Birth weight and adult health in historical perspective
Evidence from a New Zealand cohort, 1907-1922

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Introduction

Birth weight and adult height have both been shown to be related to mortality. In particular, small babies and short adults are more likely to die of coronary heart disease. The association between short stature and increased mortality risk has been shown across many countries, and for cohorts born since 1840. The relationship between birth weight and adult morbidity and mortality has been demonstrated most often for cohorts born in the twentieth century, and only rarely for nineteenth century cohorts. Because a relationship has been shown between body size at two different points of the lifespan, it is important to understand the relationship between infant and adult body size, to determine how mortality risks evolve across the lifespan.

In this paper we provide evidence from an early twentieth century cohort on the relationship between weight and length at birth, and height, weight and blood pressure in early adulthood. Our cohort was born in Wellington, New Zealand between 1907 and 1922. We use maternity hospital records linked to military enlistment records from World War II to obtain information on size at birth and health in early adulthood for males. Birth weight, and adult height and body mass index provide indirect evidence of health, while blood pressure provides a reliably measured direct measure of health.

Historical evidence on nutrition, growth and health is important for both basic science and targeting health interventions. At one extreme, if adult body composition is predestined by size at birth then health interventions should be targeted at pregnant women. A more realistic perspective views disease risk as a flexible process across the life cycle from conception to the present (Kuh & Ben-Shlomo, 2004; Kuh & Smith, 1993). Emerging evidence of epigenetic effects

create a strong rationale for understanding nutrition and health in historical populations. The epigenetic hypothesis that gene expression and inheritance is modified by environmental influences implies that the relationship between birth and adult size, and health may not be fixed over time (Godfrey et al., 2007). While modern studies go beyond body composition to more precise measures of nutrition, growth and health, in most historical populations all that is available is anthropometric data. Our study provides unique evidence from an historical cohort that combines measured, rather than recalled, birth weight and a reliable measure of blood pressure.

The relationship between size at birth and adult height and weight is also important as a point of basic science. Human growth trajectories have interested physical anthropologists and others interested in auxology for several centuries (Bogin & Kapell, 1997; Tanner, 1981). But there were few large studies of growth from birth to maturity until the twentieth century. Most early twentieth century growth studies were instead motivated by interest in how early life conditions after infancy influenced development and health outcomes in later life. In Europe and North America many of these studies began in the late 1920s and early 1930s (Young et al., 1991). The early-twentieth century interest in growth from childhood to maturity—rather than infancy to maturity—was partially driven by the greater ease of recruiting children in kindergartens and schools, rather than maternity hospitals. Nearly universal school attendance pre-dated nearly universal hospital births, so more representative samples of children could be obtained later in the life course. Until the early twentieth century many maternity hospitals served poor populations including abandoned mothers, while the middle class continued to give birth at home (Ward, 1993). While

hospital births became more common in developed countries through the twentieth century, representative and usable samples of birth records from before 1940 are valuable because of their scarcity.

Evidence from New Zealand is of particular interest because of its apparently very healthy population in the early twentieth century. New Zealand, along with Scandinavia, had one of the earliest sustained falls in infant mortality in the industrialized world. The New Zealand infant mortality decline attracted international attention as an example of what improvements in infant health were possible through medical care and social policy (Woodbury, 1922). Contemporary commentators attributed New Zealand's infant mortality decline to the establishment in 1907 of an infant welfare group called the Plunket Society. However, the infant mortality decline in New Zealand had started in the 1880s and there is not firm evidence that Plunket was the cause of the decline. A similar trajectory of decline can be seen in Scandinavian countries, which like New Zealand were relatively well-off, but where the largest cities were quite small (Edvinsson et al., 2008; Mein Smith, 1988). Despite the international interest at the time there has been little demographic research on the New Zealand case, with more attention devoted to the innovative Plunket scheme (Bryder, 2001, 2003, 2009). This paper therefore brings overdue attention to measured health outcomes among cohorts born during the New Zealand infant mortality decline.

Infant mortality can also be an informative indicator about the population health and living conditions of babies who survived. The infant mortality rate is a widely used proxy for the general infectious disease conditions in a population. Moreover, cohort patterns of infant mortality have been shown to correlate

strongly with indicators of health in later life such as height and cardiovascular disease mortality (Barker & Osmond, 1986; Bozzoli et al., 2009)(Myrskylä, 2010).

