

A Gift of Health: The Duke Endowment's Impact on Hospital Care and Mortality*

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July 9, 2021

Does access to hospital care affect infant and later-life mortality? We study this question in the context of a large-scale expansion in hospital care in North Carolina that was facilitated by funding from the Duke Endowment starting in the mid-1920s. Difference-in-differences estimates imply that counties exposed to Duke funding experienced reductions in infant mortality and mortality at older ages. These gains are qualitatively larger for African Americans compared to Whites. Plausible mechanisms include improved access to nursing care and higher-skilled physicians. On the other hand, we do not find evidence of additional gains from new pharmaceutical treatments available to hospital patients.

JEL: I14, J13, N32

Keywords: hospital access, mortality, childhood health

*We thank seminar participants at Emory University and the University of Copenhagen.

1 Introduction

The barriers that limit access to health care in the United States today - a lack of providers in rural areas, financial difficulties and poverty, insufficient insurance, and structural impediments such as a lack of transportation - were challenging patients almost 100 years ago in 1924, when James Buchanan Duke established the Duke Endowment to relieve these burdens. The Duke Endowment's Hospital Section subsidized free care and funded the construction of new hospitals and the expansion of existing ones, particularly in rural areas, throughout North Carolina.¹ It also provided funding for improved hospital equipment and increased the number of well-trained staff in existing hospitals. By the end of 1949, The Endowment had poured over \$27 million (nearly \$300 million in 2020 dollars) into hospital construction and modernization. Duke's financial resources significantly increased North Carolina's medical infrastructure in terms of both the number of hospitals and beds available. On the eve of Duke's involvement in medical care in 1925, North Carolina was served by 122 hospitals with 5,334 beds, but 49 percent of counties had no hospitals. Across these hospitals, total capacity averaged 44 beds. By the end of 1949, the number of hospitals increased 41 percent to 172 hospitals, and the number of beds increased by 410 percent to 27,178, with the average hospital having 158 beds.

In this paper, we examine the Duke Endowment's impact on access to hospital care, infant mortality, and on the longevity of individuals exposed to Duke's contributions during their pre-natal and infancy years. To study the impact of Duke Endowment spending, we construct a new data set that combines reports from the Duke Endowment with data on hospitals, physicians, and nurses from a variety of sources. We rely on vital statistics information from North Carolina death certificates and county-level information on natality and mortality from the ICPSR. To examine how Duke funding may have impacted longevity, we use the 2007 version of the Social Security Administration NUMIDENT data. In our analysis we pay particular attention to differential effects of spending by race. This is important not only because of long-standing mortality gaps

¹The Duke Endowment subsidized these efforts in both North and South Carolina, but due to limited data on outcomes for South Carolina we limit our analysis to North Carolina only.

between African Americans and Whites, but also because The Duke Endowment's Indenture of Trust specifically stipulated that contributions be paid to hospitals "...whether white or colored," in keeping with the Duke family's history of philanthropy on behalf of African Americans (Durdin 1998, 85). Econometrically, we estimate the effect of Duke Endowment funding on health outcomes by estimating two-way fixed effects difference-in-differences (TWFE DiD) regressions and event study specifications of changes in infant mortality rates and changes in the probability of death by age 65.

Our rich data set also allows us to explore whether hospitals supported by the Duke Endowment attracted more physicians and nurses, and how the human capital of these physicians compared to the existing stock of physicians in a county. Thus, we further investigate whether gains in mortality can be attributed to the number and quality of physicians and nurses who were present. Lastly, we interact Duke funding with the availability of sulfa drugs after 1937 to measure the interaction between increased medical access and medical innovations. For example, Thomasson and Treber (2008) show that increased access to hospital care did not reduce maternal mortality until the development of sulfa. Duke Endowment reports suggest that fatality rates after surgery and caesarean sections fell significantly between 1925 and 1949. In North Carolina, for example, the fatality rate following surgery fell from 4.5 percent to 2.7 percent, while the fatality rate following caesarean sections fell from 13.3 percent to 0.9 percent. By interacting Duke funding with the availability of sulfa, we can disentangle the effect of having more hospitals and physicians with the impact of adding improved technology.

We find evidence that exposure to Duke funding prenatally and in infancy reduced infant mortality by about 10 percent and reduced the likelihood of death between ages 20 to 65 by up to 4 percent. These average gains are not uniform across racial groups; infant mortality for African Americans dropped by over 13 percent, while White infant mortality fell by only about 6 percent. We do not find statistically significant estimates by race for long-run mortality, but the estimate for African Americans is almost three-times larger than for Whites. In addition, one of the reasons The Duke Endowment funded hospitals was to attract providers to rural areas by

providing adequate medical facilities ([The Duke Endowment 1925](#), 44). Our analysis confirms that Duke Endowment funding did lead to increases in the supply of more recently graduated and thus better trained physicians, as well as increases in the supply of nurses. When we control for the interaction of Duke funding and the availability of sulfa, due to lack of precision we do not find any statistically significant compounding effects. For both infant mortality and adult mortality, however, the point estimates are almost twice as large in the aftermath of the sulfa innovation.

Our analyses provide a unique set of contributions to several different strands of literature. First, our results contribute to the literature on the long-run consequences of access to health care early in life. Several studies analyze the effects of access to health insurance on morality and adult outcomes. In the U.S. context, [Goodman-Bacon \(2017\)](#) shows that childhood Medicaid eligibility during the program's introduction reduces mortality and disability, increases employment, and reduces receipt of disability transfer programs up to 50 years later. Similarly Medicaid expansions in the 1980s have been shown to have positive long-run effects on health, human capital, and earnings ([Brown et al. 2020](#); [Miller and Wherry 2019](#); [Cohodes et al. 2016](#)). However, a complicating factor in these studies is that the expansions combine medical, food and, cash benefits jointly rather than isolating the healthcare access component. Outside the U.S., [Lührmann and Wilson \(2018\)](#) show that universal healthcare, with the introduction of the NHS in the United Kingdom, not only immediately decreased infant mortality but also increased longevity later in life. [Bauernschuster et al. \(2019\)](#) study the introduction of social health insurance during the 19th century Bismarckian era of the German Empire and finds large reductions in mortality despite the lack of antibiotics and most vaccines. Importantly from our perspective, their main mechanism hinges upon access to well-trained doctors who were able to diminish deaths from infectious diseases.

