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

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ABSTRACT

We provide new historical evidence on the developmental origins of health and disease in a cohort of boys born between 1907 and 1922 in Wellington, New Zealand. Using a dataset of 1523 birth records that include birth weight and length we find 852 (58%) of the adult cohort in World War II records measuring stature, body mass and blood pressure. On average, the boys weighed 3.5 kg at birth, similar to Australian and American babies of the era, and nearly identical to full-term New Zealand babies in the 1990s. Using OLS regression models we estimate the effect of birth weight on adult stature and systolic blood pressure. We find an increase in birth weight of 1 kg is associated with an increase in stature of 2.6 cm (95% confidence interval [CI] 1.6 cm–3.6 cm), and a decrease in systolic blood pressure of 2.1 mm/Hg (95% CI −5.00 to 0.67). This is the earliest cohort by fifty years for whom the fetal origins hypothesis has been examined in early adulthood. Our estimates of the effect of birth weight on blood pressure are towards the upper end of the range of published estimates in modern cohorts.

1. Introduction

The hypothesis that conditions in early life—social, economic, environmental, nutritional, or experiencing illness—can have long run effects on individual and population health can be traced to at least the early twentieth century (Kermack et al., 1934). Social and medical scientists have given increasing attention to this idea since the late 1980s. The work of David Barker and colleagues was particularly influential in drawing attention to the potential connections between nutrition before birth, birth weight, later adult health and subsequent cardiovascular mortality (Barker et al., 1989a; Barker and Osmond, 1986; Barker et al., 1989b). Barker's influence on the field is acknowledged in the widespread use of the term "Barker hypothesis" to denote the idea that adult diseases and mortality can have fetal origins (Almond and Currie, 2011). The fetal origins hypothesis does not posit that adult health outcomes are predestined by fetal conditions, and thus closely related, but slightly broader literatures investigate the "developmental origins of health and disease" (Gluckman et al., 2010) and life course influences on health (Ben-Shlomo and Kuh, 2002).

A significant amount of the literature on the fetal origins hypothesis by Barker and others has addressed the specific question of how birth weight is associated with blood pressure in later life. Evidence from modern studies has shown that increases in birth weight of 1 kg are associated with declines in systolic blood pressure of between 1 and 4 mm/Hg (Gamborg et al., 2007). Conversely, increases in birth weight are associated with increases in adult stature (Sorensen et al., 1999). With lower blood pressure and increased stature both associated with lower adult mortality, there is strong evidence that increased birth weight is associated, at least indirectly, with improved health in later life even for babies well over the clinical low birth weight threshold of 2.5 kg.

The fetal and developmental origins literature and life course epidemiology emphasize that health conditions at any point in life are influenced by experience over a person's life from conception on. Accumulated experience will differ among individuals within a similar cohort, and at a population level across socio-economic and environmental contexts. An important and under-recognized implication of this literature is that the relationship between early life health and later life outcomes may change over time and differ across geographic areas as different cohorts are exposed to *inter alia* different environmental conditions, nutritional practices, disease exposure, and medical care. Because the fetal origins literature has largely developed since the 1980s scholars have been able

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to measure cohorts born before World War II in later life, and only been able to measure younger cohorts born since the 1960s.

This paper provides the earliest ever evidence on the fetal origins hypothesis in young adults. Our cohort was born in Wellington, New Zealand between 1907 and 1922. We use maternity hospital records linked to military enlistment records for men enlisting in World War II to obtain information on size at birth and health in early adulthood. Combining the information from these records allows us to address several closely related questions about birth weight and early-adult health. Specifically, we measure the

- Size at birth of infants in New Zealand as an indicator of child health during the New Zealand infant mortality decline,
- Association of birth weight and birth length, with adult height
- Association of birth weight and birth length, with adult blood pressure: the fetal origins hypothesis

While modern studies collecting data prospectively can go beyond body composition to more precise measures of nutrition, growth and health, only anthropometric data is available in historical populations. Our data combines accurately measured—rather than recalled—birth weight and reliable measures of blood pressure. Although other authors have examined the fetal origins hypothesis in cohorts born in the early twentieth century (Eriksson et al., 2004), our study is the first to test the hypothesis in these cohorts in young adulthood. Indeed, our cohort is born at least 45 years earlier than any group for whom the fetal origins hypothesis has been studied in young adults (Järvelin et al., 2004).