Both the early twentieth century growth studies and research since the 1970s have provided significant evidence that size at birth is related to health consequences in later life. At the very least, size at birth is a predictor of health in later life. Now that the statistical association between size at birth and poorer health has been widely demonstrated in modern cohorts, the etiological pathways are becoming clearer (Barker, 2000). The consequences of low birth weight have attracted most attention (Barker, 1998; Kuh & Ben-Shlomo, 2004; Ward, 1993). Very low birth weight has long been recognized as a risk factor for early mortality, in both full-term and premature births. The proportion of infants weighing less than 2500g at full-term birth is often used as a summary measure of population health at birth. Because historical birth weight records are often not representative of all births that occurred, it is difficult to measure the extent of low birth weight in the past. However, research using the sources that are available show that in poorer populations in North America, between 5 and 8 percent of babies were less than 2500g at birth (Costa, 1998; Goldin & Margo, 1989). Low birth weight remains a key indicator of maternal and infant health in developing countries today (Cesar et al., 2008). By contrast, in modern industrialized countries where childhood and adulthood obesity is an increasing health issue, the morbidity and mortality risks of infants being overly nourished in-utero are emerging (Debbie A. Lawlor et al., 2007; Wu et al., 2004). Infants who are overly nourished in-utero are unlikely to suffer immediate health consequences as "small and sickly" babies were in the past. The consequences of

fetal over nutrition appear to be increased risks of obesity and Type 2 diabetes (Grattan, 2008). While the etiology is not as well researched, there is evidence of long-term effects of fetal over-nutrition on health in later life. For example, recent research with a large Danish cohort shows that higher birth weight is associated with greater adult mortality. (Baker et al., 2008). Between the extremes lies the more subtle research question of how incremental variations in size at birth affect adult body composition and health.

The risks of being small or large at birth are associated with specific conditions. Being small at birth increases the lifetime risk for coronary heart disease, Type 2 diabetes, hypertension, hemorrhagic stroke, and osteoporosis (Godfrey et al., 2007; Huxley et al., 2007; Hypponen et al., 2001; Kaijser et al., 2008; Newsome et al., 2003). Being large at birth is associated with some forms of cancer and obesity (Ahlgren et al., 2007). The risk of being small at birth is compounded if there is rapid weight gain in childhood with further risk of coronary heart disease for individuals who gain weight rapidly after being born small (Barker, 2004; Eriksson et al., 1999). The increased risks from rapid weight gain after being born small are consistent with the epigenetic hypothesis of an interaction between genes and the environment. Even if a person has the genetic inheritance to be bigger in adulthood, there is a permanent effect of starting out small because of poor nutrition in-utero. Thus, while both birth weight and adult stature have a large genetic variability, there is substantial scope for both to influenced by environmental conditions

Adult or terminal height is the product of both genetic and environmental influences, and is independently associated with mortality risk (A Engeland et al., 2003b; McEvoy & Visscher, 2009; Karri Silventoinen, 2003; Waaler, 1984). In

particular short stature is associated strongly with coronary heart disease (K Silventoinen et al., 2006). The association between adult height and mortality risk reflects that adult height is a summary measure of environmental conditions, particularly caloric intake and disease burden, during the growth period. Height in itself is not the variable of interest. Rather, height summarizes influences across the growth period. Particularly in retrospective or historical data, it may be one of the few reliable variables about early life experience available.

A significant amount of research has focused on the relationship between height and coronary heart disease. 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 have found a stronger correlation between leg length and adult cardiovascular disease than total height. Significantly, more 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 & Ben-Shlomo, 2004; Debbie A. Lawlor 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 is also inversely related to stroke, respiratory disease, and stomach cancers, and the association is due to early life conditions manifesting in both reduced stature and disease (George Davey Smith & Lynch,

2004). However, height is not associated with the development of hypertension (Whincup et al., 2004).

The association between early life deprivation—reflected in shorter stature—and chronic disease onset is significant because diabetes, stroke, cancer and cardiovascular conditions contribute to increased mortality. Therefore we would expect that shorter than usual stature is associated with increased mortality. 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. 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 (A. Engeland et al., 2003a).

Other researchers have found similar results in smaller samples. Height was inversely associated with all cause, coronary heart disease, stroke, and respiratory disease mortality in a 20 year study of Scottish men (G. Davey Smith et al., 2000). Similarly using data from the Whitehall study of British civil servants, an increase in height of 15cm was associated with a 10% reduction in mortality risk (G. David Batty et al., 2009; G. D. Batty et al., 2006). A Finnish study showed even stronger support for the relationship with a 5cm increase in height being associated with 10% declines in mortality risk (Jousilahti et al., 2000). 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 (Lee et al., 2009; Song et al., 2003). Moreover, studies of identical twins have demonstrated that the shorter twin had an increased chance of coronary heart disease mortality (K Silventoinen et al., 2006). In summary, the direction of the association between short stature and increased mortality risk is well established in modern research.

The relationship between size at birth, stature, and subsequent health outcomes is unlikely to be a universal or stable biological relationship, but mediated by social and economic conditions (Almond & Currie, 2011; George Davey Smith & Lynch, 2004). Estimates from a range of countries and time periods are needed to show the distribution of the parameters of interest. However, to our knowledge there is just one modern study examining the association between birth weight and later health outcomes for a cohort born in the nineteenth century (Morley et al., 2006). Accumulating evidence from diverse social and economic conditions about the relationship between size at birth, size in adulthood, and health outcomes can help establish whether social welfare policies and medical treatments reduce the influence of poor nutrition in utero or during the growth phase.