Relatedly, Anderson, Charles, and Rees investigate a series of public health policies in the early to mid 20th century in the US. [Anderson et al. \(2021a\)](#) study the racial desegregation in Southern US hospitals in the 1960s - a period after time considered in this paper. Contrary to our findings they don't find effects of these policies on either White or African-American infant

mortality. On the other hand, [Anderson et al. \(2020\)](#) find that midwifery reforms between 1900 and 1940 in the US reduced maternal mortality but had little if any effects on infant mortality. Finally, [Anderson et al. \(2021b\)](#) document that among many public health policies implemented to fight against food- and water-borne diseases, only water filtration lead to sizable declines in infant mortality. Their estimates of about 11-12 percent are very similar to what we find in this paper for effects of improved hospital funding.

Another strand of related literature focuses on the physical infrastructure of health care and access to nurses, doctors, and preventative services. [Moehling and Thomasson \(2014\)](#) document that the Sheppard-Towner Act increased one-on-one contacts with physicians and nurses which in turn reduced infant mortality, and the effects were particularly pronounced among nonwhites for whom deprivation in access to health care was the greatest. In the Scandinavian context, both [Bütikofer et al. \(2019\)](#) and [Hjort et al. \(2017\)](#) show long-term health benefits of access to nurse home visiting programs in Norway and Denmark, respectively. Furthermore, [Bütikofer et al. \(2019\)](#) show that treated children experienced improved socioeconomic outcomes. Similarly, for Sweden, [Lazuka \(2020\)](#) finds that access to maternity wards had a short-run effect on neonatal mortality and long-run impacts on labor income and disability pensions. Some of the proposed mechanisms behind these findings include not only improved nutrition at critical stages of life, but also availability of fast referral of ill infants to a doctor. This finding motivates our investigation of improved access to doctors and hospital care. On the other hand, work by [Carrillo and Feres \(2019\)](#) in Brazil suggests that physician and nurse care, at least in a contemporary setting, could be good substitutes in the production of infant care, given that the authors do not find that the increased supply of physicians who replaced nurses lead to health gains in the short-run. Our setting is somewhat different, however, since the historical Duke Endowment funding increased supply of both nurses and doctors as well as improved the quality of hospitals.

Since we investigate an interaction between access to health care and technological change in medicine brought on by the invention of sulfa drugs, we naturally rely heavily on work exploring the consequences of this scientific breakthrough. [Jayachandran et al. \(2010\)](#) documents

that the introduction of the sulfa drugs in 1937 in the U.S., which we adopt as our definition of treatment, lead to large declines in several types of mortality from infectious diseases and an overall decrease in mortality of 2 to 3 percent. The benefits from antibiotic therapies translated into other positive long-run outcomes for children exposed in infancy but were less beneficial for African Americans ([Bhalotra and Venkataramani 2015](#)). Socioeconomic gains were observed in adult education, employment, disability, income and income mobility. One reason why African Americans did not reap the full benefits of this innovation could have been the lack of access to hospital care. This motivates the examination of exogenous variation in access to hospital care due to funding from the Duke Endowment in this study. It is also worth noting that benefits from sulfa drugs introduction were not limited to the U.S. as [Lazuka \(2020\)](#) shows similar gains in the case of Sweden.

Very few studies in the extant literature examine how policies or economic shocks interact with each other, and in particular how changes in physical and human capital can interact with technological improvements. The closest in spirit to our work is [Alsan and Goldin \(2019\)](#) who examine the interaction between clean water provision and technological innovation in sewerage disposal and its effects on child mortality in the context of the late-19th and early-20th century U.S. Two other studies investigate interactions between multiple health inputs. Both [Bütikofer et al. \(2021\)](#) and [Gunnsteinsson et al. \(2019\)](#) show that early post-natal policies, access to health checkups in the former study and vitamin A supplementation in the latter one, can offset negative prenatal health shocks. Furthermore, [Rossin-Slater and Wüst \(2020\)](#) study an interaction between nurse home visiting program and the availability of high quality preschool while research from developing countries generally focuses on the interaction between a financial shock (e.g., through rainfall among farmers) and poverty alleviation programs ([Adhvaryu et al. 2018](#); [Duque et al. 2019](#)).

Our work further complements studies examining the determinants of mortality and longevity. Previous research has focused on the relationships between education and mortality ([Clark and Royer 2013](#); [Galama et al. 2018](#)), income and mortality ([Cesarini et al. 2016](#); [Chetty et al. 2016](#)),

pollution and mortality ([Hanlon 2018](#); [Alsan and Goldin 2019](#)), or climate change and mortality ([Deschenes and Greenstone 2011](#); [Barreca et al. 2016](#)). On the other hand, we focus on improved access to hospital care and medical technology for both the immediate mortality effects as well as longer-run longevity outcomes.

Finally, our paper connects to the broader literature on the long-run consequences of prenatal and early childhood health. Evidence to date shows that prenatal health ([Black et al. 2007b](#); [Figlio et al. 2014](#); [Bharadwaj et al. 2018](#)) is important for cognitive development, labor market outcomes, and elderly health. Much less is known about health in childhood but both contemporaneous ([Currie et al. 2010](#)) and historical ([Karbownik and Wray 2019](#)) evidence suggests that it matters for both health and socioeconomic outcomes.

