Height, weight and mortality in the past
New evidence from a late nineteenth century New Zealand cohort.

Evan Roberts (author for correspondence)
Department of History and Minnesota Population Center
University of Minnesota

Kris Inwood
Departments of History and Economics
University of Guelph

Les Oxley
Department of Economics
University of Canterbury

eroberts@umn.edu

Acknowledgements: This research was carried out with funding from the Health Research Council and Marsden Fund of New Zealand. We thank the Registrar of Births, Deaths and Marriages for facilitating our research by providing access to confidential birth and death registers, and Archives New Zealand for access to World War I personnel files. Nicholas Radburn and Jackie Lindley provided research assistance.

The Victoria University of Wellington Human Ethics Committee granted ethical permission for this research.

Abstract

The positive correlation of mortality risk with early and mid-life BMI is well-documented for modern populations but largely unknown for earlier cohorts when BMI was generally lower and medical knowledge more limited (Costa 1993; Murray 1997). This paper examines birth, World War I service and death records for 2500 men born in New Zealand between 1870 and 1897. Stratification by occupation and ethnicity reveals mortality was greater for indigenous Maori, and lower for men farming prior to WWI enlistment. We find an "L-shaped" relationship rather than the "U-shaped" relationship between BMI and mortality risk identified in other studies. There is no evidence of an effect from low BMI but the relative risk of mortality rises steeply for men whose BMI in early adulthood was 27 or greater. This finding is robust to alternate specifications of premature mortality. We find weaker evidence of mortality risk varying by height (which proxies for early life conditions).

Introduction

The anthropometric history literature is at least implicitly, and often explicitly, longitudinal. Since the mid-1980s, following the pioneering studies of Waaler (Waaler, 1984), anthropometric historians have endeavored to not only measure changes in height and body mass over cohorts, but also how body composition affects health in later life, often proxied by mortality or life expectancy. Height itself is not the object of interest, but merely a well-understood proxy. Because height is a summary measure of nutritional conditions in the first two decades of life, studies of the relationship between height and health use the former to proxy for early life conditions. Research on the relationship between height and later life health revolve around the influence of environmental conditions on later health that are potentially exogenous to individual choices. Babies have no influence over their conditions in the womb, and children have very limited influence over factors that affect their net nutrition and attained height. Conversely, adult body weight may be appropriately thought of as a rational choice dependent on preferences, technology and relative prices (Lakdawalla & Philipson, 2009; Lumey et al., 2010 forthcoming; Philipson & Posner, 2003). Beyond a certain point increases in body mass are undesirable for humans, bringing an increased risk of morbidity, particularly cardiovascular conditions, and consequently increased risk of mortality (Gillman, 2004). Although as yet relatively few historical articles address this question, the first results suggest that the relationship between body mass and mortality has changed somewhat between nineteenth century and modern populations. In this paper, we bring new historical evidence to bear

on the question of body composition and health in later life, with a sample of World War I enlistees from New Zealand linked to their vital records.

Prior studies of the relationship between height, weight and mortality

Determining if the relationship between body composition and health has changed is critical to using nutritional measures of living standards over time. Studies of living standards using anthropometric measures interpret increases in average height and body mass as indicative of higher real wages (due to lower food costs), lower disease incidence, or reduced physical intensity of work. In the absence of other changes to welfare these improvements to net nutrition can be interpreted as improvements to living standards. However, if the relationship between body composition and health changes, then changes in body composition over time cannot be interpreted as pure improvements in living standards.

To make the issue more concrete, consider the hypothesis that improvements in medical technology might lower the mortality risks of obesity. There are two separate, but not mutually exclusive, ways in which the obesity-mortality relationship might have changed. First, the obesity-mortality function could shift, giving a lower mortality risk at any given BMI. Second, the shape of the obesity-health function could change, lowering the mortality risk of an increase in BMI. We emphasize that these are hypotheses about possible changes, rather than established results, which we discuss later. If the obesity-health relationship has shifted, then the implicit price of obesity has changed. People are able to have their cake and eat it too. Conversely, an unchanging

relationship between obesity and health across cohorts and countries is powerful evidence that the obesity-health relationship approximates a biological constant. If the obesity-health relationship is stable across time and place, we can compare changes in the population distribution of body composition among diverse populations, and forecast the implications of changing body composition. Conversely, if the obesity-health relationship has shifted substantially over just a century there is evidence that the obesity-health relationship is mediated by social and economic factors. Thus a key question for historical longitudinal studies in anthropometric history is whether the height–health and body mass–health relationship has changed substantially over time.

Weight and height direct our attention at different points in human life as critical in determining morbidity and mortality. While studies of height and health in later life address the relationship of early-life experience and later-life health, studies of weight and health seek to address the question of how behavior in adulthood—closer to the end of the lifespan—affect morbidity and mortality. The two perspectives are not opposed. Morbidity and mortality risk can be affected by both growth period experiences, and adult experiences and behavior. Yet the emphasis on early-life or adult experience as the primary cause of disease and mortality has shifted over the twentieth century (Kuh and Smith, 1993). Prior to World War II researchers from a variety of disciplines emphasized the early-life antecedents of adult disease and mortality. However, in the 1950s and 1960s there was a much greater influence on adult behavior—such as poor nutrition manifesting in overweight—as a primary cause of morbidity (Kuh and Ben-Shlomo, 2004). Interest in early life influences—often summarized by height in historical studies—resumed in the 1970s. The current

epidemiological consensus is that morbidity and mortality risk are determined over the life span. Moreover, the relative contributions of early life and adult experiences and behavior are likely to differ between individuals and between morbidities. The data requirements to answer this form of question are high. Conclusions are thus necessarily partial and inclusive—both early and later life experiences affect morbidity and mortality.

Height and morbidity

A significant amount of modern research on the association between height and morbidity has focused on the relationship between height and coronary heart disease. Across the population height has been shown to be inversely associated with the development of cardiovascular diseases. In other words, shorter people have a higher risk of developing cardiovascular diseases. Similarly, there is an inverse association between height and diabetes, with shorter men having a higher prevalence of diabetes, and greater levels of insulin resistance. Modern researchers who have been able to collect data on the components of height have found a stronger correlation between leg length and adult cardiovascular disease than total height. This correlation is significant because more of environmental variation in height comes through variation in leg length than in torso length. That is, people who are shorter than their genetic potential due to deprivation in childhood tend to be shorter in the legs than they could have been (Elford and Ben-Shlomo, 2004; Lawlor, Ben-Shlomo et al., 2004). The conclusion from modern research is that the inverse relationship between height and cardiovascular disease is most likely due to cardiovascular disease being a long-term consequence of conditions in childhood that are also reflected in height. Height has also been shown to be inversely related to stroke,

respiratory disease, and stomach cancers, and that the association is due to early life conditions manifesting in both reduced stature and disease (Davey Smith and Lynch, 2004). However, height is not associated with the development of hypertension (Whincup, Cook et al., 2004). The emerging and important literature examining long-term consequences of famine for children will add further evidence (Lumey, Stein et al., 2010 forthcoming). Already it seems clear that chronic malnutrition or repeated famine has much more serious impact than a single episode of food deprivation.