It is important that we measure change in the influence of early life conditions on adult health outcomes, because welfare policies since the late nineteenth century have aimed to reduce socio-economic disparities in children's life chances. If these policies were effective, we would expect the

relationship between size at birth and later health outcomes to change. For example, in New Zealand family allowances were introduced in 1926 that provided income-contingent assistance to families with children (Campbell, 1927; Nolan, 2009). In the 1930s New Zealand expanded health camps for poorly nourished children, and introduced a school milk scheme for all children (Tennant, 1996). The explicit aim of these policies was to improve childhood nutrition. At the time, the government believed that a low infant mortality rate showed that infant and maternal health problems had been addressed, and the health of school age children needed more attention. Shifting government policies about the balance between infant and child health services provides a very clear rationale for why the relationship between birth and adult size is unlikely to be constant over time.

Being small at birth and short in adulthood is likely to reflect both genetic variations between individuals—some people are just smaller because their parents are smaller—but also persistence in socio-economic conditions. In the absence of social welfare policies people whose mothers were undernourished during pregnancy (and thus small at birth) may be more likely to be undernourished across the growth period and thus short as adults. Therefore if social policies such as family allowances have been effective we would expect to see a stronger relationship between size at birth, and adult stature and health than in modern cohorts.

Our cohort also provides early evidence for a relatively large cohort on the relationship between size at birth and in adulthood. The relationship between birth size has interested scholars and lay people since at least the eighteenth century (Bogin, 1999; Tanner, 1981). The interest was diverse,

sometimes coming from eugenics for example**,** particularly in the early twentieth century (Baldwin, 1921). Contemporary research between the two world wars revealed that children's physical development was strongly influenced by environmental, particularly nutritional, influences. Many of these studies enrolled their cohorts after birth. Notable exceptions that studied growth from birth onwards were the Denver Study of Human Development (1927), Fels Longitudinal Study (1927-32), and the Berkeley Growth and Guidance Study and Harvard Growth Study (both 1929) (Jones & Bayley, 1941; McCammon, 1970; Reed & Stuart, 1959; Roche, 1992; Valadian & Porter, 1977). After World War II there was extensive investment in longitudinal research from birth to maturity in Europe, North America and Australasia (Bogin, 1999; Dye, 1987). Thus our cohort provides earlier evidence on physical growth from birth than available in the published literature, since the earliest growth studies from birth date from the late 1920s, and earlier research on childhood growth patterns focuses on school children.

Data

Our sample comes from births at the Wellington St Helens Hospital between 1907 and 1922. The St Helens Hospitals were established during an era of concern about high maternal and infant mortality. The Midwives Act 1904, although primarily enacted to establish formal midwifery training, registration and regulation, also addressed the need for safe maternity care for women who could not afford to engage a registered midwife to attend them in their home.

Between 1905 and 1920, seven St Helens hospitals were established throughout New Zealand to provide state-subsidised care for working-class women whose husbands earned less than £3 a week, and a place for training midwives. Women were attended by midwives, the medical superintendent was only called in for complicated cases, and the hospitals led New Zealand on all indices of safe maternity care.

The first St Helens Hospital was established in Wellington, the capital city of New Zealand, in May 1905. Its birth-related records appear in casebooks, admission books and maternity registers from 1907 until the hospital's closure in 1980. Although the first run of 'indoor' casebook records, from 1907 until 1922, are incomplete for the years 1907, 1910, 1911 and 1922, they provide information on 3,166 births. Two pages in the casebooks relate to each case. On admission to the hospital, the woman's name, address, age, date of admission, parity and physical appearance were noted, as well as any comments on her general health. Ethnicity and marital status were not noted – all women were given the title 'Mrs' and the great majority would have been married. The baby's presentation at birth, the length of each stage of labour, the date and time of delivery, the sex, weight and length of the infant, and any complications were added following the birth. The names of those in attendance at the birth were also recorded. The second page was mostly for midwives' comments on the mother's and baby's progress and care, and any problem or treatment that needed to be recorded. At discharge, the infant's weight and mode of feeding were recorded and any remarks about the mother's condition.

The range of detailed information provided by these casebooks was not unique in early twentieth-century maternity care. The records of other maternity

hospitals or dispensary services for working-class women hold similar information. Birth record ledgers at the Royal Women's Hospital in Melbourne also record the woman's country of birth and marital status (Morley et al., 2006). The Edinburgh Royal Maternity Hospital's 'indoor' casebooks have a single line across two pages for each case. Nuttall notes that their inclusion of the names of attendants at the birth was an exception to the usual lack of information on this aspect of care in other surviving hospital records (Nuttall, 2006). The hospital's birth registers also gave the woman's social data, including the occupation of the father (or the mother, if she was unmarried) (Nuttall, 2007). Case records of the Midwifery Dispensary on the Lower East Side of New York included the woman's ethnicity, income and occupation , which the St Helens records did not note although the upper limit of the husband's income was stipulated for admission to the hospital (Dye, 1987). New Zealand maternity hospitals did not record ethnicity because there was little ethnic diversity in the population. Thus, the New Zealand maternity hospital records are very similar to the records collected in other Anglophone countries at the same time.