2 Duke Endowment and Medical Care

James Buchanan Duke established The Duke Endowment with initial funding of \$40 million to improve access to health care, education, and spiritual services for people in North and South Carolina. He directed that 80 percent of the income from the invested funds would then be apportioned as follows: 32 percent to Duke University; 32 percent to the maintenance and construction of hospitals; 10 percent to the care of orphans and “half orphans”; 14 percent to be divided between Davidson College, Furman University, and the Johnson C. Smith University; and the remainder to Methodist ministers and the building and maintenance of rural Methodist churches.

The Duke Endowment limited assistance to nonprofit and public hospitals, stating “..sickness is too big a problem for private resources and too socially important a problem to be neglected, avoided or shirked by the community” ([The Duke Endowment 1925](#), 138). Under the provisions of the Deed of Trust, The Endowment could give one dollar per day to hospitals treating patients free of charge.² Any surplus over this expenditure could be used to build new hospitals, expand

²\$1 per day represented about 1/3 of the average daily cost of treating a patient. The Duke Endowment was reluctant to pay more because it feared that doing so would “...tend to paralyze community interest and effort” involved with paying for charity care ([The Duke Endowment 1925](#), 144). However, as this amount was fixed in nominal terms in the Deed of Trust, it did not grow over time and its real value declined as the cost of care increased.

existing ones, or add new equipment. Special attention was paid to racial equality and funding was more abundant for hospitals which accepted patients of both races. In fact, the charity would often buy shares in a White-only hospital so that they could convert it to a mixed-race institution. Duke also noted that the development of improved hospital facilities was the means to their greater objective of improving the number and quality of doctors and nurses practicing medicine in an area. In its first report, The Hospital Section noted, “Hospitals hold and attract the better type of physicians...So it is that a local hospital builds up a profession, raises professional standards and serves to improve the practice of medicine not only for patients in the hospital but for patients in the whole county” ([The Duke Endowment 1925](#), 144-45). The additional resources reached a fertile ground as North Carolina struggled with providing quality health care for their citizens. At baseline in 1925, there were 122 hospitals in 51 counties, while 49 percent of counties did not have any hospital facility. This situation has changed dramatically over the first 17 years of the endowment’s activity and by 1942 only 7% of counties did not have any hospital.³ Figure 1a depicts the roll-out for the share of counties receiving Duke funding (panel A) and total number of beds per 100,000 (panel B). A similar picture can be drawn when investigating young physician density, which increased from 26 per 1000 population in 1923 to 56 per 1000 population in 1940. The actual utilization likewise increased as the fraction of births attended by physicians increased from 1925 to 1940 as well, moving from 85.9% to 99.6% for whites and nearly tripling for blacks from 29.5% to 76.6%. Thus, due to the Duke Endowment, there is clearly an increase in coverage, utilization, and financing of hospital care in North Carolina.

The investments also had important consequences for racial disparities in access to health care. Although African Americans constituted about 30 percent of the population in the Carolinas in 1924, they could only access a far more limited set of hospitals due to segregation. The inequality in access to health care was likewise reflected in the inequality of outcomes. For ex-

³The Duke Endowment funding was actually distributed beyond 1942, and we collected data for the period up to 1962. Since one of our outcomes of interest is longevity we cut the sample in 1942 so that we can observe individuals up to the age of 65 in 2007 which is the last year for which we have information on deaths from the Social Security Administration. Post-1942 expansion was relatively smaller than the initial shock from the start of the Fund while in Section 6 we document that our infant mortality results remain very similar when we consider using the whole data span of 1925 to 1960.

ample, in 1925 only 29.5 percent of non-white births were attended by a physician as compared to 85.9 percent for white births. Likewise, African-American women in North Carolina were twice as likely as white women to die from complications of childbirth. The investment studied in this paper provided opportunity to massively narrow these racial disparities.

3 Data

Our paper draws on a new data set of hospital-level information that we compiled from Duke Endowment reports between 1925 and 1942. We link this data to historical death certificates from North Carolina as well as to individual-level vital statistics information from Numident. The three data sets allow us to combine variation in access to and quality of hospital care in infancy and early childhood with contemporaneous and long-run mortality information for the universe of births to residents of North Carolina between 1920 and 1942.

3.1 Duke Endowment Records

Our first data set consists of annual reports of the Duke Endowment between 1925 and 1942 which include detailed information about the health projects financed by the Duke Endowment. We digitized and transcribed this information from books issued annually by the foundation. Each volume contains somewhat different information and not all information is available for every year. Nonetheless, we were able to extract consistent information on hospital-level contributions for both newly built as well as existing institutions and information on hospital operations. Information from the latter set of variables includes number of beds, number of total days of care as well as number of free days of care (i.e., those funded by the Duke Endowment). Auxiliary information on these hospitals is also available to us and includes location within a county and when the hospital started its operations. We harmonize all these data across years and construct a panel of hospitals with all available information. We then aggregate this information up to the county-by-year level.

3.2 North Carolina death certificates

Our efforts to study the effects of Duke Endowment funding on mortality are complicated by incomplete birth and death registration during our sample period, especially in the U.S. South. Ideally, we would like to observe deaths by place of residence at birth. However, data on infant deaths that are available for North Carolina throughout our study period are reported by place of *occurrence*. Somewhat better are data on deaths by place of *residence*, but these are not reported until 1942 - the final year of our sample (Bailey et al. 2016). Deaths by place of occurrence are problematic for our objectives since they are endogenous to hospitalization - more deaths occur in places where more hospitals are built. Even deaths by place of residence may be problematic if families selectively migrated to locations with better health care during a child's infancy.

