Long-run Health and Mortality Effects of Exposure to Universal Health Care at Birth

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Abstract

In this paper we investigate to what extent access to universal healthcare influences later life health outcomes. We examine a fundamental re-organisation of the healthcare environment to universal healthcare in the United Kingdom, which occurred through the introduction of the National Health Service (NHS) in July 1948. Immediate large decreases in infant mortality ensued, which were focused on the neo-natal period and larger for individuals who prior to the NHS had a lower access to medical services. Using administrative data on mortality, we compare mortality outcomes above age 50 of individuals born in the immediate cohorts around the introduction of the National Health Service (NHS) in a regression discontinuity design. We additionally exploit geographical variation in the change in medical services upon the NHS introduction for identification. Our findings indicate that age-specific survival rates are systematically higher among lower class individuals whose post-natal care expanded through the NHS. We supplement these findings with analysis of hospital records, which reveal a decrease in hospitalisations for cardiovascular disease for lower class individuals. These long run impacts of birth exposure to universal healthcare coverage through the NHS are economically significant, representing a 16% reduction in mortality by age 64.

JEL classification: I12, I13, I14, J14

Keywords: Universal health care, early life intervention, life cycle impact, mortality, health

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"*The astonishing fact is that Bevan's vision has stood both the test of time and the test of change unimaginable in his day. At the centre of his vision was a National Health Service, and sixty years on his NHS – by surviving, growing and adapting to technological and demographic change – remains at the centre of the life of our nation as a uniquely British creation, and still a uniquely powerful engine of social justice.*"
(Gordon Brown, UK Prime Minister, 2008)

1 Introduction

Healthcare environments around birth substantially affect early life survival and infant health. A growing literature recognises that infancy is a key developmental period, and that adult health and mortality may be substantially shaped by adverse or beneficial infancy environments. Most investigations of this link focus on healthcare interventions that are either i) targeted towards specific population groups rather than universal, usually those at risk of experiencing adverse environments, ii) timed to specific periods such as prenatal versus postnatal care, or iii) aimed at specific health outcomes or behaviours, such as breastfeeding or nutrition programs. In contrast, the development plan of the United Nations spearheads a movement towards universal healthcare provision, i.e a program that is universal, accessible at any age, and aimed at improving general population health. A key question is whether early life exposure to universal healthcare delivers long-lasting benefits, and whether these are similar to those of targeted programmes.

In this paper, we investigate the long-run impacts of infancy exposure to universal healthcare. We examine the health and mortality outcomes of individuals 50 to 60 years after exposure to the National Health Service (NHS), a universal healthcare system introduced by the UK Government in July 1948. The inception of the NHS saw a fundamental re-organisation of the healthcare environment, introducing universal access to medical services for the entire population. Our analysis is possible due to the coincidence of two factors: first, the introduction of the NHS is a large-scale historical intervention that reaches back long enough to offer the opportunity to study health and mortality impacts at older ages. Second, there has been recent innovations in the availability of large administrative datasets, which hold information about individuals throughout their life course.

The benefits of extending the availability of healthcare to populations with inadequate access to medical services can be most readily seen in infant health. Almond, Chay, and Greenstone (2018) show that federally mandated desegregation of hospitals in the US southern states following the 1964 Civil Rights Act along with the introduction of the Medicaid and Medicare programmes in 1965 which prohibited payments to hospitals with racially discriminatory practices, improved the infant mortality rate of black infants relative to white. Analysing the introduction of Medicaid in the US between 1966-1970, Goodman-Bacon (2018) finds that states with high-eligibility rates for Medicaid saw substantial declines in both infant and child mortality rates relative to low-eligibility states, with improvements strongest for non-white children. In the 1980s the Medicaid programme was expanded, Currie and Gruber (1996a,b) find that increasing the availability of healthcare has large impacts particularly on perinatal (stillbirths and deaths under 7 days) and neonatal (deaths within 28 days of birth) mortality rates.

Infancy is a key developmental period for both mental and physical health as well as cognition. A large literature suggests that the origins of adult disease or mortality often lie in adverse or beneficial early life environments. Three factors may be at play: First, improved infancy care may prevent an early decumulation of the health stock of a newborn. Second, and a similar environment change may have larger effects in infancy than at later ages, as it is a key period of fast neurological development (Bhalotra and Venkataramani, 2011). Finally, medical research suggests that infancy environments may lead to biologically embedded adjustments in humans, triggering disease response which may take decades to manifest (Shonkoff, Boyce, and McEwen, 2009).

A growing literature examines the impact of early childhood healthcare interventions beyond infancy and into adolescence and adulthood, (for an overview see Almond, Currie, and Duque (2017)). Analysis of Medicaid expansions find mortality reductions persist into childhood and adolescence, as well as improvements in educational attainment and lower welfare dependency (Currie, Decker, and Lin (2008); Brown, Kowalski, and Lurie (2015); Wherry and Meyer (2016)). These policy interventions can now be analysed for their impacts on outcomes in early adulthood, but the earliest date at which data on their long-run impacts that reach to retirement age can be ob-

tained would be in the 2040s. The Well-Child Programmes, instituted in the 1930s in various Scandinavian countries, in contrast, do allow for a long-run investigation, and the results point to life-long impacts. These programmes were universal and targeted at infants within the first year life. In Norway and Denmark home visiting programmes led to a significant increase in infant survival attributable to a decrease in mortality from diarrhea-related causes (Hjort, Sølvsten, and Wüst (2017); Bütikofer and Salvanes (2018)), whereas for Sweden, Bhalotra, Karlsson, and Nilsson (2017) find reduced infant mortality due to prematurity. These programmes all had long-term impacts on adult health, particularly a reduction in the incidence of cardiovascular disease and age-specific mortality.

Universal healthcare is distinct from these programmes, as it is available to the entire population, regardless of age, and so is aimed at improving general population health. In our contribution we show that access to a universal healthcare system, which by definition is not targeted towards specific population groups, delivers mortality gains that persist into later life.

We identify the long-run health and mortality impacts of infancy exposure to universal healthcare using two methods. First, as the NHS was introduced nationwide on a single date, we employ a Regression Discontinuity Design, where we allow for pre-existing trends in the outcomes to be different either side of the threshold (i.e. being born in a narrow window around the NHS introduction). Additionally, we exploit geographical variation in available medical services in infancy (across counties). We develop a simple model of medical services based on competition among (a changing number of) patients for access to medical services, which predicts that areas with a larger proportion of poorer individuals, for whom healthcare could be unaffordable, should see larger reductions in mortality after the expansion of access to healthcare. We also allow for competition to adversely affect individuals who could afford healthcare prior to the NHS. We focus on mortality and the onset of cardiovascular disease at older ages in the analysis of later-life health outcomes.[1] We use age-specific mortality rates using data from the Office of National Statistics Longitudinal Study, a 1% sample of the population containing information on individuals linked between 5 successive censuses

[1]World-wide cardiovascular disease is the leading cause of death, in 2015 accounting for around 8.5 million deaths (World Health Organisation, 2017), and accounted for 11.5% of all deaths registered in England and Wales Office for National Statistics (2016).

combined with administrative death records. For the analysis of health impacts we use the UK Biobank, a rich high-quality micro-dataset linked to administrative hospital records. We observe individuals' infancy exposure to county-specific medical services through information on individual's location at birth recorded in both datasets.

Our findings indicate that survival rates between ages 52 and 64 are systematically higher among lower class individuals whose infancy care expanded through the NHS. The increase in the beneficial impact of the NHS on survival rates in this population group represents a 15-22% reduction in (age-specific) mortality. Our results further confirm the hypothesis that the benefits for this group are larger in areas where per-capital medical services increases were larger. In the parallel analysis of health records we find that the onset of cardiovascular disease amongst lower class individuals between age 52 and 64 is reduced by up to 5%. Conversely, we find evidence of crowding out between previously covered and incoming patients due to competition for local medical resources. The results indicate that the lack of expansion in the healthcare infrastructure at the inception of the NHS also has long-run implications: mortality rates around ages 50 and 60 increased for individuals with access to healthcare prior to the NHS. Mortality increases in this group are larger in areas with stronger competition. Both identification strategies proposed above yield the same qualitative result.

Our identification strategy is supported by additional evidence on the *immediate* impact of the NHS on infant health. While microdata dating back to the 1940s is not available, we have digitised detailed information on infant mortality at a low-level geographical disaggregation for this period. We find a decrease in infant mortality that was driven predominantly by improvements in neo-natal outcomes for individuals with lower socio-economic status. We also find that these improvements were larger in areas where a higher proportion of the population are of low socio-economic status.

Given this immediate increase in infant survival, which varies by social class and location of birth, later life changes in health and mortality may be the result of a combination of two factors: the long-run impacts of better childhood health outcomes, and selective survival at earlier ages. Our current estimates may underestimate the long-run impact of infancy exposure to the NHS, if infants who would not have survived prior to the NHS were, say, inherently

weaker or unhealthier than those who survived. Selective infant mortality may imply a lowering of overall population health for cohorts born (and surviving) after the NHS introduction, and may lower the average health and mortality of survivors in adulthood. Previous studies of early child interventions acknowledge this limitation, and derive bounds for their estimates of the long-run health premium under assumptions about the health distribution of those who died. In future work, we intend to extend the model in Bozzoli, Deaton, and Quintana-Domeque (2009), and implement it in estimation to separately quantify i) the health premium of enhanced health outcomes in adulthood due to improved childhood conditions, and ii) the selective mortality effect ensuing from increases in infant survival.

Our work has important policy implications for this debate. First, it suggests that improving early life healthcare provision plays a substantial part in reducing health disparities in childhood and in adulthood. Second, our findings are informative for developing countries in Latin America, Africa and Asia who are planning to, or recently have, implemented universal healthcare systems.[2] Indeed, in 2015 the United Nations launched a new development agenda comprising 17 Sustainable Development Goals (SDGs). A central aspect in this agenda is to "ensure healthy lives and promote well-being for all at all ages" (SDG3). Universal health coverage is understood as the key mechanism through which these health targets will be met (World Health Organization, 2016). Our results highlight the large and lifelong benefits for population groups with no prior access to healthcare in infancy, but also point to potential detrimental effects if these programmes are introduced without an adequate expansion of the healthcare infrastructure. Third, our analysis is one of few to be able to study impacts of early life interventions on later life outcomes. Existing evidence on a plethora of early childhood health, nutrition and cognition interventions has mostly found surprisingly large impacts on childhood outcomes, and increasing evidence pointing to lasting impacts into early adulthood and middle age. We present evidence that the benefits of early life conditions extend into later life as well. Finally, we quantify the long-run health dividend of NHS services in infancy, providing new evidence in the current discussion within the UK

[2]Maeda, Araujo, Cashin, Harris, Ikegami, and Reich (2014) present case studies on 11 countries which in recent years have adopted or are developing universal health coverage schemes.

regarding the financial pressures and potential reform of the NHS.

The remainder of the paper is structured as follows. Section 2 briefly sets out the institutional environment around the NHS introduction and discusses the impact on infant mortality. Section 3 presents the empirical methodology and data used in the mortality and cardio-vascular disease onset analysis, and discusses the results. Section 4 concludes and outlines the pathway for our subsequent analysis.

2 Institutional Setting

Access to adequate healthcare is fundamentally linked to population health. Following the 1942 Beveridge report, which highlighted the extent of social and health disparities within Britain, in July 1948 the UK Government introduced a National Health Service (NHS) whereby comprehensive health services were provided 'free at the point of use', instead being centrally funded through general taxation. The NHS was established with three main aims: i) free provision of healthcare, ii) access based on clinical need, not ability to pay, and iii) equalisation of access to medical services (see NHS Constitution). Public demand for healthcare quickly exceeded predictions. The primary beneficiaries were individuals in the lower tail of the income distribution, who were able to access previously unaffordable health services.

Prior to the NHS, healthcare was mainly provided privately for a doctor's or hospital fee.[3] Limited access to free healthcare was provided by voluntary hospitals who depended on private donations (and the agreement of such donors to sponsor a patient in need), or through hospitals operated by local authorities, based on the Poor law. Both institutions were suffering severe financing problems in the early 1940s (Rivett, 1998). Local authorities also had governance over public health programmes, providing *inter alia* tuberculosis sanatoria, treatment and prevention of infectious disease through vaccination schemes, and family health services such as ante-natal clinics, domiciliary midwifery and district nursing.[4] Following the 1944 Education

[3]Prices were set often according to a patient's ability to pay. Anecdotal evidence suggests the going rate for a doctor's visit was around 5 shillings - in context the average weekly wage was 110 shillings in 1947 (Flexner, 1947).

[4]The 1918 Maternal and Child Welfare Act mandated local authority provision of ante-natal clinics. According to Ministry of Health records 75% of expectant mothers attended

Act, local authorities also provided free medical services to children attending maintained schools.