Between 1907 and 1922 there were 3,166 births at the Wellington St Helens Hospital. Our aim was to link as many boys birth records to World War II enlistment records. Between a birth at St Helens and enlistment in World War II there were many stages at which an individual could be eliminated from the sample. In Table 1 we summarize the construction of the sample from birth to World War II.

Table 1. Sample construction from records of St. Helens Hospital and World War II enlistment files

Sample	Number	Proportion of previous line
All births, 1907-1922	3166	
Births involving boys	1566	0.49
Live births	1523	0.97
Singleton births	1496	0.98
Identified in official birth registers	1459	0.98
Identified in World War II enlistment	843	0.58

The first stage in constructing our sample was to focus only on live, singleton male births. Because these were maternity hospital records, complete records of stillbirths were kept. Although the female growth trajectory is also of interest, our record of early adult health comes from military records. While several thousand New Zealand women served in auxiliary or nursing roles in World War II, they were a small fraction of women in this birth cohort. By contrast, based on military enlistment figures and the eligible age for conscription during World War II, we expected that approximately half of the men surviving to adulthood would have enlisted in the New Zealand armed forces. As Table 1 shows, we were able to match more than half the boys whose names we could identify in birth records to military records in World War II. Because the St Helens records were hospital birth records, and recorded the characteristics of the mother and attributes of the birth, they did not record the child's name. The St Helens casebooks recorded only the mother's name. However, we were able to obtain the names of children based on the mother's name, the known location of birth (Wellington) and residence of the parents (mostly Wellington), and the exact date of birth. These details were matched to births in the official birth registers maintained by the Registrar of Births, Deaths and Marriages. Records more than one hundred years old are publicly available,

while newer records are confidential, and can only be accessed with the permission of the Registrar of Births, Deaths and Marriages. We searched publicly available (1907-31 December 1909) records ourselves through the Births, Deaths and Marriages historical records search interface. The Births, Deaths and Marriages office searched for boys born after January 1910 for us, and provided us with a spreadsheet of boys' names. We used wildcard searching to expand the chances of making links if a match was not made initially. Our match rate of 98% between the maternity hospital records and the official birth registers represents just 37 boys out of 1496 singleton, live male births that did not appear to be registered. New Zealand was regarded as having an effective and nearly complete civil registration system, and this result confirms a very high level of coverage.

Individuals were linked to their World War II records by matching on the following variables available in both maternity 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 1907 and 1922, this limited set of information was sufficient to make exact matches with a high degree of confidence. We traced individuals into all three branches of the military during World War II. All forces collected similar information on men enlisting including socio-economic information, and an extensive medical examination. In particular men enlisting detailed their family background, educational qualifications, current employment, and religion. The medical examination was designed to be thorough, but the most consistently collected information was on height, weight, and blood pressure. Recruits were asked to

provide details on significant childhood diseases they had suffered. Because responses were open-ended the definition of "significant" disease appeared to vary between men, and we do not believe the unstructured collection of this data provides usable information.

Results and analysis

Our analysis focuses first on the characteristics of the sample at birth, describing both the linked and unlinked sample, and then on the relationship between birth and adult health. While we found statistically significant differences between the weight at birth of men who enlisted and those who did not, the magnitude of the differences was small.

Birth weight and length

Babies born at St Helens Hospital between 1907 and 1922 averaged a healthy 3467 grams at birth. There was no trend towards increasing or decreasing birth weight over time. Consistent with both historical and modern research, boys at St Helens weighed slightly more than girls. Across the 15 years of records, boys at St Helens weighed 3531 grams at birth, while girls weighted 3403 grams at births (Figure 1). Despite the great interest in New Zealand infant welfare in the early twentieth century because of the reduction in infant mortality, there has been no historical evidence on how birth weights in New Zealand compared to those overseas. Comparison with contemporary and historical research on birth weight in comparable countries shows that the Wellington St Helens babies were around the same size as babies born in the United States and Australia, and larger than babies born in the United Kingdom

(Table 2). Slightly fewer of the New Zealand babies were less than 2500 grams than at the New York Lying-in Hospital studied by Costa for a similar time period (Costa, 1998). As Costa found both a higher proportion of low birth weight babies, and a higher mean weight, this indicates that the New Zealand babies were more homogenous at birth than the United States ones. Comparison with Robertson's Australian figures is instructive. The New Zealand female babies weighed slightly more than those in Adelaide, but the male babies slightly less (T. Robertson, 1915a). Given that we cannot know precisely how selectivity by socio-economic status varied between the various countries, it is important not to make too much of small differences. The evidence is clear that babies of working class parents in New Zealand were as well-nourished as those in similar hospitals in Australia and the United States. Babies in all three "new world" countries were significantly heavier than babies in the United Kingdom.

