Cohort Morbidity Hypothesis: Health Inequalities of Older Māori and non-Māori in New Zealand

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Abstract

This paper describes the mortality trends from 1948 to 2008 between Māori and non-Māori populations. Using the cohort morbidity hypothesis, we propose that health disparities between the populations can be partially explained by different levels of early-life exposure to infectious diseases. We conducted regression analysis and found strong associations between early- and old-age mortality for cohorts. Childhood mortality, rather than mid-life mortality, accounted for greater variance in older age. The mortality trend of the 1948 Māori birth cohort is similar to the 1902 non-Māori birth cohort 46 years earlier. Implications are discussed.

New Zealand has a uniquely documented history of mortality. Much can be gathered from these records about the inequalities in mortality rates and health disparities especially between the indigenous (Māori) and non-indigenous (non-Māori) populations. These two populations have had very different health histories, and older persons of today in both groups have lived in different epidemiological environments. Throughout the life course of both groups, there have been clear disparities in health outcomes, whether it is differences in obesity rates in childhood (Rush et al., 2012) or disability, cardiovascular disease and cancer incidences in adulthood (Ministry of Health, 2011). These differences are evidenced by the gap in life expectancy. For example, in 2008, life expectancy at birth for Māori was 74 years compared with 81 years for non-Māori (Human Mortality database). It is projected that the proportion of Māori population aged 50 and over will increase by 7.1 per cent compared with 3.3 per cent of the non-Māori population (Ministry of

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Health, 2011). Given the continued disparities in health outcomes, efforts in understanding and improving Māori health status remain important.

Over the past few centuries, the causes of death in almost all countries have shifted dramatically from infectious and acute diseases to chronic conditions that are often associated with ageing. This change in causes of mortality is referred to as the epidemiological transition, and it is typically characterised by a reduction in childhood mortality and an increase in life expectancy. In New Zealand, similar to other countries, chronic conditions of older age, such as cardiovascular and respiratory diseases, have replaced infectious diseases as the primary causes of death. However, this transition from acute to chronic disease has not been uniform across the Māori and non-Māori populations, and New Zealand has had two distinct epidemiological transitions: Non-Māori experienced the transition in the 19th century and Māori in the 20th century (Pool, 2013a). We intend to show that the difference in the timing of the transitions can partly explain the health disparities that influence the differences in mortality rates between the two populations.

Using Finch and Crimmins’ (2004) cohort morbidity hypothesis in describing the linkage between early and late-life mortality, we hypothesise that the difference in older-age mortality rates between Māori and non-Māori is related to the historical context in which they were born. In particular, Māori had higher infant mortality associated with infectious diseases than did non-Māori, who had relatively lower infant mortality and lower exposure to infectious conditions. In this paper, we intend to demonstrate how the higher mortality and slower decline in older Māori mortality reflects their historical past, while the current trend in non-Māori mortality reflects current trends.

**Background**

Life expectancy in many countries has increased over the last 250 years; however, this increase has not been equally distributed across all populations. For example, life expectancy of Māori has increased at a slower rate than that for non-Māori, and the gap in life expectancy between these two populations has widened due to the slower decline in cardiovascular mortality rates among Māori (Ajwani, Blakely, Robson, & Bonne, 2003). Despite improvements in accessibility to health services,
disparities in health status between Māori and non-Māori continue to exist, especially among the older population, resulting in poorer health status and higher mortality (Ministry of Health, 2011). Disparities for indigenous populations are also well documented in other countries including Australia, Canada and the United States (Paisano, Cobb, & Espey, 2003; Ring & Brown, 2002).

Mortality decline among older Māori over recent decades is likely to reflect their epidemiological history. The cohort morbidity hypothesis has been used to explain the similarity in historical decline in cohort mortality rates across young and old ages within a cohort before the introduction of vaccinations and improvement in public health, sanitation and nutrition. Specifically, the cohort morbidity hypothesis explains that the increase in life expectancy at older ages is attributed, in part, to the lifetime reduction in chronic exposure to infectious disease and inflammation (Finch & Crimmins, 2004). Thus, survivors of birth cohorts who experienced lower early-life mortality will also experience lower mortality rates in older years (Crimmins & Finch, 2006; Kermack, McKendrick, & McKinlay, 1934).