Height and mortality

The relationship between height and mortality addresses indirectly the question of whether conditions in early life—reflected in height—also affect survival in the long run. The mechanisms for how early life conditions affect mortality and life expectancy are unclear in much of this research, opening up further questions rather than resolving issues.

Waaler's pioneering research with 1.7 million Norwegians demonstrated that height was associated with increased longevity in late-adult men, and that the effects were substantial (Waaler, 1984). Among men aged 55-59 at initial screening and followed for 17 years, the mortality of men standing 185-189cm was half that of men 150-55cm. However, while the relative risk for shorter men appeared dramatic, the population at risk was very small. Less than eight percent of the population studied had an excess mortality risk of more than 50%. A subsequent follow-up of the same population for another 17 years to the year 2000, maintained Waaler's basic results. Men shorter than 165cm had an

elevated mortality risk, but above that height there was no increase in the risk of death over 25-34 years of follow-up (Engeland, Bjorge et al., 2003). British researchers have found similar results in smaller samples (Davey Smith, Hart et al., 2000, Batty, Shipley et al., 2006; Batty, Shipley et al., 2009). Recent papers using Asian and Australasian data from prospective medical studies found that a 6cm (1 standard deviation) increase in height was associated with a 3% decline in mortality risk. This finding is significant because of the extension to non-European ethnic groups. No significant differences were found between the Asian and Australasian populations, suggesting this relationship is robust across different ethnic groups (Song, Smith et al., 2003; Lee, Barzi et al., 2009). Moreover, studies of identical twins have demonstrated that the shorter twin had an increased chance of coronary heart disease mortality (Silventoinen, Zdravkovic et al., 2006). In summary, the direction of the association between short stature and increased mortality risk is well established in modern research. The estimated size of the relationship varies across countries. This would be surprising if the association was a biological one with little variation, but the risk of short stature is likely to vary across countries and time depending on social and economic conditions. Estimating how the relationship has changed and why remains a challenge for economic historians.

The extensive modern research is complemented by just two historical studies addressing the same question directly. Engaging directly with Waaler's results Costa examined the mortality risk of stature in a sample of Union Army veterans (Costa, 1993). Her findings broadly paralleled the modern research. Mortality risk declined with increasing height after 160cm and was lowest from

183 to 188cm. Mortality risk began to rise again for very tall men. Subsequent research by Murray on the mortality risk of stature in a sample of elite Amherst college students complicates the historical picture (Murray, 1997). Murray found no relation between height and mortality risk. He concluded that the influence of height on mortality was mediated by social and behavioral factors. In economically homogeneous groups, such as elite college students in the nineteenth century United States height may not be predictive of mortality. In an economically homogeneous sample the variation in height is likely to be more genetic than reflective of environmental and economic conditions in childhood. Despite Murray's null finding, the similarity of the relationship in Costa's research with the extensive modern evidence suggests the height-mortality relationship is not unchanging, but certainly long-standing. Additional research on historical populations is necessary to show how the strength of the relationship has changed across time and space.

Weight and health

Longitudinal studies involving weight are even more tightly constrained than studies of height by limitations on the availability and quality of evidence. Before the nineteenth century there was not adequate technology to accurately record human weights. While balance scales—such as the steelyard—have been used since ancient times they are of little value for measuring live weight (which moves). Fairbanks scales were perfected and mass produced in the United States by the middle of the 19th century, and were more stable and suitable for weighing people and animals. By the mid-1870s Fairbanks scales and similar

designs manufactured elsewhere were used in anthropometric studies (Bowditch, 1877; Roberts, 1879). Ironically the weight of platform scales, which were too bulky and expensive to have been used in the field and at muster stations, means we have little historical data on weight before the late nineteenth century.

Both historical and modern research show that individuals at the extremes of the weight distribution have a higher risk of mortality, with the risks mediated through specific diseases. Here we consider men only since anthropometric historical data on a longitudinal basis for women are scarce. Indeed, even modern medical studies have paid less attention to these issues for women (Kuh and Hardy, 2004). Although the mortality risk of being overweight dominates the academic and policy discussion today, being underweight was a more common health risk in before World War II.

The greater prevalence of underweight adults before World War II was related to the three contributors to net nutrition discussed above, namely caloric intake, disease burden, and physical intensity of daily living. Humans today, particularly in industrial and service work but also in agriculture, expend substantially less energy in work and transport activities than they did a century ago (Cutler, Glaeser et al., 2003; Philipson and Posner, 2003). People in the past needed more calories just to carry out their daily activities. Moreover, nutritious food was expensive relative to income, both to acquire and prepare (Logan, 2006; Logan, 2009). Mass urbanization in the nineteenth century reduced the ability of western populations to produce their own food supplies that they could consume if they were unemployed or had reduced wages. Finally common

infectious gastrointestinal and respiratory diseases led to weight loss, and were associated with significant (but declining) mortality in the nineteenth century and first half of the twentieth century. Thus, despite the current policy and academic concern about overweight as a risk factor for morbidity and mortality, historical studies also show an important relationship between being underweight and later mortality.

Weight and mortality

The recognition that individuals at the extremes of the body mass distribution have a higher risk of mortality has been widely accepted in medical research and practice throughout the twentieth century. Moreover, it is established that the mortality risk of excess weight is specific to circulatory diseases, some cancers, and diabetes. What is less clear is when in the life course excess weight is a particular risk. In the 1950s and 1960s when cardiovascular disease mortality in industrialized nations became an increasingly common cause of death, medical and epidemiological research emphasized excess weight in mid-adulthood as the most important risk factor. Since the 1970s, and particularly in the last two decades, medical and epidemiological research has paid increasing attention to other periods in the life course. Medical research has shown some mortality risk from excess weight in childhood (even if later BMI values are normal), and from fluctuations in adult weight that increase mortality risk even if an individual is in a normal BMI range much of the time (Kuh and Smith, 1993).

The inspiration for much of the economic history literature on the relationship between body composition and mortality was a large-scale Norwegian study (Waaler, 1984). Height and weight were measured between 1963 and 1975 as part of a tuberculosis screening program, and deaths of the same individuals traced from 1963 to 1979. Waaler's research showed that the U-shaped relationship between body mass and mortality flattened as the age of initial measurement increased. All individuals measured at age 85 have a high risk of dying in the next 17 years, so the *relative* mortality risk for extremely slender or obese people is lower. Conversely, for individuals in their 20s and 30s the risk of mortality in the next 17 years is low, and people with extreme BMI values have a high relative risk of dying. With an extremely large sample Waaler was able to show that the U-shaped relationship between mortality and body mass was also found after stratifying by height. Taller men with moderate BMI values had the lowest mortality risk. Again, taking advantage of the extremely large sample Waaler was able to show that the U-shaped relationship was strongest for particular diseases. Cerebrovascular (stroke) disease showed a strong U-shaped relationship with body mass, while cardiovascular diseases and diabetes had weaker U-shaped relationships. The excess mortality risk of being obese was greatest for stroke and diabetes.