Length at birth varies less than weight, and is less useful as an indicator of fetal nutrition. Under nutrition while in utero is manifested in being lighter than the potential weight, rather than being shorter. Length was also not as consistently measured at St Helens. Whereas 112 of the 3023 live, singleton births at St Helens were missing weight measurements, 263 were missing length. With this in mind, the St Helens babies averaged 52cm at birth, slightly longer than observed in other similar populations such as the New York Lying in Hospital.

Selectivity of the linked sample

Selectivity of the sample of men linked to their World War II enlistment records is a key issue in how far the results from this analysis can be generalized. If there is significant selectivity by birth weight in the men we find in World War

II, then the results are not as generalizable to a wider population. We do find differences in birth weight between men who enlisted, and those who did not. On average, men who enlisted weighed 3564 grams at birth, compared to 3487 grams for men who did not enlist in World War II. The difference is statistically significant (p=0.008), but inspection of the distribution of birth weights for both groups shows that there were not large differences between the two groups. There were slightly more men of lower birth weight in the group that did not enlist, and slightly heavier babies were more likely to enlist. A Kolmogorov-Smirnov test for equality of the distribution functions cannot reject the hypothesis that the two distributions were equal. A probit estimation of the chances of enlisting in World War II on weight at birth shows that every 1kg increase in birth weight only increases the chance of enlisting by 6.4%. A boy at the low birth weight margin of 2500g had a 52% chance of being found in the World War II records, while a boy of 4kg (one standard deviation larger than the mean) had a 60% chance of being found in the World War II records. The differences are statistically significant, but not large.

Relationship of birth weight to adult height

Consistent with modern research birth weight was strongly related to final adult height. For the 98 men who weighed less than 3000 grams at birth, the average height on enlistment in World War II was 169.6cm. For men between 3000 and 3500 grams average height was 1.5cm greater, and men weighing up to 4000 grams averaged 172.6cm when they finished growing. Controlling for maternal age, birth order, marital status of the mother, and year

of birth we estimate that for every one-kilogram increment in birth weight, a man was 2.5cm (approximately one inch) taller in adulthood (Table 3). These effects are significant, and comparable to the effects found for socio-economic differences. In a study of New Zealand soldiers in both World War I and World War II Inwood, Oxley and Roberts found that farmers were approximately one inch taller than urban manufacturing workers (K. Inwood et al., 2009; Kris Inwood et al., 2010). Similar differences in attained adult height between farmers and manufacturing workers have been found in the American Midwest (Steckel & Haurin, 1995).

Length at birth had a smaller quantitative impact on attained adult height. For every 1cm increment in birth length, a man was just 0.15 of a centimetre taller in adulthood. A man who was one standard deviation heavier than average at birth grew up to be 1.5cm taller in adulthood. By comparison, a man who was one standard deviation longer at birth was just 0.5 cm taller in adulthood. Birth weight, not length, exercised more of an influence on adult height. This finding is consistent with the modern literature that has examined size at birth and adult height (Sorensen et al., 1999).

Maternal age and parity had offsetting effects on adult height. Each additional year of a mother's age was associated with an increase in adult height for boys of 0.075cm. However, this implies that otherwise equivalent children born to mothers in their early 20s or late 30s would differ in height by a full centimeter. These estimates of weak but consistent positive effects of maternal age on adult height are consistent with modern epidemiological studies (Eide et al., 2005; Sorensen et al., 1999). Parity progression had a stronger effect on adult height. Each subsequent pregnancy was estimated to reduce adult height by just

over one-third of a centimeter. Again, these effects are consistent in direction with the modern literature (Eide et al., 2005), but of a somewhat larger magnitude. The cohorts examined here grew up during a long period of economic uncertainty in New Zealand (Greasley & Oxley, 2009). In particular, growth in New Zealand was weak during the 1920s, and economists have characterized the period from the end of World War I through the Great Depression as a long depression. While the economy recovered quickly after 1934, only the later cohorts considered here—born 1920 through 1922—would have hit their adolescent growth spurt after 1934. It is likely that the impact of maternal parity on children's height varies across social and economic conditions.

Relationship of birth weight to adult systolic blood pressure

The relationship between birth weight and blood pressure in adulthood has been central to the debate over the developmental or fetal origins hypothesis (Almond & Currie, 2011). Since Barker's early work in the 1980s there has been significant medical and epidemiological research into the connection (Barker, 1998, 2004). Recent summaries of the literature by Huxley *et al*, and Lawlor and Davey-Smith suggest the range of associations in modern studies lies between a decrease of 1.5 to 2 mm/Hg for each one kilogram increase in birth weight (Huxley et al., 2002; Debbie A Lawlor & Smith, 2005).

We find results slightly higher than the values in the modern literature. (Table 4). Without controlling for current weight, we estimate that every one kilogram increase in birth weight is associated with a decrease in systolic blood pressure of 2.4 mm Hg. Controlling for current weight or body mass index

marginally decreases the size of the inverse association to 2.3 mm Hg/kg. However, not all recruits measured in World War II provided all measurements. Estimating the relationship between systolic blood pressure and birth weight without current BMI on the sample for whom adult height and weight were measured, leads to further attenuation in the size and statistical significance of the relationship between birth weight and adult blood pressure (Table 4). Socio-economic factors such as maternal age and parity do not have a significant effect on adult blood pressure, whereas they were associated with adult height.