The origins of the hypothesis date back to as early as the 1930s when Kermack, McKendrick and McKinlay (1934) noticed that each successive cohort of improvement in childhood mortality in England and Sweden was accompanied by lower adult mortality. They proposed that both maternal health and early childhood environment were major determinants of late-life health morbidity. Jones (1956) further developed the hypothesis explaining that mortality curves of birth cohorts follow similar changes at younger and older ages. Finch and Crimmins (2004) examined historical mortality data and proposed that the reduction in lifetime exposure to infectious conditions is partially responsible for the increase in life expectancy at older ages over the past 250 years. In particular, using available mortality data on birth cohorts for Sweden (1751–1899), France (1806–1899), England (1841–1899) and Switzerland (1871–1899), they found that mortality before age 15 years accounted for substantial variability in old-age mortality after 70 years of age: 87 per cent in Sweden, 94 per cent in France, and 96 per cent in England and Switzerland (Crimmins & Finch, 2006).

Childhood exposure to an infectious environment has been strongly associated with adverse health outcomes and mortality in later ages. In
18th century Sweden, exposure to infection during infancy was correlated with mortality among survivors of the cohorts at older age (Bengtsson & Lindström, 2000). This is because exposure to early-life infection can lead to heightened inflammatory responses that promote the process of atherogenesis and accelerate the development of cardiovascular disease and ageing (Finch & Crimmins, 2004). The mechanisms that link early-life infection and late-life morbidity occur through the development of cardiovascular and respiratory disease, cancer and diabetes. In examining links between younger and older persons, infant mortality rates are often used as a proxy for exposure to infection among the survivors.²

The cohort morbidity hypothesis has been useful in explaining morbidity and mortality rates in historical cohorts; however, due to the long-term reduction in mortality at young ages, this hypothesis has seldom been applied to contemporary cohorts. Although the infant mortality rate has declined significantly in contemporary societies, including both Māori and non-Māori populations in New Zealand, the history of Māori is one of relatively high mortality for recent cohorts of older persons.

Finch and Crimmins (2004) proposed that inflammation during early life has led directly to morbidity and mortality from heart disease. This proposal has been supported by research linking inflammatory proteins such as C-reactive protein (CRP) with the increase risk for heart attack, stroke and cancer (Danesh, Collins, Appleby, & Peto, 1998; Willerson & Ridker, 2004). Even in contemporary society, cardiovascular disease is considered one of the leading causes of death in the United States and around the world (Centers for Disease Control and Prevention, 2010; World Health Organization, 2008). Although mortality from heart disease is decreasing, the slower decline in cardiovascular death among Māori compared to non-Māori is a major public health concern (Ajwani et al., 2003). For example, at age 50 and older, Māori have reported higher incidences of health conditions and chronic diseases, including cardiovascular disease, than their counterparts (Ministry of Health, 2011).³

Essentially, the Māori population lags far behind the non-Māori population on all indicators of health and well-being (Ministry of Health, 2011).⁴ The growing disparities in health between the two populations led to the creation of a “Positive Ageing Strategy” by the New Zealand Government in 2001 which continued to address these health inequalities until 2010.
Relative socio-economic disadvantage among Māori has primarily contributed to the disparities in mortality (CSDH, 2008; Ministry of Health, 2011; Sporle, Pearce & Davis, 2002). In fact, it has been posited that socio-economic determinants are more predictive of health disparities among both Māori and non-Māori than is smoking (Blakely, Fawcett, Hunt, & Wilson, 2006). However, even when controlling for socio-economic factors, studies have shown that Māori still continue to have worse physical and mental health outcomes (Blakely et al., 2006; Dulin, Stephens, Alpass, Hall, & Stevenson, 2011).

These findings point to a need to identify additional factors as sources of health inequalities between Māori and non-Māori. Among the suggested additional factors are the effects of colonisation, land confiscation and racism (Blakely et al., 2005). Dulin et al. (2011) found that the distal effects of colonisation not only influence the socio-economic factors but may also have a biological effect. For instance, low socio-economic position is associated with an increase in levels of cortisol, which could negatively affect the health of Māori population (Marmot, 2006). This effect could be aggravated by maladaptive behaviours including smoking and excessive drinking that can further compound poor health outcomes, especially for older Māori adults.