Waaler's results have been supported by many subsequent studies. The most robust result is the substantial increase in mortality risk for obese (BMI ≥ 30) individuals in mid and late-adulthood (30-64). The relative risk of mortality for obese men has generally been found to be at least 50% greater than the mortality risk for men with a BMI between 20 and 25. For example Calle and

colleagues in a prospective study of more than a million United States residents over 14 years of follow-up found that 30-64 year old men with a BMI of 32-35 had an all-cause mortality risk 2.17 times higher than men whose BMI was between 23.5 and 25 (Calle, Thun et al., 1999). Calle and colleagues found that smoking substantially modified the relationship between low BMI and mortality. Amongst non-smokers with no pre-existing major diseases, the relationship between BMI and mortality risk was J-shaped. Risk rose only slightly for very lean men (BMI below 20). A U-shaped relationship was found for smokers, both with and without a history of other diseases. Because of the significant smoking rates in industrialized countries from approximately World War I to the 1980s, the association between leanness and mortality is confounded by smoking behavior (Pierce, 1989; Giskes, Kunst et al., 2005; Preston and Wang, 2006). In populations with very low smoking prevalence, such as Seventh Day Adventists who abstained from smoking for religious reasons, a positive linear relationship has been found between BMI and mortality risk (Lindsted, Tonstad et al., 1991). Similar results have been found in other studies that controlled for smoking history in measuring the relationship between body mass and mortality (Lee, Manson et al., 1993; Solomon and Manson, 1997; Singh, Lindsted et al., 1999; Allison, Zhu et al., 2002). These studies typically showed that there was no adverse effect of a BMI as low as 19. The implications for historical anthropometric research are most profound in studying cohorts who were in adulthood during the twentieth century. The growing availability of anthropometric data from the two world wars in the twentieth century mean that increasing research will be done on these populations. It is unlikely that the available data sources for these historical populations will be able to control for

smoking behavior since what is typically available to researchers from military records are anthropometric measures in young adulthood, medical records from later in life, and death records. For cohorts living mainly through the nineteenth century when smoking prevalence was much lower the confounding effect of smoking on the BMI-mortality relationship is less of an issue.

A further issue raised by modern studies is when in the life course mortality risk is raised by excess weight. For example, a study by Hoffmans of Dutch men born in 1932, measured in 1950 for military service and followed for 32 years, found that the relative mortality risk of men overweight at 18 rose "slightly" after 15 years, and "steeply" after 20 years of follow-up (Hoffmans, Kromhout et al., 1988; Hoffmans, Kromhout et al., 1989). People can live with excess weight for several decades in childhood and early adulthood, but the risk rises over time. Because of the recent development of research interest in the links between childhood through early adult BMI and health in later life, many studies do not yet have enough deaths to test for a relationship between BMI and mortality. Thus, alternative endpoints such as the development of particular diseases are chosen. For example, there is no relationship between excess BMI in early childhood (3-6 years) and coronary heart disease in later life. However BMI in later childhood (7-18 years) and early adulthood (18-30) was positively related to later development of coronary heart disease (Owen, Whincup et al., 2009).

A frequent finding in the epidemiological literature is that the relative mortality risk of being obese diminishes in the very elderly. In an older sample everyone has a high risk of dying, so the relative risk of being obese falls (Corrada, Kawas et al., 2006). However, the difference in mortality rates between

the obese and the lean may widen in older samples. For example, Stevens found that over 11 years of follow-up 1.5% of men aged 30-39 with a BMI between 18.5 and 25 died, compared to 3.6% of obese men. Among men aged 60-69 the mortality rates were 25.3% and 31.9% respectively (Stevens, 2000). A related question to the mortality differences between groups is a comparison of the years of life lost in different BMI categories. Stevens found that on average the years of life lost for the obese was less than a year more than in the normal weight category. While obesity carries health risks and raises relative mortality, the years of life lost due to obesity may be relatively modest. Recent work by Mehta and Chang advances this argument further (Mehta and Chang, 2009). Using data from the United States Health and Retirement Study, which studied individuals born between 1931 and 1941 from 1992 to 2004, they found the mortality risk of obesity only rose significantly for BMI levels above 35. People with a BMI between 30 and 34—who have been found to have a significantly higher relative risk of mortality in many previous studies—had a mortality risk similar to people in the normal weight and overweight categories. Mehta and Chang attributed just 3% of observed male mortality to obesity, compared to 50% attributable to smoking. However, Mehta and Chang's conclusions are challenged by other recent research that forecast the life expectancy costs of obesity in the United States to rise (Stewart, Cutler et al., 2009).

The limited number of historical studies of the relationship between weight, or body mass index (BMI), and mortality confirm the findings of modern research that extreme body mass values have a greater mortality risk. Historical research shows that mortality risk for men in their fifties and sixties is lowest at a BMI between 20 and 27. World Health Organisation guidelines for healthy

weight currently define BMI values of 18.5-25 as a healthy weight (World Health Organization, 1999). However there is emerging evidence from large-scale epidemiological research that the increased mortality risk of having a BMI between 25 and 30—categorized as overweight by WHO and other public health agencies—may be diminishing (McGee and Diverse Population Collaboration, 2005). The development of medical devices and pharmaceuticals to treat cardiovascular and related conditions may have reduced the cost of being slightly overweight. The mortality risks of obesity (BMI between 30 and 35) and severe obesity (BMI over 35) remain high. The proportion of the population classified as overweight or obese has been growing in most countries since the 1970s. The mortality risks of being obese do not appear to have changed substantially in the past century, but the population at risk has increased substantially. However, there is evidence that the risks of being moderately overweight have decreased through the twentieth century.

The only historical studies of the relationship between weight and mortality for cohorts born in the nineteenth century come from the United States. With the exception of John Murray's 1997 article on Amherst College students, the Union Army data provide the sole source of evidence on this topic (Fogel, 1993; Murray, 1997). Historical research on the relationship between weight and mortality has explicitly aimed to produce results comparable with modern studies. From the first article measuring the BMI-mortality relationship in an historical population, researchers have aimed to make comparisons with the contemporary literature (Costa, 1993). Comparisons with Waaler's pioneering 1984 study of Norwegian men have been particularly important in the historical literature (Waaler, 1984).

