Exploring the gap: an analysis of mortality differentials in the New Zealand Māori and New Zealand non-Māori population

Daniëlle Eleveld
s2579952
d.eleveld@student.rug.nl

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Master Population Studies
Population Research Center
Faculty of Spatial Sciences
University of Groningen

Supervisor: Dr. A.P.P. Remund

I. Abstract

There is a large health gap between the Indigenous people of New Zealand, the Māori, and the non-Indigenous population of New Zealand. Therefore, this research investigates mortality patterns and existing gaps for both population groups using a life table analysis, which provides life expectancy and a lifespan variability measure. Māori have a disadvantaged mortality pattern compared to non-Māori. Trends in life expectancy and lifespan variability show substantial improvements from 1948 onwards. However, further improvements in averting Māori premature deaths is desirable, especially in males. In 2016, life expectancy was 6.7 years lower for Māori males and 5.5 years lower for Māori females compared to their non-Māori counterparts. For both sexes, lifespan variability is over 20% higher for Māori. The ages of 50-79 mainly explain the gaps. This corresponds with the large contribution of late-onset diseases as circulatory diseases and cancers. External causes in young-adult ages seem to explain a substantial part of the life expectancy and life disparity gaps in males. The contributions of respiratory diseases and lung cancer reflect the high smoking rates for Māori females. Opposed to the other age groups, the oldest-aged Māori have an advantage compared to non-Māori. Further research should address whether this is due to selective survival or data issues. Public health policies aiming to reduce health and mortality gaps between Māori and non-Māori should focus on decreasing Māori smoking rates while, more importantly, a focus on indirect causes of morbidity and mortality such as lower socioeconomic status and racism should not be forgotten.

Keywords: mortality, life expectancy, lifespan variability, indigenous, Māori

II. Preface

When I was 18 years old, I flew to Australia to enjoy a gap year on the other side of the world. I worked on a cattle station in northwest Queensland for a couple of months, which taught me a lot about life on an outback farm, about myself, and about other people. One of my co-workers was Max, an Aboriginal from Alice Springs. Because of his accent due to speaking in his tribal language for most of his lifetime, it took me about two weeks to be able to understand him. However, after these initial weeks, we got into some good conversations and grew fond of one another. He taught me a lot about farm life, the Australian outback, and about his family. I would have never guessed he was around 83 at that time since he had energy as if he was in the height of his life. And, even though he smoked all day long, he was never sick or ill. His mother, he told me once, was 103 at the time and still doing rather well. While I thought about my own grandfather who had passed away two years earlier at the age of 73, it struck me as remarkable that Max's family was this old and healthy.

It appeared that thought was right. It had always seemed interesting to me that Max and his family were able to enjoy such longevity, even compared to families in more developed circumstances such as my own. So, during one of my courses for the Population Studies Master, I wrote about life expectancy differences in the Indigenous and non-Indigenous Australian population. This way I learned of the existence of a gap in life expectancy between both population groups: in general, Indigenous populations had a much lower life expectancy compared to non-Indigenous populations. This was true for Australia, but also for other colonized countries like the United States, Canada, and New Zealand.

It was this train of events that led me to choose life expectancy in the Australian Indigenous population as the subject for my thesis. In addition to just the numbers, I wanted to know where the difference originated. Later, this was extended with a lifespan variation measure. Although I started with the Australian context, it appeared that the data on the Australian Indigenous population is of low quality and rather incomplete. With the help of my supervisor, Adrien, I shifted to the New Zealand context. I could have chosen Canada or the United States instead, but New Zealand sparked my interest because of a visit there and, in my thoughts, it would probably resemble the Australian context better than the other countries. The high quality of New Zealand data also worked in my favour.

This little story explains how I got inspired for my thesis subject. However, the thesis itself would not have come about without the help and (mental) support of a couple of people. I would, therefore, like to thank my supervisor, dr. Adrien Remund, for always being available for questions and feedback, for responding to my emails almost always within a day, and for the encouraging words I sometimes needed to hear. I would also like to thank Tineke for struggling in the library together: without you, it would not have been as much fun. And last but definitely not least: thank you to Casper for listening, supporting, and motivating me.

That Max and his family enjoyed such longevity seems rather unique to me, especially after writing my thesis. I know his family enjoyed living 'the old way': living in small communities, surrounded by family and with not as much social pressure as we have in some modern societies. Maybe we could all enjoy longevity if we do what we love, surrounded by the people we love. Or maybe his family was just an exception to the rule. I will probably never know for sure.

I hope you enjoy reading this thesis,

Daniëlle

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Abbreviation	Meaning
AI/AN	American Indian and Alaska Native [i.e. Indigenous population of the United States]
CANZUS	Canada, Australia, New Zealand, United States
CDC	Centers for Disease Control and Prevention [Organization of the United States Department of Health and Human Services that is assigned to detect, handle, and prevent diseases that are perceived as a public health threat.]
CRVS	Civil registration and vital statistics
DALY's	Disability Adjusted Life Years
HMD	Human Mortality Database
ICD	International Classification of Diseases
U.S./U.S.A.	United States of America
WHO	World Health Organization

1. Introduction

1.1 Background and problem description

For Indigenous populations, the colonisation era has brought severe disruptions to their way of life, social interaction, and their population and individual health. Health outcomes of Indigenous people have been suffering from the colonial oppression (Gracey & King, 2009; Paradies, 2016), with the adverse effects also visible in settler-colonies such as the CANZUS nations – that is, Canada, Australia, New Zealand and the United States (Ford, 2012). Since the colonisation era, the Indigenous population in these countries experienced rapidly changing lifestyles: from their traditional lifestyle to transitional, and finally the modern lifestyle (Gracey & King, 2009). The sudden changes in way of life, food patterns, and social interaction led them to have worse health outcomes compared to the countries' non-Indigenous population, and it is assumed that this pattern will not change drastically in the nearby future (Stephens et al., 2005; Gracey & King, 2009). Examples that indicate these disadvantages in health outcomes for Indigenous population groups include higher mortality rates (New Zealand Government – Ministry of Health, 2018c), higher diabetes rates (U.S. Department of Health and Human Services, 2016), and higher daily smoking rates (Government of Canada, 2018).

The aforementioned examples focus on physical health whereas the widely accepted definition of health is rather holistic and does not confine itself to the physical body, but also comprises mental well-being. The World Health Organization (WHO) (1946, p.1) defines health as "*a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity*". When considering health as a holistic concept, Indigenous populations are at an even larger disadvantage. Next to the elevated rates concerning physical health like the ones mentioned before, Indigenous populations also experience an excess burden of mental illnesses and mental disorders (Gracey & King, 2009; Maronne, 2007). However, often one cannot simply distinguish the two: physical and mental health are interlinked and the relationship between the two is bidirectional. Physical illness can, for example, lead to deterioration of mental health, and, the other way around, sound mental health can have a protective influence on the maintenance of good physical health (Steptoe et al., 2015). This reciprocal relationship between physical and mental health leads to an increased risk of premature mortality for individuals coping with mental disorders (Chesney et al., 2014; Walker et al., 2015).

The health discrepancies between the Indigenous and non-Indigenous population groups of the CANZUS-nations have been studied widely (see e.g. Gracey & King, 2009; King et al., 2009). In each of these countries, indicators of health status show worse health outcomes for the Indigenous population compared to the non-Indigenous population. In Canada, infant mortality rates remain more than twice as high for Indigenous people compared to the non-native population (Government of Canada, 2019). In the United States, the heart disease rate is 20% higher for American Indians and Alaskan Natives (Centers for Disease Control and Prevention, 2016). In Australia too, the distinction between native and non-native health is reflected in, among others, higher smoking prevalence (Australian Bureau of Statistics, 2017) and a higher prevalence of mental health conditions (Jorm et al., 2012; Balaratnasingam & Janca, 2019).

The case has been no different in New Zealand. The Indigenous population group of New Zealand, the Māori, have had to deal with the presence of Europeans from 1769 onwards, which caused developments in their population. The invasion of non-endemic diseases such as dysentery, influenza and tuberculosis was one of the causes for the changes in the Māori population (Pool, 1991). Since the Māori were immunologically naïve to the pathogens brought by the Europeans, a spread of diseases occurred, causing mortality rates to rise. This caused their population numbers to decline in such a way that in 1911, the Māori population was only an estimated 40% of the 1769 population size. Next to the decline in population due to diseases, the loss of resources influenced Māori health. The deliberate transfer of resources as initiated by the European settlers caused Māori to lose their land and, with it, their way of life. This loss of resources had serious negative consequences for Māori well-being, leading them to not only suffer physically from invading pathogens but also suffer mentally from a forced lifestyle change. These changes interacted with one another. Over time, the Māori acquired more resistance against the new diseases. This led to positive consequences for their population numbers. However, these positive developments were counteracted by the underdevelopment caused by the loss of resources, leading to a demographic equilibrium. Over time, the Māori got accommodated to a

changed way of life and their population numbers started recovering (Pool, 2015). Nowadays, approximately 723.500 people identify as Māori, which equals 15.4% of the total population (Statistics New Zealand, 2017). Although the survival of Māori ethnicity is no longer an issue, their health status is still lagging behind the non-Māori population. Indicators of health status clearly display this existing health gap between the Māori and non-Māori population of New Zealand. These indicators of health are measures like life expectancy at birth, mortality rates, hospitalisation rates, and disease prevalence (Anthamatten & Hazen, 2011). Some of these health status indicators and the existing differences for the New Zealand Māori and New Zealand non-Māori population are summarized in Table 1.

Table 1: Health status indicators of New Zealand Māori and New Zealand non-Māori.

Health status indicators of New Zealand Māori and New Zealand non-Māori			
	Māori	non-Māori	Ratio
Life expectancy males	73.0	80.3	7.3*
Life expectancy females	77.1	83.9	6.8*
Mortality rate (per 100,000 persons)	527.8	286.1	1.84
Infant mortality rate (per 1,000 live births)	6.8	4.5	1.51
Suicide mortality rate (per 100,000 persons)	16.9	9.1	1.86
Cancer registrations rate (per 100,000 persons)	506.3	405.8	1.25
Smoking prevalence	42%	15.5%	2.71
(Very) high probability of anxiety of depressive disorder	11.5%	7.9%	1.46

* These numbers are the difference in life expectancy in years, not ratios.

S*ource: own table based on New Zealand Government – Ministry of Health, 2015a; New Zealand Government – Ministry of Health, 2018c; New Zealand Government – Ministry of Health, 2019e.*

1.2 Relevance

Chapter **1.1** has shown the existence of health and mortality inequalities between the New Zealand Māori and New Zealand non-Māori population groups. Health inequalities are avoidable when not biologically determined (Preda & Voigt, 2015; Woodward & Kawachi, 2000) and may be unfair, unjust (Hausman et al., 2002; Woodward & Kawachi, 2000), and unethical (Ruger, 2006; World Health Organization - Commission on the Social Determinants of Health, 2008). Therefore, health inequalities need to be addressed in order to be able to improve health for every population group in a society. Although improvements have occurred and the difference between Māori health status and non-Māori health status has decreased (New Zealand Government – Ministry of Health (2015a), the gaps as observed in Table 1 are still substantial. The New Zealand Government acknowledges the improvements that are made but is also aware of the steps that still have to be taken. Therefore, the Māori Health Strategy, officially known as He Korowai Oranga, was put into place in 2002 (New Zealand Government – Ministry of Health, 2002). Equity between Māori and non-Māori health status is one of the key threads of this strategy. A part of the work by which the Government thinks to accomplish this is by "*continuing to develop good-quality ethnicity data to measure and report on health status*" (New Zealand Government – Ministry of Health, 2015b, 'Equity'). This indicates a desire of the government to increase the knowledge of Indigenous and non-Indigenous health differentials. The overview of mortality patterns that is gained by this research thus serves a goal that is formulated by the New Zealand Government and is, therefore, of societal relevance.

Multiple studies (see e.g. Ajwani et al., 2003; Anderson et al., 2006; Beaton et al., 2019; Gracey & King, 2009) have already addressed the health, morbidity and mortality inequalities in the New Zealand context. These studies have helped to bring the existence of inequalities to light and deepened the understanding of them. However, an overview of the magnitude of these mortality differentials seems absent. Such an overview, however, might be used to learn from areas (i.e. age groups or particular causes of death) that are doing relatively well and to learn which areas deserve extra attention since they are clearly lagging behind. This research aims to be of academic relevance by providing this

overview of mortality differentials. When researching mortality differentials, life expectancy can be used as a summary measure of mortality. Period life expectancy informs us on "*the average number of additional years that a survivor to age x will live beyond that age"* (Preston et al., 2001, p.39), assuming that *"current mortality rates continue to apply*" (Anthamatten & Hazen, 2011, p.88). Since life expectancy, usually life expectancy at birth, can be compared between different population groups it is an uncomplicated tool to compare mortality levels between two population groups. A novel approach of studying the Indigenous/non-Indigenous mortality gap would be to focus on lifespan variability. Whereas life expectancy educates us on the average age at death of a population, lifespan variability indicates how accessible this age is for all of the population. Examination of ethnic inequalities in lifespan variability is limited (Lariscy et al., 2016), and the research that has been done on this topic has focused on the United States (see e.g. Brown et al., 2012; Edwards & Tuljapurkar, 2005; Lariscy et al., 2016; Nau & Firebaugh, 2012). Examination of lifespan variabilities among Indigenous and non-Indigenous population groups has, to the best of my knowledge, not been conducted before and thus, gives a unique insight into the mortality gap and might be able to consider different explanations and solutions for the problem at hand. Life expectancy and lifespan variability combined provide us therefore with a general overview of mortality patterns of a population. This research into mortality patterns of the New Zealand Māori and New Zealand non-Māori is, therefore, constructed along the lines of these two concepts: life expectancy and lifespan variability.

1.3 Research objective and research questions

The aim of this research is thus to investigate the extent of difference in mortality patterns for New Zealand Māori and New Zealand non-Māori. Next to estimating this difference, this research searches for reasons for the existence of this difference. To be able to achieve this research objective, the following main research question is formulated:

"*To what extent and why do the New Zealand Māori and New Zealand non-Māori differ in their mortality patterns?*"

To help guide the research, the following sub-questions are formulated:

1. *How have New Zealand Māori and New Zealand non-Māori life expectancy levels evolved in the past?*
2. *How much does the life expectancy of New Zealand Māori and New Zealand non-Māori differ? What are the contributions of different causes of death and age groups to this difference?*
3. *How have New Zealand Māori and New Zealand non-Māori lifespan variability levels evolved in the past?*
4. *How do the New Zealand Māori and New Zealand non-Māori differ in the variability of their lifespans? What are causes and age groups that contribute to this variability in lifespan?*
5. *Which aspects of public health policies should be targeted to decrease the health gap between the New Zealand Māori and New Zealand non-Māori?*

The first and third sub-question will be answered by analysing life table data from 1948 onwards. The second sub-question will be answered by constructing and investigating period life tables for both populations groups and decomposing this data by age groups and causes of death. The fourth sub-question will be answered by adding a measure of lifespan variability to the life expectancy analysis. This measure of lifespan variability then needs to be decomposed by age groups and causes of death. Sub-question five can be addressed by interpreting the literature review and the results of the analyses. The main research question can be acknowledged by incorporating the answers of the sub-questions.

Although there are Māori living outside of New Zealand, this research is confined to those residing in New Zealand due to data availability and clarity. The units of analysis for this research are thus the New Zealand Māori and New Zealand non-Māori population groups, which will be referred to as Māori and non-Māori from this point forward. More information on the Māori and non-Māori populations is included in chapter **1.1** and chapter **3.3.2**.

1.4 Thesis outline

In chapter **1**, the area of interest is sketched and the problem described. In addition, the relevance of the study is indicated and the research objective and research questions are presented. Chapter **2** provides a literature review on ethnic health and mortality differentials and the determinants of indigenous health. In chapter **3**, the methodology of the analyses is explained and information about the used datasets is given. Chapter **4** quantifies the results of the empirical analyses, while chapter **5** discusses the outcomes in the light of previous research and the New Zealand context. Finally, chapter **6** concludes this study by answering the research questions.

2. Theoretical background

Chapter **1** shows the existence of health and mortality gaps between the Māori and non-Māori population groups. Chapter **2.1** aims to get a deeper understanding of the knowledge of existing mortality patterns for Indigenous populations and other ethnic minorities. It addresses observed patterns concerning life expectancy, lifespan variability, age patterns of mortality rates, and causes of death. To gain a sense of understanding for the existence of the gaps, one can wonder where these gaps originate. A lot of research has been done around the question "*What factors determine health outcomes?*". Over the years, the answers to this question and reasoning behind it have evolved to entail behavioural determinants and social determinants of health. Chapter **2.2** provides a non-comprehensive overview of the mostly social epidemiologic literature on the determinants of health. This literature review is summarized in chapter **2.3**, which also provides a conceptual model that envisions how certain health outcomes in Indigenous populations come about.