**A history of Māori health change**

Colonisation by European settlers has had a profound effect on Māori society. The arrival of Europeans and subsequent introduction of new diseases during the mid-18th century contributed to the decline in the Māori population. These diseases included measles, mumps, whooping cough, bronchitis and tuberculosis, which affected both adults and children and lead to severe epidemics during this period (Pool, 2013b). After the Treaty of Waitangi was signed in 1840, there was an influx of settlers demanding land. Consequently, through land confiscation following the 1860s wars and Crown purchase, many Māori found themselves displaced and without access to traditional food, a situation that lead to poor diet while living in overcrowded and unsanitary conditions which led to further spread of diseases (Pool, 2013b).

Population recovery began in the early 1900s due to increased immunity to diseases and spread of Māori-led health initiatives. Although the life expectancy of Māori started to rise during the first half of the 20th
century, Māori life expectancy still lagged behind that of the non-Māori due to their vulnerability to severe epidemics. For instance, the Māori death rate in the 1918 influenza pandemic was seven times greater than the non-Māori population’s. Epidemics occurred frequently, claiming the lives of many Māori until the 1940s (Pool, 2013c). Since then, the epidemiological transition of the Māori accelerated with the drop in mortality in all ages from infectious diseases, especially during early life. Although Māori remain disadvantaged socially and economically, the shift from rural communities to urban centres provided better access to health facilities and improved medical technology including specific health and social measures to reduce the incidence of tuberculosis (Pool, 2013c).

Despite these improvements, health disparities between the Māori and non-Māori populations including low birth weight, high prevalence of preterm birth, small size for gestational age, high stillbirth and neonatal deaths are still observed (Barnes et al., 2013; Ministry of Health, 2012, 2013). Studies have shown that key stages of fetal and early-life development, with critical timing and exposures, have long-term health implications (Bengtsson & Lindström, 2000; Davis & Sandman, 2010). To this end, this study hypothesises that the highly infectious environment into which the current older Māori population was born and lived their formative lives can, in part, explain the current health inequalities in trends and levels of older Māori and non-Māori mortality.

**Data Source and Analysis**

New Zealand has information on death registration and census data for both the indigenous and non-indigenous people, thus allowing comparison of both populations. Cohort and period mortality data for Māori and non-Māori are available in the Human Mortality Database for varying years: Māori from 1948 to 2008 and non-Māori from 1901 to 2008. While there may still be some undercount of Māori death registration because the system is somewhat newer compared with non-Māori, the current data represent the most complete mortality data.

We compare trends in childhood and old-age mortality among the Māori and non-Māori populations. While childhood mortality was examined at all ages, the age interval of 50–54 was chosen for older-age mortality for several reasons. First, it allowed us to link older-age
mortality to mortality among Māori cohorts born in 1948 and after. Second, there are higher levels of under-registration and age uncertainties among older Māori, thus limiting the analysis to those under 55 years. Third, due to the lower life expectancy and greater health disparities among Māori compared to non-Māori, ageing studies tend to examine older adults as 50 years and older, rather than the traditional 65 years and older (Ministry of Health, 2011). Lastly, age 50 is the time in life when most chronic conditions, such as cardiovascular disease, cancers and diabetes, begin to have significant effect on morbidity and mortality (Bengtsson & Lindström, 2000).

Because cohort data for Māori began in 1948, we were able to examine cohort mortality in childhood for the cohorts born from 1948–1958 as well as their mortality at age 50–54 in 2008. The average cohort life expectancy of Māori at birth for the 1948–1958 birth cohorts was low, at 56 years compared with 71 years for non-Māori of the same birth cohort. This estimation of life expectancy is partially projected, as the 1948–1958 birth cohorts are not 100 years old yet. In addition, the infant mortality in the 1948–1958 birth cohorts was more than three times higher for Māori than non-Māori. The low life expectancy and high infant mortality among the 1948–1958 Māori birth cohorts imply that these cohorts were exposed to high levels of infection and inflammation earlier in the life course, which may contribute to the increasing health disparities in New Zealand.