Instead, we use a database containing the universe of North Carolina death certificates from 1906 to 1976 (Cook et al. 2014, 2016).⁴ Importantly, the data include place of birth. Thus, we identify the county of residence at birth corresponding to the reported birth location and then aggregate the individual deaths to the county-by-year of birth level. Given that we link these cohorts to county-by-year variation in Duke Endowment funding, our assumption is that individuals did not move across county lines between birth and death during infancy. However, in the presence of migration, we can interpret our estimates as intention-to-treat effects for the county of birth. As infant mortality rates are typically expressed as the number of infant deaths per 1,000 live births - either in levels or in logs - our analysis requires not only data on infant deaths, but also on births.⁵ We use data on births by year and county of occurrence from Bailey et al. (2016) to construct infant mortality rates and to use as weights in regressions with data at the county-by-year level.

⁴We thank John Parman for sharing these data.

⁵Our later-life mortality rate variables are also constructed as the ratio of mortality counts to birth cohort size.

3.3 Numident

We face two primary challenges when it comes to estimating the effect of Duke Endowment funding on longevity. First, we do not observe completed lifespan seeing as some individuals from birth cohorts in our sample period, especially those towards the tail end, remain alive today. Second, to our best knowledge, publicly-available data on mortality for the cohorts of interest do not extend to the present day. Thus, we turn to the public-use version of the Social Security Administration (SSA)'s Numerical Identification Files (NUMIDENT) that is provided by the National Archives and Records Administration (NARA).

The NUMIDENT series contains records for every social security number (SSN) assigned to individuals with a verified death or who would have been over 110 years old by December 31, 2007. With 1942 births being the latest cohort used in our analysis, the right-censoring of the NUMIDENT in 2007 implies that for all cohorts in our sample we can observe mortality by age 65 (i.e. a death at age 66 from the 1942 birth cohort would have occurred in 2008 and would only be found in the restricted-use data). An additional limitation with the public-use version is that it excludes so-called State reported deaths, which consist primarily of deaths of children who had not collected Social Security benefits, such as in the case of disability. Given these data features, we use deaths between the ages of 20 and 65 as a measure of later-life mortality.

The NUMIDENT also enables us to link exposure to Duke Endowment funding to county-by-year birth cohorts as it contains date and place of birth. However, since the place of birth text string is truncated at the 20th position, we use a crosswalk file that maps the text strings to a geocoded location and collapse the data by county and year of birth ([Black et al. 2015](#)).

3.4 Physicians and Nurses

One of our hypothesized mechanisms for the improvement in outcomes is that more and better qualified physicians and nurses moved to countries that received the Duke funding. We explore this possibility using two data sources. First, we use data on U.S. physicians from the

American Medical Directories which are available for 1914, 1918, 1923, 1927, 1931, 1936 and 1940. They include the physician’s name, their place of practice, the medical school from which they graduated, their year of birth, year of medical school graduation, and year of licensing, as well as detailed information on their specialization. We limit our analysis to doctors working in North Carolina. After cleaning the data we construct indicators for physician quality: experience proxied by age and years from graduation as well as admission requirements in their medical school for a respective cohort of graduates. We attach this information to our baseline data at the county-by-year level.

As a measure of access to nurses, we turn to the complete count U.S. census data for the 1910 to 1940 censuses. We use the IPUMS occupation and industry codes to identify nurses and collapse the data at the county-by-census-year level to obtain counts of medical practitioners for each census year. The census data have the advantage of including pre-treatment years prior to the introduction of Duke Endowment funding and having information on nurses which the AMD data lack. The downside is that the data are lower frequency at only the decennial level.

4 Methods

We use a difference-in-differences framework to estimate the causal effects of exposure during the in utero and infancy periods to improved access to medical care. In our baseline specification we assign treatment based on the timing of the first appropriation for capital expenditure from the Duke Endowment to a county. Thus, the first difference compares cohorts born before versus after the first appropriation in a county (time variation), while the second difference contrasts counties that received funding at a given point in time with those that did not (spatial variation). We assign exposure based on a child’s place of birth because this is the geographic unit at which our mortality variables are measured. We first estimate the following regressions:

$$Y_{ct} = \alpha_0 + \alpha_1 \text{First appropriation}_{ct} + \gamma_c + \delta_t + \Theta \mathbf{X}_{ct} + \epsilon_{ct} \quad (1)$$

where c indexes county of birth while t indexes birth year. Y_{ct} is one of the two dependent

variables: (1) the natural log of the county-by-birth year infant mortality rate defined in section 3.2 and (2) the probability of death between ages 20 and 65 as defined in section 3.3. The vector \mathbf{X}_{ct} , included in some specifications, contains the following control variables: percent of the population that is illiterate, percent of the population that is black, percent of the population that is of other (non-white) race, percent of the population residing in urban areas, retail sales per capita, and manufacturing wages per capita. Furthermore, all specifications include county of birth (γ) and year of birth (δ) fixed effects. We cluster the standard errors (ϵ_{ct}) by county of birth to account for correlated errors within a county.

Our variable of interest is $\text{First appropriation}_{ct}$ which takes the value of one for the first year, and all the years thereafter, that a county received an appropriation from the Duke Endowment. Thus, in this regression model, the coefficient (α_1) measures the effects of exposure to additional financial resources for health care on the two mortality outcomes. This is an intent to treat effect, and it does not account for either utilization of the financial resources (i.e., actual spending size) or allocation of funding (i.e., what the money was spend on). We also present even-studies of the following form:

$$Y_{ct} = \alpha_0 + \sum_{j=-7}^{-2} \beta_j \text{First appropriation}_{ct} + \sum_{j=0}^7 \beta_j \text{First appropriation}_{ct} + \gamma_c + \delta_t + \Theta \mathbf{X}_{ct} + \epsilon_{ct} \quad (2)$$

Here coefficients β_{-7} to β_{-2} capture trends prior to the start of the funding while coefficients β_0 to β_7 capture effects of access to Duke Endowment resources. The omitted category is one year prior to the start of the treatment. Our event study plots omit the left-most and right-most coefficients which are binned such as the coefficient on β_{-7} includes time periods between -20 and -7 while the coefficient on β_7 includes time periods between 7 and 15 . We weight observations by county birth cohort size and do not include additional controls for ease of interpretation.