Costa and subsequent authors have drawn on datasets created of men who served in the Union Army during the American Civil War, and were born in the first half of the nineteenth century, when mean stature of American men was declining—a phenomenon known as the antebellum puzzle (Haines, Craig et al., 2003). Using an early version of the Union Army dataset Costa was able to study the relationship between BMI and mortality for 377 men who were weighed between the ages of 45 and 49 as part of pension exams (Costa, 1993). Compared to modern populations, the Union Army veterans were light, with an average BMI of 22.8 compared to a BMI of 25 among the Norwegian men measured in Waaler's research (Waaler, 1984). Over a 25-year follow-up period the risk of mortality was lowest for men with a BMI between 21 and 28. With a small sample Costa was not able to examine the risk of BMI for specific causes of death, but found that the relationship between weight and mortality was the same when men described as "very sick" were not included in the estimation. Costa concluded that the shape of the BMI-mortality relationship was the same in historical and modern populations, and that the location was similar with BMI risk lowest between 21 and 28. Using the same sample Costa also found that reduced stature was associated with higher mortality. With both stature and BMI rising in modern populations, the question is which effect dominates? Over the past century in the United States, at a population level the effects of increases in stature, reflecting better net-nutrition in childhood, have dominated the effects of increased BMI (Fogel and Costa, 1997).

Subsequent research with an expanded Union Army dataset has modified, but not overturned Costa's conclusions. The U-shaped relationship between weight and mortality remains, but the slope of the curve has changed. Costa

began with a sample of 1447 men, and the analysis of BMI and mortality risk included just 322 men. Recent research with the Union Army data has used samples of up to 10,000 men depending on sample restrictions, and compared the results to modern health surveys. For example, Henderson was able to use a sample of 9,509 Union Army veterans examined after 1865 (Henderson, 2005). Henderson compared the Union Army cohorts born before 1845 with men in the first wave of the National Health and Nutrition Examination Surveys (NHANES I) carried out between 1971 and 1975. Henderson also found that the relationship between BMI and mortality was U-shaped and reached a minimum for men with a BMI between 20 and 30 in both cohorts. With greater sample size, Henderson was able to make finer conclusions on changes in the shape and location of the BMI-mortality curve. Henderson found that the relative mortality risk of extreme BMI values—both underweight and obese—was smaller in the nineteenth century. The overall BMI-mortality curve was flatter for the Union Army cohort. In the Union Army cohorts men with a BMI of 30-35 were approximately 26% more likely to die within 20 years of examination. In the NHANES I samples, men in the same BMI range were 49-60% more likely to die within 20 years of examination. Similarly, the mortality risk of having a BMI under 20 was greater in the twentieth century cohorts. Yet Henderson also found that moderately overweight men with a BMI of between 25 and 30 had reduced mortality risk in the twentieth century. Henderson concludes that twentieth century cohorts had higher fat-free mass for the same BMI—equivalent to a lower body fat percentage—increasing the chances of survival at BMI values in the normal and moderately overweight range (20-30).

Other authors have confirmed Henderson's conclusion that fat-free mass at a given BMI has increased since the nineteenth century (Costa, 2004; Su, 2005). Su compared a sample of 1238 men from the Union Army dataset followed up over 18 years, with a sample of 861 from the NHANES follow-up sample. The incidence of obesity increased from 2.5% amongst the Union Army cohorts in 1890 to 28% in the NHANES sample measured in the early 1970s. Su found that in the Union Army cohorts underweight men had the greatest risk of death over 18 years, compared to men who were overweight. The difference was greatest in the first 9 years after follow-up—with a 20% difference in survival rates between underweight men and their heavier peers. But after 18 years the survival gap between BMI categories had narrowed significantly to just 12%. More of the underweight men died soon after their initial examination because some were already suffering from gastrointestinal diseases. While the same order of mortality risks was found in the NHANES cohorts, the mortality risks diverged over time. In the first 9 years the gap between the BMI categories was no more than 10%, but grew to 29% at 18 years. Just 40% of men who were underweight at initial examination in the NHANES survived 18 years, compared with 69% of overweight men. The optimal BMI for survival increased from a range of 20.6 – 23.6 in the Union Army cohorts to 22.7 – 27.3 in the NHANES cohorts. However, in both groups the optimal BMI for survival was in the middle of the distribution, the BMI-mortality relationship was again U-shaped. Using a larger sample with different age restriction, Linares and Su also examined survival rates over 20 years (Linares and Su, 2005). They also found a U-shaped relationship between body mass and mortality. Similarly to Henderson, they categorized body mass into the modern BMI categories of underweight (< 20),

normal weight (20-25), overweight (25-30) and obese (30+). Mortality was minimized with a normal BMI between 20 and 25, paralleling the results that Henderson obtained with the same dataset. BMI was less strongly associated with deaths more than 9 years after measurement, emphasizing that current weight is most predictive of mortality.

The results obtained in the Union Army datasets are broadly paralleled by findings on Amherst College students (Murray, 1997). Men who were extremely thin or fat had a significantly elevated mortality risk, compared to men with a normal weight. Because the men were weighed at college in their early 20s, Murray's research examined the effect of early-adult body composition on survival to age 70. Even after controlling for height, men with a BMI below 18 were one-third more likely to die before age 70 than men of normal weight or moderate underweight (a BMI between 18 and 20). Men who were overweight (BMI > 25) were 45% more likely to die before age 70 than normal or moderately underweight men. Like the authors using the Union Army dataset, Murray concluded that the BMI-mortality relationship was not significantly different in historical populations than contemporary ones.

A final piece of historical evidence on the weight-mortality relationship comes from a study of Scottish university students born in the 1920s, and followed until 2002 (Jeffreys, McCarron et al., 2003). The men, who were students at Glasgow University in 1948, were weighed in-person in 1948 and reported their weight in a postal questionnaire in the mid-1960s. Although the two weight measures were not collected in the same way, the independent timing of the two weight measures distinguishes this research from the Union Army research, where men who visited the doctor more frequently were

weighed more frequently. Unlike the Union Army based research Jeffreys *et al* found that weight in early adulthood was more strongly predictive of later-life mortality than mid-adult weight. Weight was most strongly predictive of cardiovascular disease mortality, and only moderately predictive of cancer mortality. Weight gain between early and mid-adulthood was not predictive of mortality. The failure to find an association between weight gain and mortality is inconsistent with the majority of the epidemiological evidence, and is likely to be due to mid-adult weight being self-reported.

Data

The New Zealand Defence Force (NZDF) in 2005 transferred 122,000 WWI service files to Archives New Zealand, which has been making them available gradually in response to genealogical and other public interest.[1] To date, we have collected the first 21,905 personnel records to become available in this way. Additionally, in order to increase the count of Maori soldiers, we secured permission to examine the files of men with indigenous surnames and with names falling in sections of the alphabet with significant indigenous representation. Some Pakeha or European-descent men were acquired in this process. Thus we begin with a roughly 17% sample of WWI records selected in an ad hoc way albeit with no detectable bias and an oversample of men with surnames of seemingly indigenous origin.

[1] The personnel files for another 6,000 servicemen and women who served after 1920 are not yet accessible.