In addition, workers were partially covered through a highly fragmented network of ca. 6,000 Approved Societies, established under the National Insurance Act 1911 (Carpenter, 1984). Compulsory cover was provided to employed persons aged 16 to 70 with annual earnings below £420 (and all manual labourers).[5] Approved Societies were not-for-profit private organisations who could refuse to provide insurance coverage to workers.[6] Contributions, deducted from wages by the employer, amounted to seven (six) pence a week for men (women), of which the employer contributed 3 pence, and were topped up with government subsidies of about two pence per worker. Insured workers were entitled to rudimentary medical care from doctors who received a fixed 'capitation fee' per patient.[7] The non-comprehensive scheme often did not cover drugs nor hospital treatment; most importantly, these schemes did not cover medical services to their dependents such as women or children. National Health Insurance provided these limited services to between a quarter and a third of the population between 1911 and 1948.

Proposals for the new health service arrangements were initially met with strong opposition by the British Medical Association. After fraught negotiations, family doctors (known as General Practioners or GPs) agreed to participate in the new health service on 28th May 1948. In a large scale national information campaign, the population was encouraged to sign up on doctors lists in June 1948, ahead of the 'appointed day', 5th July 1948, that the saw the inauguration of the NHS. By July 6 (July 31), 84% (91%) of the population had signed up on doctors lists. By the end of 1948, 96% of the population had enlisted for a GP.

On the supply side, hospitals were taken into public ownership and vested to the Minister of Health in a centralised hospital service.[8] GPs became

a local authority ante natal clinic, and over half of births took place in the home (MoH, 1959).

[5]Self-employed persons could insure voluntarily.

[6]Workers who could not find an insurer paid into a private healthcare fund which was drawn down upon need.

[7]Additional entitlements were: sickness pay of 10 shillings per week for 26 weeks and disability benefits of 5 shillings a week beyond the 26 weeks; maternity benefits of 30 shillings per child.

[8]2,835 voluntary and local hospitals, with a total of 388,000 staffed beds were taken into the Ministry of Health. 277 hospitals, the majority belonging to religious communities,

independent contractors that were paid fixed fees per treatment set by Executive Councils, which decided on contracts and payments. By September of 1948 18,165 out of 21,000 GPs had signed up to become contractors of the NHS. As previously, local authorities continued to administer family health services such as maternity and child welfare clinics, midwives, and other services. Hence, while the NHS resulted in a fundamental change in accessibility to patients, medical services supply remained under administration by several entities. More importantly, the re-organisation of the healthcare environment wrought by the introduction of the NHS was neither immediately accompanied by large investments into healthcare infrastructure (Rivett, 1998),[9] nor by a discontinuous expansion in medical professionals.[10]

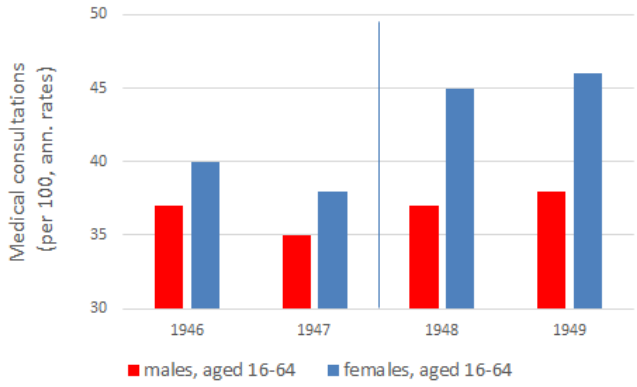
In summary, the introduction of the NHS provided, for the first time, universal access to health care in the UK. Before the NHS, the healthcare system mostly relied on private financing through fees, thus restricting access to those able to pay for such services. Public healthcare provision before 1948 was rudimentary and focused on working men, with no coverage for the elderly, for women or young children. From the inception of the NHS there was an immediate increase in the use of medical services, with an increase of 13% in medical consultations, and a doubling of prescriptions for proprietary medicines (MoH, 1949). Figure 1 shows that the rise in adult medical consultations was far greater for women (absolute and relative to men). At the time it was observed that "there can be little doubt that before the start of the New National health Service, many women [...] were deterred from seeking medical advice by economic reasons. Now that the financial barrier has been removed, women [...] are able to consult their doctor more often than they did before." (Logan, 1950).

remained outside the centralised service. Access to these were not denied to patients, as if it was deemed necessary the NHS could arrange for patients to be admitted to these disclaimed hospitals on a contractual basis (MoH, 1948).

[9]In the first five years of the service no new hospitals were built (MoH, 1951). Abel-Smith and Titmuss (1956) observe that capital expenditure on hospitals during this period was approximately one third of the pre-war level of expenditure.

[10]In 1946 15,484 midwives were employed, attending 93.1% of deliveries. By 1954 the number of midwives had decreased to 15,105, but attended a slightly higher proportion (96.4%) of births (CMB, 1957). During the 1940s the number of registered medical practitioners increased at rate of around 2% annually, however there was a virtual standstill in the number of GPs over the same period (MoH, 1955).

Figure 1: Medical consultations prior to and after the introduction of the NHS, by gender



Source: Survey of Sickness, The Wellcome Library.

As we will show in the next section, increased access to maternity healthcare and pediatric services had an immediate impact on infant mortality rates, which is particularly salient as compared to immediate mortality effects at older ages.[11]

2.1 The impact on infant mortality

Our analysis of the immediate impacts of birth exposure to the NHS is based on aggregate data on mortality in infancy, obtained from the Registrar General's statistical review of England and Wales and the Ministry of Health Annual Reports[12]. We digitised this historic datasource to illustrate the immediate impact of the NHS introduction in 1948 on infant mortality. We

[11] An examination of annual (period) mortality rates from the Human Mortality Database reveals a decline in deaths within the first year of life of approximately 20% between pre and post-NHS cohorts. In contrast there is no evidence of a clear and systematic drop in the mortality of older individuals (aged 50 and older) around or following the introduction of the NHS in 1948. This is consistent with US evidence of the increase in health insurance coverage at age 65 through Medicare eligibility, although Card, Dobkin, and Maestas (2008) find a reduction in health inequalities, but only a small impact on self-reported health and no impact on mortality rates.

[12] Both these series are held in the archives of the Wellcome Library. The data contain detailed mortality rates at different ages. However, these are period rather than cohort mortality rates. For example, death rates under age 1 in 1948 may contain deaths from babies born in 1947. Thus, we only consider mortality rates during the first year of life as under age 5 mortality rates in 1948 would contain a large fraction of children born prior to the NHS introduction.

mainly use nationally aggregated data for England and Wales to present our results, and complement this with data aggregated to the regional level.

Table 1: Mean mortality rate in infancy by time of death, 1940 to 1947

Period of death	Mortality rate (per 1000 total births)	Percent of mortality under 1 year	
		period	cumulative
Stillbirths	27.33	.3865	.3865
Under 1 day	35.76	.1193	.5058
Under 1 week	44.46	.1234	.6292
Under 1 month	50.93	.0917	.7209
Under 3 months	58.49	.1066	.8275
Under 1 year	70.72	.1725	1.000

Notes: The table shows cumulative deaths per 1,000 total births, including stillbirths in England and Wales.
Source: Registrar General's Annual report 1940-1955, The Wellcome Library.

First, we identify critical pre- and postnatal periods up to the end of the first year of life with high mortality rates in the period 1940 to 1947, the year before the NHS was introduced (see Table 1). For this purpose, we construct deaths per 1000 total births, thus including prenatal deaths. On average, we find that stillbirth[13] was the largest driver of early life mortality with 27 deaths per 1,000 total births, or about 39% of the total deaths recorded during pregnancy and up to the end of the first year of life. The first day also carried a high death rate of about 7 per 1,000 total births (approximately an additional 12%). By the end of the first week, these amounted to 44 deaths per 1,000 total births (accounting for 63% of total deaths within one year). 72% of total infant deaths occurred within the neonatal period[14].

Table 2: Mean neonatal mortality by week of death, 1940 to 1947

Period of death	Mortality rate (per 1,000 live births)	Percent of infant mortality rate
In week 1	17.61	0.3958
In week 2	3.01	0.0677
In week 3	2.05	0.0460
In week 4	1.55	0.0347
Total	24.26	0.5454

Notes: The table shows deaths per 1,000 live births, and mortality rates in percent of the infant mortality rate (defined as deaths from birth to under 1 year per 1,000 live births).
Source: Registrar General's Annual report 1940-1955, The Wellcome Library.

[13]Stillbirths are defined as births after 28 or more weeks completed gestation which did not, at any time, breathe or show signs of life

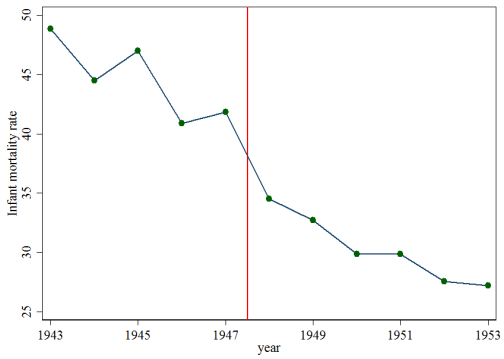
[14]The neonatal period is defined as the period between birth and one month. Neonatal mortality rates are calculated as the number of deaths per 1,000 live births

A closer look at the critical neonatal period, (see Table 2), shows that about 18 deaths per 1,000 live births occur in the first week of life, and death rates in the first 4 weeks of life are 24 per 1,000. The neonatal mortality rate is almost as high as prenatal mortality and accounts for 54% deaths within one year of birth. After these first 4 weeks, mortality drops significantly to under 8 per 1,000 live deaths per 3 month period.

Overall the data show that the pre-NHS period was characterised by very high mortality rates in infancy. Death rates amounted to 71 per 1,000 total births up to the end of the first year of life, and infant mortality rates amounted to 45 deaths (per 1,000 live births)[15]. Bringing these figures into context: infant mortality rates in the pre-NHS period were as high as modern day infant mortality rates in Sudan, Zambia or Turkmenistan, while neonatal mortality was as high as it is on average in South-East Asia in 2015.[16] In 2014, less than 9 deaths per 1,000 total births were recorded in England and Wales with an infant mortality rate of 3.6 per 1,000 live births.

Figure 2 shows infant mortality rates for cohorts born around the NHS introduction. There is a sharp reduction of approximately 17% between 1947 and 1948, coinciding with the introduction of the NHS.

Figure 2: Infant mortality by time of death



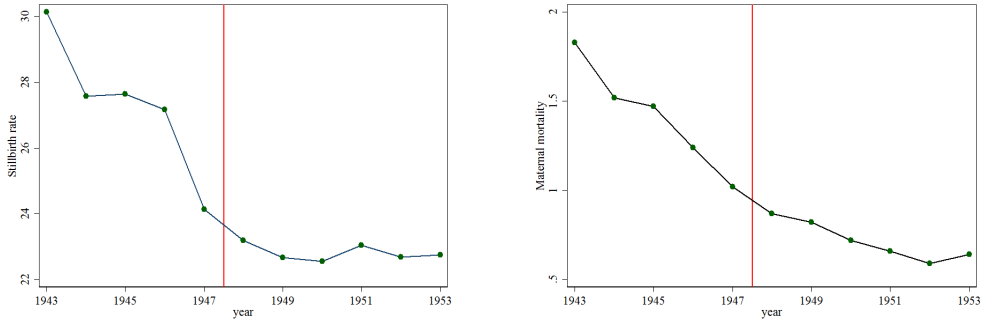
Notes: The graph shows deaths per 1,000 live births in England and Wales.
Source: Registrar General's Annual report 1940-1955, The Wellcome Library.

[15]Infant mortality rates describe mortality within the first year of life, thus excluding stillbirths, and are calculated as deaths within the first year of life as a proportion of live births.

[16]2015 Estimates Developed by the UN Inter-agency Group for Child Mortality Estimation (UNICEF, WHO, World Bank, UN DESA Population Division) at childmortality.org. Projected data are from the United Nations Population Division's World Population Prospects; and may in some cases not be consistent with data before the current year.

In contrast, although stillbirth rates declined substantially during the pre-NHS period, there is no evidence of a discontinuous change in antenatal mortality in 1948, measured though stillbirth rates per 1,000 total births around 1948 (see Figure 3a). Similar to stillbirth, we find no effect of the NHS introduction on maternal mortality per 1,000 total births (see Figure 3b).

Figure 3: Stillbirths and Maternal Mortality per 1,000 total births

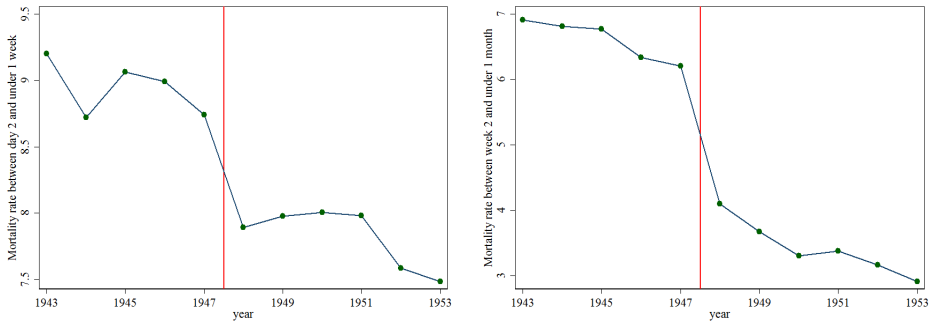


(a) Stillbirth (b) Maternal Mortality

Notes: The graph shows rates per 1,000 total births in England and Wales.
Source: Registrar General's Annual report 1940-1955, The Wellcome Library.