We are also interested in the interaction effect between improved access to health care facilities and medical technology. Therefore, we overlay our Duke Endowment difference-in-differences estimation strategy with cohort-level variation stemming from the introduction of sulfa antibiotics. We estimate the following equation for both infant mortality as well as for the mortality

rate at ages 20 to 65:

$$\begin{aligned}
 Y_{ct} &= \alpha_0 + \alpha_1 \text{First appropriation}_{ct} + \alpha_2 \text{First appropriation}_{ct} \times \text{Sulfa innovation}_t \\
 &+ \gamma_c + \delta_t + \Theta \mathbf{X}_{ct} + \epsilon_{ct}
 \end{aligned} \tag{3}$$

where the variables and fixed effects are identical to those defined in Equation (1), but we add the term $\text{First appropriation}_{ct} \times \text{Sulfa innovation}_t$. This variable interacts our Duke exposure (i.e., an indicator for the first year, and all the years thereafter, that a county receives an appropriation) with a dummy variable that takes a value of one for all cohorts born in or after 1937, the year when sulfa drugs were introduced to the U.S. market. In this setting, α_1 estimates the effect of Duke funding prior to sulfa innovation while α_2 estimates the additional effect from Duke funding when sulfa drugs became available and were used in medical practice. Since this treatment is only time-varying we do not need an additional term for $\text{Sulfa innovation}_t$ as this variable is collinear with our birth year fixed effects δ .

We also implement a stacked regression estimator following [Cengiz et al. \(2019\)](#) to ensure that no previously treated counties are included as part of the control group. We do so by creating treatment-timing group specific data sets that include counties first exposed to Duke Endowment funding in a particular year and “clean control” counties that have not yet been treated in the estimation window of $t = -6$ to $t = 6$. We stack these data sets and estimate DiD regressions and event study specifications that modify equations 1 and 2, respectively, by saturating the county and year fixed effects with indicators for each of the stacked data sets. Lastly, we apply the [Callaway and Sant’Anna \(2021\)](#) estimator using only never-treated counties as controls to estimate event study specifications and an aggregate treatment effects.

5 Results

We present our main results in Table 1. Columns (1) and (2) present results for infant mortality while columns (3) and (4) present effects for our measure of longevity. We find that exposure to financial resources from the Duke Endowment in the county of birth during the prenatal period

reduced infant mortality by 9.4 to 10.4 percent. Additionally, it reduced the mortality rate between ages 20 to 65 by 3.4 to 4.3 percent. In line with our quasi-experimental setting, the effects are very similar whether or not we control for additional time-varying variables. Furthermore, in Figures 2 and 3, we present event studies for the two variables of interest. In each case we observe parallel trends prior to the start of Duke funding that then turn into negative mortality effects afterwards. This not only provides support for our identifying assumptions but is also consistent with findings presented in Table 1.

The findings presented in Table 1 suggest statistically significant and economically meaningful effects on mortality and longevity, but it is worth comparing our effect sizes to other reduced form effects presented in the relevant literature. [Anderson et al. \(2020\)](#), who study midwifery laws in the U.S. during roughly the same period of time as this paper, find reductions in infant mortality rates of about 2.6 percent on average which is roughly 4 times smaller than our estimates. On the other hand, [Anderson et al. \(2021b\)](#) show that water filtration in U.S. cities between 1900 and 1940 lead to 11-12 percent declines in infant mortality. However, they do not find any positive effects from other sanitation methods. More directly related to hospital access, [Anderson et al. \(2021a\)](#) do not find any mortality effects of the federal desegregation of hospitals.

Similar to [Anderson et al. \(2021a\)](#), we are also interested in understanding differential effects of Duke funding by race. We present the results for infant mortality and longevity in Table 2. Since our data for mortality by race are available for years 1920 to 1939 only, we first replicate the main result from Table 1 on this restricted sample. The results, presented in column (1) and (5), are somewhat smaller but nonetheless remain statistically significant at conventional levels. Then, in columns (2) and (6), we restrict to White and African-American mortality. Our finding for infant mortality rate remains largely unchanged, but the longevity estimate is now smaller in magnitude and no longer statistically significant at conventional levels. Columns (3) to (4) and (7) to (8) present results separately for White and African-American mortality. We find larger health benefits accruing to African Americans compared with whites, but neither longevity estimate is significant. On the other hand, for infant mortality we find a reduction of 13.4 percent for African

Americans while only 6.1 percent reduction for Whites. This implies a racial gap in mortality gains of 7.3 percentage points which contrasts with findings in [Anderson et al. \(2021a\)](#).

What explains our positive mortality and longevity effects? We propose that a major driver of improvements in health care quality due to Duke Endowment funding is the ability of the hospitals to attract a workforce of physicians and nurses in larger numbers and of higher quality. We test this proposition by studying the effects on the number of nurses and doctors practicing in counties that received Duke funding. Columns (1) and (2) in Table 5 present results for the number of nurses based on U.S. Census data while columns (3) to (12) present results for quality of doctors. We posit that younger doctors were better educated and of higher quality given the changes induced by the Flexner report - this is explored in columns (3) to (6). Alternatively we measure doctor quality based on their graduation date and graduating institution. We label doctors as high quality if they were licensed more than two years after the medical school they attended introduced a two-year degree requirement as an admission pre-requisite.

Our results suggest statistically significant increases in the number of nurses as a result of the additional funding. In particular, treated counties have between 40 and 65 more nurses per 100,000 population. Furthermore, we find increases in number of young and higher quality doctors as well as simultaneous decreases in number of old and lower quality doctors practicing in these counties in the aftermath of increased funding. These findings are consistent with our proposed mechanism of improvements in the human capital of medical staff.