For these men we have information on name, place and date of birth, enlistment date, occupation at enlistment, marital status, educational achievement and religion, military identification number, and height and weight. Heights were measured to the quarter inch. The New Zealand military had measured men without shoes since the South African War of 1899-1902 if not before.[2] Photos from WWI suggest that balance weight scales were used in at least the four largest cities (Auckland, Wellington, Christchurch and Dunedin). We do not yet have clear evidence how recruits were weighed in the smaller centres. Other medical and health information was not uniformly collected. Many of the men were assessed as having 'good' health along various dimensions of health. If any aspect of a man's health was poor, further details from medical tests are sometimes given. Thus, while detailed quantitative health information is available selectively for the less fit recruits, it is not easily used in the analysis of overall population health.

We have set aside the records in our sample of women who served as nurses because their numbers do not support useful analysis. We also exclude from the analysis men who enlisted before the age of 21 years because many of them were still growing. In itself, the observation that men were still growing in their late teens is an indicator of living standards below current ones. In well-nourished modern populations many men attain adult height before age 20 (Bogin, 1999). We exclude men older than 49 years in order to minimize any complication arising from the diminution of height at advanced ages.

---

[2] Attestation of William Eli Johnston, 1902. AABK/18805/W5515, Box 29, Record 2872. Archives New Zealand, Wellington.

We also discard men born outside New Zealand. This is 30% of our sample - roughly the same as the foreign-born share of men at appropriate ages in the 1911 census (32%).[3] Roughly 1 in 8 of our men were born in Great Britain. British migration to New Zealand peaked in the early-1860s and mid-1870s (Phillips & Hearn, 2008). An immigrant may have arrived at a young age, in which case his attained height would reflect the New Zealand nutritional environment. However, without longitudinal data, we cannot distribute childhood influences into a part reflecting experience elsewhere and another part reflecting experience in New Zealand.

The New Zealand-born personnel divide equally between the North Island and the South Island, reflecting the approximately equal populations of the two main islands in the late nineteenth century. There was no appreciable difference in stature between men born in the two islands.

Distinguishing men who were entirely or largely of European descent (Pakeha) from the Maori population indigenous to New Zealand is complex. There is no unambiguous method apart from reconstructing ancestry for the entire sample, which is impractical (and incomplete insofar as it would ignore cultural practice). Thus, we rely on Maori names, principally surnames, to indicate Maori ethnicity - a strategy that brings with it all the ambiguities associated with self-identification *(Axelsson* and *Sköld 2011)*. In our case, after more than a century of interaction some men would have had both European

---

[3] Government Statistician, *Results of a Census of the Dominion of New Zealand Taken for the Night of 2nd April, 1911,* (Wellington: Registrar General's Office): *xii*, 228-229.

and Maori ancestry. Moreover, a genetically 'pure' Maori could adapt a European name, and a European might adopt a Maori name. And regardless of name someone might chose to live in ways consistent with Maori but not European cultural practice (or vice-versa). Our understanding of New Zealand society suggests these possibilities were relatively uncommon and that name-based ethnic identification usefully points to men of substantially indigenous ancestry who, at least as children, lived as Maori. At present our Maori sample is too small to distinguish statistically between different iwi (tribes).

Construction of linked sample

Our construction of a sample of men linked to vital registration records began from our dataset of New Zealand World War I enlistment records. We excluded from the universe of men eligible for the linked sample the following categories of recruits:

- Men known to be born outside New Zealand
- Men known to have died during World War I or in 1919

We then drew a sample of 2500 men stratified into the following categories

- Māori working as farmers or farm laborers
- Māori working in non-agricultural occupations
- Pākehā working as farmers or farm laborers
- Pākehā working in non-agricultural occupations

The stratification was designed to ensure we had an equal balance of rural and urban workers, and as many identified Māori men as we had found in

the World War I records to date (Table 1). Individuals were linked to their birth and death records by matching on the following variables available in both civil registration and military enlistment records: full given name, exact date of birth, and location of birth. In a country as small as New Zealand with between 24,000 and 30,000 annual births (thus approximately half that number of male births) each year between 1870 and 1897, this limited set of information was sufficient to make exact matches with a high degree of confidence.

Overall we were able to link about three quarters of the sample sent to the Registrar General's office for linking to birth and death certificates (Table 2). Our link rate was substantially higher for Pākehā than for the indigenous Māori population. We were only able to link half the Māori sample to their death certificates, while for Pākehā our linkage rate was 77%. Within each strata there were some differences in the height, weight, and BMI of the linked and non-linked groups (Table 3 and 4). With a relatively large sample size, these differences were statistically significant. Within strata and within race, the differences were not large. However, the differences in BMI between races were substantial with mean Māori BMI over 24, compared to 22-23 for Pākehā. Although based on relatively small numbers, these racial differences are striking considering contemporary concern about current Māori rates of obesity, and debate over whether those differences are long-standing and genetic (Ministry of Health, 2008).

Results and analysis

Our analysis focuses first on the remaining years of life lived by World War I survivors, and then on the relationship between early adult health and

mortality. Our primary interest in this preliminary paper is the shape of the relationship between mortality and anthropometric measures. The historical studies reviewed earlier suggest that in the nineteenth century there was a U-shaped relationship between both height and weight, and mortality. To date this evidence rests on two quite different samples from the United States, using records of Union Army soldiers and Amherst College students (Costa, 1993; Henderson, 2005; Murray, 1997). To facilitate comparison with previous results, we follow the divisions for height and weight suggested by Murray in his analysis of Amherst College students. Our other covariates necessarily differ because of the New Zealand setting, but the pattern of height, weight, and the relationship to mortality can be compared directly.

Mortality experience of World War I survivors

On average this group of World War I survivors lived a long time after the war. Among the 1879 men successfully linked to their death certificates and not dying between 1915 and 1919, mean years lived from military enlistment was 44.3 years, with an average age at death of 69.9 years (Table 6). This compares favorably with the average life expectancy of New Zealand men born in 1891 {Statistics New Zealand, 2006 #6316}. For men born in 1891 (the average man in our sample was born in early 1890) life expectancy at birth was 51 years, at 1 year of age 57 years, and at 25 years of age (the average age of enlistment in this sample), life expectancy was 41 years. Thus, the men in our sample are, if anything, slightly longer-lived than the average New Zealand man born at the

same time .[4] The pattern of survivorship was similar in the first 10 years after the war for all strata in the sample (Figure 1). However, by the early 1930s differences begin to emerge between the different strata. For the majority of these men's lives, mortality was lowest for Pākehā who had been working in farming at enlistment, and highest for Māori.

Our analysis of the mortality experience of the survivors is in this paper restricted to an analysis of mortality between the ages of 55 and 70 (cf. Costa 1993). External causes of death—suicide, and accidents involving vehicles or agricultural or industrial equipment—were more common before age 55. Restricting our analysis to men surviving to 55 also allows us to abstract from any effects of injuries suffered in World War I causing mortality during the 1920s. We have detailed cause of death information for our sample, which will in future allow us to refine the exclusion restrictions.