Figures 2 and 3 suggest that the decrease in infant mortality was driven by post-natal rather than ante-natal conditions. Examining neonatal mortality in detail, we find no NHS effects in the first 30 minutes or 24 hours of life, that would be suggestive of improved delivery methods through better health care access. However, we do find a significant reduction of 11% in deaths occurring between 1 day and 1 week of birth, and a one-third decrease in mortality between the second and the fourth week (see Figure 4). Overall, this amounts to a reduction in neonatal mortality of around 13%.

Figure 4: Neonatal mortality, by period of death



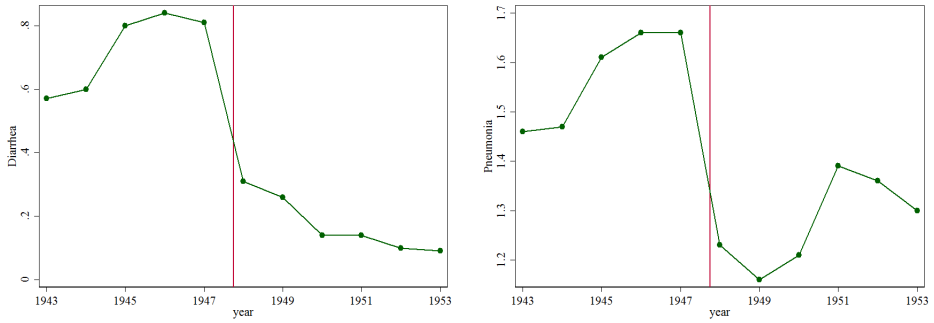
Notes: The graph shows deaths per 1,000 live births in England and Wales.
Source: Registrar General's Annual report 1940-1955, The Wellcome Library.

The Ministry of Health Annual reports also contain information on neonatal mortality according to certified cause.[17] Figure 5 shows large declines in two cause of death categories: a decline of approximately 0.35 deaths per 1,000 due to diarrhea (ca. 50% decrease); and a decline of 0.4 deaths per 1,000 due to pneumonia (roughly a 25% fall). Therefore these two causes alone contribute to approximately one third of the reduction in neonatal mortality. Both declining death causes, pneumonia and diarrhea, relate to conditions developed after birth, and both are particularly dangerous in the neonatal period where we observe the largest NHS-related decrease in infant mortality. As both pneumonia and diarrhea could be successfully treated during the 1940s, but require timely intervention, this suggests that the NHS resulted in more timely treatment or access to treatment when a critical health shock manifests.

Our conjecture is that these early life impacts of NHS exposure around birth differ by pre-NHS access to health care. More precisely, we expect stronger reduction in mortality rates among children of lower social class whose mothers would not have had health insurance before the NHS was introduced. This conjecture is consistent with the study of Dykes (1950) who finds a strong social class gradient in a case study of 2,000 infants born in an English town in 1946, and suggests that higher mortality in the lower social class groups is related to delay in accessing medical care. Unfortunately mortality statistics are not available by maternal (or childrens') social class or other

[17]Eight specific death causes common to this period, are listed: Asphyxia and Atelectasis, Bronchitis, Congenital Malformations, Diarrhea, Immaturity, Infective Parasitic Disease, Pneumonia and Other.

Figure 5: Neonatal mortality by cause of death

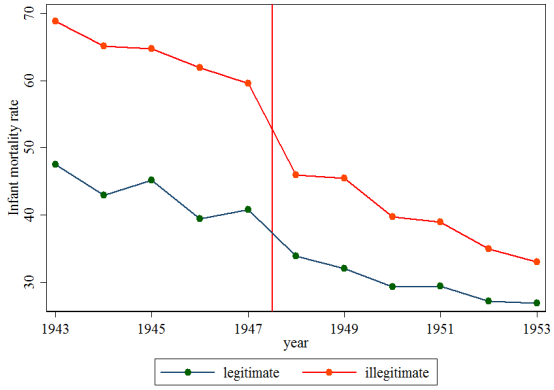


(a) Diarrhea (b) Pneumonia

Notes: The graphs show the neonatal (within 28 days) death rate per 1,000 related births by cause of death over the period 1943-1953).
Source: Ministry of Health Annual Reports, The Wellcome Library.

relevant socio-demographic metrics such as education or income. As auxiliary evidence, we therefore use the available distinction of mortality rates by the legitimacy of the child.[18] Figure 6 confirms our hypothesis, illustrating particularly large mortality declines among illegitimate children relative to legitimate children and a consequent narrowing of the (large) mortality gap between illegitimate and legitimate children. Overall, the mortality gap about halves.

Figure 6: Infant mortality by legitimacy



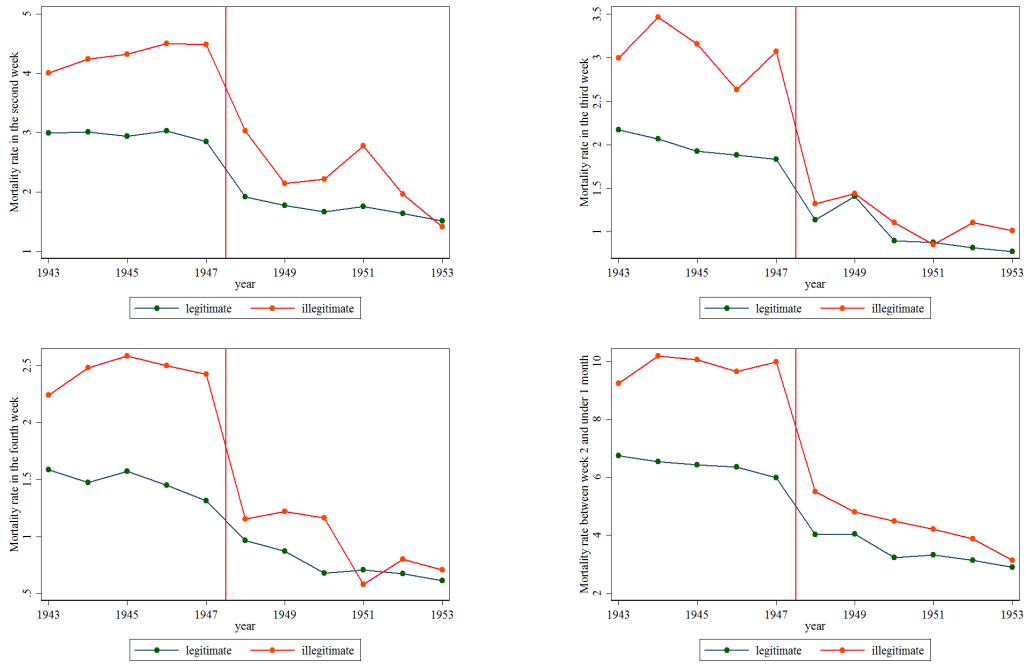
Notes: The graph shows infant mortality in England and Wales by legitimacy of child.
Source: Registrar General's Annual report 1940-1955, The Wellcome Library.

In Figure 7, we again focus on the neonatal period. The narrowing of the mortality gap by legitimacy status is particularly pronounced, indeed it is

[18] Wright (1973) finds that illegitimate births in Britain are highly concentrated among mothers of lower social class.

practically eliminated in the second, third and fourth week after birth.

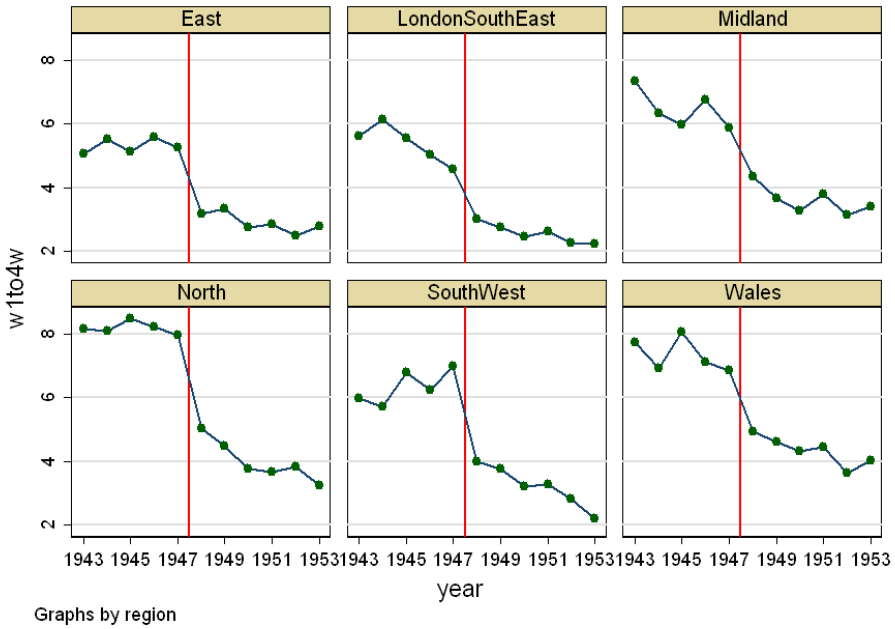
Figure 7: Infant mortality by time of death and legitimacy



Notes: The graph shows infant mortality in England and Wales in the neonatal period.
Source: Registrar General's Annual report 1940-1955, The Wellcome Library.

We examine geographical differences in the impact of the NHS, finding similar effects at the regional level. Figure 8 illustrates regional differences in not only the levels of the neonatal mortality rate, but also in the magnitude of the decline between 1947 and 1948. In the North, and South West regions, areas traditionally with a relatively high proportion of the population in lower socio-economic groups, neo-natal mortality both before and after the NHS introduction is higher than in London and the East regions, areas traditionally with a relatively low proportion of the population in lower socio-economic groups. However, the decline in neo-natal mortality around the NHS introduction is higher in areas with a higher proportion of low social class individuals, consistent with the findings of Heady and Norris (1955) who suggest that areas with relatively poor social class conditions tend to have poorer medical services and consequent higher mortality rates.

Figure 8: Neonatal mortality by time of death and region



Notes: The graph shows neonatal mortality in England and Wales.
Source: Registrar General's Annual report 1940-1955, The Wellcome Library.

In summary, we find no evidence that would suggest improved mortality before or around birth. Rather, neonatal mortality decreases strongly. This has important effects on cumulative mortality under 4 weeks, leading to a reduction in infant mortality rates by about 17% between 1947 and 1948. While we find throughout that female mortality in infancy is significantly lower in all considered time periods, we find no differential NHS effect by gender (not shown). The strong declines in infant mortality are particularly evident for illegitimate children and in areas with a high proportion of lower class individuals.

In order to gauge the robustness of our assertion that this decrease in mortality was driven by the introduction of the NHS, we examine several possible confounding influences: the impact of food availability, as the UK was still experiencing food shortages in 1948 and so had maintained food rationing; the influence of birth rate fluctuations and potential changes in the composition of mothers; and whether the severe winter of the preceding year influenced infant mortality trends. We find no evidence that these factors played a role, see Appendix A.

3 Empirical analysis of long-run impacts of infancy exposure to the NHS

In Figure 2 we demonstrated an immediate and discontinuous drop in infant mortality by ca. 20% among birth cohorts who benefitted from health care coverage through the introduction of the NHS at birth, relative to those born just before its introduction. We now seek to identify the impact of infancy exposure to universal health care (UHC) over the life course in two dimensions - health and mortality.

Infancy is a key developmental period for both mental and physical health as well as cognition. A large literature documents the important influence of early childhood environments in fostering this developmental process. In this paper, we explore the impact of a fundamental change in the healthcare environment in infancy which may affect health and mortality outcomes in later life through several channels. First, improved infancy care may prevent an early decumulation of the "health stock" of a newborn, with cumulative impacts over time. Second, early life decumulation of health is more severe than similar environmental shocks at later ages due to the fast neurological development in infancy. Early life infections, for example, may inhibit neurological development and brain plasticity in infancy, with the effect of an accelerated ageing process (Bhalotra and Venkataramani, 2011). In terms of the Grossman model, this would imply a larger depreciation rate. Finally, medical research suggest that the immature organism adapts to key environmental characteristics and retains the initial programming even when the environment changes.[19] In an adverse early environment the organism prioritises short-term survival at a potentially significant cost to longer term health, leading to a potential lag in disease response which may take decades to manifest. This latter mechanisms implies biological embedding of infancy environments (Shonkoff et al., 2009). All three factors present reasons to expect that the origins of adult disease or mortality are often to be found in adverse or beneficial early life environments.

We proceed as follows: In section 3.1, we describe the empirical method and identification strategy used in both parts of the analysis. Section 3.2 describes the data used to establish the impact of infancy exposure to the

[19] A similar argument has been made for in-utero conditions in the Barker hypothesis.

NHS on mortality beyond age 50, and presents the results. Section 3.3 does the same for the onset of cardiovascular disease, a key driver of mortality during this life period.