Evidence from New Zealand is especially interesting because of its apparently very healthy population in the early twentieth century. New Zealand, like Scandinavia, had the earliest sustained infant mortality decline in the industrialized world (Edvinsson et al., 2008; Woodbury, 1922). Contemporary commentators attributed New Zealand's decline to the establishment in 1907 of the infant welfare group: the Plunket Society (Bryder, 2003). But the New Zealand infant mortality decline started in the 1880s, and there is no demographic evidence Plunket caused the decline. A similar trajectory of decline was seen in Scandinavia, which like New Zealand was wealthy with low population density (Edvinsson et al., 2008; Mein Smith, 1988). Thus, this paper also brings overdue attention to measured health outcomes among cohorts in the New Zealand infant mortality decline.

1.1. Results of prior studies: 1. Association of birth weight and length with adult height

The relationship between birth and adult size has interested physical anthropologists and others for several centuries (Baldwin, 1921; Bogin and Kapell, 1997; Tanner, 1981) But there were few large studies of growth from birth to maturity until the twentieth century (Young et al., 1991). Many early twentieth century studies of childhood growth recruited samples in schools, rather than at birth. Both the early twentieth century growth studies, and research since the 1970s have provided significant evidence that weight and length at birth are correlated with adult height. For example, a study of Danish conscripts born in the 1970s found that average stature of men weighing 2.5–3 kg at birth was 4 cm less than men who weighed 3.75–4 kg at birth (Sorensen et al., 1999). Similar results were observed in a large study of Norwegian conscripts (Eide et al., 2005). In the 1958 British birth cohort an increase in birth weight of 1 kg was associated with a 2 cm increase in adult height (Li et al., 2004).

The association between size at birth and adult stature is important because stature is associated with morbidity and mortality (Engeland et al., 2003a, 2003b; Silventoinen, 2003; Waaler, 1984). The association between height and mortality risk reflects that adult height summarizes environmental conditions—particularly caloric intake and disease burden—experienced during the growth period. A significant amount of research has focused on the inverse relationship between height and coronary heart disease (Paajanen et al., 2010; Silventoinen et al., 2006). 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 (Asao et al., 2006). Height is also inversely related to stroke, respiratory disease, and stomach cancers (Smith and Lynch, 2004). But if stature itself is associated with birth weight, then analyses of stature and health outcomes that do not control for birth weight will overestimate the effect of conditions in childhood (measured by stature) compared to conditions in-utero (measured by birth weight).

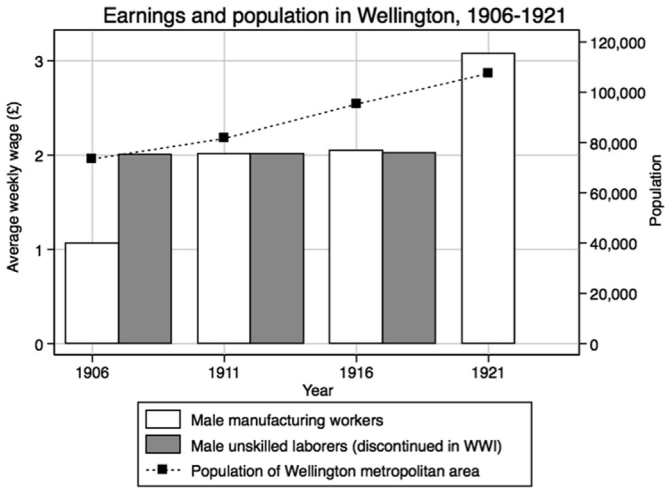

Fig. 1. Earnings and population in Wellington, 1906–1921.

1.2. Results of prior studies: 2. Association of birth weight and length with blood pressure

The relationship between birth weight and blood pressure in adulthood is central to research on the developmental or fetal origins hypothesis (Almond and Currie, 2011). Since Barker's early work in the 1980s there has been significant medical and epidemiological research into the connection (D.J.P. Barker, 1998). Recent comprehensive summaries of modern literature by Huxley et al., and Lawlor and Davey-Smith suggest that for each one kilogram increase in birth weight systolic blood pressure decreases of 1.5–2 mm/Hg (Huxley et al., 2002; Lawlor and Smith, 2005). Much of the modern literature studies the relationship when subjects are aged 50 or older, significantly older than our sample aged 18–35 at enlistment. Because blood pressure changes throughout the life course it is unclear whether the association between birth weight and blood pressure changes over time (Davies et al., 2006; Huxley et al., 2002).

2. Data

Our sample comes from births at Wellington's St Helens Hospital between 1907 and 1922. The St Helens Hospitals were established during an era of concern about high maternal and infant mortality. Following the 1904 Midwives Act seven St Helens hospitals were established in New Zealand cities between 1905 and 1920. They provided state-subsidized maternity care for working-class women whose husbands earned less than £3 (1905–1912)