2.1 Ethnic mortality differentials

2.1.1 Life expectancy

It is interesting to study the literature on life expectancy for different Indigenous populations to understand what patterns are generally observed, and thus if the New Zealand context fits within this pattern. Life expectancy provides an average measure of health for a population and is, therefore, a quick measure for observing differences (Anthamatten & Hazen, 2011) (for more information on the life expectancy measure see **3.1.1**). Countries with indigenous populations, such as Australia, Canada, and the United States, have been studying the mortality of their indigenous populations. In 2017, the Australian Aboriginal and Torres Strait Islander people had a life expectancy of 71.6 (males) and 75.6 (females) years. This means Indigenous Australians had a life expectancy 8.6 (males) and 7.8 (females) years lower compared to their non-Indigenous counterparts. While the gap has substantially decreased over the years, Indigenous people still experience a life expectancy that is approximately 10% lower than that of non-Indigenous people (Australian Institute of Health and Welfare, 2019b). In Canada, the numbers are a bit more delicate due to data issues, but estimates for 2011 show a life expectancy gap ranging from 4.5 to 11.4 years for males and 5.0 to 11.2 years for females, the difference depending on being First Nation, Métis, or Inuit (Tjepkema et al., 2019). The American Indians and Alaskan Natives experience a life expectancy that is 5.5 years lower than the total American population (U.S. Department of Health and Human Services – Indian Health Service, 2019). Indigenous populations outside the CANZUS-nations also experience lower life expectancy compared to their non-Indigenous counterparts. In 2010, Guatemalan Indigenous people experienced 13 years lower life expectancy, the Indigenous population in Panama 10 years, and the number for Mexico was set at 6 years (United Nations, 2010). For all Indigenous populations, life expectancy thus appears to be lower compared to the non-Indigenous population. In New Zealand, this has been true as well: from the moment population numbers and deaths were documented in 1948, Māori have had a lower life expectancy than the non-Māori population (New Zealand Government – Ministry of Social Development, 2016). The exact numbers and differences are calculated in chapter **4**.

2.1.2 Lifespan variability

Lifespan variability is a measure that informs us about mortality from a different point of view, and it is recently becoming more prominent in the literature. Whereas life expectancy has generally been increasing for all population groups throughout the world, the pattern is less uniform when considering lifespan variability (Edwards & Tuljapurkar, 2005). Differences in lifespan variability for indigenous and non-indigenous populations have, to the best of my knowledge, not been published to this point. Little is known about the relationship between ethnicity and lifespan variability.

Lariscy and colleagues (2016) are one of the few researchers who addressed ethnic differences in lifespan variability. Their research focuses on lifespan variability among the Hispanic and white ethnicities of the United States (U.S.). Alongside a higher life expectancy, lifespan variability for Hispanics is found to be lower compared to whites. The case for Hispanics in the United States is known

as the 'Hispanic paradox' since Hispanics experience a mortality advantage while they are socioeconomically disadvantaged. While the case of the Hispanics and whites in the United States might be an exception, it does fit the general pattern of a negative relationship between life expectancy and lifespan variability (see also **chapter 3.2.1**). Research into lifespan variability for different races has also been conducted by Edwards & Tuljapurkar (2005). Their research, conducted with U.S. data, indicated that lifespan variability was 15-20% higher for African Americans compared to whites. Edwards & Tuljapurkar (2005, p.658) suggest that *"…racial differences in U.S. mortality are thought to serve as a proxy for socioeconomic differences*." Therefore, research into the relationship between lifespan variability and socioeconomic status directly has been conducted.

Brown and colleagues (2012), Edwards & Tuljapurkar (2005), and Van Raalte and colleagues (2011) all seem to find a link between a lower socioeconomic status and a higher lifespan variability. People in the lowest income quintile experienced a higher lifespan variability compared to people in the highest income quintile. The same pattern was found when focusing on educational attainment (Edwards & Tuljapurkar, 2005). The research by van Raalte and colleagues (2011) also concluded that, for 10 European countries, educational attainment is reflected in one's life expectancy and lifespan variability: a lower educated population is linked with a lower life expectancy and a higher lifespan variability. Brown and colleagues (2012) confirmed that the case was similar in the United States. When considering occupational class, another measure for socioeconomic status, Van Raalte and colleagues (2014) found higher levels of lifespan variability among manual workers compared to non-manual workers. These three measures of socioeconomic status – income, education, and occupation – thus illustrate the negative relationship between socioeconomic status and lifespan variability.

When taking the research on racial differences by Edwards & Tuljapurkar (2005) into account and considering the relationship between socioeconomic status and lifespan variability, it can be expected that Māori exhibit a larger lifespan variability compared to non-Māori due to their lower socioeconomic status. Also, the link that generally persists between life expectancy and lifespan variability (see chapter **3.2.1**) makes it plausible to expect a higher lifespan variability for Māori compared to non-Māori.

2.1.3 Causes of death

When looking at mortality measures such as life expectancy and lifespan variability, a predominant disadvantage for ethnic minorities is revealed. To gain a deeper understanding of the mortality patterns, one can wonder of what causes these people die and if these are much different from the non-Indigenous population or the ethnic majority.

In the Australian context, Wiemers and colleagues (2018) point out that the difference in the mortality gap between the Indigenous and non-Indigenous population can be largely explained by a higher burden of ischaemic heart disease, which is a cardiovascular disease. Woods et al. (2012) confirm the excess burden of heart failure in Indigenous Australians. Chronic diseases in general, especially cardiovascular and respiratory diseases, are explanatory for the Australian mortality gap (Anderson & Kowal, 2012). Vos et al. (2009) studied Disability Adjusted Life Years (DALY'S) and consequently added diabetes and mental disorders to this list. An exception is cancers: 27% of all non-Indigenous deaths could be ascribed to this cause, whereas this was 15% for the Indigenous population. Next to internal causes of death, external causes of death such as accidents, intentional self-harm and assault are causing relatively more deaths in the Indigenous compared to the non-Indigenous population (Australian Bureau of Statistics, 2008). Zhao & Dempsey (2006) indicate that the improvements in life expectancy, in combination with the excess burden of chronic diseases, might challenge the decrease of the Australian Indigenous - non-Indigenous mortality gap.

Prominent in the case of Canada is the high suicide rate of the Indigenous Canadian population, with the suicide rates for the Inuit being among the highest in the world, mainly due to people under 25 years of age (Chachamovich, 2015; Government of Canada, 2014). Mortality rates for external causes of death in general, and suicide and injury in particular, are higher among the Indigenous Canadian population compared to the non-Indigenous Canadian population (British Columbia Provincial Health Officer, 2009; Tjepkema et al., 2011). Chronic diseases are a more prevalent cause of death for Indigenous people, with cancers again being the exception: cancer mortality in Canada is lower among Indigenous than among non-Indigenous people (British Columbia Provincial Health Officer, 2009).

In contrast to the Australian and Canadian Indigenous populations, the American Indian and Alaska Native (AI/AN) population in the United States experience slightly higher cancer mortality rates compared to the non-Indigenous population (Espey et al., 2014). AI/AN also experience higher death rates from injuries and diabetes mellitus (Chang et al., 2016), and have higher rates for homicide (Espey et al., 2014) and chronic liver disease (Heron, 2012). Mortality rates for heart diseases are slightly increased in comparison to the non-Indigenous population. Just like in Canada, suicide is a problem in the Indigenous population, especially among those younger than 25 years of age (Centers for Disease Control and Prevention, 2014; Heron, 2012).

An official national publication researching 2010-2015 mortality data for Māori and non-Māori illustrates an excess burden in terms of cardiovascular diseases, cancers, and respiratory diseases for Māori. Mortality rates for external causes, too, contributed to the gap: mortality rates for suicide were two times as high for Māori compared to non-Māori, 2.5 times as high for assault and homicide and 1.5 times as high for injuries (New Zealand Government – Ministry of Health, 2015a).

One trend in all the CANZUS-nations seems to be the elevated mortality rate for suicide, also called intentional self-harm. More generally, external causes of death seem to emerge as an excess burden of mortality for the Indigenous populations in these countries. Cardiovascular diseases also contribute to the mortality gaps in all CANZUS-nations. Respiratory disease claims excessive Indigenous lives in Australia and New Zealand, and, whereas cancer mortality rates for Indigenous people are lower in the Australian and Canadian context they are higher in the United States and New Zealand. The gaps thus seem to be partly overlapping, but they are also conditional upon geographical context and lifestyle. A clear-cut list of causes that this research will find as contributing to the Māori/non-Māori mortality gap can thus not be provided, although it will most likely at least include external causes and cardiovascular diseases as large contributors.

2.1.4 Age pattern of mortality rates

The trend of a disadvantaged Indigenous population or ethnic minority becomes a bit more complex when observing age-specific mortality rates for different population groups. There appears to be a standard of ethnic minorities exhibiting higher mortality rates. However, in older ages, ethnic minorities seem to be doing rather well. In fact, the oldest-aged of ethnic minorities are often found to be doing better compared to the ethnic majority in terms of mortality rates.

This mortality crossover is mostly studied in the context of blacks and whites in the United States (see e.g. Eberstein et al., 2008; Elo & Preston, 1997; Manton & Stallard, 1997; Nam, 1995), where blacks experience lower life expectancy and higher mortality rates at younger ages, but a mortality crossover occurs around age 85. The mortality crossover is also observed in other population groups within the United States: African Americans and whites invert their mortality pattern between age 75 and age 80 (Roth et al., 2016). The Hispanic population too, which experiences a disadvantaged socioeconomic status, has lower mortality rates at older ages compared to the white population (Black et al., 2017). In theory, mortality crossovers can influence a population's lifespan variability. This is explained in chapter **3.2.1**.

The literature on the subject of mortality crossovers has provided two possible explanations for this phenomenon: selective survival and data quality (Lariscy, 2017). Manton & Stallard (1981) proposed that ageing populations cannot be considered homogenous. That is, when mortality rates in the younger ages are relatively high, the weakest individuals of that population group will not survive these younger ages. This would leave a relatively robust older-age population residual. The relatively high age at which a mortality crossover takes place – usually not before the age of 75 – can be explained in the same way: even individuals who survive to retirement age survive selectively: the frailest individuals will not survive to ages above approximately age 75, leaving a robust old-age cohort after this age. Selective survival thus implies heterogeneity, even when individuals belong to the same ethnicity or race.

The second explanation for mortality convergence and crossovers concerns data quality. Two types of biases can distort the data quality in this sense: age misstatement and ethnicity misstatement. Age misstatement occurs when the age in the vital statistics (i.e. the census) does not correspond with the age on the death certificate. Preston and colleagues (1999) stated that the effects of age misstatement are often encountered as lower mortality rates. When using adjusted mortality rates for blacks, Preston

and colleagues (1996) found the mortality crossover with the white population disappeared. Next to age misstatement, misstatements concerning race or ethnicity can distort data quality. Whereas ethnicity in the census is self-reported, ethnicity on the death certificate is decided upon by the funeral director and family of the deceased, both in the United States (United States Census Bureau, 2017; Arias et al., 2016) and New Zealand (New Zealand Government – Ministry of Health, 2019b). Mismatched ethnicity can thus cause inaccuracies in the numerator and denominator of the mortality rate equation.

The difference between the explanations - selective survival and data quality - lies in the fact that whether or not the mortality crossover is a real or an artificial phenomenon. When populations indeed survive selectively and the more robust population is still alive at the oldest ages, then the mortality crossover is real. However, when a mortality crossover is caused by low-quality data, the mortality crossover can be considered artificial. Whereas some literature states that low data quality is the reason behind mortality crossovers (see e.g. Black et al., 2017; Elo & Preston, 1997), the majority of researchers (see e.g. Eberstein et al., 2008; Lariscy, 2017; Manton & Stallard, 1997; Masters, 2012) appears to conclude that selective survival is the reason for mortality crossovers and thus that mortality crossovers are a real phenomenon on the population level.

In the context of the New Zealand Māori, mortality crossovers could possibly be observed too. If survival selection is indeed the reason for the observed crossovers, this selection might take place in any human population (Manton & Stallard, 1981). Moreover, if low data quality is the reason for the observed crossovers, one can still expect to observe mortality crossovers in the New Zealand mortality data. Whereas misstated ethnicity did not appear to be a problem for distinguishing between blacks and whites in the United States (Arias et al., 2016), this might pose a problem that influences mortality rates in New Zealand, since Māori ethnicity on death certificates might be undercounted (Houghton, 2002). The United States mortality data of blacks seems to encounter age misstatement (see e.g. Preston et al., 1996). However, similar encounters are not reported for New Zealand. Mortality crossovers will thus most likely not be caused by age misstatement in the New Zealand context, although they can be expected to appear due to selective survival or ethnicity misstatement.

2.2 Determinants of health

Chapter **2.1** provides an insight into the existing mortality patterns in Indigenous populations and ethnic minorities. These patterns help to give an understanding of what is likely to be expected in the context of the New Zealand Māori. However, when studying health outcomes and mortality differentials, it is important to understand how these come about. The literature on health outcomes and where these originate indicate two broad groups of determinants that are frequently found: behavioural determinants of health and social determinants of health. Chapter **2.2.1** and **2.2.2** explore these determinants and what is known about their influence on health. Chapter **2.2.3** visualizes the findings in a conceptual model and incorporates the findings in ecosocial theory.

2.2.1 Behavioural determinants

Over the last decades, the field of epidemiology has provided evidence for the existence of a link between certain health behaviours and their consequent outcomes on health status. Since these behaviours determine health outcomes they are termed '*behavioural determinants of health*'. The effects of smoking, diet, physical activity and alcohol consumption are among the health behaviours that are most studied.

In the 1950s and 1960s, a lot of research into the effects of smoking was conducted (see e.g. Anthony & Thomas, 1970; Mandelbaum & Mandelbaum, 1952). The enormous increase in lung cancer mortality started this new wave of investigating tobacco smoking (Newcomb & Carbone, 1992). From then on, people are being informed and warned of the negative effects smoking can bring about. The negative effects mainly result in elevated cancer rates, especially lung cancer rates, elevated rates in diseases of the circulatory system, respiratory diseases, and issues during pregnancy and giving birth such as miscarriages (West, 2017).

A more recent line of research focusses on nutrition and its effects on health. Although there is still debate about which type of diet causes most harm and which type is most beneficial, researchers agree on the negative effects of diets containing mostly highly processed and industrialized foods and

the positive effects of diets containing a lot of fruit and vegetables (Hu et al., 2000; Joshipura et al., 2001; Law & Morris, 1998). Unhealthy diets can cause a multi-faceted array of health disadvantages, including obesity (An, 2015), mental ill-health (Jacka et al., 2014), cardiovascular diseases (Whatnall et al., 2016), and it can have adverse effects for the baby when pregnant (Ramos et al., 2018). Healthy diets, by contrast, provide a lower risk of premature mortality due to heart disease (Hu et al., 2000; Joshipura et al., 2001; Law & Morris, 1998), cancer (Schwingshackl et al., 2018; Vieira et al., 2016), and diabetes (Wang et al., 2016).

Next to smoking behaviour and (un)healthy diets, physical activity can influence one's health. Thirty minutes of moderate physical activity on most days of the week is commonly advised to gain health benefits from engaging in physical activity (World Health Organization, n.d.). Oguma et al. (2002) found that adhering to these guidelines has indeed a positive effect on postponing mortality. Next to decreasing the risk for all-cause premature mortality, physical activity is linked with reducing the risk for multiple chronic conditions. Especially cardiovascular diseases, circulatory diseases, cancers, and diabetes type 2 are found to be of decreased risk when the individual engages in regular physical activity (Rhodes et al., 2017).

The risk for digestive diseases, cardiovascular diseases, and cancers is also increased by harmful alcohol consumption. Alcohol consumption thus increases the risk of chronic diseases. Premature death can be caused by the high risk of injury that is involved with harmful alcohol consumption (World Health Organization, 2018). Whereas people in the Blue Zones of the world, the regions where the population enjoys remarkable longevity, usually drink small amounts of alcohol daily (Legrand et al., 2019), Burton & Sheron (2018) found that no level of alcohol consumption promotes health outcomes. However, increased amounts of alcohol lead to higher mortality risks (Jayasekara et al., 2014), and excessive drinking is, therefore, more harmful compared to drinking only small amounts of alcohol.

Diseases like obesity and diabetes mellitus type 2 are examples of lifestyle diseases since they are mainly caused by an unhealthy diet, a lack of physical activity, and other adverse health behaviours. These health behaviours or lifestyles are all influencing certain health outcomes on an individual level. However, they do not necessarily lead to worse health immediately: they cause individuals to be at higher risk for worse health outcomes. Whereas a certain share of the individuals performing unfavourable health behaviour(s) actually experience the negative consequences, there is also a share that does not experience them. Therefore, certain health behaviours or lifestyles are a risk factor for diseases. Since health behaviours like the ones mentioned before take place on an individual level and influence individuals' health outcomes, they can be considered individually-based risk factors for disease.

Indigenous populations generally display more unfavourable health behaviours compared to the non-Indigenous population (Vos et al., 2009). However, research into the habits of the New Zealand Māori has indicated that, on average, this population group does not have a lifestyle that is very contrasting to the non-Māori population. Nevertheless, when elaborating on the four health behaviours mentioned above, overall, Māori are disadvantaged over the non-Māori population. While the dietary habits and levels of physical activity of Māori are comparable to that of the non-Māori population, the adopted smoking rates in the Māori population do not promote favourable health outcomes. Smoking rates are over two times as high for Māori males compared to non-Māori males, and over three times as high for Māori females compared to non-Māori females. The higher smoking rates for Māori lead to an increased risk for certain lifestyle diseases and are thus likely to translate in elevated (lung) cancer rates and an increased share of respiratory diseases in the comparison with non-Māori. Concerning alcohol consumption in general, Māori and non-Māori can be considered comparable. However, compared to non-Māori, Māori are less likely to drink four or more times per week but are more likely to drink large amounts of alcohol when they do (New Zealand Government – Ministry of Health, 2018b). A convergence in health outcomes of Māori and non-Māori is to be expected based on the equality in dietary patterns and physical activity levels. However, the effect of the higher smoking rates and the increased likeliness of excessive alcohol consumption leads to a divergence in health outcomes between Māori and non-Māori. Thus, for behavioural determinants overall, Māori are expected to have a disadvantage over non-Māori.