3.1 Empirical method

As the NHS was implemented nationally in July 1948 there is a clearly defined threshold, and the analysis therefore proceeds using a regression discontinuity approach. We define infancy exposure to universal health care exploiting the timing of the scheme's introduction combined with date of birth information. We allow for cohort trends in mortality either side of the threshold. Our analysis considers cohorts born within a narrow window of maximally 4 years either side of the threshold, the introduction of the NHS in July 1948. In the following, we describe two strategies that we use to identify the causal impact of infancy access to universal healthcare on later life mortality and health.[20]

Given the differential access to healthcare before universal coverage was provided by the NHS, as described in Section 2, we do not expect a uniform expansion in available infancy care across all individuals born after the introduction of the NHS. Instead, we apply a fuzzy design. The probability of an increase in pre- and postnatal care is larger for individuals born by mothers of lower social class than for those whose mothers could afford to purchase private maternity care. In consequence, we expect the potential benefits of access to universal healthcare to be concentrated among individuals who absent of the treatment would have had limited availability of healthcare in infancy, i.e. we expect that mothers of lower social class, and their children, have disproportionately benefited from the introduction of universal health care. Indeed, given the lack of investment in healthcare infrastructure at inception, healthcare access of mothers who could afford medical services prior to the NHS may be adversely affected due to crowding out by the influx of new patients. In consequence, we separately estimate the impacts of UHC

[20]To be precise, all individuals had access to UHC from 1948 onwards throughout their lives. Yet, depending on their birth date they differ in terms of infancy exposure. Hence, we seek to identify the impact of infancy healthcare access in a setting where all individuals have access to healthcare beyond infancy.

on these two groups, using the following model:

$$y_{ica} = \alpha + \gamma_1 T_c + \gamma_2 T_c LC_{ic} + \delta LC_{ic} + \beta_1 C_c + \beta_2 C_c LC_{ic} + X'_{ic}\eta + \mu_g + \epsilon_{ic} \quad (1)$$

where y_{ica} denotes the mortality or health outcome of interest of individual i born into cohort c at age a. X_{ic} denotes a set of socio-demographic characteristics. μ_g capture fixed effects for county of birth, reflecting potential heterogeneity in childhood environments and healthcare infrastructure as well as other relevant geographical variation that are constant over time. T_c denotes the time and cohort discontinuity that we exploit for identification, and $T_c LC_{ic}$ captures the differential increase in healthcare access that the introduction of the NHS afforded to newborns whose mothers were of lower social class. C_c is the cohort trend, while $C_c LC_{ic}$ is the cohort-specific trend for those with mothers of lower social class.

In our second identification approach, we take into account that the impact of infancy exposure to UHC may not only depend on gaining access to healthcare, but also on the amount of medical services available. We posit that the degree of expansion in medical services upon introduction of the NHS varies by county of birth. To show this we construct a simple, stylised model of medical services before and after the introduction of the NHS. Our model incorporates the three core principles of the NHS, i.e. i) equalisation of access to medical services, ii) medical services are free at the point of delivery, and iii) access is based on clinical need, not ability to pay. We derive three model features from these principles. First, all individuals obtained the same access to (and quantity of) medical services per person once universal healthcare coverage was rolled out. Secondly, we assume that medical services are consumed free of charge.[21] We further treat healthcare capacity as fixed at pre-NHS levels in the short run. This assumption relies on evidence from section 2 that the inception of the NHS was not immediately accompanied by investment into expanding the pre-existing healthcare infrastructure.

Our model has two sources of heterogeneity: first, individuals differ in access to medical services m_i prior to the existence of the NHS. Second, individuals live in different counties g. Population composition differs across counties, such that the fraction of individuals with access to medical care prior to the

[21] We do not model the taxes raised to finance the NHS, as these will not affect consumption of medical services at the margin.

NHS, I_g, is county-specific. Since pre-NHS healthcare was privately financed, doctors (and other healthcare service) provision followed private demand, and supply was larger in areas where a larger fraction of the population could afford healthcare. Given the lack of investment and the cessation fo free movement of doctors at the inception of the NHS, supply remains fixed in the short run. For simplicity, we abstract from other sources of heterogeneity in healthcare provision at the county level in the model, assume a fixed population and that pre-NHS, per patient medical services are equal across counties (for those individuals who have access to them). We normalise per-person medical services to 1.

In consequence, medical services of individual i in county g prior to the NHS are:

$$m_{ig} = \begin{cases} 0 & if \quad i = NI, \\ 1 & if \quad i = I \quad \forall g \end{cases} \tag{2}$$

Previously covered individuals, I_g, enjoy one unit of medical services, while those previously uninsured (denoted as NI_g) obtain no medical services. Initial county-level capacity M_g then varies with the number of previously covered individuals living in the geographical area previous to the NHS, I_g, and amounts to $M_g = I_g$.

In the short-run the capacity of medical services is fixed. The introduction of universal healthcare coverage therefore leads to increased demand for a fixed resource, as newly eligible patients compete with pre-existing patients for a fixed level of medical services M_g. The gain in medical services for previously uncovered individuals NI varies with the ratio of new to old patients in each county g. Hence, the increase in per-capita medical services (from initially zero) for each previously uncovered individual is:

$$\Delta(m_{NI,g}) = \frac{I_g}{N_g} \tag{3}$$

where N_g denotes the population of county g.

Similarly, per-capita medical services enjoyed by the previously covered fall upon the start of the new healthcare system due to increased demand for the fixed resource:

$$\Delta(m_{I,g}) = \frac{I_g}{N_g} - 1 \tag{4}$$

This implies cross-county variation in per-capita medical services provision. To illustrate: County A has a large fraction of pre-existing patients, with I_g close to N_g, therefore the NHS introduction does not add many new patients, and $m_{NI,g}$ is high and close to 1. Similarly, per-capita medical services enjoyed by the previously covered change little. In contrast, County B has a small fraction of pre-existing patients and therefore a large fraction of the population gain access to medical service for the first time with the NHS introduction. As the supply of medical services is fixed at the pre-NHS level, and there are now more users, per capita medical services in County B will be lower than in County A. Overall, the (per capita) level of medical services in a county increases with the proportion of pre-existing patients, $\frac{I_g}{N_g}$, after the introduction of the NHS.

This simple model has several implications for our empirical strategy: first, geographical variation in the fraction of previously insured (and reversely the fraction of new patients), and in the amount of medical services available to new patients may explain the regional variation in the magnitude of infant mortality rate reductions that we presented in section 2.1.[22] More importantly, the model informs our analysis of long-run effects. If the amount of medical services provided per person varies with the county-level proportion of previously covered individuals, then we can exploit such county-level variation in addition to temporal variation in our identification strategy. The higher $\frac{I_g}{N_g}$, the lower the proportion of new patients competing over the fixed resource, and the higher are medical services enjoyed by newly eligible individuals, $m_{NI,g}$. Hence, we expect larger NHS infancy exposure effects for those newly gaining access in areas with a high proportion of previously insured residents.

We estimate the following second model:

$$\begin{aligned} y_{ica} = \alpha &+ \gamma_1 T_c + \gamma_2 T_c LC_{ic} + \gamma_3 T_c HIGHarea_g + \gamma_4 T_c LC_{ic} HIGHarea_g \\ &+ \gamma_5 LC_{ic} HIGHarea_g + \delta LC_{ic} + \zeta HIGHarea_g \\ &+ \beta_1 C_c + \beta_2 C_c LC_{ic} + X'_{ic}\eta + \epsilon_{ic} \end{aligned} \tag{5}$$

where $HIGHarea_g$ denotes whether the individual was born in an area with

[22] We test our medical services model and find empirical evidence supporting it. These are available form the authors upon request.

a high proportion of previously insured. We interact this variable with our Treatment indicator T_c, the social class of the individual LC_{ic}, and include a full set of interactions to saturate the model. Our parameters of interest are γ_1 to γ_4, while γ_5 allows for health externalities in areas with a high proportion of previously insured individuals that may benefit individuals with such access, e.g through vaccination externalities or charitable giving to provide more free health services for the poor.

In the model, we assumed homogeneity of counties in terms of population and pre-existing levels of pre-capita medical services in our model. When we take the model to the data, county fixed effects will sweep up these pre-existing differences in model 1 (and any other relevant county-level differences that are constant over time), and $HIGHarea_g$ in model 2. To link individual health and mortality outcomes around ages 52 to 64 with information on social class composition at birth, we use the individual-level information on county of birth contained in both microdatasets. We estimate linear probability models and cluster standard errors at the county level throughout.

3.2 Results: long-run impacts on mortality

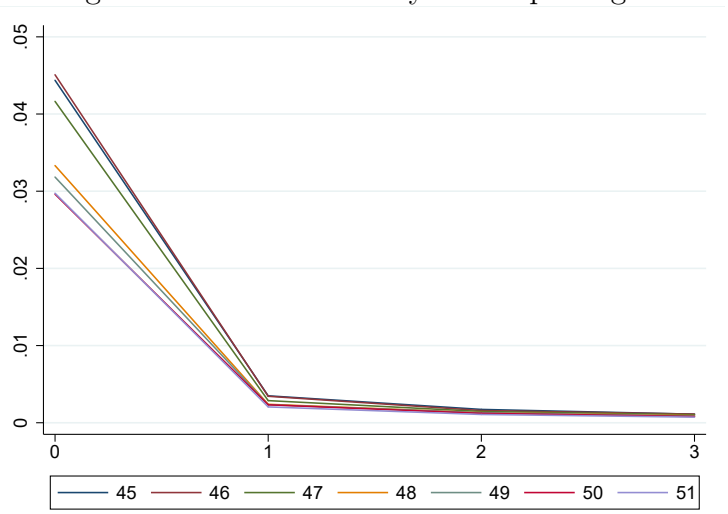
The analysis in this section is based on microdata from the ONS Longitudinal Study, an approximate 1% sample of the population of England and Wales. The study contains census records of individuals born on four specific days of the year, the LS members, which are linked between five successive censuses (1971 to 2011). The census data is also linked to key event records from administrative data, such as birth, death, and emigration records.[23] New LS members enter the sample through birth or immigration, whereas attrition occurs only through emigration or death. Approximately 500,000 LS members are enumerated in any given census year, and the LS currently holds information on around 1 million sample members collected over the 40 years since its inception.

Since the first available census dates from 1971, we conduct our analysis conditional on an individual's survival up to 1971.[24] The data contains a rich set

[23]The linkage with these event records are periodically updated. Currently the study holds information for deaths occurring until December 2016.

[24]Conditioning on survival to 1971 does not result in significant survivorship bias as the cohorts we consider in our analysis are between ages 20 and 26 in 1971, and mortality

Figure 9: Cohort mortality rates up to age 3



Notes: The graph shows mortality rates for cohorts born between 1945 and 1951 from birth to age 3. Source: Human Mortality Database.

of socio-economic characteristics collected at each census wave, supplemented with geographical information on place of birth,[25] as well as information on the time, cause and place of death where appropriate. The resultant dataset allows us to follow individuals into the later stages of their life-cycle to analyse mortality patterns.

We consider a narrow window of cohorts born prior (1945-1947) and after (1948-1951) the introduction of the NHS in 1948.[26] We focus our analysis on mortality from age 52 onwards, since mortality rates were high in infancy but very low between ages 3 to around 52, see Figures 9 and 10.[27] Above age 52, a time where critical health shocks begin to manifest, we see the mortality rates begin to 'fan out' indicating mortality differentials by cohort. In Figure 10 we show descriptive evidence that there is a widening mortality gap between pre- and post-NHS cohorts.

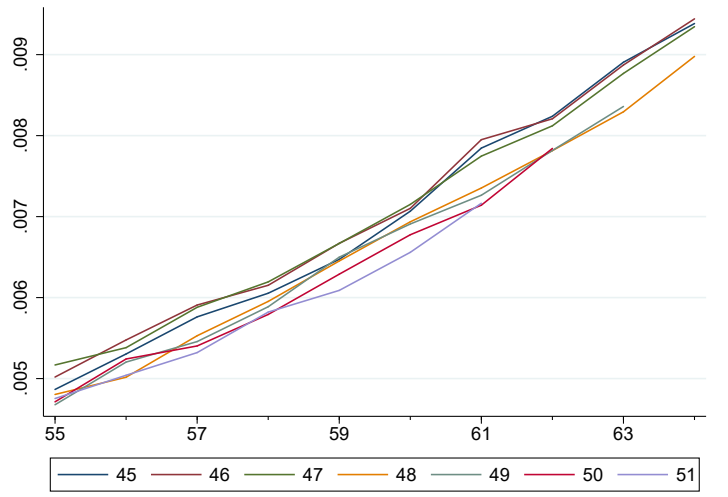
rates are close to zero after infancy.

[25]Place of birth information is derived from the information in identifying codes within NHS records, which contains a geographic identifier of a person's place of birth. For individuals born prior to 5th July 1948 the NHS identifier is comprised of their National Registration number, which is similarly coded with a geographic identifier of natality for those born after September 1939. For individuals born prior to September 1939 place of enumeration on the National Registration day is recorded.

[26]Cohort and treatment exposure variables in the ONS Longitudinal Study are defined based on the year of birth, as month of birth is unattainable due to the sampling strategy of the data.

[27]Our results do not change if we start at age 50. We choose age 52 for comparability, as it is the earliest possible age in the analysis of health outcomes.

Figure 10: Cohort mortality rates from age 55 to 65



Notes: The graph shows mortality rates for cohorts born between 1945 and 1912 from age 55 to age 65. Source: Human Mortality Database.