**Evidence of cohort mortality between infant and old ages**

The trend and level of mortality at ages 1 and 50 of successive cohorts by year of birth up to 1958 showed an association between infant and older-age mortality for both the Māori and non-Māori populations (Figure 1). The levels of mortality rates vary between the two populations with Māori having higher infant and old-age mortality rates than non-Māori. The cohort mortality at age 50 (m₅₀) followed similar patterns to the infant mortality rate age at 1 (m₀), 49 years earlier, suggesting that infant and old-age mortality for the 1948–1958 cohort are closely associated.
To empirically examine the association between infant and old-age mortality and to test the hypothesis that a highly infectious environment, as evidenced by high infant mortality, is associated with increased risk for mortality in later age, this study replicated Crimmins and Finch’s (2006) study by conducting regression analysis between mortality at older age, \( m_{50-54} \), and mortality in three stages of childhood: infancy (\( m_0 \)), early childhood (\( m_{1-4} \)) and later childhood (\( m_{5-9} \)). We choose these ages because we believe the mortality rate before the age of 10 provides a good index of exposure to inflammation.

Most of the variance over time in Māori old-age mortality is associated with mortality rates before age 10 (\( R^2 = 85 \) per cent). This can be compared to 71 per cent for non-Māori (Table 1). The higher explanation of variance among Māori supports the idea that where mortality is high in childhood, there are strong relationships between infant infections (i.e. \( m_0 \)) and old-age mortality (i.e. \( m_{50-54} \)). The coefficients indicate that only infant mortality is significantly related to cohort mortality at the older ages.
The cohort analysis linked childhood mortality from 1948–1958 and old-age mortality about 50 years later in order to examine the impact of earlier life infection and inflammation on mortality change over time. However, to compare the contemporary period relationships between childhood and old-age mortality and examine the association between contemporary and developmental plasticity linked to early-life infections, the study also replicated Crimmins and Finch’s (2006) study by conducting regression analysis between the annual childhood and old-age mortality at the same calendar year from 1948 to 1958.

The results indicated that the proportion of variance accounted for across periods is lower than the cohort relationships for the Māori population. In particular, the results indicate that contemporary old-age mortality is not predicted by childhood mortality in the same calendar year as is the cohort old-age mortality. In fact, none of the coefficients is significant. For the non-Māori population, the proportion of variance explained in older-age mortality is higher for the period relationships. In addition, the trends in childhood mortality (i.e. m5–9) are negatively related to contemporary trends in old-age mortality. Together, these differences reinforce the importance of the impact of early infectious environment prior to modern medicine on late-age mortality.
To further examine the hypothesis on the strong linkage between childhood and old-age mortality due to infectious environment, the study investigates whether this linkage also exists between childhood (m₀, m₁–₄, m₅–₉) and early adulthood mortality (m₂₅–₂₉). Table 2 shows the relationship between childhood and early adulthood mortality. Since most early adulthood mortality is caused by crime, accidents and violence, and not by the chronic conditions of older age, the cohort mortality trends in childhood and early adulthood are not expected to follow the same patterns. Hence, as Crimmins and Finch (2006) hypothesised, the cohort mortality in early adulthood should be more closely associated with the contemporary childhood conditions.

Table 2 illustrates that the relationships between childhood and early adulthood mortality for both the Māori and non-Māori populations are weaker than when compared with those for old-age mortality in Table 1. In particular, for Māori, the cohort variance explained between childhood and early adulthood is about 59 per cent, whereas for non-Māori the $R^2$ is about 47 per cent. Different patterns emerged for the contemporary period change between childhood and early adulthood mortality in the same calendar year. First, for both the Māori and non-Māori populations, the change is stronger in the contemporary than the

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<td>$N= 11$</td>
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<td><strong>Total Non-Māori (1948–1958)</strong></td>
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<td>Intercept</td>
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cohort ($R^2 = 78$ per cent versus $59$ per cent for Māori, and $47$ per cent versus $63$ per cent for non-Māori). Second, the contemporary association between childhood and early adulthood mortality is generally stronger, especially for Māori, than compared with the cohort association between childhood and old-age mortality (see contemporary $R^2$ in Tables 1 and 2).