2.2.2 Social determinants

While the behaviour-based determinants explain a part of the health difference found between different population groups, they do not explain the entire difference. In 1994, Marmot (1994) found that health behaviours such as smoking and drinking alcohol account for only one-third of the observed health difference between Britons with different employment levels. Cutler & Lleras-Muney (2006) found a similar number, and Khaw et al. (2008) found evidence for the cumulative impact of (un)healthy behaviours on mortality. This means that evidence has shown that the more health-promoting behaviours an individual performs, the lower the mortality risk, although these behavioural factors will still not explain the entire difference that is found between population groups. So even though individual health behaviours can lead to worse health outcomes they are not the sole culprit.

Previous research in the field of social epidemiology and medical sociology has established a paramount position for social factors as a cause of deteriorating health outcomes (Preda & Voigt, 2015). To express the association between social factors and health outcomes, academics speak of 'the social gradient in health'. The social gradient in health reflects the findings of a relationship between social factors and health: in general, the socially disadvantaged individuals are suffering deprived health outcomes and the socially advantaged individuals experience privileged health outcomes. These social factors can be divided into socio-economic factors and psychosocial factors.

Socio-economic factors are often quantified by means of three measures that indicate socio-economic status: education, income, and occupation. A lot of research has been done on the link between socio-economic status and health (see e.g. Dow & Rehkopf, 2010; Herd et al., 2007; Williams et al., 2010), and the general trend is to conclude that the lower educated, less earning, and lower-skilled workers experience the worst health outcomes. Due to minimal education, opportunities for high-skilled jobs and a corresponding higher income are lower (Braveman et al., 2011). Lower education levels are also associated with lower health knowledge and a lower probability of adapting health-related recommendations (Cutler & Lleras-Muney, 2006) and healthy behaviours (Braveman et al., 2011). A lower income reflects worse access to nutritious and healthy food (Booth et al., 2005), and results in lower-quality housing conditions in disadvantaged neighbourhoods, where air and water quality might be low (Braveman et al., 2011), industry or other toxic environments might be proximate (Bullard, 2000), and sports facilities might be fewer (Gordon-Larsen et al., 2006). Occupation in a lower-skilled profession can lead to higher exposure to chemicals (Braveman et al., 2011), higher rates of getting injured (O'Neil et al., 2001), and yields lower income.

These indicators of socio-economic status have a cross-sectional link: when an individual has a minimal education, he or she is more likely to have a low-skilled job and an associated low income. Thus, when an individual is disadvantaged in one of these indicators - education, income, or occupation -, he or she is likely to experience disadvantages in other fields as well. Additionally, these indicators appear to have a longitudinal link (Blane, 2005). This longitudinal link sheds light on the influence of childhood social organization and health outcomes in later life. For example, health outcomes in later life are associated with childhood social class (Cohen et al., 2010). In addition, a lower socioeconomic status during childhood is associated with higher levels of premature mortality (Galobardes et al., 2004). The influence of the life course appears thus to be important in studying health outcomes and premature mortality, and the impacts on health can be cumulative (Robson & Harris, 2007).

It appears that the social gradient in health is visible within ethnicities: disadvantaged blacks have poorer health compared to more advantaged blacks, and the same pattern can be observed for Hispanics and whites (Braveman et al., 2011). However, there are differences between ethnicities visible that show a disadvantage in socio-economic status for ethnic minorities compared to ethnic majorities. Hispanics in the United States, for example, attain lower education compared to the white population (U.S. Department of Education, 2017), and the same pattern can be seen for the Indigenous population of Canada (National Collaborating Centre for Aboriginal Health, 2017). The Indigenous population in Australia too, has a weekly income that is on average 33% lower than the weekly income of non-Indigenous Australians (Australian Institute of Health and Welfare, 2019a). Considering the concentration of people in low-paid jobs, the Hispanic and African-American population in the United States are disadvantaged over the white population (Alonso-Villar et al., 2012), and unemployment rates are higher for the Spanish Roma compared to the rest of the population (La Parra Casado et al., 2016).

Also in New Zealand, this link with socioeconomic status and ethnicity is clearly visible: on average, Māori score lower on all three indicators. Whereas 64.3% of the non-Māori complete school at the high school level, this is only 45.1% for Māori. This leads Māori to have a higher percentage of people earning less than 10,000NZD and, compared to non-Māori, more than twice as much Māori are on income support (New Zealand Government – Ministry of Health, 2015a). Socioeconomic status thus influences health outcomes through access to resources, connections and knowledge. Due to the ever-present pattern of lower socioeconomic status and worse health outcomes, socioeconomic status is seen as a fundamental cause of health. Even though the origin of disadvantaged health outcomes for people in lower social classes may vary over time, the difference in health outcome by socioeconomic status endures (Link & Phelan, 1995; Van Raalte et al., 2014).

However, the social determinants of health include more than socio-economic measures like education, income, and occupation. Psychosocial factors are also important social determinants that influence health outcomes. These psychosocial factors include feelings of social support, the existence of a social network, and feelings of discrimination and racism.

Previous research has provided evidence for a link between the absence of a social network and adverse health outcomes, and, the other way around, for a positive relationship between feelings of social support and health outcomes (see e.g. Kemp et al., 2017; Rook & Charles, 2017). A study by Holt-Lunstad and colleagues (2010) showed that the influence of a social network was equally important as smoking cessation or adopting more physical activity in reducing mortality risk. In this sense, psychosocial factors are just as important as behavioural factors in explaining health outcomes. For the New Zealand Māori, feelings of loneliness appear not to be significantly more common compared to the non-Māori population. In 2014, all ethnic groups in New Zealand reported rates of loneliness of around 15% (New Zealand Government – Ministry of Social Development, 2016). When looking at statistics of people living alone, the Europeans are the ethnic group with the highest percentage of people living alone (approximately 11.5%), followed by Māori (9%) (Statistics New Zealand, 2016). These figures would point to equality in terms of social support and a social network, or even a slight advantage for Māori, and would, on its own, not cause a higher mortality risk for the Māori population.

Next to the presence or absence of a social network and social support, racism affects one's psychological state and consequently one's health. When an individual experiences racial discrimination, this affects one's mental health state, health behaviours (Paradies, 2006), and, thus, one's overall health outcomes. But racism works in more indirect ways too: when experiencing racism in applying or keeping a job, one's socioeconomic status might not increase when it could have if racism did not take place, which affects one's housing situation, resources to buy healthy food and all previously mentioned ways in which socioeconomic status influences health (Harris et al., 2006). The impacts of racism might be even more pronounced when considering indigeneity. Research has found that Māori, along with other Indigenous populations of the CANZUS nations, experience racism in everyday life and especially in the health care sector (see e.g. Durey & Thompson, 2012; Harris et al., 2006; Hippolite & Bruce, 2010; Reid et al., 2019). An example of such racism can be found in health care practices, where Māori often suffer from being treated differently and being misunderstood. A holistic treatment of disease is important for Māori, and this includes spirituality (New Zealand Government – Ministry of Health, 2017). However, the modern healthcare system does not provide such a spiritual treatment (Walker et al., 2008), nor do most nurse and medical practitioners understand the need for such treatment (McCreanor & Nairn, 2002). Research has also proven that Māori are less frequently referred for surgery (Westbrooke & Baxter, 2001) and are offered preventive care less often compared to non-Māori. Misunderstanding and previous unfavourable encounters can lead to non-admission or late-admission to medical care for Māori, leading to no treatment at all or treating an illness or disease too late (Ellison-Loschmann & Pearce, 2006). This (unintended) discrimination might thus indirectly cause elevated mortality at premature ages. In general, the percentage of people that experience racism is higher for Māori than for people of European ethnicity, which is the largest non-Māori ethnicity. Pacific people experience approximately equally as often racism as Māori, and Asian people encounter racism most often (Statistics New Zealand, 2012). Harris and colleagues (2006) found that the self-reported experience of racial discrimination is strongly associated with poor health outcomes, even when corrected for socioeconomic factors. The health differences between Māori and non-Māori might thus (partly) be explained by these experiences of racism. The experiences of racial discrimination can occur

on a personal level between two individuals – interpersonal racism – or it can be ingrained in policies and everyday practices – institutionalized racism (Karlsen & Nazroo, 2002). When racism is institutionalized, people can be unconsciously racist, since they might not recognize the action or non-action as racist.

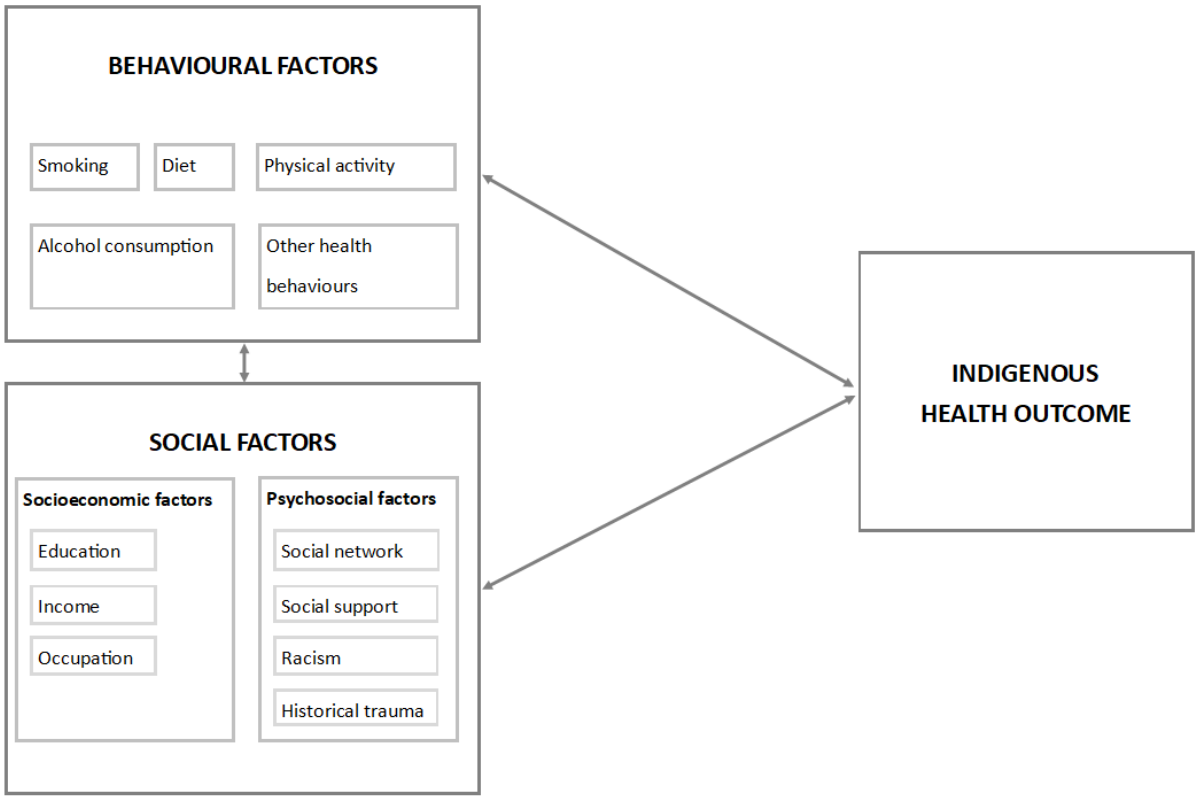
While racism might be unintentional, "…*current policies and practices are products of colonial processes that have shaped White people's sense of the way things are and should be, and which incorporates their own entitlement, superiority and established systems...*" (Durey & Thompson, 2012, p.3). In this sense, Indigenous populations are still suffering from the colonial past. This thought is reflected in the concept of historical trauma, which was first addressed by Brave Heart (2003). Studies into historical trauma, which is "*the cumulative emotional and psychological wounding over the lifespan and across generations, emanating from massive group trauma experiences*" (Brave Heart, 2003, p.7), link occurrences in previous generations to the health of the contemporary population (group) (see e.g. Evans-Campbell, 2008; Walters et al., 2011). Walters et al. (2011, p.183) mention that "*trauma can literally become embodied, manifesting as poor mental and physical health outcomes in descendant generations*". The findings of the study by Brave Heart (2003) show that historical trauma, a burden of the native population of the CANZUS-nations, contributes to the existing health inequalities. Even more so, the impact of historical trauma on health outcomes appears to be even bigger than contemporary aggravations (Walls & Whitbeck, 2011), such as interpersonal violence (Walters et al., 2011), and racism (Paradies, 2016). Moreover, the effect of historical trauma is intensified by contemporary aggravations, also called present trauma. Both historical and present trauma can thus have an incremental effect on the discrepancy in health outcomes between the Māori and non-Māori.

2.3 Conceptual model

To sum up, health outcomes are influenced by behavioural factors that have a more direct influence on health and by social factors that often have a more indirect influence. Behavioural factors can, however, be influenced by social factors. Having a socioeconomic disadvantage, for example, limits the access to resources such as adequate money and knowledge to prevent disease or minimize the consequences of a disease once it occurs (Phelan & Link, 2013). Since Māori experience a lower level of socioeconomic measures than non-Māori, this might seem a plausible explanation. Nevertheless, Pollock (2018) found that even when correcting for socioeconomic factors, Māori suffer poorer health. This might mean that psychosocial factors play a paramount role in inducing Māori poor health outcomes. This idea corresponds with the study by Harris and colleagues (2016) which acknowledges that racial discrimination is strongly associated with poor health outcomes, even when corrected for socioeconomic factors. Not only racial discrimination but also historical trauma leads to a disadvantage in the psychosocial state. The importance of psychosocial factors does not negate the detrimental position of Māori when considering socioeconomic status or when evaluating certain health behaviours. In fact, all these factors are interlinked and hard to fragmentize. As Nancy Krieger (2001, p.673) noted: "*Simplistic divisions of the social and biological will not suffice*." Favourable psychosocial determinants are, for example, found to improve the frequency of health-promoting behaviours (Boehm & Kubzansky, 2012; Dubois et al., 2012). A link between behavioural determinants and socioeconomic determinants is simply visualized when considering the effects of consuming too much alcohol on keeping one's job and subsequent income. Losing one's job can lead to a deterioration of one's well-being and thus psychosocial state. Contrarily, enjoying a high education might lead one to adopt a healthy lifestyle because one is aware of the consequences of unhealthy behaviour. All determinants of individual health, in this chapter summarized as behavioural determinants and social determinants (consisting of socioeconomic determinants and psychosocial determinants), are thus interlinked and can be cumulative over time. These interlinkages are reflected in the ecosocial theory that was introduced by social epidemiologist Nancy Krieger (1994). In addressing the interlinkages of determinants of health, ecosocial theory reflects what the field of social epidemiology is about: combining the medical and behavioural sciences. Since behavioural, socioeconomic and psychosocial factors all seem to influence health outcomes, ecosocial theory advocates for a combination of the factors: it is the accumulation and integration of behavioural and social factors that lead to certain health outcomes (Krieger, 2001). For Indigenous populations such as the Māori, historical trauma and racism due to ethnicity seem to play an important role in the web of determinants of health as well. Figure 1 summarizes the determinants of

health mentioned in this chapter and the interlinkages that are at play that ultimately lead to certain health outcomes for Indigenous people.

Figure 1: Conceptual model.



Source: own illustration.

This chapter has illustrated which factors determine health outcomes and that these factors are not operating separately but are interlinked within all population groups. This interlinkedness allows for health (dis)advantages to be cumulative, which might explain the large differences between certain population groups. Ethnic minorities are on many measures at a disadvantage when comparing mortality measures. One bright spot is to be observed for the oldest age category. Previous research has observed mortality crossovers in these ages. All in all, the New Zealand context can be expected to reflect general trends in health outcomes and ethnic mortality differentials. This would include lower life expectancy and higher lifespan variability for the Māori population, the gaps likely being explained by cardiovascular diseases and external causes of death, and possible mortality crossovers for the older age groups. Since both behavioural and social determinants of health work in an interlinked and cumulative way, it is hard, if not impossible, to identify sole determinants of the mortality gap. It has become clear that strategies achieving to improve health should not focus on one (part of a) determinant of health, but should encompass a wide range of tools to address multiple factors leading to ill-health. Nevertheless, it is a matter of social justice to try to improve Māori health and narrow the existing health gaps. To quantify and decompose these gaps, chapter **3** will now focus on the methodology of how this is done.

3. Methodology

The empirical analyses necessary to answer the research questions consist of two methods: construction of a life table and a decomposition thereof. Two summary measures of mortality are derived from the life table, namely life expectancy and lifespan variability. The decomposition of these measures indicates age groups and causes of death that contribute to differences in the measures between the two population groups. The analyses are applied to two different sets of data: an all-cause mortality dataset from 1948-2008, and a cause-specific dataset from 2016. First, to provide an overview of the current mortality patterns of both the Māori and non-Māori population groups, a life table is computed using the 2016 data. This is extended by an overview of life expectancy for both population groups from 1948 until 2008. By providing this information, it will become clear whether there is a trend visible for one or both population groups. A trend will show whether the inequality, if it exists, is enlarging or not and whether the necessity for the implementation or adjustment of public policy is required. Additional information on the computation of life tables and the decomposition of its measures is provided in chapter **3.1**.