Our outcome variables are age-specific mortality rates up to age 64, the maximum age for which we observe all study cohorts in the data.[28]

County-level social class composition data, used to estimate model 2, is contained in the Great Britain Historical Database (Southall, Aucott, and Dorling, 2004). The Database presents county-level statistics of social class composition aggregated from the 1951 census, based on the county proportion of males above age 15 in 5 social classes.[29] We use county-level social class composition data to proxy for the proportion of previously covered individuals in a county, $\frac{I_g}{N_g}$, which drives the level of medical services available after the birth of the NHS. Social class proxies for healthcare coverage status prior to the NHS, as low income individuals had no previous access to healthcare while individuals of higher social class could pay for healthcare

[28]Death records are currently available up to the end of 2016. The oldest cohort we consider is born in 1945, and thus 71 in 2016, the cutoff year for the death registers. The youngest cohort we consider is born in 1951 and thus 65 in 2016. We exclude 2016 as some death records are updated with delay, and consider deaths up to age 64. While death registers consider all death of individuals in the sample, the earliest we observe an individual is in the 1971 census, so we only consider deaths after age 26, the lowest age at which we observe all cohorts.

[29]To our knowledge, data from the 1951 census is the closest available date to 1948 that records this information at the county level. It is not accessible as a microdataset. More aggregated data at the level of government office regions is available for the pre-NHS census from 1931, and we find little difference in social class composition between the 1931 and 1951 census.

Table 3: Descriptive statistics, ONS Longitudinal Study

Variable	Mean	Std. Dev.
Mortality rate by age ...		
52	0.0340	0.1812
54	0.0415	0.1994
56	0.0512	0.2203
58	0.0614	0.2400
60	0.0729	0.2600
62	0.0859	0.2801
64	0.0996	0.2994
Cohort (in % of sample)		
1945	13.04	
1946	15.32	
1947	16.40	
1948	14.54	
1949	13.97	
1950	13.30	
1951	13.42	
Female	.4913	0.4999
Low Social Class	0.2276	.4193
Mid Social Class	0.5690	.4952
High Social Class	0.2035	0.4026
Population in HIGH areas (%)	0.1809	0.3849
by social class (% of total sample):		
	low	middle & high
	3.47	14.62
Population in LOW areas (%)	0.8191	
by social class (% of total sample):		
	low	middle & high
	19.29	62.62
Observations	*44,122*	

Source: ONS Longitudinal Study

privately. We construct county-level population proportions in high (consisting of those in Professional and Intermediate occupations) and middle class (Skilled and Party Skilled Occupations), and create dummy variables for counties in which the proportion of inhabitants of high social classes is in the upper tertile.

Summary statistics of the resulting sample are presented in Table 3. 3.40% of the sample died by age 52[30], with the mortality rate increasing with each year of age. By age 64, 9.96% of individuals first observed in 1971 have died. When first observed in the 1971 census, individuals have a mean age of

[30]All death rates based on ONS Longitudinal Data are defined conditional on surviving to age 31.

around 23 years, 49.1% are female; 22.8% are of low, 56.9% of middle, and 10.3% of higher social class. We do not observe the social class of the mother at the time of her child's birth, but instead use the individual's social class in early adulthood, as reported in 1971, the earliest available census year.[31] Although a degree of intergenerational social mobility is to be expected, it is probable that social class in early adulthood is highly correlated with social class at birth.

Table 4 shows the estimation results for the probability of death by ages 52 to 64 using a linear probability model and the empirical specification from equation 1.[32] We find that the introduction of UHC impacts on both - the previously uncovered and the previously covered. Mortality rates of individuals born to mothers of lower social class who would not have been able to afford healthcare before the NHS fall throughout from age 52 to 64. F-tests for joint significance of the parameters γ_1 and γ_2 confirm statistically significant impacts at the 1-5% level. In the lower panel of Table 4, we show the well-documented mortality differential by social class, and calculate the mortality changes implied by our estimates. We find that newly gained access to UHC reduces mortality between ages 52 and 64 by 15-22%. These represent large long run impacts which manifest 50 to 60 years after infancy exposure. Reductions in later life mortality are particularly large at ages 52 and 54, where they amount to around a 20% fall, and decline with age, suggesting that infancy exposure to UHC prevents early death.

We also find large increases in mortality for those born into middle or higher class households, denoted as HC. These are less precisely estimated than the mortality gains for the previously uncovered, but of a similar order of magnitude of 10-24%, albeit applied to a lower baseline rate. They are consistently observed for all age-specific mortality rates that we consider. This is suggestive of crowding out effects, where new patients absorb part of the medical services previously consumed exclusively by those previously covered. Furthermore, this point towards a health production function where

[31] This classification is based on the Office of National Statistics 7-category social class code which assigns categories according to occupation/employment duties. We define classes I-II (Professional, Intermediate) as "High Social Class", III-IV as middle class (Skilled nonmanual, Skilled Manual) and classes V-VII (Partly Skilled, Unskilled, Armed Forces) as "Low Social Class". Since class is always missing for non-working women, we use her household's social class, and fill in missing data using individual and household social class information from 1981.

[32] We re-estimated these using a probit model with similar results.

Table 4: Estimates of mortality rates by ages 52 to 64

	Mortality rate by age ...						
	52	**54**	**56**	**58**	**60**	**62**	**64**
$T_c * LC_{ic}$	-0.0173**	-0.0223**	-0.0187**	-0.0249**	-0.0279***	-0.0272**	-0.0313***
	(0.00763)	(0.00874)	(0.00875)	(0.00998)	(0.0100)	(0.0104)	(0.0112)
T_c	0.00678*	0.00897**	0.00560	0.00697	0.0102*	0.00935*	0.00816
	(0.00392)	(0.00426)	(0.00482)	(0.00512)	(0.00536)	(0.00530)	(0.00617)
LC_{ic}	0.0249***	0.0319***	0.0332***	0.0400***	0.0444***	0.0479***	0.0581***
	(0.00522)	(0.00617)	(0.00568)	(0.00676)	(0.00716)	(0.00640)	(0.00793)
Observations	44,121	44,121	44,121	44,121	44,121	44,121	44,121
F-test for joint significance of T_cLC_{ic} and T_c coefficients							
p-value	0.0790*	0.0391**	0.1057	0.0509*	0.0244**	0.0347**	0.0262**
Mean mortality rate prior to NHS inception, by social class							
LC	0.0488	0.0606	0.0730	0.0884	0.1029	0.1209	0.1421
HC	0.0306	0.0367	0.0462	0.0558	0.0657	0.0783	0.0899
Lower-class mortality reduction in percent (relative to mean)							
LC	-21.56	-22.00	(-17.95)	-20.28	-17.20	-14.76	-16.28
HC	22.16	24.44	(12.12)	(12.49)	15.53	11.94	(9.08)

*Notes: The table shows estimates of equation 1. All specifications control for additional characteristics determined at birth (gender, birth location), current region of residence, and include cohort trends. Robust standard errors are reported in parentheses. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.*
Source: ONS Longitudinal Study

not only access matters but also the amount (or quality) of medical services provided.

In Table 5, we present the analysis using the empirical specification described in equation 5, where we include the social class composition in county of birth to proxy geographical variation in medical services available during infancy. Consistent with our expectation we observe larger NHS birth exposure effects specifically for low class individuals born in areas with a larger fraction of high social class, as shown in the lower panel of the table. While mortality reductions are between 11 and 17% in areas with fewer residents of high social class, i.e. a smaller healthcare infrastructure and much pressure on existing resources, they amount to 30-44% in areas with few new patients and higher medical services. Again, we find that the highest mortality reductions are in early onset by age 54.

In line with our findings using specification 1, mortality worsens for those with previous access to health care. Conversely, in areas that experience a larger expansion in demand for medical services, i.e. those with more incoming patients, mortality rates increase by more, reflecting a larger reduction in medical services available for this group. Mortality rates in these areas

increase by 13.43 to 29.86%, again with larger increases in the early 50s. In areas with a high fraction of higher social class residents, i.e. those where pre-capita medical service reductions are small, mortality increases are small, amounting to a maximum of 11.92% by age 54.

Appendix B reports sensitivity analysis, and shows that our findings are robust to i) narrowing the window of birth cohorts around the discontinuity (to 1946 to 1950 cohorts), ii) variations of the study area, i.e. we leave out Wales, iii) accounting for the systematically lower mortality of women between 52 and 62 by producing gender-specific estimates.

In summary, we find consistent results im models 1 and 2. Infancy exposure to universal healthcare reduces mortality rates substantially among those who gained access to healthcare for the first time. Access or quantity of medical services declined for those who enjoyed healthcare prior to the inception fo the NHS, with negative impacts on survival in later life, between ages 52 and 64. Our results support the hypothesis that the benefits of UHC depend on the amount of medical services provided, as mortality gains are higher in areas with a smaller expansion in demand for services that were fixed in the short-run.

3.3 Results: long-run impacts on health

We now proceed to the analysis of health impacts, using a rich, high-quality micro-dataset, the UK Biobank. The data comprises a sample of approximately 500,000 individuals. It contains detailed self-reported health information, as well as objective measures recorded by a healthcare professional. The data was collected between 2006 and 2010, and therefore provides a snapshot of individual health around age 60 for those born in the 1940s. It is linked to all hospital spells occurring between 1997 and 2016, generating a 19-year panel of health trajectories at older ages which we use to estimate impacts on health and morbidity.

Again, we consider a narrow window of cohorts around the introduction of the NHS in 1948, i.e. cohorts born between 1945 and 1951.[33] As the Biobank provides information on month and year of birth, we define cohort

[33]For comparability with the ONS Longitudinal Study data, we exclude data from Scotland.

Table 5: Estimates of mortality rates by ages 52 to 64

	Mortality rate by age ...						
	52	**54**	**56**	**58**	**60**	**62**	**64**
$T_c * LC_{ic}$	-0.0119	-0.0110	-0.0128	-0.0227*	-0.0224	-0.0303**	-0.0271
* HIGHarea	(0.0124)	(0.0118)	(0.0125)	(0.0118)	(0.0140)	(0.0150)	(0.0196)
$T_c * LC_{ic}$	-0.0158**	-0.0211**	-0.0172*	-0.0217**	-0.0243**	-0.0225**	-0.0272**
	(0.00751)	(0.00854)	(0.00861)	(0.0102)	(0.0101)	(0.0106)	(0.0111)
T_c* HIGHarea	-0.00825**	-0.00598	-0.0108**	-0.00763*	-0.00453	-0.00344	-0.00254
	(0.00318)	(0.00361)	(0.00520)	(0.00441)	(0.00428)	(0.00473)	(0.00529)
T_c	0.00845**	0.0102**	0.00770	0.00852	0.0110**	0.0101*	0.00873
	(0.00412)	(0.00433)	(0.00480)	(0.00521)	(0.00532)	(0.00526)	(0.00619)
LC_{ic}	0.0229***	0.0301***	0.0309***	0.0372***	0.0410***	0.0435***	0.0537***
	(0.00546)	(0.00651)	(0.00600)	(0.00725)	(0.00787)	(0.00714)	(0.00848)
HIGHarea	0.00395	0.000127	0.00170	0.00208	0.00103	-0.00213	-0.000594
	(0.00410)	(0.0045)	(0.0046)	(0.0054)	(0.0053)	(0.0057)	(0.0063)
LC_{ic}* HIGHarea	0.0134	0.0125	0.0162*	0.0187*	0.0212*	0.0283**	0.0283*
	(0.00824)	(0.0080)	(0.0090)	(0.0108)	(0.0113)	(0.0131)	(0.0166)
Observations	44,121	44,121	44,121	44,121	44,121	44,121	44,121
F-tests of joint significance (p-values)							
LC in HIGHarea	0.0519*	0.0838*	0.0208**	0.0169**	0.0532*	0.0429**	0.0808*
LC in LOWarea	0.0751*	0.0338**	0.1275	0.0988*	0.0397**	0.0700*	0.0534*
HC in HIGHarea	0.0280**	0.0493**	0.0628*	0.1200	0.0943*	0.1488	0.3607
Mean mortality rate by area and social class							
LC in HIGHarea	0.0624	0.07	0.0868	0.1035	0.1187	0.14	0.1613
LC in LOWarea	0.0429	0.0556	0.0659	0.0814	0.0955	0.1126	n/a
HC in HIGHarea	0.0326	0.0354	0.0462	0.0552	0.0639	0.074	0.0858
HC in LOWarea	0.0283	0.0312	0.0426	0.0527	0.0631	0.0752	n/a
Mortality change in percent (relative to mean mortality rate)							
LC in HIGHarea	-44.07	-39.83	-38.13	-42.04	-33.89	-32.96	-29.83
LC in LOWarea	-17.13	-19.60	-14.42	-16.19	-13.93	-11.01	n/a
HC in HIGHarea	0.61	11.92	-6.71	(1.61)	10.13	(9.00)	7.21
HC in LOWarea	29.86	32.69	(18.08)	(16.17)	17.43	13.43	n/a

*Notes: The table shows estimates of equation 5. All specifications control for additional characteristics determined at birth (gender, birth location), current region of residence and include cohort trends. Robust standard errors are reported in parentheses. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.*
Source: ONS Longitudinal Study

in year-months, and define July 1948 as the cutoff. In contrast to the ONS Longitudinal Studies data, our cohorts of interest are interviewed at a much older age, between age 55 and 65, with cohorts born prior to the NHS more likely to be interviewed at an older age. To account for cohort-specific survivorship bias, we conduct sensitivity analysis on a subsample of individuals surviving up to age 65.