**Gompertz Curves for Māori and non-Māori**

Figure 2 illustrates the mortality rates on semi-logarithmic plots for Māori and non-Māori populations for the 1948 through 1958 birth cohorts. The figure demonstrates that the mortality rates displayed a typical Gompertz curve, indicating similar rates of mortality acceleration across the birth cohorts. Yet, despite the similarity in shape of the curves, Figure 2a shows differences across cohorts in morbidity and mortality rates for Māori, reflecting the epidemiological change in mortality rates for the 1948 to 1958 birth cohorts. In contrast, for non-Māori, Figure 2b depicts smaller cohort differences, as evidence by the overlapping of mortality curves over the same time period. The overlaps in the slopes suggest that there is little to no change in mortality during the time period.

**Figure 2a: Māori cohort mortality**

![Māori cohort mortality graph](image-url)
Finally, given the differences in mortality rates between Māori and non-Māori, we show the cohort mortality curves for an earlier non-Māori cohort in order to compare similar levels in cohort mortality rates. Figure 3 illustrates the similarity of Māori 1948 birth cohort mortality and the non-Māori 1902 birth cohort, this similarity indicating that the Māori population is essentially 46 years behind the non-Māori population in mortality change.
Discussion

The disparities in health outcomes and mortality rates among older Māori and non-Māori populations have been the subject of much discussion. While social and economic factors, as well as health behaviours, play an important role in influencing health outcomes, they do not account for the variance in explaining health disparities (Blakely et al., 2006). This study found that mortality at age 50 followed similar patterns of mortality at age 1 for both Māori and non-Māori populations, suggesting a close association between early- and late-life mortality. However, for the non-Māori population, the association is more related to the lack of variability in mortality rates. This is because the non-Māori population has low infant mortality and longer life expectancy reflecting a different epidemiological environment.

This study also provided support that cohort effects, for the current generations of older adults in New Zealand, could partially explain the health disparities between the Māori and the non-Māori populations. In particular, it found a strong association between infant and old-age mortality. This relationship is much stronger for older-age than early-adulthood mortality. The relationship between childhood and early-adulthood mortality is more related to contemporary factors such as violence and accidents than the relationship to older ages, which is more
related to the chronic conditions of older age. This relationship is stronger for Māori compared to the non-Māori population, suggesting the differences in epidemiological transition and the historical effect of colonisation.

Specifically, in all instances, Māori have higher mortality rates, and mortality under the age of 10 years accounted for greater variance in old-age mortality for Māori than for non-Māori (85 per cent compared with 71 per cent). All these findings suggest that survivors of childhood mortality from exposure to infectious diseases and inflammation have a direct linkage to late-life mortality. The differences between the two populations suggest a difference in the level of exposure to infectious environment reflecting differences in epidemiological environment, such that the current mortality rate of Māori from the 1948 birth cohort is similar to the mortality of non-Māori from the 1902 birth cohort.

These findings are consistent with the cohort morbidity hypothesis and the literature that links early-life infection with late-life mortality in a historical context in 18th-century Sweden (Bengtsson & Lindström, 2000). Using the Health and Retirement Survey in the USA, Blackwell, Hayward, and Crimmins (2001) found that, even after controlling for socio-economic status, childhood health has long and enduring health consequences in later life in terms of cancer, cardiovascular and respiratory disease and arthritis. Given the strong association with early- and late-life mortality, Costa (2000) found that the decline in infectious disease during the 20th century accounts for up to 24 per cent of the decline in late-life morbidity and mortality. One mechanism to explain early- and late-life mortality is that survivors of highly infectious environments, as indicated by high infant mortality, carry a high lifetime burden of inflammation (Crimmins & Finch, 2006).