Secondly, the lifespan variability metric will provide another perspective on the distribution of ages at death. Where life expectancy provides an average of age at death, lifespan variability considers the variability of when deaths occur (Edwards & Tuljapurkar, 2005), and whether the average level is equally accessible for all people. Further information on lifespan variability and how this metric is derived is provided in chapter **3.2**.

Lastly, chapter **3.3** focuses on the data used for this particular study, which is derived from the Human Mortality Database (Barbieri et al., 2015) and official statistics made publicly accessible by the New Zealand Government – Ministry of Health (2019a). This subchapter addresses both datasets and their limitations. Also, the adapted typology of causes of death is shown and elaborated upon.

3.1 Life table analysis

3.1.1 The life table

A life table is an often-used tool in demographic studies because it provides an overview of the mortality pattern of an observed or hypothetical cohort. A life table provides adequate information on mortality including, but not limited to, age-specific death rates, the probability of dying and surviving an age group, and remaining life expectancy at any age. Life expectancy is defined as "*the average number of additional years that a survivor to age x will live beyond that age*" (Preston et al., 2001, p. 39). Most often, life expectancy at birth is used to summarize mortality conditions. Life expectancy at birth, denoted as e^0, equals the average age at death for the cohort (Preston et al., 2001). Life tables are an effective tool for comparing populations since they standardize differences in population size and age composition across populations (Nau & Firebaugh, 2012). Since no two population groups, including the Māori and non-Māori of New Zealand, are exactly equal in size and composition, a life table analysis is a convenient method to be able to compare the groups.

A life table provides mortality information about a cohort. This cohort can be a real cohort, for example, every person that was born in a certain year. The use of a real cohort implies that the life table can only be completed when the full cohort is extinct since it reflects what happened to this particular cohort. The construction of these cohort life tables can be useful for analysing data of extinct populations but poses a problem for researching populations that are currently alive (Hinde, 1998; Preston et al., 2001). In most occasions, including this research, it is advantageous to compose period life tables. Period life tables reflect "*what would happen to a cohort if it were subjected for all of its life to the mortality conditions of that period*" (Preston et al., 2001, p. 42). Mortality conditions consist of the age-specific death rates for the current period (Preston et al., 2001). Since health conditions, technology, and health policies might change in the future, the use of current mortality conditions in a period life table does not reflect the reality of individual cohorts (Hinde, 1998). However, a period life table is useful in that it illustrates "*the mortality experience of a population during a particular period*" (Hinde, 1998, p. 38), and it gives "*an excellent indication of the overall health performance of a society at a specific point in time*" (Smits & Monden, 2009, p. 1115).

Age is placed in the rows of a life table. In theory, age is a continuous quantity, since people age every day, every minute and even every second, and not just on their birthday. However, in practice age is discretized to single years or age groups. Age groups containing multiple ages are used to limit the number of rows in a life table and to be able to give a clearer overview of the mortality pattern compared to a full-length life table (i.e. one that uses single years) that can consist of over 100 rows (Hinde, 1998). In this research, 5-year age groups are used, leading to abridged life tables in the analysis. The oldest age group is treated differently, leading to the following format: 0-4, 5-9, 10-14, 15-19, …, 80-84 and 85+. Since 5-year age groups are provided by the cause-specific dataset, this is the most specific format available. The use of these intervals, although preferably with a separate age group for 0-1 and 1-4, is considered "*the most conventional format*" according to Preston et al. (2001, p.39). The youngest and oldest intervals mentioned above require extra attention. Mortality in the youngest age group of 0-4 years of age is often concentrated in the first year, leading to distorted measures of life expectancy when making calculations for the entire 0-4 age group (Preston, 2001). Therefore, when possible, infant mortality is calculated separately using a different calculation for ${}_na_x$, which is the average years lived in the age interval (Hinde, 1998). However, infant population data is not provided by the cause-specific dataset (Government of New Zealand – Ministry of Health, 2019a) and is, therefore, not treated separately in this research. Next to the young ages, the open-ended interval at the end of the life table necessitates extra attention. The last interval in a life table will always be open-ended since it is unknown at what age the last person of that cohort will die, i.e. there is no final age at which it is known that everyone will have died. Therefore, the open-ended interval lasts, theoretically speaking, to infinity (Hinde, 1998; Preston, 2001). However, ${}_nq_x$, the probability of dying within the last age interval, will be one, since everyone has to die at some point within this interval. This also implies that the number left alive at the beginning of the age interval (l_x) is equal to the number dying within this interval (${}_nd_x$), and thus that L_x can be calculated by dividing l_x over m_x to complete the life table (Preston, 2001). This is not a very accurate measure since it assumes that all deaths are evenly distributed, which often appears not to be true for the oldest age group. However, it is considered a sufficient method (Hinde, 1998). In the case of this research, the open-ended interval contains all data above the age of 85, since this is the most accurate data available in the cause-specific dataset.

3.1.2 Decomposition of life table measures

Since life expectancy at birth happens to be different for the Māori and non-Māori population groups (Statistics New Zealand, 2015), it is the aim to investigate where this difference originates. The decomposition of this difference can be achieved by focusing on age groups: How much does each age group contribute to the observed difference? Zooming in on causes of death is another point of view: How much does each cause of death contribute to the observed difference? Also, these views can be combined to get an idea which causes of death contributed to the observed difference in a particular age group.

In the 1980s, multiple scholars introduced methods to decompose differences in life expectancy (Andreev, 1982; Arriaga, 1984; Pollard, 1982; Pressat, 1985) but these methods are considered to be qualitatively indistinguishable and can, therefore, be regarded similar (Edwards & Tuljapurkar, 2005). The formulae composed in the 1980s are specific for the decomposition of differences in life expectancies by ages. To be able to decompose other aggregate measures as well, such as measures in lifespan variability (e.g. Gini coefficient, e†, interquartile range), Andreev and colleagues (2002) composed a more general algorithm. For the sake of consistency, and since the applied algorithm of Andreev et al. (2002) leads to the same formula as the ones by Andreev (1982), Arriaga (1984) and Pressat (1985), this method was also applied for the decomposition of the difference in life expectancies.

The algorithm of Andreev et al. (2002) makes use of the method of stepwise replacement. This involves a replacement of age-specific death rates in one population by age-specific death rates from another population so the effect on life expectancy can be calculated. This can also be done for causes of death, making use of cause-specific death rates instead of age-specific death rates. When incorporating age- and cause-specific rates, the effects of the causes of death in each age group are calculated (Shkolnikov et al., 2019). Computing life tables and decomposing differences in life expectancy and lifespan variability can be done using Excel, however, this can be slow and it limits the causes of death that can be taken into account (Andreev & Shkolnikov, 2012). The statistical software

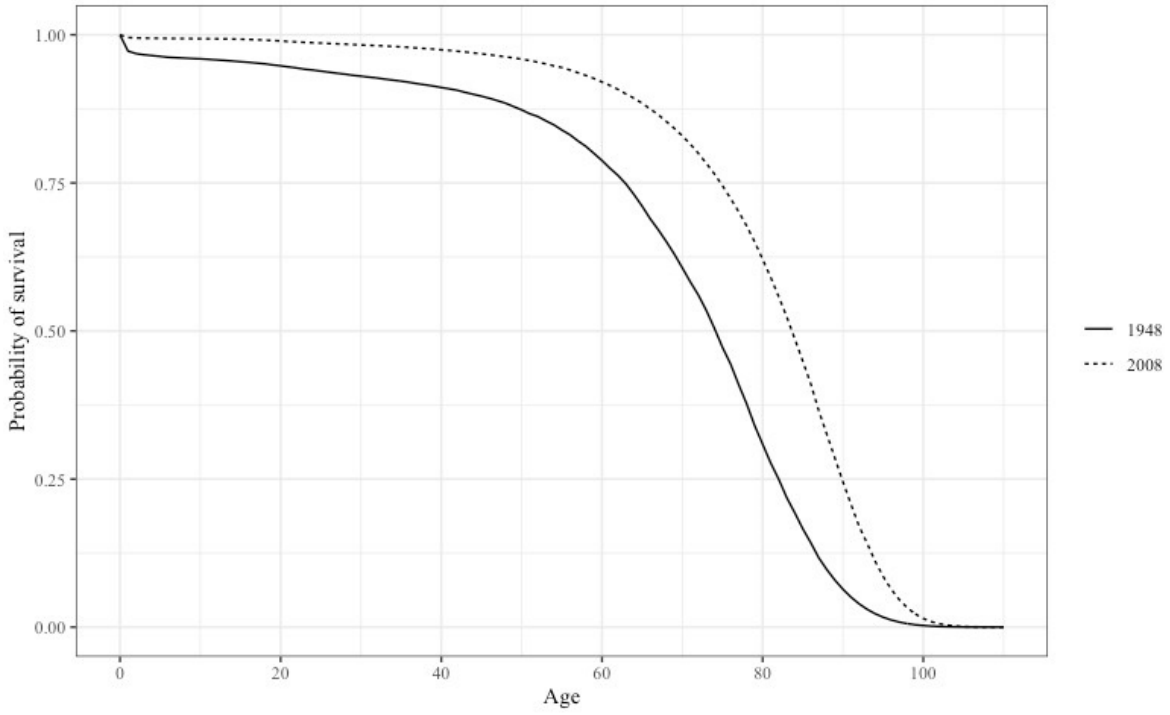
programme R, established by Ihaka & Gentleman (1996), has dedicated packages available that allow doing these calculations quickly and unrestrictedly. The statistical language of R was therefore opted as a means of performing the analyses. DemoDecomp (Riffe, 2018a), developed by Tim Riffe, is an R-package that can be used for decomposition analyses. The DemoDecomp package offers two methods for decomposing data, namely the stepwise replacement method as introduced by Andreev and colleagues (2002) that is mentioned above and the pseudo-continuous decomposition method as introduced by Horiuchi and colleagues (2008). The former is chosen for these analyses, although its results are essentially equal to the pseudo-continuous method.

3.2 Lifespan variability metric

3.2.1 Lifespan variability

Life expectancy provides information about the average length of life of a population. Another measure that provides knowledge about mortality is lifespan variability. Lifespan variability explains the "*variation in longevity*" (Van Raalte & Caswell, 2013, p. 1616). In other words, it shows how accessible the average age at death is for all people (Shkolnikov et al., 2003). When studying different populations or subgroups of a population, life expectancy might be relatively equal, while lifespan inequality can be high. This is true because life expectancy only presents us the average length of life of the population, whereas lifespan variability informs us on the heterogeneity in this data, thus giving another perspective on the mortality schemes of populations (Edwards & Tuljapurkar, 2005). The more concentrated the ages at death of a population are, and, thus, the lower the lifespan variability, the more rectangular the shape of the survival curve. The rectangularization of the survival curve is also called the compression of mortality since most deaths are compressed into older age (Wilmoth & Horiuchi, 1999). For means of illustration, Figure 2 shows the survival curve for the total population of New Zealand for 1948 and 2008. The survival curve has become more rectangular through the years, indicating compression of deaths at the older ages. For the total population of New Zealand, the lifespan variability has thus decreased over these years.

Figure 2: Survival curve for the total population of New Zealand for 1948 and 2008.



Source: own graph based on data from Demography Division - Statistics New Zealand (1948-2013).

Lifespan variability and life expectancy have a history of being linked by a negative relationship: an increasing life expectancy generally corresponds with a decreasing lifespan variability (Aburto et al., 2020; Shkolnikov et al., 2009; Smits & Monden, 2009). While this has been the case for most populations (see e.g. Smits & Monden, 2009; Wilmoth & Horiuchi, 1999), this relationship appears to be variable across populations and over time. For example, Edwards & Tuljapurkar (2005) found both life expectancy and lifespan variability to be increasing for several developed countries, but lifespan variability did so at a much slower pace. Therefore, progress in life expectancy does not always directly relate to the same level of progress in avoiding premature mortality (Vaupel et al., 2011).

To be able to define which deaths are premature and which ones are not, Zhang & Vaupel (2009) introduced a threshold age: reducing mortality below this age decreases lifespan variability while reducing mortality above this age increases lifespan variability. When considering rectangularization of the survival curve, this threshold age makes sense: when more deaths occur in the earlier years in life, the curve gets less rectangular, and lifespan variability thus increases. When more deaths occur in the later years of life, the shape of the curve gets more rectangular and lifespan variability decreases. The threshold age is generally just below life expectancy and is often used to determine which deaths can be considered as premature deaths and which deaths can be considered as late deaths (Vaupel et al., 2011).

The existence of mortality crossovers (see **2.1.4**) can, in theory, influence lifespan variability. When a population group that is disadvantaged for young and adult age groups and a population group that is advantaged for these age groups invert their mortality patterns, a mortality crossover is observed. The survival curve of the population group with the higher young-age mortality and lower high-age mortality is thus less rectangular compared to the other population group. The mortality before the threshold age is thus higher and mortality after the threshold age is lower, both leading to a higher lifespan variability.

3.2.2 Measures of lifespan variability

Multiple metrics have been developed to measure lifespan variability. Several analyses have explored which of these indices is the most appropriate (see e.g. Shkolnikov et al., 2003; Van Raalte & Caswell, 2013; Vaupel et al. 2011; Wilmoth & Horiuchi, 1999). The conclusions of these researches differ, although they all agree on the high correlation between the measures. All indices will roughly provide the same conclusions and results and can, therefore, be considered interchangeable (Colchero et al., 2016; Wilmoth & Horiuchi, 1999).

Life disparity, denoted as e†, is one of these indices. It measures the number of life-years lost due to death and is defined as "*the average remaining life expectancy at the ages when death occurs*" (Vaupel et al., 2011, p. 2). Life disparity is a measure that is easily understood and can intuitively be interpreted (Zhang & Vaupel, 2009), which is less so for other indices. The use of the same unit of measurement (i.e. years of life) as life expectancy aids the ability to understand the concept. Life disparity is, therefore, chosen as the measure of lifespan variability in this study. Since e† measures life-years lost due to death, it is computed as the summed product of life expectancy at the age at death (determined by the distribution from the life table) and the number of people dying within that interval. In mathematical notation, life disparity is calculated as:

$$e\dagger = \sum_x e_x \cdot d_x$$

When calculating life disparity for an entire population, e† displays the number of years of life that are on average lost due to death for this population (Vaupel et al., 2011). It is, therefore, both a measure of life disparity and a measure of premature mortality, emphasizing the direct relationship between these two concepts. Indeed, if people do not die at the same age (i.e. there are inequalities), this means that those who died earlier have died too young compared to what could have been achieved in a more equalitarian society, thus they have died prematurely. In other words, inequalities in the ages at death reflect the inability of the health care system to avoid people dying prematurely.

Because of the notable life expectancy gap between the Māori and non-Māori population groups, lifespan variability is likely to display a gap between both groups as well. To investigate where a difference in lifespan variability originates, the gap in life disparity is to be decomposed. To decompose e†, the method by Andreev and colleagues (2002) as described in **3.1.2** will be used.

3.3 Data

For performing the empirical analyses, data is extracted from two sources: the Human Mortality Database (Barbieri et al., 2015) and a mortality dataset published by the New Zealand Government – Ministry of Health (2019a). The Human Mortality Database (HMD) data, which is an all-cause mortality dataset, is used for constructing the historical trends in life expectancy and life disparity. The cause-specific Ministry of Health dataset was used for the construction of the life table, calculation of life disparity, and the decompositions of both these measures.

3.3.1 Human Mortality Database

The Human Mortality Database (Barbieri et al., 2015) is an open-access database presenting mortality data on 46 populations. The data provided consists of but is not limited to, birth counts, death counts, population estimates, and life tables. For data to be included in the HMD, it needs to be of adequate quality: data need to be practically complete and detailed to at least 5-year age intervals with a separate category for infants, and including high ages. Raw input data is handled to reflect standard output data formats. Since all data in the HMD is prone to these quality checks and are handled in the same manner, the data is comparable between populations (Barbieri et al., 2015). Using the HMDHFDPlus package (Riffe, 2018b), data can be loaded directly into R.

For New Zealand, data on three (sub-)populations is available: the total population, the Māori population and the non-Māori population. Most mortality data for these populations goes back to 1948 and is available in six different formats considering age intervals and year intervals. It is, therefore, possible to construct historical trends in life expectancy and life disparity for both the Māori population and the non-Māori population using HMD data. For the historical trends, data by single calendar years was used to provide the most precise picture of time trends since 1948 possible.