We focus our analysis on cardiovascular disease for three reasons: first, it has relatively high onset rates in the age range that we consider (ages 52 to 64). Second, we expect childhood environments to matter if at all, most in terms of cardiovascular disease. Third, cardiovascular disease is the leading cause of death worldwide (World Health Organisation, 2017), and is the second largest cause of death in England accounting for 22% of deaths by age 75 (Department of Health, 2014).[34]

To minimise measurement error from self-reported outcomes, we use the linked hospital spell data to compute age-specific onset rates. Hospital spells are linked from 1997, and are currently updated to 2016 (with partial records for 2015), so we observe hospital spells for all cohorts between ages 52 and 64.[35] The data records the date of onset, and primary and secondary diagnosis, classified using the 9th and 10th revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-9 and ICD-10). The same classification is used for main causes of death, which will allow us to link disease onset to mortality caused by cardiovascular disease. We use primary diagnosis codes and aggregate across all types of cardiovascular disease, e.g. ischaemic heart disease, hypertensive heart diseases etc. For each individual, we compute the age of first onset between ages 52 and 64 (if any onset occurs). We then construct incidence rates by ages 54, 60 and 64.

[34]The largest cause of death is cancer which accounts for 42% of deaths up to age 75. However, as cancer onset results from gene mutations, we expect that its onset is driven less by childhood environment and to a larger extent by genetic disposition and lifestyles in adulthood such as drinking, smoking, exercise and nutrition.

[35]In this age range, we observe onsets for all cohorts in our window of analysis.

Table 6: Descriptive statistics, UK Biobank

Variable	Mean	Std. Dev.
Cardiovascular condition by age...		
54	0.0166	0.1278
56	0.0344	0.1822
58	0.0526	0.2233
60	0.0719	0.2583
62	0.0930	0.2905
64	0.1151	0.3192
Low social class	0.3590	0.4797
Female	0.5438	0.4981
Observations	*126,078*	

Notes: Source: UK Biobank

As in the ONS Longitudinal Study, we do not observe the social class of the mother at the time of her child's birth. Instead we use the individual's school leaving age and the age at which they start working as indicators of social class. An individual is considered of lower class if they leave school before age 16, have no qualifications or if they start working before age 17.[36] Additional covariates are gender, and current location of residence to control for contemporaneous cross-county variation, e.g. in medical service provision. In model 1, we control for county of birth fixed effects to capture differences in local health infrastructure in infancy and other relevant cross-county differences in infancy socio-economic environments. Summary statistics for our estimation sample are shown in Table 6. Our estimation sample contains more than 126,000 individuals. The incidence of cardiovascular disease increases with age, from 1.66% at age 54 to 11.51% at age 64.

Table 7 shows the estimation results for the probability of onset of cardiovascular disease between age 52 and 64 using a linear probability model and the empirical specification from equation 1. Columns 1,3 and 5 report our main estimates, while columns 2, 4 and 6 report estimates conditioning on survival up to age 65, thus avoiding selective mortality at older ages. Results are qualitatively and quantitatively very similar across the two sets of estimates.

As with the results for overall mortality we find the parameter estimate $\hat{\gamma}_2$

[36]The minimum school leaving age for these cohorts was age 15.

Table 7: Estimates of incidence of cardiovascular disease by age

	Incidence by ...					
	age 56		age 60		age 64	
	(1)	(2)	(3)	(4)	(5)	(6)
$T_c LC_{ic}$	-0.0091**	-0.0082**	-0.0091*	-0.0095*	-0.0159**	-0.0168***
	(0.004)	(0.004)	(0.005)	(0.005)	(0.007)	(0.006)
T_c	0.0068***	0.0067***	0.0074*	0.0073*	0.0100**	0.0106**
	(0.002)	(0.002)	(0.004)	(0.004)	(0.004)	(0.004)
LC_{ic}	0.0166***	0.0161***	0.0259***	0.0250***	0.0340***	0.0340***
	(0.004)	(0.004)	(0.004)	(0.004)	(0.005)	(0.005)
Cond. on survival[a]	N	Y	N	Y	N	Y
Obs.	126,078	124,134	126,078	124,134	126,078	124,134
F-test for joint significance of $T_c LC_{ic}$ and T_c coefficients						
p-value	0.0020***	0.0040**	0.0925*	0.0781*	0.0151**	0.0064***
Mean incidence rate prior to NHS inception by social class						
LC	0.0426	0.0419	0.0872	0.0863	0.1399	0.1383
HC	0.0294	0.0292	0.0630	0.0625	0.1036	0.1024
Incidence change in percent (relative to pre-NHS mean)						
LC	-5.40	-3.58	-1.95	-2.55	-4.21	-4.48
HC	23.13	22.95	11.75	11.68	9.65	10.35

*Notes: The table shows estimates of equation (1). All specifications control for gender, birth location, home location. We include cohort trends, and allow for specific cohort-trends for lower social class individuals, and allow for a trend break post NHS. Standard errors are clustered at county of birth level and reported in parentheses. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. [a]: For robustness, we condition on surviving to at least age 65 to avoid bias from cohort-specific sample selection due to the late interview year in which the Biobank was started.*
Source: UK Biobank

increases in magnitude and statistical significance with age. The reduction in the onset of cardiovascular disease between ages 52 and 63 is 0.0023 percentage points for individuals from lower social class by age 56, and the F-test confirms that it is statistically significant at the 1% level. This represents a reduction of 5.40% in the incidence of cardiovascular disease relative to the pre-NHS mean for this population group. We can see that these reductions are largest up to age 56. Similar to our mortality results, incidence of cardiovascular disease increases among those with healthcare access prior to the NHS. Depending on age, incidence rises by 10 to 23%, pointing to potentially large crowding out effects from the influx of new patients.

Yet, when we repeat the estimation of model 2 in the UK Biobank, we do not observe additional reductions in the onset of cardiovascular disease for low class individuals born in areas with a larger high social class population composition (in contrast to the analogous results for mortality presented in

Table 5). Neither do we observe higher increases of cardiovascular disease incidence for the previously covered in areas with more incoming new patients. Yet, the estimated coefficients for γ_1 (γ_2), capturing low versus middle and higher class impacts of infancy exposure to the NHS, remain statistically significant and have a positive (negative) sign.

Again, we i) vary the window around the threshold, cutting it approximately in half by restricting to 20 months either side of the threshold, ii) produce estimates for England only and for England without the capital London, and iii) omit current location fixed effects. Our results are robust to all of these alternative estimates. We further split the sample by gender and produce gender-specific results, and find our qualitative results confirmed for both sexes.

4 Conclusion

We have analysed the long-run impacts of infancy exposure to one of the oldest universal healthcare systems worldwide, the UK National Health Service. We demonstrate that in addition to the large immediate effect of infancy exposure to universal health care on infant mortality, there is evidence of long-term effects on health and mortality that manifest 50 to 60 years later. Beneficial impacts on survival and cardiovascular health are concentrated among individuals who had low socio-economic status at birth, for whom the introduction of the NHS would have provided a salient increase in the access to healthcare services. Yet, we additionally find that there is a second group whose later life mortality is adversely affected by infancy exposure to the NHS - those with previous healthcare access. This suggests that when universal healthcare is rolled out without accompanying investments in healthcare infrastructure, increased competition among patients can lead to crowding out between patients. We show that survival gains for those who newly gain access are larger and mortality increases for those with previous access to healthcare lower in areas with less patient competition.

Our results are consistent with Grossman (1972) who models health as a stock that depreciates with age and increases in health investments. Around age 50, depreciation accelerates stochastically via the onset of stochastic health

shocks such as cardiovascular disease, stroke, cancer and other conditions.[37] While many of these conditions can be treated, most are hard to reverse. Hence, a better health stock at age 50, or a lower probability of experiencing such a shock, will positively affect health in subsequent years. This way, small (and statistically insignificant) positive treatment effects at ages 50, 51 and so forth accumulate over time, yielding lower disease onset rates and hence stronger survival gains when individuals approach their sixties. In our analysis, we find that mortality reduction from infancy exposure to the NHS manifest mainly in prevention of death in the earlier 50s for lower class individuals. These mortality reductions become larger for those individuals born in areas where the NHS introduction resulted in increased medical services per capita. We find smaller, but statistically significant decreases in the onset of cardiovascular disease for low class individuals, although there is no evidence of geographical variation according to social class composition.

Overall, our empirical results point to long-run health and mortality premia from infancy exposure to universal healthcare. The long-run effect manifests about 60 years after the intervention, at a time of life characterised by increasing onset rates of disease and rising mortality. However, our current estimates may underestimate the long-run impact of infancy exposure to the NHS, if infants who would not have survived prior to the NHS were, say, inherently weaker or unhealthier than those who survived. Selective infant mortality may imply a lowering of overall population health for cohorts born (and surviving) after the NHS introduction, and may lower the average health and mortality of survivors in adulthood. We outline our future research intentions below, where we propose a strategy that will allow us to separate selective mortality effects from long-run health and mortality premia of universal healthcare coverage in infancy.

[37] Smith (2004) finds that the onset of major conditions approximately doubles with each decade of age, with an onset rate of 12.9% in the 51-61 age group as compared to 7.2% in the 41-50 age group.

5 Outlook

5.1 The role of selective mortality in infancy

Our current estimates may underestimate the long-run impact of birth exposure to the NHS, if infants who would not have survived prior to the NHS were, say, inherently weaker or unhealthier than those who survived. Selective infant mortality may imply a lowering of overall population health for cohorts born (and surviving) after the NHS introduction, and may lower the average health and mortality of survivors in adulthood. Given that we find a substantial reduction in infant mortality of 17% following the introduction of the NHS, such selection may significantly bias our estimates.

The tension between selective infant (or fetal) mortality and the impact of early life interventions on later outcomes has been recognised by several authors in the literature. Currie and Gruber (1996a) use information on infant mortality rates to provide bounds for the magnitude of the bias from selective mortality. Almond (2006) and Bozzoli et al. (2009) develop models that take into account the impact of selection effects on the health of survivors. Both authors either lack the necessary data or have to make strong assumptions to determine the magnitude of bias from selective mortality. Bozzoli et al. (2009) conclude that selection effects may dominate for high levels of infant mortality.

To account for this in estimation, we adapt the Bozzoli et al. (2009) model to separately identify i) the "health premium" of enhanced health outcomes in adulthood due to improved childhood conditions, and ii) the "selective mortality" effect ensuing from increases in infant survival. We combine their model with our simple medical services model to reduce the number of parameters that need to be calibrated. We sketch this model in the following.

Bozzoli et al. (2009) find a strong inverse relationship between postneonatal (one month to one year) mortality and the mean height of those children as adults. The authors interpret infant mortality as an indicator of early childhood disease and nutritional environment, and develop a model that separates the long-run scarring effect of such early childhood conditions on health in adulthood from the impact of infant mortality on the probability to survive to adulthood. Simulations using the model predict that an increase in

infant mortality may lead to a selection effect that may increase the average height of survivors, and may dominate the long-run scarring effect. In a pseudo cohort panel of birth-cohort data for 12 countries, they show that postneonatal mortality in the year of birth accounts for more than 60 percent of the variation in adult height.

The model assumes that individuals are born with an innate level of (adult) health which follows a distribution $F(h_{it})$. Individuals born with health h_{it} less than or equal to a threshold value z die in infancy. A change in the childhood environment acts as a shock v_t which moves the threshold value z. Infant mortality can thus be expressed as:

$$Inf_t = F(z + v_t) \tag{6}$$

Thus, the probability of surviving depends on the threshold value z and the childhood shock v_t.

This truncates the distribution of health for survivors which is:

$$\tilde{h}_{it} = \frac{\int_{F^{-1}(Inf_t)}^{\inf} h_i dF(h)}{1 - Inf_t} + \theta v_t \tag{7}$$

The first term captures the above described selection in the health status of survivors. The last term θv_t captures the long run scarring effect of the shock which moves the distribution of health to the left.

Bozzoli et al. (2009) then assume that $F(.)$ is a normal distribution with mean μ and variance σ^2. Substituting in equation 6, integrating and converting to a standard normal distribution gives:

$$\frac{\tilde{h}_{it} - \mu}{\sigma} = \frac{\phi\Phi^{-1}(Inf_t)}{1 - Inf_t} - \theta\left(\Phi^{-1}(Inf_t) - \frac{z - \mu}{\sigma}\right) \tag{8}$$

where ϕ and Φ are standard normal density and distribution functions. The equation expresses the z-score of h_{it} in terms of mortality rate Inf_t, survival cutoff z (expressed in standard deviations from the mean) and the stunting parameter θ. Taking their model to the data, the authors make assumptions about the parameters of the normal distribution, μ and σ, and the threshold value z. They then estimate θ using data on Inf_t and the height of survivors (their indicator of adult health).