Discussion and conclusion

Along with the consistency between our results and modern ones on the size of the association between birth weight and blood pressure, this suggests that the relationship between birth weight and adult height is mediated by socio-economic factors. Conversely, the relationship between birth weight and blood pressure may be measuring a relatively stable biological relationship that does not change much in different socio-economic conditions.

The fetal origins hypothesis has attracted significant attention from social and medical scientists in the past two decades, but has not been adequately tested on historical birth cohorts. Using a unique data set of measured birth weights and health measurements in early adulthood, we examine the relationship of size at birth with adult height and blood pressure in an earlier cohort than hitherto studied. The successful linking of maternity records to military enlistment records opens up new possibilities for historical research on early life conditions and later life health. Consistent with modern epidemiological studies we find that birth weight has a statistically significant

relationship with adult height and blood pressure. Our estimates are consistent with the range of values given by modern epidemiological research. Some of our estimates suggest greater sensitivity of health to socio-economic differences in this historical cohort compared to modern ones. This suggests that while the association between size at birth and adult health may vary over time and space with social and economic conditions, it does so in a biologically determined range. Being smaller at birth had consequences in the past, and continues to have consequences to the present day.

Table 2. Comparison of St Helens and international birth size

Sample	Years	Female		Male		Less than 2500g
		Weight (grams)	Length (cm)	Weight (grams)	Length (cm)	
Wellington St Helens	1907-1922	3403		3531		4.2%
Stanford University Medical School Baby Clinic (Faber, 1920)	1906-1919	3305		3495		
New York Lying-in Hospital (Costa, 1998) (Medians, not means)	1910-1931	3430	51	3500	51	5.5%
Minneapolis General Hospital (Brenton, 1922)	1917	3282		3419		
Lambeth Lying Hospital (Pearson, 1899)	c. 1900	3208	51	3312	52	
Westminster Health Society, London (T. Robertson, 1916)	1914	3210		3310		
Leeds Babies Welcome (T. Robertson, 1916)	1914	3200		3300		
Birmingham Maternity Hospital (T. B. Robertson, 1915b)	c. 1914	3217		3257		
Queens Home, Adelaide, South Australia (T. Robertson, 1915a)	1909-1913	3410		3590		
Japan (Misawa, 1909)	c. 1905	2870	49	3040	49	

Table 3. Effect of birth weight on adult height

	Coefficient	Standard error	t	P > |t|
Birth weight (kg)	2.470	0.499	4.951	0.000
Length at birth (cm)	0.351	0.218	1.605	0.109
Mother married	-1.309	4.429	-0.296	0.768
Maternal Age	0.075	0.039	1.922	0.055
Number of pregnancies	-0.369	0.129	-2.853	0.004
Born in 1907 (reference)				
Born in 1908	-0.373	3.336	-0.112	0.911
Born in 1909	1.124	3.274	0.343	0.731
Born in 1910	-2.669	3.378	-0.790	0.430
Born in 1911	-1.205	4.552	-0.265	0.791
Born in 1912	0.510	3.223	0.158	0.874
Born in 1913	0.577	3.260	0.177	0.859
Born in 1914	0.266	3.220	0.082	0.934
Born in 1915	-0.137	3.227	-0.043	0.966
Born in 1916	0.650	3.190	0.204	0.839
Born in 1917	1.644	3.160	0.520	0.603
Born in 1918	1.293	3.160	0.409	0.682
Born in 1919	1.237	3.177	0.389	0.697
Born in 1920	0.844	3.159	0.267	0.790
Born in 1921	1.172	3.181	0.369	0.713
Born in 1922	1.675	3.309	0.506	0.613
Constant	155.398	6.087	25.530	0.000
R^2	0.090			
N	705			

Table 4. Effect of birth weight on blood pressure.

	Model 1 No control for current BMI	Model 2 Controls for current BMI	Model 3 No controls for current BMI if BMI measured
	(b/se/t)	(b/se/t)	(b/se/t)
Birth weight (kg)	-2.443	-2.325	-2.160
	1.250	1.413	1.406
	-1.954	-1.645	-1.536
Adult BMI at enlistment		0.299	
		0.261	
		1.148	
Length at birth (cm)	-0.029	-0.155	-0.166
	0.213	0.240	0.239
	-0.136	-0.648	-0.692
Born in 1908	-1.223	-0.046	-1.155
	8.116	8.320	8.266
	-0.151	-0.006	-0.140
Born in 1909	-6.029	-5.525	-6.630
	7.870	8.172	8.118
	-0.766	-0.676	-0.817
Born in 1910	2.575	4.234	3.021
	8.156	8.480	8.416
	0.316	0.499	0.359
Born in 1911	-17.083	-5.059	-6.419
	12.326	16.014	15.976
	-1.386	-0.316	-0.402
Born in 1912	-4.230	-4.422	-5.175
	7.816	8.025	8.000
	-0.541	-0.551	-0.647
Born in 1913	-6.330	-5.710	-6.777
	7.834	8.119	8.069
	-0.808	-0.703	-0.840
Born in 1914	-1.284	-0.482	-1.423
	7.755	7.938	7.898
	-0.166	-0.061	-0.180
Born in 1915	-5.671	-5.046	-6.086
	7.781	8.016	7.967
	-0.729	-0.630	-0.764
Born in 1916	-4.910	-3.673	-4.680
	7.693	7.891	7.845
	-0.638	-0.466	-0.597
Born in 1917	-3.453	-2.382	-3.605
	7.610	7.806	7.736
	-0.454	-0.305	-0.466