Table 7 shows the results of several models relating mortality to anthropometric measures. Race and occupation are controlled for by the sample stratification, and do not appear explicitly as covariates. Following Murray (1997) we develop our models from sparse "anthropometric only" specifications to full models that eventually control for height, body mass and covariates. Models 1 and 2 show the effect of BMI alone and with controls for covariates, while Models 3 and 4 examine the effect of height independent of BMI. Finally, Models 5 and 6 are the preferred models that control both for height and BMI simultaneously adding in covariates for birth cohort.

[4] The life expectancy estimates are calculated excluding the heavy impact of wartime casualties.

The models which control for height and BMI together are preferred for both empirical and theoretical reasons. By definition any research that uses the body mass index has information on stature. It follows that the relationship between height and mortality, and between body mass and mortality, are interdependent since height squared enters as a term in the definition of body mass. Treating the relationships independently implies that the BMI-mortality relationship is stable across the height distribution. This assumption should be tested, and empirically it has been found that the relationship is not stable (Fogel, 2009). Put slightly differently, we need to investigate if each increment of height may require different increments of weight to maintain constant health.

Empirically, a key issue is whether people who are short but adequately nourished—as measured by BMI—are at a higher risk of death. People living through periods of rapid economic change—such as in contemporary developing countries, or now developed countries in the late nineteenth and early twentieth centuries—may be poorly nourished in childhood and thus short, but well nourished in adulthood and thus have a BMI in the healthy range. In a longitudinal perspective the question is whether early life deprivation reflected in short stature could be compensated for by adequate nutrition later on (Fogel, 1994; Fogel and Costa, 1997).

Because previous research on historical populations has found that the extremes of both height and BMI are at elevated risk for mortality we specify our models with dummy variables for categories of both measures, omitting a category in the middle of the distribution to test for a U-shaped curve. However, we find weaker evidence of an effect of body composition on mortality than in previous studies of historical populations. We find no evidence that people with

low BMI values were at an elevated risk of mortality (Models 1 and 2), and that they may in fact have had a lower risk of mortality (Table 7). The size of the effects is not altered by controlling for sample composition. There is somewhat stronger evidence that individuals with a BMI of over 25 at enlistment are at a slightly elevated risk of mortality. Mortality risk for these individuals was elevated 10% over individuals in the bottom of the normal range (20-25) for BMI, but the statistical significance of the effect was low ($t \approx 1.4$ in preferred models). 15% of our sample had a BMI of over 25 at enlistment, significantly greater than the proportion of overweight men in Murray's study of Amherst College students. Thus, our failure to detect a significant effect of being overweight is not related to a low proportion of men being overweight. We also found limited evidence that height had any impact on mortality risk. The hazard ratio for height categories varied from 0.95 to 1.04 in all models, and was not affected by controlling for weight or sample composition.

Following Costa and Murray we also examined the relative risk of mortality between ages 55 and 70 within BMI categories, by dividing the proportion of men in the BMI group who died by the overall proportion of men in the study group who died (Figure 2). Consistent with our hazard models we found weak evidence of any relationship between being underweight at enlistment and an elevated risk of mortality in later life. However, men who were overweight at enlistment had a substantially elevated relative risk of mortality. The relative risk of mortality rose sharply when the BMI exceeded 25. While the exact shape of the risk curve is sensitive to the numbers within each BMI category, the overall pattern is similar to what has been observed by previous

authors — being overweight even in early life has significant consequences for mortality in adulthood.

The evidence that the shape of this relationship holds in another nineteenth century cohort is significant. If we take seriously the hypothesis that environmental, social and economic conditions can influence health we are also obliged to consider that "influence" is itself a variable. Parameters can shift, and co-efficients can change. This evidence from New Zealand suggests that the elevated mortality risk of being overweight in the late nineteenth and early twentieth century could have been broadly similar across developed countries. Reductions in the relative mortality risk of being overweight appear to have occurred in recent years with the relative risk of having a BMI over 30 being less than 2 in some modern studies, and the relative risk of being moderately overweight (25-30) being less than 1.5. Identifying the precise timing and cause of this shift is an important unresolved question.

Bibliography

Bogin, B. (1999). *Patterns of human growth*. Cambridge: Cambridge University Press.

Costa, D. (1993). Height, weight, wartime stress, and older age mortality: evidence from the Union Army records. *Explorations in Economic History,* 30, 424-449.

Gillman, M.W. (2004). A life course approach to obesity. In D. Kuh, & Y. Ben-Shlomo (Eds.), *A life course approach to chronic disease epidemiology* pp. 189-217). Oxford: Oxford University Press.

Henderson, R.M. (2005). The bigger the healthier: Are the limits of BMI risk changing over time? *Economics and Human Biology,* 3, 339-366.

Lakdawalla, D., & Philipson, T. (2009). The growth of obesity and technological change. *Economics & Human Biology,* 7, 283-293.

Lumey, L.H., Stein, A.D., & Susser, E. (2010 forthcoming). Prenatal Famine and Adult Physical and Mental Health. *Annual Review of Public Health*.

Ministry of Health (2008). *A Portrait of Health: Key results of the 2006/07 New Zealand Health Survey*. Wellington: Ministry of Health.

Murray, J.E. (1997). Standards of the present for people of the past: Height, weight, and mortality among men of Amherst College, 1834-1949. *Journal Of Economic History,* 57, 585-606.

Philipson, T., & Posner, R. (2003). The long-run growth in obesity as a function of technological change. *Perspectives in Biology and Medicine,* 46, 87-107.

Phillips, J., & Hearn, T. (2008). *Settlers: New Zealand Immigrants from England, Ireland & Scotland 1800-1945*. Auckland: Auckland University Press.

Waaler, H. (1984). Height, weight and mortality. The Norwegian experience. *Acta Medica Scandinavica,* Supplementum 679, 1-56.

Table 1. Selection for linking by Registration Office

Strata	Not selected		Selected for linking		Total	
	No.		No.	%	No.	%
Pakeha farm	5,086	26.3%	1,092	43.0	6,178	28.2
Pakeha non-farm	13,912	71.8	1,134	44.7	15,046	68.7
Maori farm	153	0.8	136	5.4	289	1.3
Maori non-farm	215	1.1	177	7.0	392	1.8
Total	19,366	100.0	2,539	100.0	21,905	100.0

Table 2. Linking success

Strata	Not in linked sample		Linked		Failed link		Linkage rate
	No.	%	No.	%	No.	%	
Pakeha farm	5,086	26.3%	860	45.9%	232	34.9%	78.8%
Pakeha non-farm	13,912	71.8%	859	45.8%	275	41.4%	75.7%
Maori farm	153	0.8%	70	3.7%	66	9.9%	51.5%
Maori non-farm	215	1.1%	86	4.6%	91	13.7%	48.6%
Total	19,366	100.0%	1,875	100.0%	664	100.0%	73.8%