Cautiousness for inconsistencies and limitations is crucial when handling and analysing demographic data (Preston, 2001), and, therefore, the known issues and limitations of this dataset are discussed. The first point of attention concerns the estimated population counts throughout the years. The estimates of the population that the HMD uses are derived from Statistics New Zealand (Demography Division - Statistics New Zealand, (1948-2013). Statistics New Zealand measured the population estimates differently before and after 1991. Up until 1991, the *de facto* population was measured meaning that all individuals that were present in New Zealand were included. From 1991 onwards, the *de jure* population was the new norm, which means that New Zealand residents only are included (Alho & Spencer, 2005; Wilmoth, 2004), although New Zealand residents who happen to be overseas are not included in the dataset, except for those serving overseas (e.g. peacekeepers) (Government of New Zealand – Ministry of Health, 2019a). Since the HMD uses Statistics New Zealand data, this measuring difference is directly reflected in the HMD dataset used for the historical trends in life expectancy and life disparity. On a national level, such a change in definition does not reflect major changes. However, it does have substantial implications when considering geographical areas, due to a high level of non-resident population in certain places (e.g. universities, military stations, tourist-dense areas, etc.) (Perz, 2004). Since this study is set to measure mortality patterns on a national level, the change in definition can be disregarded.

Next to a difference in inclusion, the moment of estimating the exposure was different. Before 1991, the average population of a year was used (so the average of the population estimation of December 31st of the previous year and December 31st of the applicable year), whereas, from 1991 onwards, the population residing in New Zealand on June 30th counted as the population estimate (Jasilionis et al., 2017). Taking the midyear population as approximate for the average population in a particular year is an often encountered method in studies on population dynamics (McGehee, 2004), and is therefore assumed to not cause any limitations to this study.

A prominent limitation concerns the ethnicity qualification in the 1980s and 1990s, resulting in a numerator-denominator bias and, consequently, underestimated Māori mortality. In these decennia, the measured ethnicity on the death registration form was different from the complement question in the census. Whereas the ethnicity qualification required a percentage of (non)-Indigenous blood on the death registration form, the census was based on the cultural affiliation of the individual. Also, ethnicity was mainly measured for Māori and Pacific deaths, leading the deaths with missing ethnicity data to be

automatically classified as non-Māori. Obviously, a call for alignment of the concepts came into being. This was enhanced by the restriction in the old definition to two possible ethnicities: 1) Māori or Pacific Islander and 2) non-Māori and non-Pacific Islander. Information on the ethnic descent of the parents of the deceased person led to a classification of the deceased. Since information on the descent of the parents was often unknown, and since only two ethnicities were recognized, this method led to a large group of non-Māori/non-Pacific. The definition of ethnicity in the death registration was finally modified in September 1995. The death registration form changed to include five ethnicities: Māori, Pacific, European, Asian, and Other. To which ethnicity the deceased belongs is decided upon in collaboration with the family of the deceased person. This adjustment led to an increase in people identifying as Māori and/or Pacific. With the number of deaths for Māori at a lower level due to the 'old' definition and Māori population at a higher level due to the 'new' definition, death rates were distorted. In the 1980s and early 1990s, Māori and Pacific mortality was thus seriously undercounted. This leads to an invalid high life expectancy for these years. The HMD-researchers have tried to resolve this problem, but an unexpected increase in Māori deaths in 1996 is still visible. In the analysis of the trend in life expectancy, these years should thus be interpreted with caution. In 2006, the ethnicity MELAA (Middle Eastern, Latin American, and African) was added as ethnicity, but this does not affect this research since it only leads to a shift within the non-Māori population group. (Ajwani et al., 2003; Jasilionis et al., 2017; Jasilionis & Jdanov, 2017; Statistics New Zealand, n.d.)

One more data limitation is concerned with incomplete registration and needs to be taken into account when interpreting the results of the analyses using the HMD dataset. Due to incomplete registration, the Māori data is considered unreliable until 1947. The HMD, therefore, publishes Māori and thus total population data from 1948 onwards. 1948 is thus the starting point of the analysis concerning the life expectancy trend. However, there are suspicions that Māori data might not be complete until the beginning of the 1960s (Jasilionis & Jdanov, 2017). In 1961, the separate system for registering Māori vital events was abolished and from then on, all events were registered in one system (Hutching, 2007). Until 1961, mortality rates at old ages appear "*implausibly low*" (Jasilionis et al., 2017, p. 8). This presumed undercount of Māori deaths might lead to imperfect Māori data as well as total population data. The life expectancy trend might thus be distorted (i.e. underestimation of deaths and overestimation of life expectancy) for Māori from 1948-1961, but the extent and seriousness of this discrepancy are unknown.

3.3.2 Ministry of Health dataset

The second dataset used in this study is the 2016 cause-specific mortality dataset released by the Ministry of Health of the Government of New Zealand (New Zealand Government – Ministry of Health, 2019a). The information on the number of deaths is derived from vital statistics. New Zealand's civil registration and vital statistics (CRVS) can be regarded as of good quality, as indicated by Mikkelsen et al. (2015). Mikkelsen et al. (2015) showed that New Zealand consistently scored around 93 out of 100 points for six measures (e.g. completeness, internal consistency, level of cause-specific detail) indicating the quality of the CRVS.

The New Zealand mortality collection uses the tenth revision of the International Classification of Diseases (ICD-10; see also **3.3.3**) since the 1st of July, 1999. Some small updates occur every couple of years, and the exact scheme used for the 2016 dataset is the ICD-10, 8th edition (New Zealand Government – Ministry of Health, 2019d). To ensure that the classification is compatible with Australian practice, Australia set up an extended version of the ICD-10 called the ICD-10-AM (i.e. Australian Modification) (Independent Hospital Pricing Authority, 2019). This Australian modification is also suited for New Zealand clinical practice and is, therefore, adopted in New Zealand as well. When a person has deceased, New Zealand nurse practitioners or medical practitioners have to fill out the Medical Certificate of Cause of Death, which is compatible with the ICD-10. This certificate informs on the direct cause of death, antecedent causes leading to death, and the underlying cause of death. These forms have to be submitted to the Ministry of Health since they are the body handling the cause of death data (New Zealand Government – Ministry of Health, 2019c). In order to pair deaths to causes of death, the death registrations from vital statistics are matched with the information on the submitted death certificates (New Zealand Government – Ministry of Health, 2019b). The cause of death forms are

manually checked before they are uploaded to the database (New Zealand Government – Ministry of Health, 2011).

The used dataset provides mortality information by age, sex, cause of death, and ethnicity. There is also information on regions, but this is irrelevant for the scope of this research. Age is provided in 5-year age intervals up to 85 with the first 5 years of life given in single-year intervals. For information on causes of death, see **3.3.3**. Considering ethnicity, the data shows death counts for the total population, the Māori population, the Pacific population, the Asian population, and the non-Māori population (New Zealand Government– Ministry of Health, 2019a). The non-Māori population includes all population groups that are not Māori and is thus equal to the total population minus the Māori population. The non-Māori population group consists mainly of individuals identifying as European (75-80%), followed by individuals identifying as Asian (10-15%) and Pacific Islander (5-10%) (New Zealand Government – Ministry of Social Development, 2016). Ethnicity is decided upon by the funeral director in consultation with a member of the deceased's family (New Zealand Government – Ministry of Health, 2019b).

The dataset is part of the Mortality Collection of the Ministry of Health. It includes all deaths registered in 2016 in New Zealand. This means that foreigners that have died while in New Zealand are included but overseas deaths of New Zealand residents are excluded, with the exception of those serving New Zealand while oversea (New Zealand Government– Ministry of Health, 2019a). In addition, the dataset includes registered deaths in 2016 and not the actual 'acts of dying' in 2016, which can lead to misplacement of an event in time: a person who was registered as deceased in the first days of January 2016, could have actually died in the last days of December of 2015, and vice versa (New Zealand Government - Ministry of Health, 2019b). The extent of this type of error is unknown, but since the consistency of measuring in this manner leads to the occurrence of this error for every year, the consequences of this error are negligible.

The dataset presents the underlying cause of death. The Ministry of Health of the New Zealand Government (2016, p.6) defines the underlying cause of death as "*the train of events leading to the death*". Especially in the older age categories, multi-morbidity is often present (Rosenberg, 1999). Therefore, in many cases, there might be more than one cause that contributes to eventual death. For this reason, Redelings et al. (2006) advise presenting both the underlying cause of death and multiple causes of death statistics whenever possible. However, data on multiple causes of death are not accessible for New Zealand and can thus not be included in this study. Due to the absence of multiple causes of death data, some causes of death might be underreported. The analysis by Redelings et al. (2006) shows that more than 90% of deaths due to malignant neoplasms, 99% due to intentional injuries and 73% of accidents and unintentional injuries are correctly explained by the underlying cause of death data only. For these causes, the attribution of causes of death to this category will be adequate. For causes such as diabetes mellitus, cerebrovascular diseases and respiratory diseases some caution is advised when interpreting an analysis that made use of underlying cause of death data only since these might be underreported (Redelings et al., 2006).

Not all registered deaths of 2016 are included in the dataset. For 141 deaths, it was not yet clear which cause of death was assigned by the coroner (New Zealand Government – Ministry of Health, 2019a). 21 of these had no known cause of death and were, therefore, uniformly distributed among the different causes by the publishers. In causes with little death counts, this might have disproportionally distorted the numbers, leading to a higher contribution of these causes than occurred in reality. In later publications of the dataset, the 120 deaths might be included, but in the dataset used in this study, they were ignored.

3.3.3 Typology of causes

The International Classification of Diseases (ICD) is a coding scheme used to classify underlying causes of death for individuals. It is used as the "*foundation for the identification of health trends and statistics globally, and the international standard for reporting diseases and health conditions*" (World Health Organization (WHO), 2019a, 'ICD purpose and uses'). The 2016 New Zealand mortality database uses the 10th revision of the ICD, which is adopted in over half of the world's countries to classify mortality. The ICD-10 is used in thousands of academic studies and has proven its validity (see e.g.: Hodge et al., 2017; Lauridsen et al., 2015). The ICD-10 classification contains over 14,000 codes (WHO, 2019b)

which can make it confusing. The New Zealand mortality dataset, therefore, presents data in multiple formats:

- Three-character classifications, which provide the most specific causes of death;
- Subgroups, which group several three-character classifications into a more general cause; and
- Chapter headings, which group several subgroups into one overarching cause.

Table 2. Leading causes of death in New Zealand, 2010-2012.

	Males	Females
Māori	Ischaemic Heart Disease	Lung cancer
	Lung cancer	Ischaemic Heart Disease
	Suicide	Chronic obstructive pulmonary disease
	Diabetes	Cerebrovascular disease
	Motor vehicle accidents	Diabetes
Non-Māori	Ischaemic Heart Disease	Ischaemic Heart Disease
	Suicide	Breast cancer
	Lung cancer	Cerebrovascular
	Cerebrovascular disease	Lung cancer
	Motor vehicle accidents	Colorectal cancer

Source: Own table based on New Zealand Government – Ministry of Health (2018a).

For this research, a dedicated typology is realized based on the ICD-10 chapter headings, subgroups, and leading causes of death of New Zealanders in 2010-2012. These leading causes of death, indicated by population group and sex are shown in Table 2. Taking the leading causes of death into account for classifying deaths is often performed in the literature (see e.g. Lariscy et al. 2015; Nau & Firebaugh 2012). Considering this information and the numbers in the dataset, 11 groups were identified (see Appendix **I**). After running the analyses, some causes appeared to have rather low significance in the results as can be seen in the results of these analyses in Appendix **I**. Therefore, these causes were grouped with other causes or transferred to the overarching *Other causes*. The ex-post typology, therefore, consists of seven causes of death and is summarized in Table 3.

Cancers is chosen because it is one of the largest contributors to mortality for Māori and non-Māori and both males and females in the dataset. *Lung cancer* and *Diabetes* were chosen because of the appearance in the list of 2010-2012 leading causes of death (New Zealand Government – Ministry of Health, 2018a). *Diseases of the circulatory system* is chosen because it is a relatively large contributor to mortality. This is also the case for *Diseases of the respiratory system* (New Zealand Government – Ministry of Health, 2019a). Transport accidents was initially grouped separately due to the appearance in the leading causes of death in 2010-2012, but was added to *External causes* since they were of greater significance together. The last category, *Other causes*, contains all causes of death that are not mentioned in the other groups. An overview of this typology, along with the corresponding ICD-10 codes and numbers and percentages of deaths are stated in Table 3.

Table 3: Ex-post typology.

Short name	**Cause of death**	**Chapter**	**Subgroup**	**Number of deaths** (total/male/female)						
				Percentage of deaths (total/male/female)						
				Māori				**non-Māori**		
	TOTAL			3465	1870	1595		27919	14023	13896
Cancers	All cancers except lung cancer	C Malignant neoplasms	C00-C33 + C35-C96	724	365	359		6907	3645	3261
				20.9	19.5	22.5		24.7	26	23.5
Lung cancer	Lung cancer	C Malignant neoplasms	C34	343	154	189		1414	786	629
				9.9	8.2	11.8		5.1	5.6	4.5
Diabetes	Impaired glucose regulation and diabetes mellitus	E Endocrine, nutritional and metabolic disorders	E09-E14	188	112	76		655	335	320
				5.4	6	4.8		2.3	2.4	2.3
Circulatory	Diseases of the circulatory system	I Diseases of the circulatory system	I00-I99	1013	584	429		8792	4339	4453
				29.2	31.2	26.9		31.5	31	32
Respiratory	Diseases of the respiratory system	J Diseases of the respiratory system	J00-J98	302	139	163		2601	1269	1332
				8.7	7.4	10.2		9.3	9	9.6
External	External causes of morbidity and mortality	U – Y External causes of morbidity and mortality	U50-Y98	362	250	112		1587	1002	585
				10.4	13.4	7		5.7	7.2	4.2
Other	Other causes			533	266	267		5963	2647	3316
				15.4	14.2	16.7		21.4	18.8	23.9

Source: Own table based on New Zealand Government – Ministry of Health, 2019a.

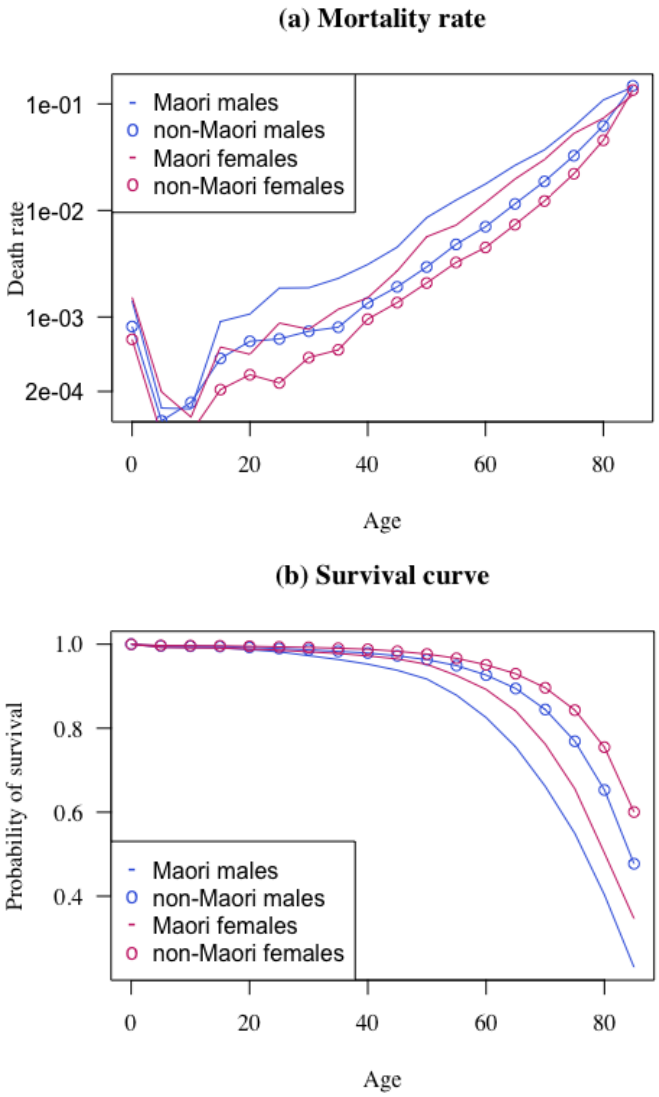
4. Results

To investigate the general mortality patterns of both the Māori and non-Māori population groups, a set of graphs was composed using information from the life table (Figure 3). Figure 3a shows the mortality rate for both population groups and both sexes. The most favourable line, i.e. the line indicating most deaths concentrated at the oldest ages and least deaths in the younger ages, is the line for non-Māori females, followed by non-Māori males. Nevertheless, in the oldest ages, Māori females seem to be doing rather well, with the line intersecting both non-Māori lines just after age 80. When taking a closer look (see Appendix **II**), the numbers seem to confirm this mortality crossover in the older ages. The crossover exists for both sexes, although it is less pronounced for males. Even though the last age group of Māori is doing rather well, Figure 3a reveals that Māori, compared to non-Māori, are, on average, dying earlier.

This same pattern can be seen in Figure 3b. The survival curves of both non-Māori males and non-Māori females show a more rectangular shape compared to their Māori counterparts. This higher level of compression of deaths at older ages for non-Māori indicates a higher lifespan variability for the Māori population compared to the non-Māori population.

Thus, when studying these curves, the non-Māori appear to have a more advantaged mortality pattern over Māori, suggesting an inequality in the mortality patterns as was expected. The rest of this chapter shows the quantification of these differences.

Figure 3: Two life table functions, 2016.



Source: own figure based on New Zealand Government – Ministry of Health, 2019a.