While on the one hand, those who have a rapid acute inflammatory response are able to fight off childhood infection and survive infant mortality, on the other hand, elevated levels of circulating CRP and other inflammatory markers can promote the development of atherosclerosis and cardiovascular disease (Finch & Crimmins, 2004). Hence, what appears to be an adaptive response earlier in the life course can became maladaptive in later life in an evolutionary hypothesis called antagonistic pleiotropy (Rose, 1982). The stronger associations between early- and late-life mortality for Māori compared with non-Māori can also be observed in the
difference in health outcomes. For instance, compared with the older non-Māori population, older Māori adults have higher all-cancer mortality rates and incidences of tuberculosis, heart and respiratory diseases (Ministry of Health, 2011). This suggests the effects of acute inflammatory response, which speeds up the development of chronic conditions and ageing.

The findings of the study are consistent with other studies indicating high levels of inflammatory markers among survivors of contemporary societies that have widespread parasitism in infectious environments. In particular, it has been found that the Tsimane of Bolivia has a significant higher level of CRP at age 35 than Americans at age 55 (Gurven, Kaplan, Winking, Finch, & Crimmins, 2008). Even among the surviving Tsimane children, 13 per cent of them have ≥ 5 mg/litre of serum CRP, indicating higher exposure to pathogens (McDade et al., 2005). The elevated levels of CRP among children exposed to infectious conditions are also found in Kilimanjaro, Tanzania (Wander, Brindle, & O’Connor, 2012) and India (Vikram, Misra, Pandey, Dwivedi, & Luthra, 2004). A prospective study found that the elevated levels of CRP robustly predicted future coronary heart disease and type 2-diabetes (Misra, 2004).

Despite improvements in the overall health status and accessibility of health services for New Zealanders, disparities continue to exist between the Māori and non-Māori populations. These differences in health status and mortality rates are especially pronounced in the older age groups who were exposed to different environments with varying degrees of infectious conditions earlier in the life course. The arrival of Europeans to New Zealand led to a major decline in the Māori population (Pool, 2013b). One of the major reasons for the decline was the Māori population’s lack of immunity from the diseases introduced by the European settlers (Pool, 2013a).

Current generations of older Māori adults (i.e. aged 50+) were born during times of infectious conditions and poor maternal health and nutritional diets. The linkages between an early infectious environment as well as poor maternal health on health consequences have long been established (see Barker, 1995; Bengtsson & Lindström, 2000; Mazumder, Almond, Park, Crimmins, & Finch, 2009). Even in contemporary times, infant mortality rate, sudden infant death syndrome and low birth weight are higher among Māori than non-Māori (Ministry of Health, 2012, 2013).
All these suggest that the effect of adverse environments on early life will continue to persist throughout the life course and will further contribute to health disparities in New Zealand.

Finch and Crimmins (2004) argue that both inflammatory infection and nutrition are complementary in linking the cohort morbidity hypothesis between early- and late-life mortality. This is because infection and nutrition are intimately related since it is possible that well-fed babies can also be vulnerable to infection triggering inflammatory response as well as impairing nutrient absorption causing malnutrition (Barker, 1995; Barker, in Kannisto, Christensen, & Vaupel, 1997; Barker, Gluckman, Godfrey, Harding, Owens, & Robinson, 1993). Although both infection and nutrition contribute to late-life mortality, Bengtsson and Lindström (2000) found that nutrition among infants has a weaker influence on late-life mortality than the level of infection. Despite many ongoing discussions, the causal paths between inflammation and chronic conditions, such as cardiovascular disease, have not been fully resolved since inflammatory markers, such as CRP and interleukin-6, can be both causes and outcomes to the development of chronic conditions (see Crimmins & Finch (2006) for a more in-depth explanation on the directional problems between inflammation and chronic diseases).

While this study found stronger linkage between early-life infections and late-life mortality for Māori, it does not claim that this linkage is the only mechanism to explain the health differences. Lifestyle choices and behaviours, such as smoking, and socio-economic factors, as well as public health policies, all contribute to the health of Māori (Blakely, Fawcett, Hunt, & Wilson, 2006; Blakely et al., 2006; Dulin, Stephens, Alpass, Hall, & Stevenson, 2011). Furthermore, as has already been noted, the non-Māori population is not a homogeneous population. It is constituted of several ethnic groups and is heavily influenced by immigration.