Table 3. Height (cm) and weight (kg) of linked and non-linked groups

	Pākehā		Māori	
Linking success	Farm	Non-farm	Farm	Non-farm
Not in linked sample	173.17	172.19	171.66	171.93
	0.09	0.06	0.49	0.4
	68.69	67.28	72.48	70.95
	0.11	0.07	0.81	0.57
Linked	172.9	171.93	172.28	172.42
	0.22	0.23	0.7	0.61
	68.16	66.6	72.11	71.87
	0.28	0.29	1.24	0.89
Failed link attempt	172.91	172.03	171.66	172.97
	0.39	0.37	0.76	0.65
	68.91	67.64	72.21	73.29
	0.57	0.52	1.04	1.12

Table 4. BMI of linked and non-linked groups

	Pākehā		Māori	
Linking success	Farm	Non-farm	Farm	Non-farm
Not in linked sample	22.92	22.70	24.57	24.04
	0.03	0.02	0.21	0.16
Linked	22.80	22.53	24.27	24.13
	0.08	0.08	0.34	0.23
Failed link attempt	23.08	22.88	24.54	24.45
	0.16	0.16	0.31	0.32

Table 5. Characteristics of linked sample

Variable	Mean
Height	
height < 1.65m	0.10
1.65m ⩽ height < 1.70m	0.21
1.70m ⩽ height < 1.75m	0.30
1.75m ⩽ height < 1.80m	0.24
height ⩽ 1.80m	0.11
BMI	
BMI < 18	0.01
18 ⩽ BMI < 20	0.06
20 ⩽ BMI < 22	0.27
22 ⩽ BMI < 25	0.44
25 ⩽ BMI	0.15
Birth cohort	
1870-74	0.02
1875-79	0.06
1880-84	0.10
1885-89	0.20
1890-95	0.33
1895-99	0.28
Birthplace	
Auckland	0.07
Wellington	0.05
Christchurch	0.07
Dunedin	0.06
Provincial city	0.14
Rural area or small town	0.58
Unknown	0.03
Place of death	
Auckland	0.15
Wellington	0.08
Christchurch	0.09
Dunedin	0.05
Provincial city	0.25
Rural area or small town	0.39
Unknown	0.00
Maori	0.08

Table 6. Death cohorts for New Zealand World War I survivors

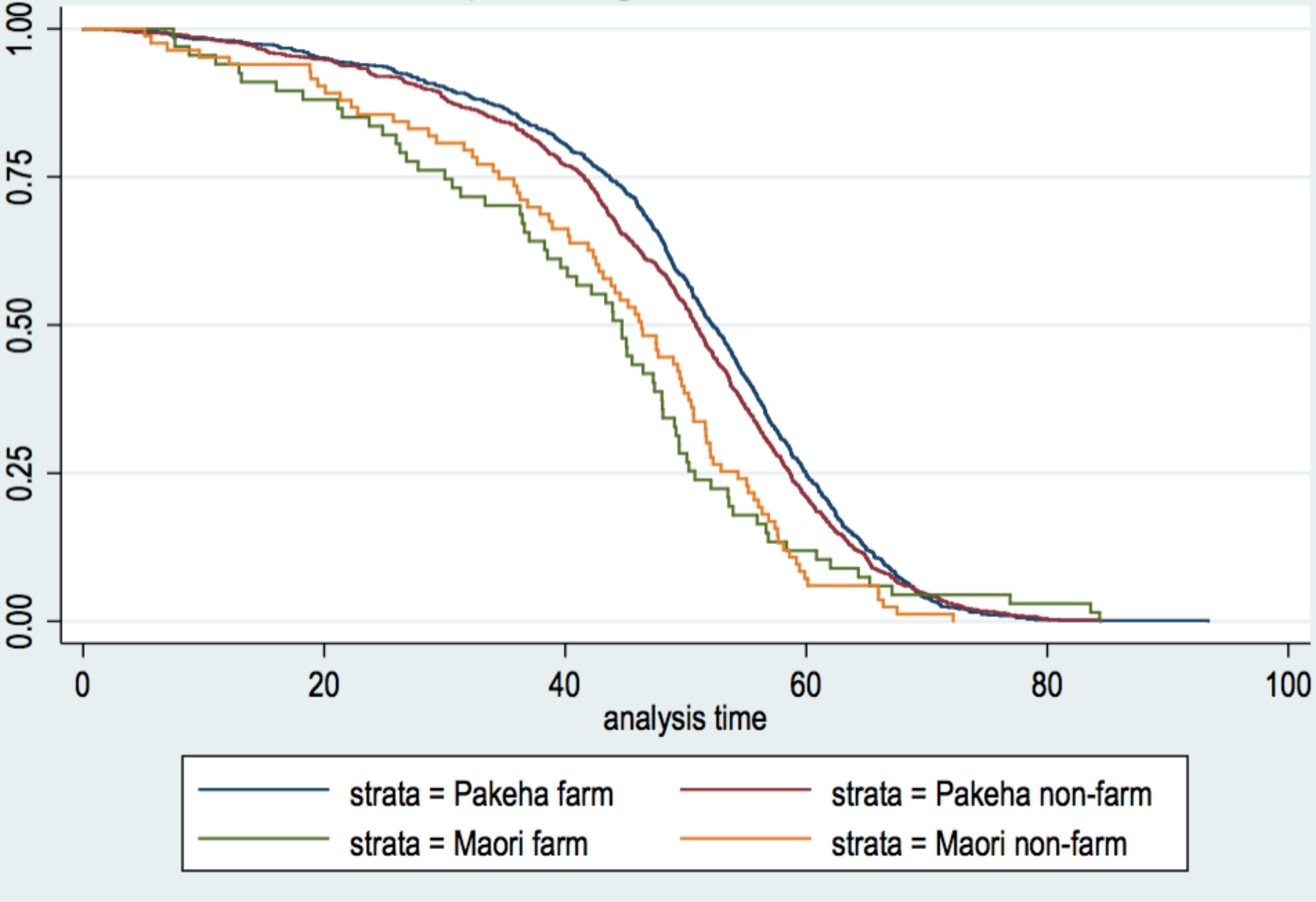
Death cohort	Number	%
1920-1924	48	2.6
1925-1929	51	2.7
1930-1934	40	2.1
1935-1939	71	3.8
1940-1944	83	4.4
1945-1949	118	6.3
1950-1954	182	9.7
1955-1959	231	12.3
1960-1964	253	13.5
1965-1969	272	14.5
1970-1974	204	10.9
1975-1979	162	8.6
1980-1984	109	5.8
1985-1989	37	2.0
1990-1994	15	0.8
1995-1999	1	0.1
Total	1877	100.0