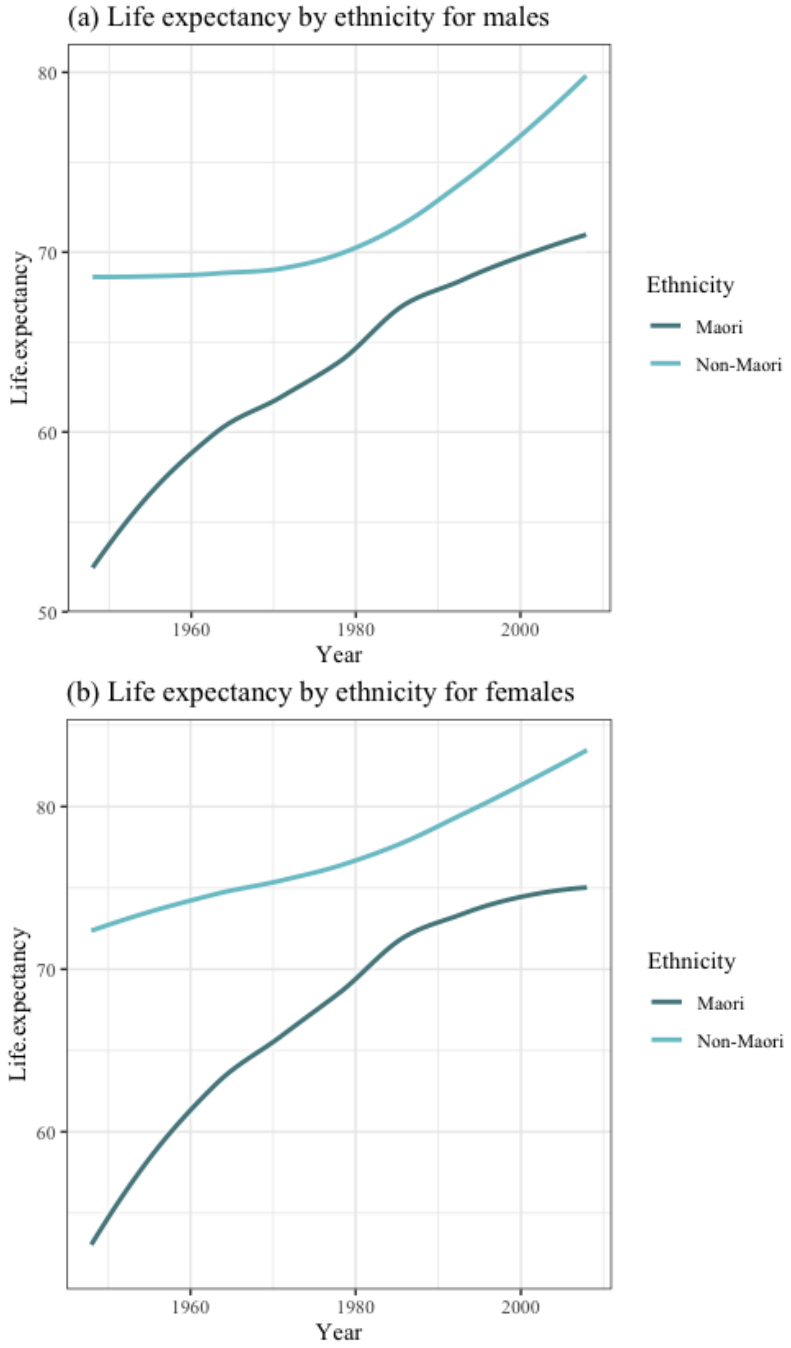
4.1 Life expectancy

4.1.1 Life expectancy trends

Figure 4 shows the trend in life expectancy for both population groups for the 1948-2008 period. Figure 4a presents the life expectancy trend for males and shows that the gap between the two population groups was as large as 16.14 years in 1948. Over the years, this gap narrowed to 3.01 years in 1987. This narrowing was mainly due to a stagnation in the increase of life expectancy for non-Māori males, which plateaued around age 69 from the early 1950s until the late 1970s. In the meantime, the life expectancy for Māori males kept increasing, leading to a narrow gap in the mid-1980s. For non-Māori males, life

expectancy started rising again from 1977 onwards. Life expectancy for Māori in the 1980s and early 1990s should be interpreted with caution due to limitations in the ethnicity qualification (**see 3.3.1**). The observed hump in Māori life expectancy in the mid-1980s is thus the result of a numerator-denominator bias. When the definition was equalised in both systems, this caused a drop in Māori life expectancy from 69.96 in 1995 to 66.38 in 1996, leading to an increase in the gap of 4.29 years in just one year time. This shift in life expectancy for Māori and the sudden increase of the gap should be interpreted with caution, since it is a matter of definition, and is, therefore, not likely to reflect actual changes. From 1996 onwards, data is considered trustworthy and a divergence of the life expectancies can be observed, caused by the slower progress in Māori males compared to non-Māori males.

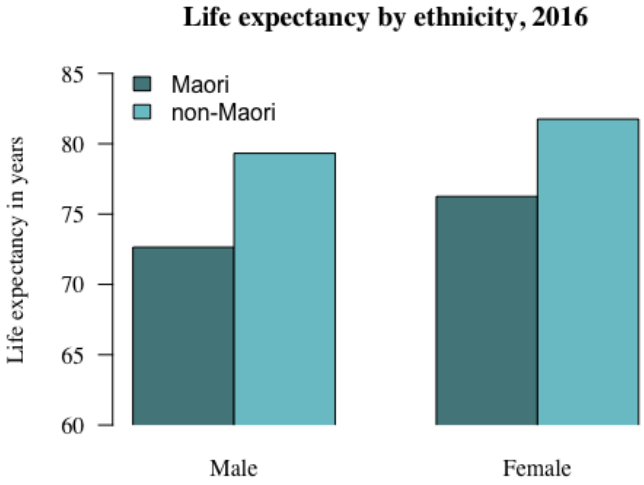
Figure 4: Life expectancy trends, 1948-2008.



Source: own figure based on Demography Division - Statistics New Zealand (1948-2013).

For females, this divergence can also be seen from 1996 onwards. The increase of non-Māori life expectancy occurs more rapidly compared to the increase of Māori life expectancy, leading to this divergence. Just as is the case for males, the definition-bias leads to a hump in Māori female life expectancy in the mid-1980s. One difference can be observed when comparing both non-Māori trends: whereas the stagnation of life expectancy in the 1950s, 1960s and partly 1970s was visible for males, their female counterpart does not seem to have experienced this stagnation. Aside from this difference, life expectancy trends for both Māori and non-Māori behave rather similarly for males and females. However, the life expectancy gap is of different magnitude. Whereas the gap was 16.14 years for males in 1948, the gap in the same year was 19.15 for females, with the female gap reaching its maximum of 21.98 years in 1949. In 2008, the gap for males was 6.54 years, whereas the gap for females was 7.42 years. The difference between the gaps of both sexes thus seems to be decreasing, and when considering the 2016 data (see **4.1.2**), the gap for females has dropped below that of males.

Figure 5: Life expectancy levels, 2016.



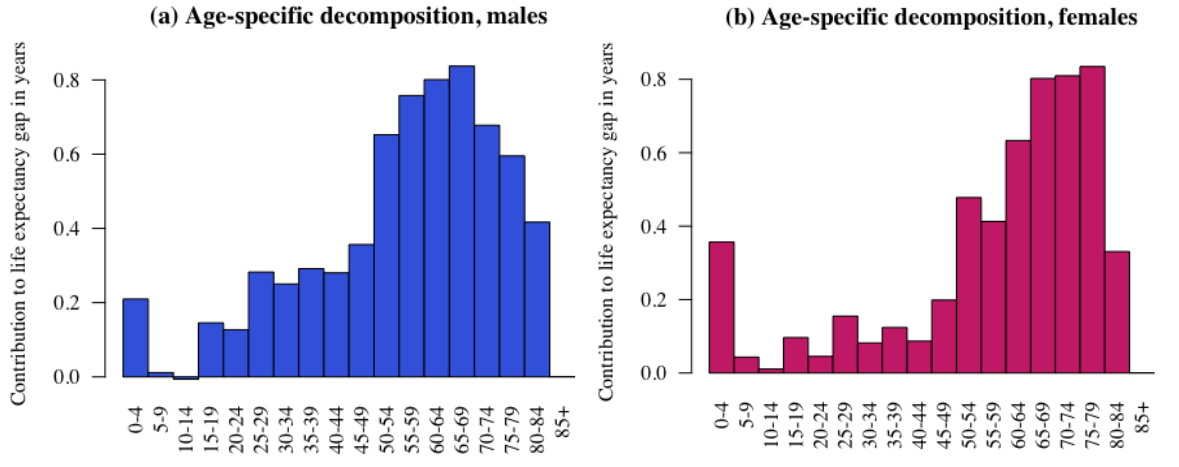
Source: own figure based on New Zealand Government – Ministry of Health, 2019a.

In 2016, the gap in life expectancy amounts to 6.68 years for males (e^0 for Māori is 72.64 compared to 79.32 for non-Māori) and 5.50 years for females (e^0 for Māori is 76.25 compared to 81.75 for non-Māori) according to official statistics (New Zealand Government – Ministry of Health, 2019a) (see Figure 5).

4.1.2 Decomposition by age groups

The contributions of each age group to these gaps are shown in Figure 6. For males, the 65-69 age group contributed most to the gap, whereas the ages from 10 to 14 had a small negative value, indicating that Māori males from 10-14 have a little advantage compared to non-Māori males of the same age. For females, the same 10-14 year age group contributed least to the gap, and the 75-79 age group was responsible for the largest contribution. A relative large contribution can be observed for the age group 0-4 for both sexes. Also, when comparing the graph for males and the graph for females, the young adult/working-class ages from approximately 20-49 years seem to have a rather large contribution for males, whereas this is less pronounced for women.

Figure 6: Age-specific decomposition of the life expectancy gaps.



Source: own figure based on New Zealand Government – Ministry of Health, 2019a.

4.1.3 Decomposition by causes of death

The gaps in life expectancy can also be decomposed by causes of death. The results of this decomposition can be seen in Table 4. For males, over one-third of the life expectancy gap can be ascribed to circulatory diseases. For females, this is over 28%. Another major contributor is cancers: more than 1.5 years of the life expectancy gap for males can be explained by cancers (including lung cancer). For females, this is even 1.8 years or 32.79%. It is well-studied that, compared to young females, young males generally die more often because of external causes, especially due to accidents, suicide, and homicide (Kruger & Nesse, 2004). However, for Māori males, this seems to be worse than for non-Māori males, with 17.16% of the gap being explained by external causes. Even for Māori women, external causes shorten the life expectancy by half a year. Two other relatively large contributors are diabetes, especially for males, and respiratory diseases, especially for females.

Table 4: Cause-specific decomposition of the life expectancy gaps.

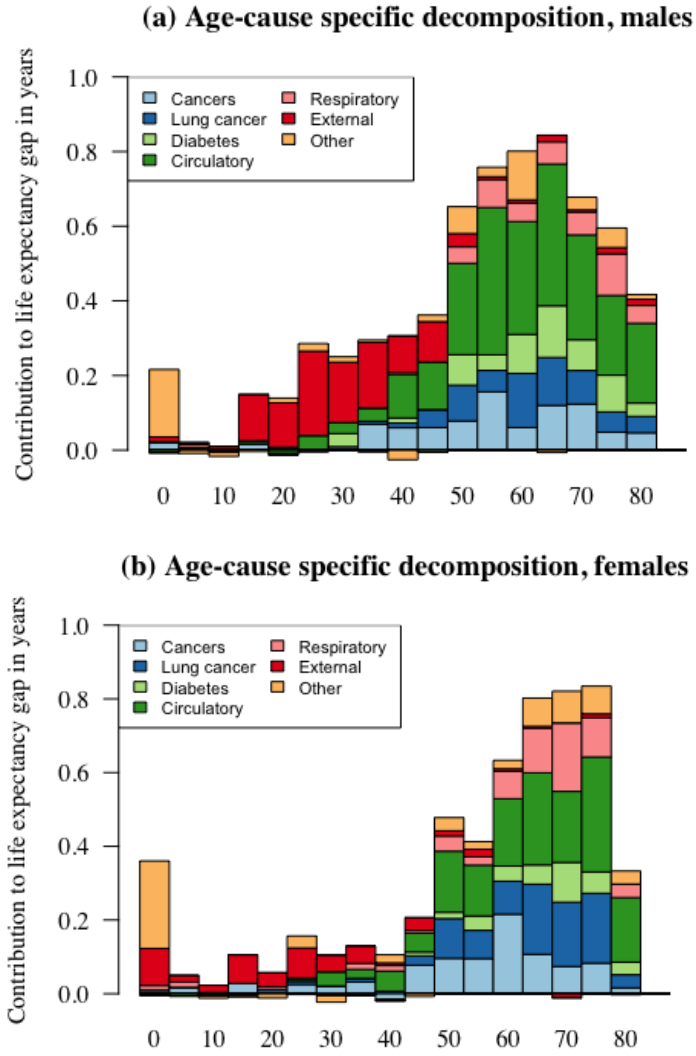
	Males			Females	
	Years	Percentage		Years	Percentage
Cancers	0.89	13.29		0.88	16.04
Lung cancer	0.68	10.22		0.92	16.75
Diabetes	0.62	9.34		0.35	6.41
Circulatory	2.37	35.40		1.58	28.68
Respiratory	0.45	6.76		0.66	11.96
External	1.15	17.16		0.51	9.34
Other	0.52	7.84		0.60	10.82

Source: own table based on New Zealand Government – Ministry of Health, 2019a.

4.1.4 Decomposition by age groups and causes of death

The results of the decomposition for both age groups and causes of death can be seen in Figure 7. Figure 7a shows the result for males, with the contributions of each cause displayed for each age-group. What catches the eye is the change that can be observed from age 50 onwards. The contribution of external causes diminishes significantly, whereas circulatory diseases now appear to be a rather large contributor. For females (Figure 7b), the contributing causes seem to mainly lump together in the ages from 50 to 80. In males, this pattern is less pronounced due to the contribution of the external causes in the ages 15-49, which explain much less of the gap in females. Circulatory diseases and cancers, including lung cancer, are relatively large contributors to the Māori female life expectancy disadvantage. What stands out in both the male and female figure is the high level of other causes in the 0-4 age group. Most causes in the typology are lifestyle diseases, which take time to develop. Therefore, it makes sense that these causes do not apply to the very young ages and that the gap in this age group is mostly explained by causes that belong to the group of other causes.

Figure 7: Age- and cause-specific decomposition of the life expectancy gaps.



Source: own graphs based on New Zealand Government – Ministry of Health, 2019a.

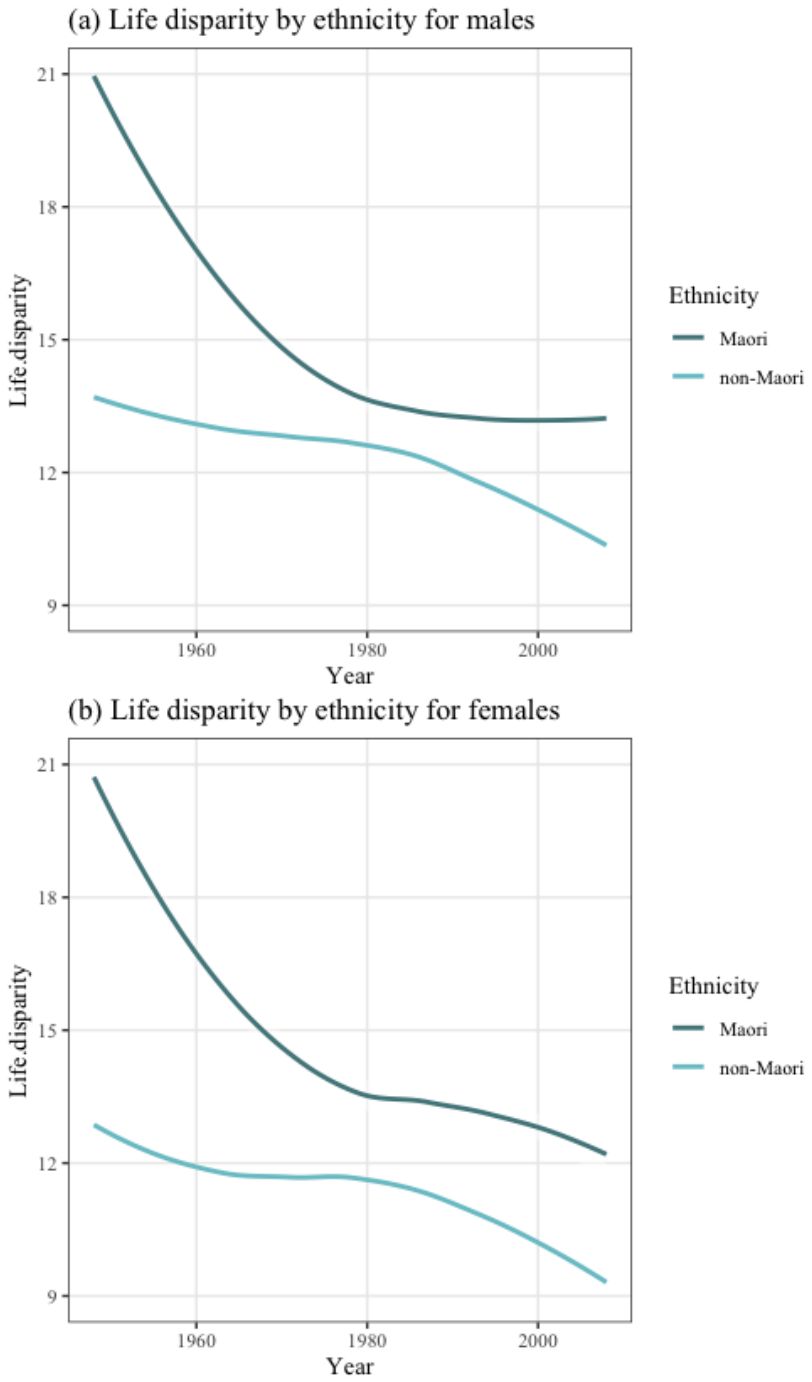
4.2 Lifespan variability

4.2.1 Life disparity trend

Lifespan variability was measured using the life disparity metric (e†). Figure 8 displays the trend in life disparity from 1948 to 2008. Prodigious progress was made in decreasing the lifespan variability until the 1980s for both Māori males and Māori females. From the early 1980s onwards, this progress seems to have stagnated. Conversely, the moderate progress for the non-Māori population does not seem to diminish after the 1980s and still goes on until at least 2008. Especially the life disparity for Māori males seems to have plateaued in the last decennia, whereas life disparity for women is still slowly progressing. The gap that exists between the population groups has narrowed from 7.1 and 7.7 years for males and females respectively in 1948 to 2.4 and 2.9 years in 2008. Or, in other words, whereas life disparity was 51.9% (males) and 58.0% (females) higher for Māori in 1948, in 2008 this was 22.7% (males) and 31.3% (females). Figure 8 shows a pattern that can partly be linked to the life expectancy trend: whereas the early years of the trends of both measures show tremendous improvement for the Māori population and

a slight improvement for the non-Māori population, the most recent decennia behave differently. Concerning life expectancy, a slight increase for Māori males is observed and a minor increase that seems to reach a plateau can be observed for Māori females. Concerning life disparity, it is the male Māori population that is stagnating and the female Māori population that shows slight progress. Whereas Māori males are thus increasing their life expectancy, life disparity is lagging behind. This indicates that in the male Māori population people are on average reaching older ages, but averting premature deaths is still a challenge. For Māori females, life expectancy seems to remain relatively stable while lifespan variability is still slightly decreasing. This indicates that averting premature deaths in the female Māori population is showing promising results.