Our final set of results explores the possibility that hospital care could have been more effective in the aftermath of the sulfa drugs invention. We present the results of this analysis based on Equation (1) in Table 6. We do not find any statistically significant interaction effects for either outcome of interest. At the same time, the point estimates suggest potentially large mortality gains in the aftermath of this technological change. In that, point estimate on the interaction term in column (3) is only 25 percent smaller than the effect of additional funding in pre-sulfa cohorts. Similarly, for longevity, it is almost identical to Duke exposure indicator. Although we lack statistical power, the near doubling of hospital funding effects in the aftermath of antibiotic

introduction, suggests that new medical technologies can be potentially beneficial in both the short- and the longer-run.

6 Robustness Checks

We explore the robustness of our main results to the functional form of the dependent variables, the weighting of observations and the inclusion of control variables in the regressions, as well as the set of relative time indicators fitted in the event study specifications. Given concerns about potential bias to the TWFE DiD estimates due to the staggered timing of exposure to Duke Endowment funding and the lengthy roll-out period, we then examine the sensitivity of the results to panel length and implement the [Goodman-Bacon \(2019\)](#) diagnostic to decompose the ATT estimate into its component using 2x2 DiD comparisons. Additionally, we compare our TWFE DiD estimates to estimates from a stacked regression setup ([Cengiz et al. 2019](#)) and the [Callaway and Sant’Anna \(2021\)](#) estimator.

Table [A1](#) examines the sensitivity of the main results to weighting as well as changing the functional form of the dependent variable from logs to levels of the mortality rate. For infant mortality, the estimates from unweighted regressions with log outcomes are slightly smaller than the weighted estimates in [Table 1](#), while the effects on levels of the mortality rate are comparable, regardless of weighting or the inclusion of controls. Thus we conclude that our infant mortality results are not driven by the choice of functional form for the outcome variable. On the other hand, estimates of the effects on mortality at ages 20 to 65 are substantially smaller in levels, as well as in logs without weighting, and are no longer statistically significant.

While it has been a relatively standard practice in the literature to estimate event study specifications which bin relative time periods further away from the introduction of treatment ([Anderson et al. 2020, 2021b,a](#)), others caution against this approach and suggest that practitioners fit indicators for each unique relative time period, with the exception of the period prior to the introduction of the treatment, which is traditionally excluded ([Baker et al. 2021](#)). Our choice to

estimate binned indicators for the time periods $t = -20$ to $t = -7$ and $t = 7$ to $t = 15$ is motivated by Figure A1, which shows a drop-off in the number of counties that contribute to the estimation of relative-time indicators further away from the introduction of treatment. However, this restriction may be problematic as it imposes a constant treatment effect assumption for the binned time periods. In Figure A2 we relax this specification choice and show event study estimates from specifications that include separate indicators for all unique event time periods. We suppress the display of these additional coefficients for expositional clarity. The results are substantively unchanged in comparison to the main estimates in Figures 2 and 3.

The extended roll-out period of Duke Endowment funding implies that additional concerns may follow from Goodman-Bacon (2019) and be relevant to our empirical setting. Namely, that the overall DiD estimate may be sensitive to the panel length and places greater weight on treatment groups that are treated closer to the middle of the panel (Baker et al. 2021). In figure A3 we examine the robustness of our main results to changing the panel length, which will shift a subset of counties between the ever-treated and never-treated groups. Panels (a) and (b) indicate that the effects on the natural log of the infant mortality rate and the mortality rate at ages 20 to 65 that were presented in columns 2 and 4 of Table 1 are unchanged when shortening or lengthening the number of pre-treatment periods included in the sample. Panel (c) shows that the infant mortality results are also unaffected when shortening or lengthening the treatment period by up to 10 years, to 1933 and 1952 respectively. Additionally, the stability of the estimates across these alternate specifications suggests that the overall TWFE DiD estimate is not biased by dynamic treatment effects that vary across treatment timing groups or by selection into the never-treated group. The results are also unaffected by including the World War II period in the sample. When it comes to long-run mortality, we can only show in panel (d) that the results are robust to shortening the treatment period as early as 1933, since deaths by age 65 are only observed for cohorts born up to 1942. Taken together, these specification checks strongly suggest that our main results are not driven by choice of panel length.

Yet, concerns about bias to the TWFE DiD estimate may remain due to the staggered roll-out

of the Duke Endowment funding. Thus, we conduct a [Goodman-Bacon \(2019\)](#) decomposition in [Table A2](#) and [Figure A4](#) and find that the overall TWFE DiD estimate is composed primarily of 2x2 DiD comparisons of treated vs. untreated units and that these comparisons with high weight are close to the overall average DiD estimate. Furthermore, we find evidence that the early vs. late and late vs. early treated comparisons are also negative albeit smaller in magnitude compared to the overall DiD estimate, but these comparisons receive much lower weights. These findings ease concerns about bias in the TWFE DiD results. We also obtain DiD ATT estimates and implement event study specifications using a stacked regression design ([Cengiz et al. 2019](#)) and the [Callaway and Sant’Anna \(2021\)](#) estimator. The stacked regression results use later-treated and never-treated counties as the control group and are shown in [Table A3](#) and [Figure A7](#), while the [Callaway and Sant’Anna \(2021\)](#) event study results use never-treated counties as the control group and are presented in [Figure A8](#). In the latter case, we also estimate aggregate treatment effects by relative event time of 11.0 percent for infant mortality and 5.4 percent of mortality at ages 20 to 65.⁶ Our estimates by race, for infant mortality, also hold when using specification proposed by [Callaway and Sant’Anna \(2021\)](#) as documented in [Figure A9](#). Using these alternate estimators, we find strong treatment effects that are similar to or larger in magnitude compared to the main TWFE DiD estimate, in addition to evidence of parallel pre-trends.

