pattern, due to higher improvement at ages above threshold age. Some research evidence showed that males experienced

the mortality transition later than females (Cheung et al., 2009; de Beer & Janssen, 2016; Janssen & de Beer, 2019).

Furthermore, findings of the study clarified that increasing rate of communicable diseases are great obstacle for further gain in life expectancy. In addition to infectious diseases, increase of genitourinary diseases and neurological conditions at older ages among females prevent to expand older age improvement to further ages. Decomposition of sex-gap in life expectancy showed that while gap in life expectancy generally narrowed below age 65, this gap is widened for age 65 and above, this means that with the improvement below threshold age males achieved to narrow gap on the other hand, with the improvement above threshold age females achieved to widen the gap.

This study evaluated the recent trends and patterns in causes of death in Turkey during pre-Covid period by considering the theories of societal and biological constraints human mortality. Overall, findings suggest ed that there are some health policy targets to be prioritized. First of all, ill-defined cardiovascular diseases in cause of death data should be decreased. As we remember from the first essay, the highest share of ill-defined cardiovascular diseases was seen in older age groups. Especially in older age groups, the underlying cause of death is very difficult to understand due to the intertwined causes of death. Regarding this problem, training physicians to determine the underlying causes and chain of events leading to death correctly is necessary. Furthermore, the findings clearly indicated that CDs created a high burden for elderly population. So, CDs among elderly should be targeted as an emergent health policy. As we saw from the results, age at death in Turkey are moving older ages for both sexes. Parallel to this improvement, perception of premature and old age mortality is also changing (Janssen & de Beer, 2019). For example, age 80 was perceived as “living long” in 2000s, however this age is now perceived as a normal length of life for males and premature mortality for females. So, information of threshold age is very important in policy-making process to reduce premature mortality and target causes of death in this context. Furthermore, as more people live longer, chronic diseases can be more problematic. A 70-year-old patient with diabetes

mellitus is not in the same situation as an 80-year-old patient. The fact that people are living longer with chronic diseases also increases vulnerability to infectious diseases in later life.

Overall, the findings of the study reveal that the burden of infectious diseases was increasing even before the Covid-19 period. This burden particularly affects the elderly population. Male population achieved to increase life expectancy more than females due to the notable improvement at young and adult ages. The main reason why female's life expectancy has increased less than male's is that lower gains at young and adult ages are compounded by an increase in infectious diseases at older ages. It appears that the pre-existing burden of infectious diseases has been combined with the Covid-19 pandemic, and both have placed an extra burden on the elderly population. These results suggest that Turkey needs to make some improvements in health care services for the elderly. Another important finding of the study is the proportion of garbage codes in pre-covid period. Only garbage cardiovascular diseases increased during examined period, while other garbage types decreased or remained stable. It is quite possible that Covid-19 pandemic will also cause a steep increase in the coding of garbage cardiovascular diseases.

This is the first study in many aspects. Firstly, this study assesses the quality of death registration data by identifying garbage codes and redistributing them to well-defined causes. Secondly, this dissertation contributes to the literature by applying the Markov chain model, which is a little-used method in demography, and line integral decomposition method, which has recently become popular in cause-of-death analyses to analyze the age, sex, and cause pattern of mortality. The third aspect is that threshold age which can be good guide for public health policies is firstly measured from the registration data for Turkey. Lastly, the theoretical framework of human longevity largely explains the cause-and-effect relationship of improvement in mortality or deterioration in mortality. Therefore, the study's findings are well suited for policy-making purposes.

The strength of the study is that mortality data of examined period has high quality. This study did not use any estimation or assumption. Findings of the study are based on the observed and reliable registration data. Nevertheless, this study has several limitations. The most important limitation is that registration data does not include migrant population. As we know that there is significant extent of migrant population in Turkey. Moreover, the migrant population has a different age and sex composition than the native population in Turkey. Therefore, the lack of information on the migrant population in the mortality data prevents us from making a definitive judgment on Turkey's mortality pattern. Another limitation is that the period analyzed was too short to draw any definitive conclusions for the epidemiological transition in Turkey. Epidemiologic transition studies generally use 20 years and above to clarify the epidemiologic trends in specified time interval.

For further studies, cause of death analysis by population subgroups such as region, level of education, working status etc. would provide more effective public policy targets. Furthermore, instead of all causes of death, focusing on specific causes may show interesting results. Projections on population, mortality or causes of death might be instructive for policy makers. Covid-19 effect on data quality and mortality pattern and mortality pattern and trend among migrant groups are other suggestions for further studies.

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