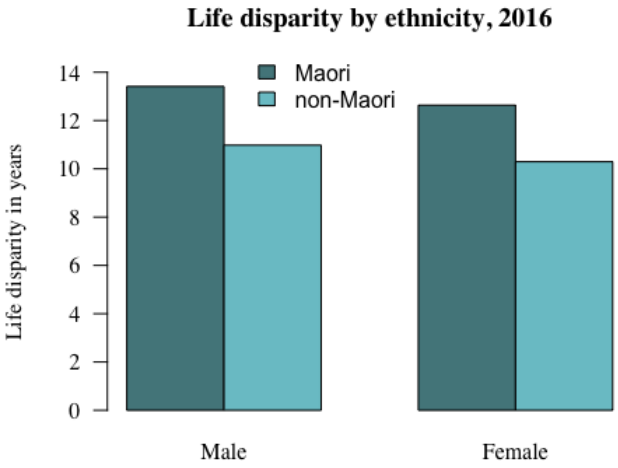
Figure 8: Life disparity trends, 1948-2008.



Source: own figure based on Demography Division - Statistics New Zealand (1948-2013).

Figure 9 shows the level of life disparity for males and females, Māori and non-Māori in 2016. The life disparity was, for both males and females, higher for the Māori population compared to the non-Māori population as was to be expected from the trend visible in Figure 8. The difference in males appeared to be 2.4 years (e† of 13.4 for Māori compared to 11.0 for non-Māori), whereas the difference in females was 2.3 years (e† of 12.6 for Māori compared to 10.3 for non-Māori). Māori males and females thus experience a life disparity that is respectively 21.8% and 22.3% higher compared to the non-Māori males and females

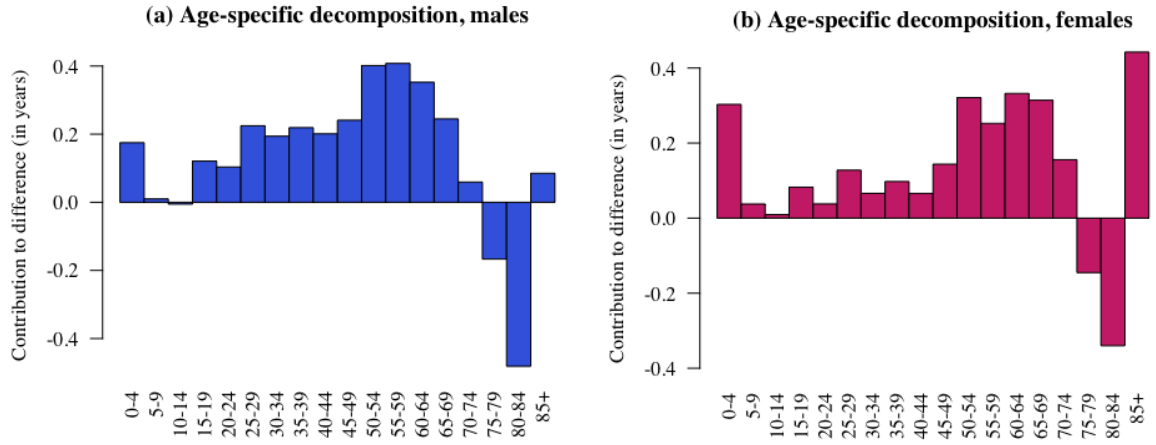
Figure 9: Life disparity levels, 2016.



Source: own figure based on New Zealand Government – Ministry of Health, 2019a.

4.2.2 Decomposition by age groups

Figure 10: Age-specific decomposition of the life disparity gaps.



Source: own figure based on New Zealand Government – Ministry of Health, 2019a.

When decomposing the gap in life disparity by age groups, the graphs for males and females look rather similar (see Figure 10). The threshold ages seem to be around the same moment: around 74 to 75 years of age. The threshold age is important when interpreting the data since a reduction in mortality before the threshold age decreases the lifespan variability, whereas a reduction in mortality after the threshold age increases the lifespan variability. Thus, values above the zero line before the threshold age and values below the zero line after the threshold age increase lifespan variability and values below the zero line before the threshold age and values above the zero line after the threshold age decreases lifespan

variability. Considering this, it appears that, for both sexes, Māori are disadvantaged up until age 84. The 85+ age group, however, behaves in the opposite direction: in this age group, Māori seem to have an advantage over non-Māori, reflecting the mortality crossover as seen in Figure 3a and Appendix **II**. The oldest age group is thus decreasing the lifespan variability of Māori while the other age groups indicate an increase of Māori lifespan variability compared to non-Māori. This effect is larger for females compared to males, which corresponds with chapter **4.2.1**, which stated that averting premature deaths is more of a challenge for the male Māori population compared to the female Māori population.

4.2.3 Decomposition by causes of death

The contributions of each cause of death to life disparity can be observed in Table 5. Some of the same patterns as in the cause-specific decomposition of the life expectancy arise again. However, especially for men, external causes seem to have an even larger effect than is observed in the life expectancy analysis. External causes explain approximately 10 months of the inequality observed between Māori males and non-Māori males. For females, this is over 4 months. For males, circulatory diseases and cancers explain another large part of the gap in life disparity. For females, cancers have the same disadvantaging effect, while other causes seem important as well. Both sexes display one negative cause, which should be interpreted as an advantage for Māori over non-Māori. However, these numbers are rather small and could be caused by the randomness of small numbers in the dataset.

Table 5: Cause-specific decomposition of the life disparity gaps.

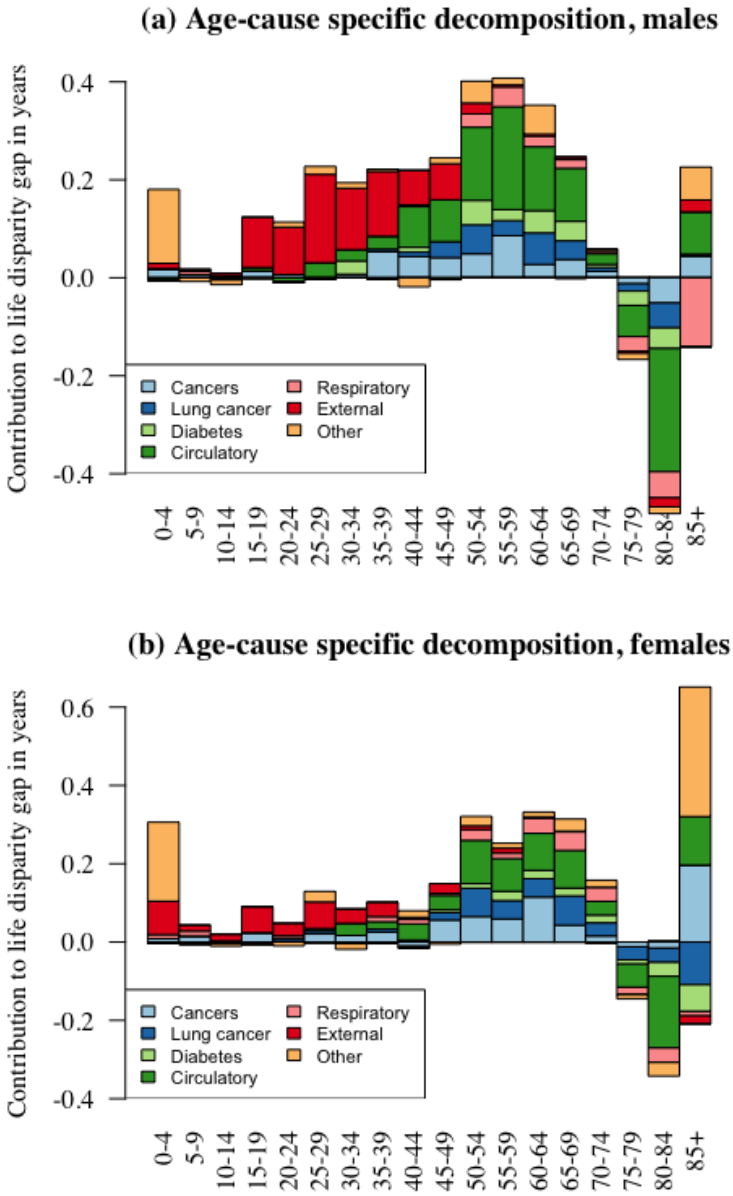
	Males			Females	
	Years	Percentage		Years	Percentage
Cancers	0.38	16.11		0.62	26.99
Lung cancer	0.18	7.69		0.14	6.05
Diabetes	0.13	5.34		-0.02	-0.70
Circulatory	0.63	26.41		0.42	18.35
Respiratory	-0.11	-4.53		0.15	6.69
External	0.84	35.23		0.39	17.09
Other	0.33	13.74		0.59	25.54

Source: own table based on New Zealand Government – Ministry of Health, 2019a.

4.2.4 Decomposition by age groups and causes of death

As expected from paragraphs **4.2.2** and **4.2.3**, for males, external causes of death play an important role in the ages from 15-49, whereas circulatory diseases explain most of the disadvantage from age 50 and onwards (see Figure 11a). A disadvantage due to respiratory diseases in the ages above 85 is cancelled out by advantages due to multiple other causes. For women (Figure 11b), the causes appear less pronounced, although cancers and circulatory diseases both seem to increase in importance from age 45 onwards. Interesting is the fact that these same causes of death cause an advantage of Māori females over non-Māori females in the oldest age group. However, the largest part of this old-age mortality crossover is explained by other causes of death.

Figure 11: Age- and cause-specific decomposition of the life disparity gaps.



Source: own graphs based on New Zealand Government – Ministry of Health, 2019a.

5. Discussion

5.1 Reflection on the research results

The findings of this study can be summarized into five main themes that characterize the mortality patterns and differences therein for New Zealand Māori and New Zealand non-Māori in relation to existing literature:

- **Relationship life expectancy and lifespan variability**: A negative relationship between life expectancy and lifespan variability is generally observed in previous research (see chapter **3.2.1**). This relationship is mostly confirmed in this study: Māori experience a lower life expectancy while having a higher lifespan variability compared to non-Māori. This indicates a '*double burden of inequality*' (Van Raalte et al., 2018, p.1003) in mortality for the Māori population. The positive side of this relationship is that life expectancy and lifespan variability often develop hand in hand, and progress can thus be achieved simultaneously. Or, in the words of Vaupel and colleagues (2011, p.1), *"Greater longevity and greater equality of individual's lifespans are not incompatible goals"*. When investigating the trends in life expectancy and life disparity from 1948-2008, Māori females seem to comply with this pattern, whereas, for Māori males, life expectancy is increasing while life disparity has in the more recent decennia been stagnating. Vaupel et al. (2011) distinguished between premature (i.e. deaths before the threshold age) and late-deaths and their impact on life expectancy and lifespan variability and found that gains were mainly made because of averting premature deaths. The combination of the trends in life expectancy and life disparity has shown that averting premature deaths is still a challenge in the male Māori population, whereas the female Māori population is improving in both life expectancy and life disparity and are thus right on track in averting premature deaths. Aburto and colleagues (2020, p.1) concluded that "*Saving lives at ages below life expectancy is the key to increasing both life expectancy and lifespan equality"*. A focus on averting Māori premature deaths, and especially male Māori premature deaths, can thus lead to gains in both life expectancy and lifespan variability. Averting Māori premature deaths would require developments in multiple areas including but not limited to:
    - Reductions of racism in the health care system leading Māori to visit a medical practitioner earlier so morbidity can be prevented or halted in an early stage;
    - Improvements in educational attainment for Māori, which will cause an increase in their socioeconomic status and, following the social gradient in health, will improve their health and postpone mortality;
    - Encouragement of healthy behaviours and, more importantly, discouragement of unhealthy behaviours (see also the following theme).
- **Smoking rates Māori females**: Chapter **2.2.1** mentions the high smoking rates for Māori, and especially Māori females. A substantial part of the mortality gap between Māori and non-Māori can be explained by lung cancer and respiratory diseases (17% of the life expectancy gap for males and 28.7% for females), indicating the negative consequences of the high smoking rates. To decrease the impact of smoking, smoking should be discouraged so smoking rates will eventually go down.
- **External causes of death Māori males**: The analysis found, in line with research into other Indigenous populations, an excess level of external causes of death for Māori, especially for males. This corresponds with the high levels of suicide and assault that are mentioned in chapter **2.1.3** of this research. Deaths due to external causes are mainly premature deaths since they most often occur before the threshold age (see also the age- and cause-specific decompositions). In line with recommendations by Lariscy et al. (2016), a focus on these causes of death is necessary when improvements in life expectancy *and* lifespan variability are to be made.
- **Late-onset diseases**: When decomposing the gaps by age groups, the age groups from 50 to 79 appear to contribute most to the gaps. The excess late-onset diseases that Māori experience seem to confirm the longitudinal link between social factors and health as mentioned in chapter **2.2.2**: when an individual experiences lower social status in youth this has an adverse effect on later-

life health outcomes. Of course, this effect is, among others, strengthened by lower socioeconomic status in the adult years and by behavioural factors.

- **Mortality crossover**: The mortality rates and the findings of the decomposition of the lifespan variability measure indicate a mortality crossover in the oldest ages. This leads to an inversion of the generally observed pattern: whereas in younger ages Māori are disadvantaged over non-Māori, the oldest aged Māori have an advantage over the oldest aged non-Māori. This corresponds with findings of studies into ethnic mortality differentials in the United States.

5.2 Synthesis and future research

Chapter **2** shows that interventions on health behaviours only will not solve health inequalities: there should also be a focus on improving Māori socioeconomic status and psychosocial status. This will be a tough challenge since this requires actions on how people are treated, awareness of the country's past, and getting all population groups to achieve high educational levels – to name just a few. The fact that this requires an effort for almost all in society makes this task an arduous and demanding one. However, when the goal is to eradicate or, at a minimum, decrease these health differences and to achieve social justice, efforts on only one front appear not to be sufficient. When taking a step back, the observation of general mortality patterns teaches us that health and mortality outcomes are not a simple mathematical formula. It is not just the high smoking rates of Māori that lead them to have a disadvantaged mortality pattern over non-Māori. It is also not just the lower socioeconomic status that causes the differences. While this research obviously did not research causality leading to the observed outcomes, it seems very unlikely that the observed patterns, age groups and causes of death designate just one behaviour or social factor as disadvantageous for Māori mortality outcomes. The observed differences between the population groups are most likely due to behaviour, socioeconomic status *and* psychosocial factors. This indicates there is not just one key to decreasing mortality differences: efforts and attention need to be directed towards all these fields if the Māori health outcomes are to be enhanced.

The findings of this study lead one to think that there is still a long way ahead. And while this is true, the improvements that have been accomplished already (see for example chapter **4.1.1**) should not be forgotten. Hans Rosling (2019, p.74) describes this as "*distinguishing between a level and a change of direction*". While the health and mortality gaps indicate a disadvantaged situation for Māori, the change of direction indicates that things are improving for this population group. Narrowing the life expectancy gap of a population group by as much as 12 years in a couple of decades is progress worth celebrating. To be able to reflect on this sort of numbers in the future, it is important to keep track of health and mortality data. While this will undoubtedly be done for life expectancy, my advice would be to focus on lifespan variability too. Providing a trend in (decomposed) lifespan variabilities might over time teach us what improvements are achieved and in what areas improvement is lacking behind. This measure can deepen our understanding of the observed mortality patterns and might thus contribute to the noble job of achieving health equity.