Advances in medical development and improved accessibility to health services have been vital to the decline in mortality across all populations in New Zealand. In fact, almost all older Māori adults currently have access to health practitioners regularly and are equally as likely as non-Māori to have a health practitioner (Ministry of Health, 2007). With the introduction of infant immunisation and antibiotics in New Zealand, infant mortality declined, thus reducing the lifetime exposure to
inflammation and an increase in life expectancy for successive cohorts. Over the past decade, improvements have been made to increase childhood immunisation, particularly for Māori. For example, full immunisation coverage for two-year-old Māori children increased from 69 per cent in 2005 to 78 per cent in 2010. Further progress in access of public health services across the lifespan could close the health disparity gaps between Māori and non-Māori in the future.

**Conclusion**

The differences in cohort mortality trends between Māori and non-Māori populations suggest different rates of exposure to past infectious conditions. New Zealand has undergone two major epidemiological transitions in the reduction of childhood mortality and increase in life expectancy. The non-Māori experienced this transition early in the 20th century while Māori experienced it much later. This difference in timing of the epidemiological transition relates to the accessibility to medical interventions and public health services contributing to the lifetime reduction in inflammation and consequently changes to the mortality rates. Furthermore, the differences in the timing of the epidemiological transition partly explain recent health disparities between the older age groups among the indigenous and non-indigenous populations.

In addressing the current and future health of Māori, two general approaches are needed. First, improving accessibility to health services while being sensitive to the cultural, traditional and spiritual needs will enhance quality of life by delaying the onset and promoting management of chronic conditions of old age. Towards this goal, a revisit of actions on New Zealand’s *Positive Ageing Strategy* (2001 to 2010) to increase the number of quality services provided by and for Māori that are culturally appropriate is of paramount importance. The findings of the study imply that improved public health conditions and medical interventions, especially for children, have immediate influence on infant mortality and long-term benefits that may deter early onset of chronic conditions. Hence, given the strong linkages of prenatal and early-life health with later health, the second approach should concurrently focus on investing in maternal health and early childhood intervention. Such investments are crucial to help change the trajectories and health trends of the future Māori population. In doing so, these approaches will ensure that Māori are
able to live longer, healthier lives and to fulfil their potential to participate in New Zealand society.

Notes

1 We note that the non-Māori population is not a closed homogeneous population but one that has changed markedly over time and is heavily influenced by immigration.

2 In Norway, Forsdahl (1977) found that high infant mortality is strongly associated with arteriosclerotic deaths among the survivors 40 to 69 years later for the 1896–1925 cohorts. Moreover, Buck and Simpson (1982) found that infant mortality from diarrhoea and enteritis among the 1917–1921 birth cohorts in the USA is significantly associated with respiratory cancer in men and arteriosclerosis heart disease for both men and women in later life.

3 In 2006 the life expectancy for the Māori population at age 50 was six years less than the non-Māori population. In particular, the mortality rate from ischemic heart disease was almost six times higher for Māori women than non-Māori women. Furthermore, for Māori men, ischemic heart disease mortality rate was twice as high as that for non-Māori men. Similarly, infectious disease rates such as tuberculosis remain higher for the Māori than the non-Māori populations across age groups and gender (Ministry of Health, 2011).

4 This includes cancer survival rates such as colorectal, breast, lung and cervical cancer (Jeffreys et al., 2005).

5 The Human Mortality Database was created to provide detailed mortality and population data to researchers, students, journalists, policy analysts and others interested in the history of human longevity. The project began as an outgrowth of earlier projects in the Department of Demography at the University of California, Berkeley, USA, and at the Max Planck Institute for Demographic Research in Rostock, Germany. See www.mortality.org.

6 In New Zealand, a person is classified as Māori if any one of their recorded ethnicities is Māori (Ministry of Health, 2011).

7 While the examination of the levels of inflammatory markers for Māori and its connection with chronic condition is beyond the scope of this study, there is substantial evidence on the influence of inflammation on major chronic conditions in older age. In addition to CRP, some of the inflammatory markers include interleukin-6, tumor necrosis factor-α, and fibrinogen. Typically, the acute inflammatory response is activated during an infection, trauma or internal tissue injury, which triggers the complement system and increases the uptake of low-density lipoprotein by macrophages that further accelerate atherosclerosis (Finch & Crimmins, 2004).
References