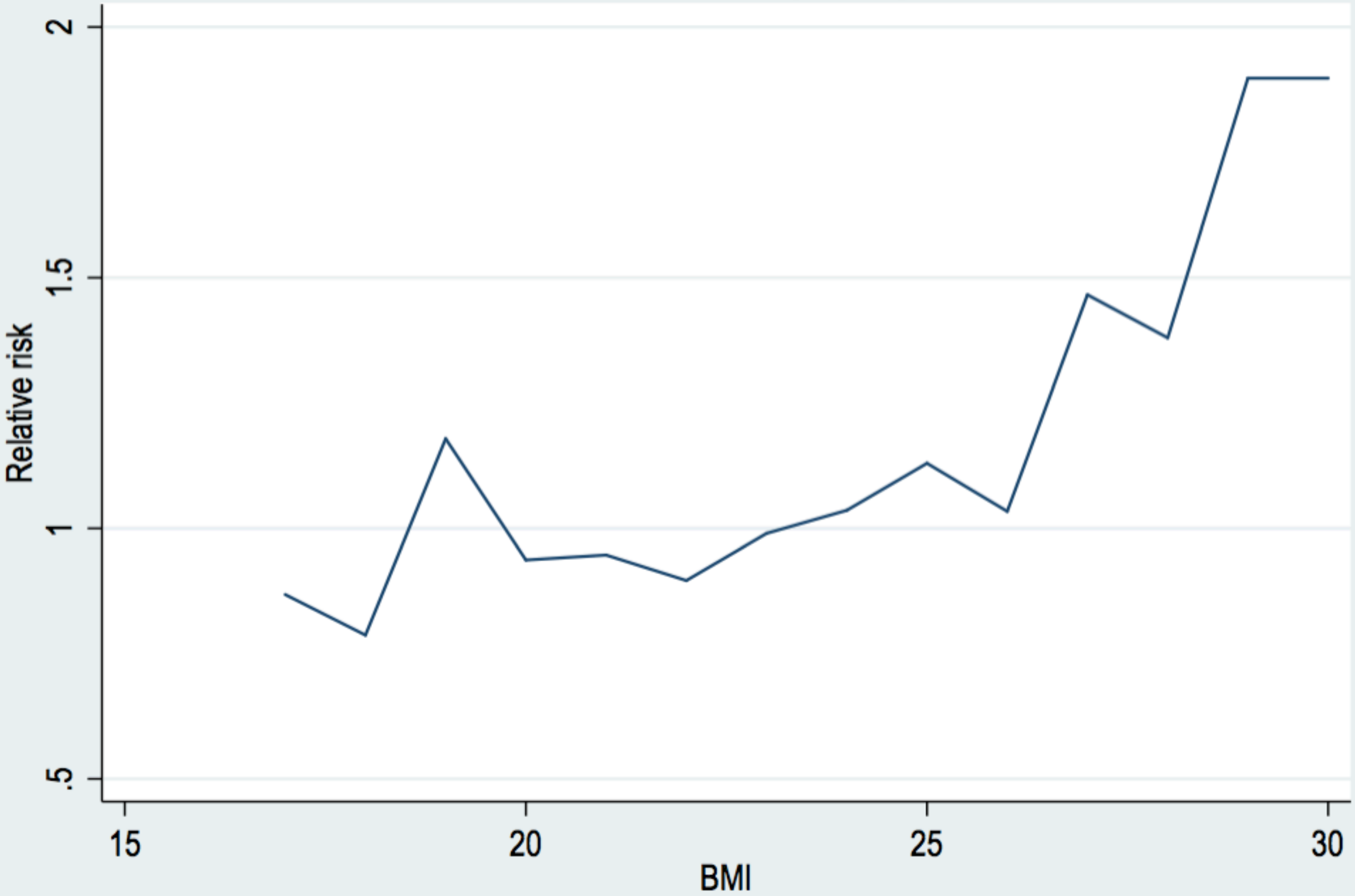
Table 7. Hazard ratios for mortality of New Zealand World War I survivors

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	Hazard ratios / (t-statistics)					
BMI < 18	0.88	0.89			0.90	0.90
	(-0.58)	(-0.56)			(-0.51)	(-0.50)
18 ⩽ BMI < 20	1.04	1.04			1.03	1.04
	(0.31)	(0.36)			(0.30)	(0.37)
22 ⩽ BMI < 25	0.99	0.99			0.99	1.00
	(-0.24)	(-0.10)			(-0.20)	(-0.05)
25 ⩽ BMI	1.09	1.12			1.10	1.12
	(1.11)	(1.36)			(1.13)	(1.39)
height < 1.65m			1.04	1.02	1.03	1.00
			(0.40)	(0.20)	(0.30)	(0.05)
1.65m ⩽ height < 1.70m			1.03	1.02	1.02	1.01
			(0.35)	(0.21)	(0.27)	(0.09)
1.70m ⩽ height < 1.75m			0.96	0.96	0.95	0.95
			(-0.67)	(-0.61)	(-0.73)	(-0.72)
height ⩽ 1.80m			0.96	0.96	0.96	0.96
			(-0.43)	(-0.41)	(-0.50)	(-0.49)
Born in						
Auckland (omitted)		1.00		1.00		1.00
		(.)		(.)		(.)
Wellington		0.86		0.88		0.87
		(-1.00)		(-0.88)		(-0.94)
Christchurch		0.87		0.87		0.87
		(-1.00)		(-0.95)		(-1.00)
Dunedin		0.88		0.89		0.89
		(-0.83)		(-0.81)		(-0.82)
Provincial city		0.87		0.87		0.87
		(-1.18)		(-1.12)		(-1.13)
Provincial city		0.90		0.90		0.90
		(-1.06)		(-0.97)		(-1.02)
Rural area or small town		0.83		0.85		0.83
		(-1.04)		(-0.90)		(-1.04)
Died in						
Auckland (omitted)		1.00		1.00		1.00
		(.)		(.)		(.)
Wellington		1.09		1.08		1.08
		(0.74)		(0.65)		(0.67)
Christchurch		1.16		1.16		1.15
		(1.32)		(1.32)		(1.31)
Dunedin		1.20		1.18		1.19
		(1.31)		(1.21)		(1.23)
Provincial city		1.02		1.01		1.01
		(0.20)		(0.15)		(0.16)
Provincial city		1.13		1.13		1.13
		(1.57)		(1.52)		(1.53)
Rural area or small town		3.03		2.95		2.98

		(1.10)		(1.07)		(1.08)
Born 1870–1874		1.25		1.27		1.26
		(1.09)		(1.16)		(1.13)
Born 1875–1879		0.96		0.98		0.96
		(-0.34)		(-0.23)		(-0.35)
Born 1880–1884		0.97		0.98		0.98
		(-0.31)		(-0.20)		(-0.25)
Born 1885 – 1889		(omitted)		(omitted)		(omitted)
Born 1890–1894		1.05		1.04		1.05
		(0.66)		(0.54)		(0.62)
Born 1895–1899		1.08		1.07		1.08
		(1.03)		(0.90)		(1.00)
N	1591	1591	1591	1591	1591	1591
chi2	-9693.01	-9687.60	-9693.39	-9688.55	-9692.28	-9687.14
Log likelihood	2.25	13.05	1.48	11.16	3.70	13.99

Survivorship after age 21 for NZ WWI enlistees




Relative risk of mortality at ages 55-70
Relative risk
2
1.5
1
.5
15
20
25
30
BMI