To better understand the finding of a mortality crossover in this study, future research could focus on whether this finding is caused by selective survival or by data issues. When selective survival appears to be the case, the mortality crossover for the Māori population would confirm itself as a real phenomenon, which might provide another opening for future research on learning from this part of the population group that is doing particularly well. When the mortality crossover appears to be caused by data issues and the phenomenon is thus artificial, this is an indication that improvement is possible in the corresponding classification of ethnicity in the census and on the death-certificate.

Another recommendation concerning future research would involve qualitative research on the way health and mortality are viewed by Māori. While this research uses quantifiable methods to research health and mortality, it also indicated that health and mortality are not inherently the same for Māori and non-Māori. The non-Indigenous meaning of health is often the absence of physical or mental disease and is measured in prevalence and incidence rates, and eventually in mortality rates. For the Indigenous population, however, health is more than the absence of physical or mental disease. Emotional and spiritual health is also evaluated when considering one's overall health in an Indigenous context. When only researching health and mortality from a non-Indigenous point of view, only one side of the coin is highlighted. Researching health and mortality differentials from the Māori viewpoint might reveal their

thoughts and feelings about measuring and experiencing health and mortality and might generate helpful insights in changing the health care system to be most beneficial for all population groups.

While the previous recommendations indicate future research, this study brought up possible additions to the New Zealand health policy too. The current He Korowai Oranga – New Zealand's Māori Health Strategy – is the public health policy aimed at reducing health gaps between Māori and non-Māori. The New Zealand Government acknowledges Māori health is at a disadvantage when compared to non-Māori health, and He Korowai Oranga states four pathways for action to close this gap (New Zealand Government – Ministry of Health, 2015b). The pathways seem to focus on getting more Māori in the health workforce and improving access to health services for Māori by providing tailored services. All in all, the knowledge of the situation and the realisation that Māori health needs to be improved are present and progress is also already made. However, the focus seems to lie only in improving the situation immediately. While this is, by all means, advantageous, more improvements can still be made in preventing disease in the future. This includes encouraging healthy behaviours and increasing Māori socioeconomic status. While this is a broad statement that takes time to achieve, focussing on education might be a start. Increased knowledge has been proven to lead to thoughtful decision-making considering behaviour. When getting educational attainment for Māori and non-Māori at a similar level, occupation and income will likely follow. The social gradient in health will still be visible, but more likely within the total population instead of between population groups. So, while keeping up the good work that is done already, a focus on broader factors influencing health might strengthen the health of Māori in the future.

6. Conclusion

This research focussed on health and mortality of the New Zealand Māori and New Zealand non-Māori. More specifically, the extent and reasons for differences in the mortality patterns of both population groups were addressed. In this final chapter, the research questions as formulated in chapter **1.3** will be answered. The sub-questions will be answered before addressing the main research question.

1: How have New Zealand Māori and New Zealand non-Māori life expectancy levels evolved in the past?
Figure 4 shows the trend in life expectancy for Māori and non-Māori between 1948 and 2008. The trend starts in 1948 since there is relatively reliable data starting from this year onwards. The figure shows that life expectancy for both Māori and non-Māori have increased in the years from 1948-2008. The gap that exists between the population groups has narrowed from 16.1 and 19.1 years for males and females respectively in 1948 to 6.5 and 7.4 years in 2008. For males up until 1980, this is mainly due to the stagnation of life expectancy increase for non-Māori. From 1980 onwards and for all years for females, the main reason for the decrease of the gap seems a larger increase for Māori life expectancy compared to non-Māori. The life expectancy gap appears to be smaller for both sexes around the 1980s and 1990s, but the data for these years is distorted due to issues with the definition of ethnicity in the census and on death certificates and can, therefore, not be considered trustworthy.

2. How much does the life expectancy of New Zealand Māori and New Zealand non-Māori differ? What are the contributions of different causes of death and age groups to this difference?
In 2016, the gap in life expectancy amounts to 6.7 years for males (e^0 for Māori is 72.6 compared to 79.3 for non-Māori) and 5.5 years for females (e^0 for Māori is 76.2 compared to 81.7 for non-Māori). For males, this gap is largely explained by circulatory diseases (35.4%), cancers (13.3%) (when including lung cancer 23.5%), and external causes of death (17.2%). Together, these causes thus explain 76.1% of the gap in life expectancy between Māori and non-Māori males. For females, the gap is largely explained by cancers (16.0%) (when including lung cancer 32.8%), circulatory diseases (28.7%), and respiratory diseases (12.0%). Together, these causes thus explain 73.5% of the gap in life expectancy between Māori and non-Māori females. For both sexes, the ages from 50-79 contribute most to the gap. This reflects the findings concerning causes of death since these are diseases that take time to develop and thus present later in life. For males, the ages of 15-49 contribute more to the gap compared to the same ages in females. This reflects the finding that external causes have a large contribution to the gap in males while this is lower for females.

3. How have New Zealand Māori and New Zealand non-Māori lifespan variability levels evolved in the past?
Figure 8 shows the trend in life disparity for Māori and non-Māori between 1948 and 2008. The figure shows that life disparity for both male and female Māori has improved substantially from 1948 until the early 1980s. From then on, moderate progress is made for Māori females, while life disparity for Māori males seems to stagnate. For both non-Māori sexes, a relatively stable slight increase is the case for the entire period. The gap that exists between the population groups has narrowed from 7.1 and 7.7 years for males and females respectively in 1948 to 2.4 and 2.9 years in 2008. Or, in other words, whereas life disparity was 51.9% (males) and 58.0% (females) higher for Māori in 1948, in 2008 this was 22.7% (males) and 31.3% (females).

4. How do the New Zealand Māori and New Zealand non-Māori differ in the variability of their lifespans? What are causes and age groups that contribute to this variability in lifespan?
As the life disparity levels from 1948-2008 have already shown, the variability in lifespan is higher for Māori compared to non-Māori. Life disparity is, in 2016, 21.8% higher for Māori males and 22.3% higher for Māori females. This means that Māori mortality suffers from a larger burden of premature deaths, i.e. deaths happening at younger ages. For males, the causes of death that contribute most to this gap are the same as found for the life expectancy gap, but the magnitude is different: 35.2% external causes of death, 26.4% circulatory diseases, and 23.8% cancers (when including lung cancer). For females, the causes that contributed most to the gap in lifespan variability are cancers (including lung

cancer) (33.0%), other causes of death (25.5%), and circulatory diseases (18.3%). Concerning age groups, the same pattern is found as in the life expectancy analysis. One feature the life expectancy analysis did not show is the mortality crossover in the oldest age category (85+) for both males and females, indicating that the oldest aged Māori are better off compared to the oldest aged non-Māori.

5. Which aspects of public health policies should be targeted to decrease the health gap between the New Zealand Māori and New Zealand non-Māori?

Concerning lifespan variability, the decomposition by age shows that a large part of the gap is due to premature deaths (i.e. deaths before the threshold age). A focus on averting Māori premature deaths can increase both life expectancy and lifespan variability. Existing theory and this research agree that public health policies should not just target one cause of death or one particular health behaviour. Underlying causes that do not have a direct link with health such as lower socioeconomic status and racial discrimination are likely to cause the inequalities for a substantial part. Therefore, these underlying causes of health and mortality should not be left unattended when trying to improve health outcomes and decrease mortality gaps. Nevertheless, a focus on the promotion of healthy behaviours might also help to reduce the gaps when this leads to lower smoking rates in the Māori population.

Main research question: To what extent and why do the New Zealand Māori and New Zealand non-Māori differ in their mortality patterns?

New Zealand Māori have a less advantaged mortality pattern when compared to New Zealand non-Māori. When considering the trends in life expectancy and life disparity, substantial improvements have been made from 1948 onwards. However, whereas Māori males are still slowly increasing their life expectancy, life disparity is lagging behind. This indicates that in the male Māori population people are on average reaching older ages, but averting premature deaths is still a challenge. For Māori females, life expectancy seems to remain relatively stable while life disparity is still slightly decreasing. This indicates that averting premature deaths in the female Māori population is showing promising results. In 2016, life expectancy is 6.7 years lower for Māori males and 5.5 years lower for Māori females compared to their non-Māori counterparts. For both sexes, lifespan variability is over 20% higher for Māori. The ages from 50-79 mainly explain the gap which corresponds with the large contribution of diseases that take time to develop and thus appear later in life such as circulatory diseases and cancers. External causes in the young-adult ages seem to explain a substantial part of both the life expectancy and life disparity gap in males. Overall, Māori have a disadvantaged mortality pattern, except for the oldest age groups, where Māori seem to have an advantage compared to non-Māori. Further research should address whether this is due to selective survival or due to data issues.

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8. Appendices

Appendix I: Ex-ante typology and results of the respective analysis

Table 6: Ex-ante typology.

Short name	**Cause of death**	**Chapter**	**Subgroup**	**Number of deaths** (total/male/female)						
				Percentage of deaths (total/male/female)						
				Māori				**Non-Māori**		
	TOTAL			3465	1870	1595		27919	14023	13896
Cancers	All cancers except lung cancer	C Malignant neoplasms	C00-C33 + C35-C96	724	365	359		6907	3645	3261
				20.9	19.5	22.5		24.7	26.0	23.5
Lung c.	Lung cancer	C Malignant neoplasms	C34	343	154	189		1414	786	629
				9.9	8.2	11.8		5.1	5.6	4.5
Endocr.	All endocrine, nutritional and metabolic disorders except diabetes	E Endocrine, nutritional and metabolic disorders	E00-E08 + E15-E99	54	30	24		207	89	118
				1.6	1.6	1.5		0.7	0.6	0.8
Diabet.	Impaired glucose regulation and diabetes mellitus	E Endocrine, nutritional and metabolic disorders	E09-E14	188	112	76		655	335	320
				5.4	6.0	4.8		2.3	2.4	2.3
IHD	Ischaemic Heart Disease	I Diseases of the circulatory system	I20-I25	513	335	178		4150	2326	1824
				14.8	17.9	11.2		14.9	16.6	13.1
CBVD	Cerebrovas-cular disease	I Diseases of the circulatory system	I60-I69	166	75	91		2156	837	1319
				4.8	4.0	5.7		7.7	6.0	9.5
Circ.	Other diseases of the circulatory system	I Diseases of the circulatory system	I00-I19 + I26-I59 + I70-I99	334	174	160		2486	1176	1310
				9.6	9.3	10.0		8.9	8.4	9.4
Resp.	Diseases of the respiratory system	J Diseases of the respiratory system	J00-J98	302	139	163		2601	1269	1332
				8.7	7.4	10.2		9.3	9.0	9.6
Ext.	All external causes except transport accidents	U – Y External causes of morbidity and mortality	W00-Y98	271	191	80		1270	765	505
				7.8	10.2	5.0		4.5	5.5	3.6
Transp.	Transport accidents	U - Y External causes of morbidity and mortality	V00-V99	91	59	32		317	237	80
				2.6	3.2	2.0		1.1	1.7	0.6
Other	Other causes			479	236	243		5756	2558	3198
				13.8	12.6	15.2		20.6	18.2	23.0

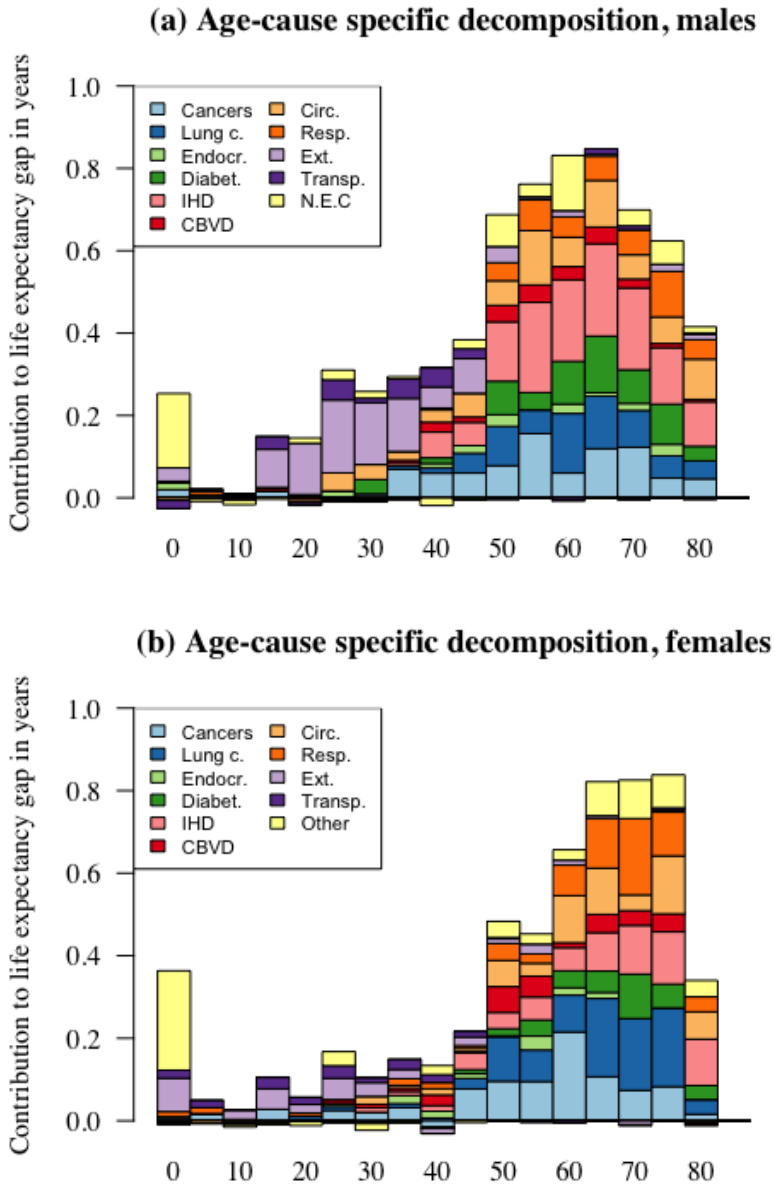
Source: Own table based on New Zealand Government – Ministry of Health, 2019a.

Table 7: Ex-ante cause-specific decomposition of the life expectancy gaps.

	Males			Females	
	Years	Percentage		Years	Percentage
Cancers	0.89	12.88		0.88	15.59
Lung c.	0.68	9.90		0.92	16.27
Endocr.	0.16	2.37		0.11	1.99
Diabet.	0.62	9.04		0.35	6.22
IHD	1.35	19.59		0.67	11.95
CBVD	0.24	3.47		0.30	5.26
Circ.	0.77	11.22		0.60	10.64
Resp.	0.45	6.54		0.66	11.62
Ext.	0.94	13.71		0.34	5.99
Transp.	0.20	2.95		0.17	3.10
Other	0.57	8.34		0.64	11.37

Source: own table based on New Zealand Government – Ministry of Health, 2019a.

Figure 12: Ex-ante age- and cause-specific decomposition of the life expectancy gaps.



Source: own graphs based on New Zealand Government – Ministry of Health, 2019a.

Appendix II: Age-specific mortality rates, 2016

Table 8: Age-specific mortality rates, 2016.

	Males			Females		
	Maori	non-Maori	Difference	Maori	non-Maori	Difference
0-4	0.0014171	0.00081386	**0.00060**	0.00152691	0.00061694	**0.00091**
5-9	0.0001397	0.00010598	**0.00003**	0.00019822	7.734E-05	**0.00012**
10-14	0.00013717	0.00015769	**-0.00002**	0.00011498	8.2576E-05	**0.00003**
15-19	0.0009061	0.00040816	**0.00050**	0.00052204	0.00020813	**0.00031**
20-24	0.00106617	0.00059124	**0.00047**	0.00044686	0.00028702	**0.00016**
25-29	0.001868	0.00062256	**0.00125**	0.00087687	0.00024032	**0.00064**
30-34	0.00188071	0.00073489	**0.00115**	0.0007668	0.00041594	**0.00035**
35-39	0.00230208	0.00080103	**0.00150**	0.00118036	0.00049134	**0.00069**
40-44	0.00310854	0.00135243	**0.00176**	0.00151515	0.00095407	**0.00056**
45-49	0.00451681	0.00191758	**0.00260**	0.00269899	0.00136446	**0.00133**
50-54	0.00855408	0.00294488	**0.00561**	0.00565667	0.00208362	**0.00357**
55-59	0.01249183	0.00479837	**0.00769**	0.00727686	0.00324872	**0.00403**
60-64	0.01771379	0.0070338	**0.01068**	0.0118451	0.00449401	**0.00735**
65-69	0.02648456	0.011514	**0.01497**	0.0196875	0.00734489	**0.01234**
70-74	0.03705104	0.01872318	**0.01833**	0.03001658	0.0122449	**0.01777**
75-79	0.06130952	0.03263177	**0.02868**	0.0527845	0.02206089	**0.03072**
80-84	0.10875	0.06214345	**0.04661**	0.07345133	0.04553491	**0.02792**
85+	0.14390244	0.14783465	**-0.00393**	0.12108844	0.13488929	**-0.01380**

Source: Own table based on New Zealand Government – Ministry of Health, 2019a.