We apply this model to our setting with *decreasing* infant mortality which may result in negative selection of survivors and a positive long-run health premium θ. In contrast to Bozzoli et al. (2009) who do not observe what may have caused the shock in childhood disease environments, and have no information on v_t, we use the medical services model described in section 3.1 to put additional structure on the childhood environment shock. We posit that the childhood shock is a function $g(m_{gt})$ of county-level medical services.

In consequence, the health of survivors can be expressed as

$$\tilde{h}_{igt} = \frac{\int_{F^{-1}(Inf_{gt})}^{\inf} h_i dF(h)}{1 - Inf_{gt}} + \theta g(m_{gt}) \tag{9}$$

This implies that data on county-level infant mortality rates Inf_{gt}, medical services m_{gt} and indicators of health (or later life mortality) $\tilde{h_{igt}}$ of survivors, combined with assumptions about the normal distribution parameters μ and σ[38], will be sufficient to estimate the health premium that ensues from birth exposure to universal healthcare coverage. We will additionally use information on the differential infant mortality rates of population groups who had access to medical services before the NHS introduction, and those that newly gained access upon the introduction of the NHS in our estimation.

[38]Bozzoli et al. (2009) fix these parameters according to the height distribution of Danes between 1976 and 1980 when infant mortality rates were close to zero. We will use a similar approach.

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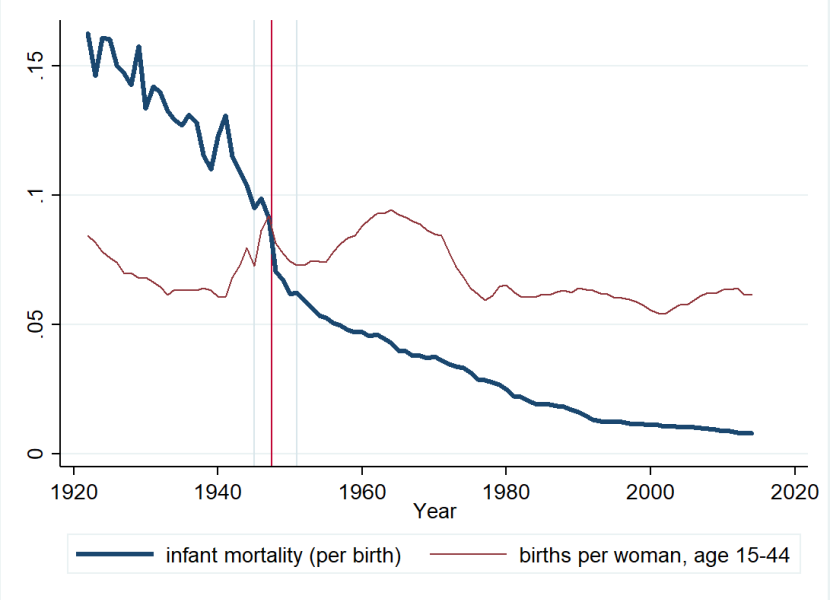
A Potential confounding effects

A.1 Trends in Births and Infant Mortality

We abstract from fertility choices which may a) have changed around the introduction of the NHS due to other factors, or b) may have changed due to the introduction of the NHS. First, we explore live births and their composition around the time of the NHS introduction. Large contractions (expansions) in the number of live births may decrease (increase) infant mortality through a lower (higher) demand for short-run fixed medical services and a consequent increase (decrease) in medical services per insured infant. Between 1940 and 1960 there was a great degree of fluctuation in the birth rate within the UK. Indeed the year prior to the introduction of the NHS saw the peak of the 'mini-baby-boom' following the demobilisation of servicemen at the end of WWII. An immediate concern is that the sharp decline in infant mortality in 1948 may be related to changes in the birth rate. We show this is not the case with two pieces of evidence.

Figure A.1: Infant mortality and births, UK, 1920-2016



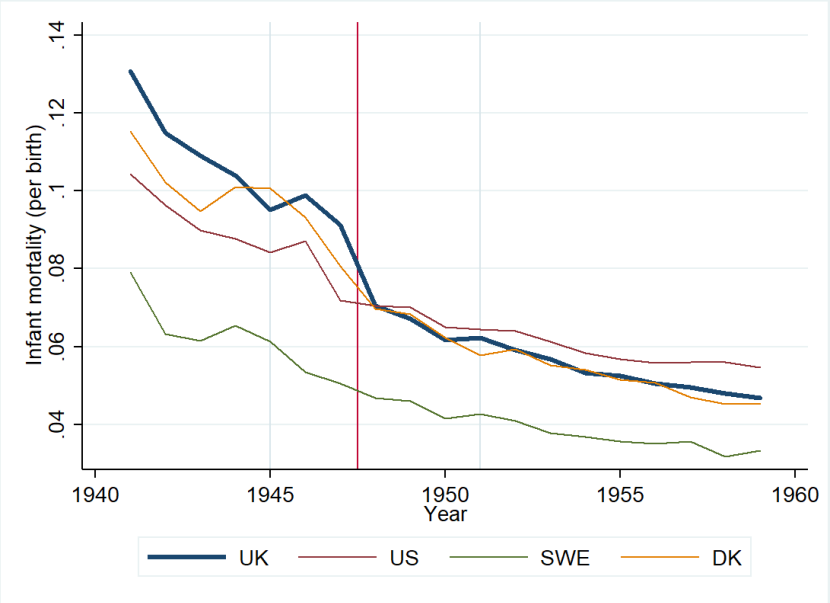
Notes: The graph indicates the infant mortality and fertility rates between 1920 and 2016. Source: Human Mortality Database

First, using UK data we compare the trends in infant mortality and births between 1920 and 2016, see Figure A.1. It is only in 1948 that we observe the sharp decline in infant mortality. Indeed there does not appear to be any

systematic link between birth and infant mortality rates.

Second, we compare the trend in infant mortality across different countries.[39]. Figure A.2 reveals that it was only the UK which experienced the sharp decline in infant mortality in 1948.

Figure A.2: Infant mortality in selected countries, 1941-1959



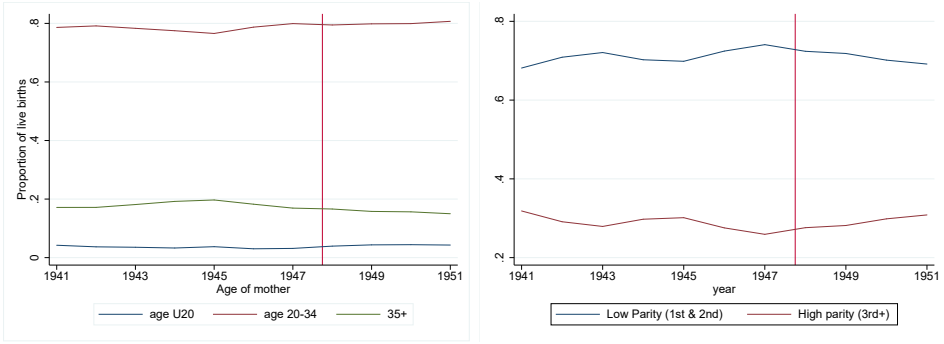
Notes: The graphs indicate infant mortality per live birth. Source: Human Mortality Database

Following the findings of Chevalier and Marie (2017), we posit that fertility composition may change over time with economic circumstances. We explore this hypothesis by looking at the composition of birth i) the age of the mother, ii) the birthorder of the child and iii) by legitimacy (i.e. whether the child was born within or outside wedlock) as a proxy for socio-economic status. Figure A.3(a) shows that there is no sharp discontinuity in the proportion of births in each age group. The proportion of prime-age mothers is relatively constant over the sample period at around 80%. There is a slight upward (downward) trend in the proportion of young (old) mothers, but no sharp change around the NHS introduction year. Similarly, Figure A.3(b), there is no discontinuity in the proportion of low or high parity births in the threshold year. Figure A.3(c) shows that although there was a small increase (decrease) in the proportion of within (outside) wedlock births during the WWII years,

[39]In this analysis we did not consider countries which experienced direct war operations during WWII because of the potential confounding influence

there is no change in the proportion of births by wedlock status from 1946 onwards.

Figure A.3: Birth composition, 1941-1951



(a) Age of mother

(b) Birth parity

(c) Legitimacy

Notes: The graphs indicate the proportion of births in each age/parity/legitimacy group Source: ONS Historic Birth Statistics

A.2 Rationing

Food was rationed in the UK between 1940 and 1954. The Ministry of Food prioritised maintaining the caloric intake of the population, although other important nutrients were monitored. Rationed foods included dairy products, meat, sugar, jam, tea, preserved fruit, bread and cereals. Many foods, including fresh fruit and vegetables, were available 'off-ration' and instead strict price controls were enforced. In addition, Government subsidies were provided for foodstuffs consumed by low-income households. In 1941 the Welfare Food Scheme was introduced which provided extra rations of milk, cheese and vitamins (in the form of concentrated orange juice and cod liver oil) for pregnant/nursing mothers, young children and heavy labourers.

Table A1: Energy value of food consumption 1945-49

	1945		1946		1947		1948		1949	
	Cals	% total	Cals	% total	Cals	% total	Cals	% total	Cals	% total
Bread/flour	721	30.4	690	29.9	716	31.0	759	31.8	723	29.8
Other cereals	247	10.4	232	10.1	223	9.7	245	10.3	244	10.1
		40.8		40.0		40.7		42.1		39.9
Potatoes	184	7.7	197	8.5	193	8.4	191	8.0	189	7.8
Other foods	153	6.4	164	7.1	157	6.9	158	6.6	148	6.1
Meat	296	12.5	270	11.7	257	11.1	229	9.6	224	9.3
Sugars	197	8.3	199	8.6	185	8.0	158	6.6	159	6.6
Milk	225	9.5	220	9.6	225	9.7	229	9.6	246	10.1
Fats	281	11.8	263	11.4	250	10.8	280	11.7	343	14.1
Other veg/fruit	41	3.0	67	2.9	73	3.2	70	2.9	75	3.1
Total	2,375	100.0	2,307	100.0	2,308	100.0	2,387	100.0	2,425	100.0

Calories per head per day
Source: National Food Survey

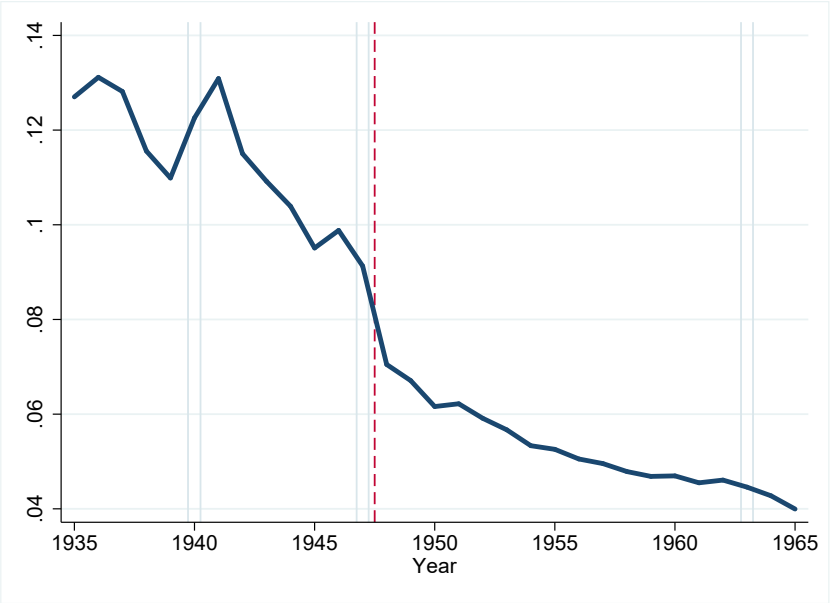
In the period around the introduction of the NHS there were only minor changes to food availability. The tea ration was slightly lowered, the bacon ration was reduced - although there was a compensating increase in the meat ration. Due to the poor harvest in 1945, bread came onto ration for the first time in 1946 and remained on ration for the next 2 years. Due to the harsh winter of the previous year, potatoes were rationed over the winter of 1947/8. However these periods coincided with an increase in availability of some 'off-ration' foods. As a result, caloric intake did not change substantially over the period of our analysis, as shown in Table A.2

A.3 Weather Shocks

Finally we investigate the impact of weather. The winter of 1946/47 was the harshest winter in the UK for almost a decade. The prolonged cold snap resulted in fuel shortages and disruption to the electricity supply in February, and potatoes were briefly rationed after their stores were destroyed by frost. The concern is whether potential excessive mortality during the winter months inflated the infant mortality rate in 1947 and as such provides a confounding influence to the impact that we attribute to the introduction of the NHS. Figure A.4 shows that this is not the case, and indeed the decrease

in infant mortality remained on trend in 1947. We go further and examine the other closest harsh winters to the period of our analysis. The winter of 1939/40 was the coldest in 45 years, and the "Big Freeze" of 1962/63 remains the coldest winter in the UK since 1895. We see that the infant mortality rate stayed on trend for all three periods, suggesting no systematic relationship between the infant mortality rate and the severe weather periods.