7 Conclusions

There is ample evidence in the literature that increased access to health insurance during prenatal period and in infancy is beneficial for children in short- as well as long-run. Much less is known, however, about potential consequences of improved access to medical facilities or specific treatments available in these facilities. We contribute to this literature by studying the effects of increased access to hospital care in the first half of the twentieth century in the U.S.

We obtain exogenous variation in access to hospitals stemming from Duke Endowment fund-

⁶We do not have enough timing group-specific variation (e.g. first treatment in 1927) to plot timing group specific event-studies as in [Callaway and Sant’Anna \(2021\)](#).

ing which provided money for building of new and expansion of existing hospitals between 1925 and 1942 in North Carolina. We document that these additional financial resources indeed lead to increases in hospital capacity as well as improvements in their staffing. At the same time, our difference-in-differences estimates imply that counties exposed to Duke funding experienced reductions in infant mortality while the surviving babies were also less likely to die between ages 20 to 65. These health gains are particularly pronounced for African-American babies compared with White babies, and the estimate for the former group is over twice the size of that for the latter. Finally, we find some suggestive evidence that gains from new pharmaceutical treatments available to hospital patients lead to further improvements in their outcomes albeit we lack statistical power to estimate these interaction effects precisely.

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8 Tables

Table 1: Two-way fixed effects DiD estimates in logs

	Ln(infant mortality)		Ln(mortality at 20 to 65)	
	(1)	(2)	(3)	(4)
=1 if Duke exposure	-0.104*** (0.031)	-0.094*** (0.030)	-0.043*** (0.016)	-0.034** (0.015)
N	2,300	2,300	2,300	2,300
Controls	No	Yes	No	Yes

Notes: Each cell represents an estimate from a separate OLS regression using a balanced panel of North Carolina counties from 1920 to 1942. The dependent variables are natural logs of deaths per 1,000 live births of individuals born in county c and year t : infant deaths in columns 1 and 2 and deaths at ages 20 to 65 in columns 3 and 4. All regressions control for county fixed effects and year fixed effects. All regressions are weighted by average the number of live births in county c . Regressions in columns 2 and 4 include controls for the percent of the population illiterate, black, other (non-white) race, and urban, as well as retail sales and manufacturing wages per capita. Standard errors are clustered at the county level.

Point estimates marked ***, **, and * are statistically significant at the 1, 5, and 10 percent levels, respectively.

Table 2: Two-way fixed effects DiD estimates by race in logs

	Ln(infant mortality)				Ln(mortality at 20 to 65)			
	(1) All	(2) Pooled	(3) White	(4) Black	(5) All	(6) Pooled	(7) White	(8) Black
=1 if Duke exposure	-0.086*** (0.026)	-0.079*** (0.027)	-0.061** (0.028)	-0.134*** (0.046)	-0.014 (0.014)	-0.014 (0.014)	-0.005 (0.020)	-0.014 (0.021)
N	2,000	2,000	2,000	2,000	2,000	2,000	2,000	2,000

Notes: Each cell represents an estimate from a separate OLS regression using a balanced panel of North Carolina counties from 1920 to 1939. The dependent variables are natural logs of deaths per 1,000 live births in county c and year t : infant deaths in columns 1 to 4 and deaths at ages 20 to 65 in columns 5 to 8. Mortality rates are reported for all races in columns 1 and 5, and pooled for blacks and whites in columns 2 and 6. Mortality rates are computed separate for whites in columns 3 and 7, and for blacks in columns 4 and 8. All regressions include county fixed effects and year fixed effects, and controls for the percent of the population illiterate, black, other (non-white) race, and urban, as well as retail sales and manufacturing wages per capita. All regressions are weighted by average the number of live births in county c . Standard errors are clustered at the county level.

Point estimates marked ***, **, and * are statistically significant at the 1, 5, and 10 percent levels, respectively.

Table 3: Two-way fixed effects DiD estimates for infant mortality by hospital type in logs

	Log infant mortality rate per 1,000 births			
	(1)	(2)	(3)	(4)
=1 if Duke funding for black-only hospital	-0.147*** (0.042)			-0.107** (0.051)
=1 if Duke funding for white-only hospital		-0.178*** (0.065)		-0.120* (0.072)
=1 if Duke funding for mixed hospital			-0.090** (0.036)	-0.062* (0.034)
N	2,300	2,300	2,300	2,300

Notes: Each column represents a separate OLS regression. The dependent variables are natural logs of infant deaths per 1,000 live births in county c and year t . All regressions include county fixed effects and year fixed effects, and controls for the percent of the population illiterate, black, other (non-white) race, and urban, as well as retail sales and manufacturing wages per capita. All regressions are weighted by average the number of live births in county c . Standard errors are clustered at the county level.

Point estimates marked ***, **, and * are statistically significant at the 1, 5, and 10 percent levels, respectively.

Table 4: Two-way fixed effects DiD estimates for infant mortality by funding type in logs

	Log infant mortality rate per 1,000 births				
	(1)	(2)	(3)	(4)	(5)
=1 if Duke funding for new hospital	-0.043 (0.047)				-0.038 (0.041)
=1 if Duke funding for addition to hospital		-0.184*** (0.048)			-0.182*** (0.053)
=1 if Duke funding for equipment			-0.055 (0.049)		-0.007 (0.045)
=1 if Duke funding for hospital purchases				-0.006 (0.084)	0.014 (0.057)
N	2,300	2,300	2,300	2,300	2,300

Notes: Each column represents a separate OLS regression. The dependent variables are natural logs of infant deaths per 1,000 live births in county c and year t . All regressions include county fixed effects and year fixed effects, and controls for the percent of the population illiterate, black, other (non-white) race, and urban, as well as retail sales and manufacturing wages per capita. All regressions are weighted by average the number of live births in county c . Standard errors are clustered at the county level.