Born in 1918	-4.763	-2.934	-4.063
	7.602	7.798	7.738
	-0.627	-0.376	-0.525
Born in 1919	-4.746	-3.544	-4.723
	7.659	7.851	7.785
	-0.620	-0.451	-0.607
Born in 1920	-6.516	-4.788	-6.077
	7.604	7.829	7.751
	-0.857	-0.612	-0.784
Born in 1921	-5.909	-4.334	-5.648
	7.657	7.894	7.813
	-0.772	-0.549	-0.723
Born in 1922	-4.974	-3.672	-5.235
	7.980	8.309	8.199
	-0.623	-0.442	-0.639
Maternal Age	0.081	0.119	0.114
	0.097	0.107	0.107
	0.828	1.112	1.065
Number of pregnancies	0.552	0.260	0.279
	0.323	0.355	0.354
	1.706	0.733	0.786
Mother married	17.965	16.353	17.730
	10.652	10.887	10.824
	1.686	1.502	1.638
_cons	128.087	127.732	134.218
	14.917	16.946	15.981
	8.587	7.538	8.398
R^2	0.041	0.041	0.038
N	654	537	537

Figure 1. Birthweight by sex

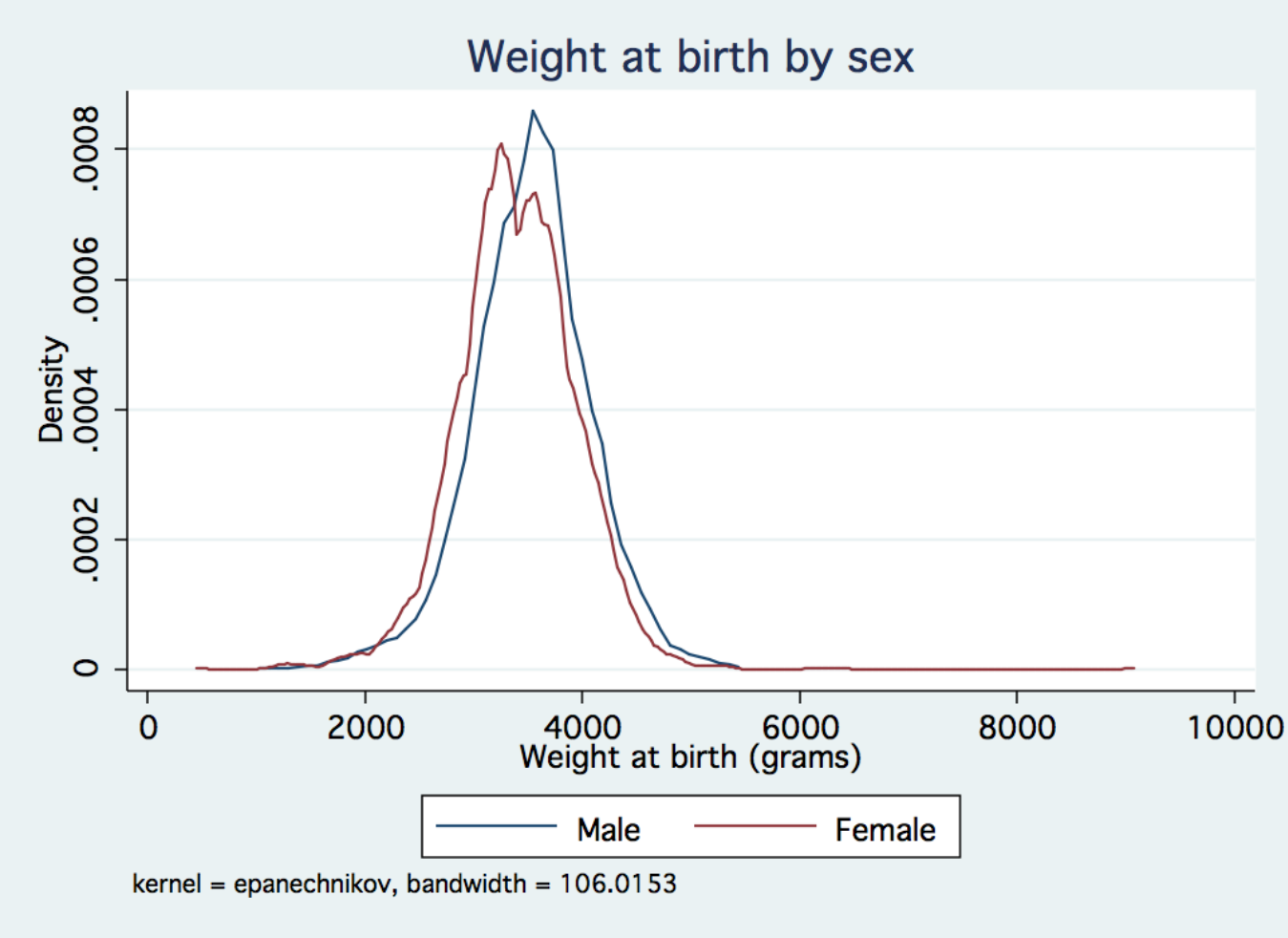
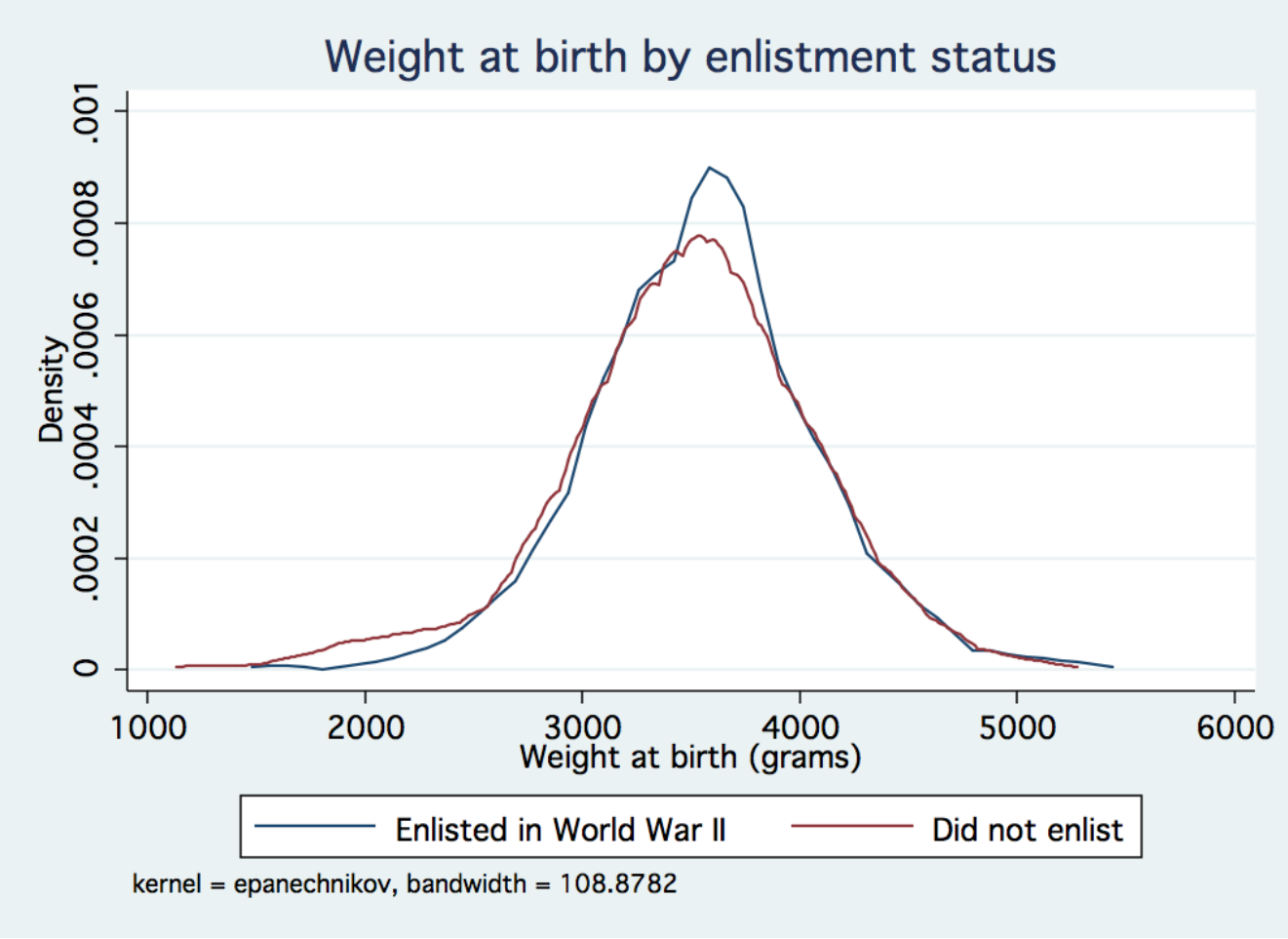


Figure 2. Birthweight and enlistment status



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