Figure A.4: Infant Mortality and Harsh Winters



Notes: The graph shows the UK infant mortality rate between 1935 and 1965. The solid vertical lines indicate the winters of 1939/40, 1946/47 and 1962/63. The dashed vertical line delineates the pre- and post-NHS periods. Source: Human Mortality Database

B Sensitivity analysis of mortality impacts

In this section, we present sensitivity analysis regarding the impacts of infancy exposure to the NHS on mortality. We first re-estimate model 1 omitting current region of residence fixed effects (see Table C1), with very little change in the estimates.

Secondly, we leave out Wales (see Table C2). We find that our estimates are not driven by differences between the two countries.

Since we use a regression discontinuity design, we next investigate whether our results are sensitive to our choice of cohort window. To check this, we narrow the window used in our main specification to the cohorts 1946 to 1950, omitting one cohort either side of the threshold. Our results remain qualitatively unchanged (see Table C3).

Finally, we account for differential mortality dynamics between men and women. Women's mortality rates between ages 52 and 64 are almost half those of men, We therefore split the sample by gender and estimate coefficients separately by gender (see Table C4). We find that infancy exposure impacts to the NHS are mainly concentrated among men.

Table C1: Estimates of mortality rates by age, no current location FE

	Death by age ...						
	52	**54**	**56**	**58**	**60**	**62**	**64**
$T_c * LC_{ic}$	-0.0175**	-0.0225***	-0.0189**	-0.0250**	-0.0279***	-0.0273***	-0.0313***
	(0.00736)	(0.00845)	(0.00837)	(0.00963)	(0.00965)	(0.0100)	(0.0109)
T_c	0.00741*	0.00971**	0.00635	0.00767	0.0108**	0.0100*	0.00881
	(0.00375)	(0.00408)	(0.00462)	(0.00491)	(0.00519)	(0.00514)	(0.00601)
LC_{ic}	0.0251***	0.0321***	0.0334***	0.0402***	0.0446***	0.0481***	0.0583***
	(0.00488)	(0.00580)	(0.00536)	(0.00662)	(0.00677)	(0.00642)	(0.00773)
Observations	44,122	44,122	44,122	44,122	44,122	44,122	44,122
R^2	0.004	0.005	0.006	0.006	0.007	0.008	0.009
F-test for joint significance of T_cLC_{ic} and T_c coefficients							
p-value	0.0561*	0.0256**	0.0862*	0.0409	0.0168**	0.0242**	0.0214**
Mean mortality rate prior to NHS inception, by social class							
LC	0.0488	0.0606	0.0730	0.0884	0.1029	0.1209	0.1421
HC	0.0306	0.0367	0.0462	0.0558	0.0657	0.0783	0.0899
Mortality reduction in percent (relative to mean), by social class							
LC	-20.68	-21.11	-17.19	(-19.60)	-16.62	-14.31	-15.83
HC	24.22	26.46	(13.74)	(13.75)	16.44	12.77	(9.80)

Notes: The table shows estimates of equation 1. All specifications control for additional characteristics determined at birth (gender, birth location, and parental origin)
*and include cohort trends. Robust standard errors are reported in parentheses. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.*
Source: ONS Longitudinal Study

Table C2: Estimates of mortality rate by age, excluding Wales

	Death by age ...						
	52	54	56	58	60	62	64
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
T_cLC_{ic}	-0.0178**	-0.0231**	-0.0209**	-0.0273***	-0.0293***	-0.0289***	-0.0351***
	(0.00777)	(0.00889)	(0.00868)	(0.0101)	(0.0101)	(0.0105)	(0.0112)
T_c	0.00839**	0.0104**	0.00702	0.00829	0.0111**	0.0102*	0.00979
	(0.00393)	(0.00423)	(0.00480)	(0.00508)	(0.00542)	(0.00535)	(0.00621)
LC_{ic}	0.0258***	0.0331***	0.0349***	0.0421***	0.0462***	0.0499***	0.0613***
	(0.00512)	(0.00605)	(0.00546)	(0.00680)	(0.00692)	(0.00653)	(0.00770)
Obs.	41,826	41,826	41,826	41,826	41,826	41,826	41,826
F-test for joint significance of T_cLC_{ic} and T_c coefficients							
p-value	0.0574*	0.0279**	0.0641*	0.0322**	0.0172**	0.0238**	0.0109**

*Notes: The table shows estimates of equation (1). All specifications control for gender, birth location, home location. We include cohort trends, and allow for specific cohort-trends for lower social class individuals. Standard errors are clustered at county of birth level and reported in parentheses. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.*
Source: ONS Longitudinal Study

Table C3: Estimates of mortality by age, cohorts 1946 to 1950

	Death by age ...						
	52	54	56	58	60	62	64
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
$T_c LC_{ic}$	-0.0261***	-0.0307***	-0.0220*	-0.0280**	-0.0282**	-0.0218	-0.0205
	(0.00965)	(0.0115)	(0.0113)	(0.0125)	(0.0126)	(0.0142)	(0.0134)
T_c	0.00648	0.00886*	0.00553	0.00549	0.00670	0.00428	0.000652
	(0.00455)	(0.00513)	(0.00563)	(0.00614)	(0.00613)	(0.00603)	(0.00673)
LC_{ic}	0.0326***	0.0395***	0.0372***	0.0448***	0.0482***	0.0488***	0.0577***
	(0.00599)	(0.00710)	(0.00661)	(0.00754)	(0.00746)	(0.00761)	(0.00814)
Obs.	32,444	32,444	32,444	32,444	32,444	32,444	32,444
F-test for joint significance of $T_c LC_{ic}$ and T_c coefficients							
p-values	0.0251**	0.0335**	0.1548	0.0797*	0.0898*	0.3104	0.3076

*Notes: The table shows estimates of equation (1). All specifications control for gender, birth location, home location. We include cohort trends, and allow for specific cohort-trends for lower social class individuals. Standard errors are clustered at county of birth level and reported in parentheses. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. [a]:*
Source: ONS Longitudinal Studies

Table C4: Estimates of mortality rate by age, by gender

	Death by ...							
	age 52		age 56		age 60		age 64	
	M	F	M	F	M	F	M	F
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
$T_c LC_{ic}$	-0.0315**	-0.0013	-0.0347**	-0.0013	-0.0496***	-0.0044	-0.0555***	-0.0043
	(0.0128)	(0.0084)	(0.0146)	(0.0097)	(0.0175)	(0.0139)	(0.0165)	(0.0152)
T_c	0.0088	0.0060	0.0097	0.0032	0.0198**	0.0020	0.0209**	-0.0033
	(0.0060)	(0.0039)	(0.0063)	(0.0057)	(0.0085)	(0.0074)	(0.0100)	(0.0078)
LC_{ic}	0.0376***	0.0107*	0.0486***	0.0161***	0.0646***	0.0221**	0.0824***	0.0312***
	(0.0079)	(0.0057)	(0.0085)	(0.0060)	(0.0103)	(0.0083)	(0.0102)	(0.0088)
Obs.	22,445	21,677	22,445	21,677	22,445	21,677	22,445	21,677
F-test for joint significance of $T_c LC_{ic}$ and T_c coefficients								
p-values	0.0531*	0.2898	0.0666*	0.8541	0.0172**	0.9391	0.0050***	0.7665

*Notes: The table shows estimates of equation (1). All specifications control for gender, birth location, home location. We include cohort trends, and allow for specific cohort-trends for lower social class individuals. Standard errors are clustered at county of birth level and reported in parentheses. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.*
Source: ONS Longitudinal Study

C Sensitivity analysis of health impacts

In this section, we present sensitivity analysis regarding the impacts of infancy exposure to the NHS on the incidence and onset of cardiovascular disease. We first re-estimate model 1 leaving out Wales (see columns 1, 3 and 5 in Table D1), and find qualitatively identical and quantitatively similar results. Furthermore, columns 2,4,6,and 8 of the tables display estimates excluding individuals born in London. We find that our estimates are not driven by dynamics in the population-rich London.

Since we use a regression discontinuity design, we next investigate whether our results are sensitive to our choice of cohort window. To check this, we narrow the window used in our main specification, i.e. roughly 40 months either side of the July 1948 threshold, to 20 months either side of the threshold. Our results remain qualitatively unchanged (see Table D2). Finally, our results are also robust when we separately estimate impacts among men and women (see Table D3).

Table D1: Estimates of incidence of cardiovascular disease by age, specific regions

	Incidence by ...					
	age 56		age 60		age 64	
	(1)	(2)	(3)	(4)	(5)	(6)
$T_c LC_{ic}$	-0.0106***	-0.0105***	-0.0103*	-0.0094*	-0.0157**	-0.0153**
	(0.003)	(0.003)	(0.005)	(0.006)	(0.007)	(0.007)
T_c	0.0068***	0.0066***	0.0062	0.0059	0.0087**	0.0082*
	(0.002)	(0.002)	(0.004)	(0.004)	(0.004)	(0.004)
LC_{ic}	0.0172***	0.0167***	0.0265***	0.0261***	0.0342***	0.0327***
	(0.004)	(0.004)	(0.004)	(0.004)	(0.005)	(0.005)
Estimates exclude:						
Wales	Y	Y	Y	Y	Y	Y
London	N	Y	N	Y	N	Y
Obs.	119,587	111,668	119,587	111,668	119,587	111,668
F-test for joint significance of $T_c LC_{ic}$ and T_c coefficients						
p-values	0.0013	0.0040	0.1215	0.200	0.0325	0.0568
Mean incidence rate prior to NHS inception by social class						
LC	0.0426	0.0428	0.0872	0.0877	0.1399	0.1405
HC	0.0294	0.0299	0.0630	0.0642	0.1036	0.1051
Incidence change in percent (relative to pre-NHS mean)						
LC	-8.92	-9.11	-4.70	-3.99	-5.00	-5.05
HC	23.13	22.07	(9.84)	(9.19)	8.40	7.80

*Notes: The table shows estimates of equation (1). All specifications control for gender, birth location, home location. We include cohort trends, and allow for specific cohort-trends for lower social class individuals, and allow for a trend break post NHS. Standard errors are clustered at county of birth level and reported in parentheses. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. [a]: For robustness, we condition on surviving to at least age 65 to avoid bias from cohort-specific sample selection due to the late interview year in which the Biobank was started.*
Source: UK Biobank

Table D2: Estimates of incidence of cardiovascular disease by age, ± 20 months around the threshold

	Incidence by age ...		
	56	60	64
	(1)	(2)	(3)
$T_c LC_{ic}$	-0.0115**	-0.0115	-0.0190**
	(0.005)	(0.007)	(0.009)
T_c	0.0061*	0.0081	0.0113*
	(0.003)	(0.005)	(0.006))
LC_{ic}	0.0142***	0.0282***	0.0340***
	(0.005)	(0.006)	(0.009)
Obs.	67,231	67,231	67,231
F-test for joint significance of $T_c LC_{ic}$ and T_c coefficients			
p-values	0.0499	0.1929	0.0797
Mean incidence rate prior to NHS inception by social class			
LC	0.0424	0.0857	0.1098
HC	0.0275	0.0616	0.10160
Incidence change in percent (relative to pre-NHS mean)			
LC	-12.74	(-3.97)	-7.01
HC	22.18	(13.14)	11.12

*Notes: The table shows estimates of equation (1). All specifications control for gender, birth location, home location. We include cohort trends, and allow for specific cohort-trends for lower social class individuals, and allow for a trend break post NHS. Standard errors are clustered at county of birth level and reported in parentheses. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. [a]:*
Source: UK Biobank

Table D3: Estimates of incidence of cardiovascular disease by age, by gender

	Incidence by ...					
	age 56		age 60		age 64	
	M	F	M	F	M	F
	(1)	(2)	(3)	(4)	(5)	(6)
$T_c LC_{ic}$	-0.0104*	-0.0086*	-0.0096	-0.0092	-0.0213*	-0.0117
	(0.006)	(0.004)	(0.009)	(0.006)	(0.012)	(0.008)
T_c	0.0064*	0.0072**	0.0101	0.0055	0.0148*	0.0063
	(0.003)	(0.004)	(0.007)	(0.004)	(0.008)	(0.005)
LC_{ic}	0.0195***	0.0145***	0.0298***	0.0230***	0.0403***	0.0289***
	(0.006)	(0.004)	(0.007)	(0.006)	(0.010)	(0.005)
Obs.	57,512	68,566	57,512	68,566	57,512	68,566
F-test for joint significance of $T_c LC_{ic}$ and T_c coefficients						
p-values	0.1036	0.0460**	0.3282	0.1908	0.1601	0.1761
Mean incidence rate prior to NHS inception by social class						
LC	0.0522	0.0349	0.1096	0.0692	0.1773	0.1097
HC	0.0352	0.0242	0.0778	0.0498	0.1304	0.0798
Incidence change in percent (relative to pre-NHS mean)						
LC	-7.66	-4.01	(0.46)	(-5.34)	-3.67	-4.92
HC	18.18	29.75	(12.98)	(11.04)	11.35	7.89

Notes: The table shows estimates of equation (1). All specifications control for gender, birth location, home location. We include cohort trends, and allow for specific cohort-trends for lower social class individuals, and allow for a trend break post NHS. Standard errors are clustered at county of birth level and reported in parentheses. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. [a]*: For robustness, we condition on surviving to at least age 65 to avoid bias from cohort-specific sample selection due to the late interview year in which the Biobank was started.*
Source: UK Biobank