Point estimates marked ***, **, and * are statistically significant at the 1, 5, and 10 percent levels, respectively.

Table 5: Mechanisms: Effects on numbers of doctors and nurses

	Doctors									
	Nurses		Young		Old		High quality		Low quality	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
=1 if Duke exposure	62.057*** (14.847)	40.210*** (13.777)	11.428*** (4.219)	7.451** (3.152)	-8.135*** (2.727)	-4.931** (2.084)	8.772*** (2.548)	6.953*** (2.136)	-7.609*** (1.773)	-5.575*** (1.422)
N	388	388	700	700	700	700	700	700	700	700
Controls	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes

Notes: Each column represents an estimate from a separate OLS regression. The dependent variables are the number of nurses (columns 1 and 2), young doctors (columns 3 and 4), and old doctors (columns 5 and 6), respectively, per 100,000 population. Column 1 and 2 are based on complete count population census data from IPUMS for 1910, 1920, 1930, and 1940. Individual doctors and nurses are identified from occupation and industry codes and aggregated to the county level for each census year. Columns 3 to 10 are based on the *American Medical Directory* for the years 1914, 1918, 1923, 1927, 1931, 1936, and 1940. A young doctor is defined as having graduated from medical school or received a medical license after 1910 or being born after 1885. All others are considered “old” doctors. Doctor are considered high quality if they were licensed more than two years after the medical school they attended introduced a two-year degree requirement as an admission pre-requisite. Low quality doctors were licensed earlier. All regressions control for county fixed effects and year fixed effects. Control variables are listed in Table 1. Regressions are weighted by total population in county c and year t . Standard errors are clustered at the county level.

Point estimates marked ***, **, and * are statistically significant at the 1, 5, and 10 percent levels, respectively.

Table 6: Interaction of Duke exposure with sulfa

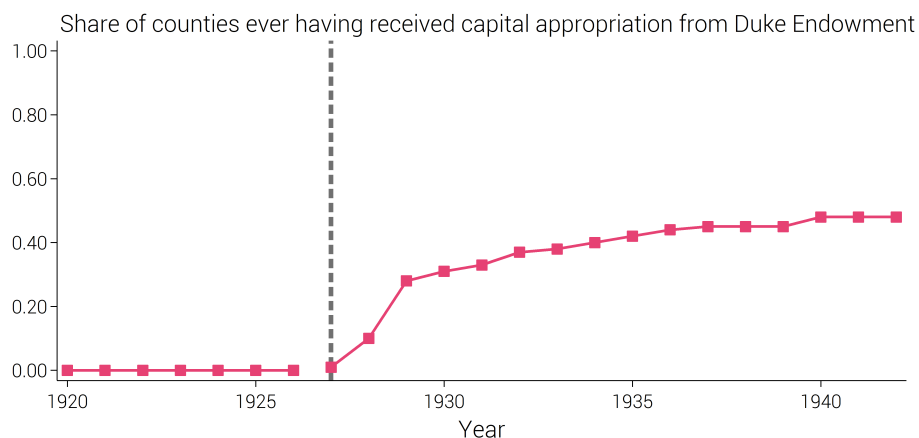
	Ln(infant mortality)		Ln(mortality at 20 to 65)	
	(1)	(2)	(3)	(4)
=1 if Duke exposure	-0.082*** (0.026)	-0.073*** (0.027)	-0.034** (0.015)	-0.025* (0.015)
Duke exposure \times sulfa	-0.058 (0.055)	-0.056 (0.050)	-0.024 (0.018)	-0.023 (0.018)
N	2,300	2,300	2,300	2,300
Controls	No	Yes	No	Yes

Notes: Each cell represents an estimate from a separate OLS regression. The dependent variables are natural logs of deaths per 1,000 live births of individuals born in county c and year t : infant deaths in columns 1 and 2 and deaths at ages 20 to 65 in columns 3 and 4. Sulfa exposure is measured by an indicator that equals to one for all years from 1937 onward. All regressions control for county fixed effects and year fixed effects. All regressions are weighted by average the number of live births in county c . Regressions in columns 2 and 4 include controls for the percent of the population illiterate, black, other (non-white) race, and urban, as well as retail sales and manufacturing wages per capita. Standard errors are clustered at the county level.

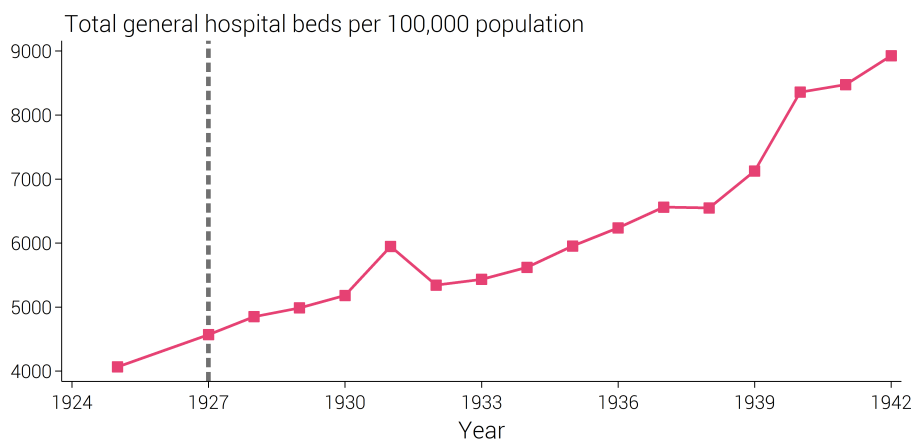
Point estimates marked ***, **, and * are statistically significant at the 1, 5, and 10 percent levels, respectively.

9 Figures

Figure 1: Hospital access in North Carolina, 1927-1942



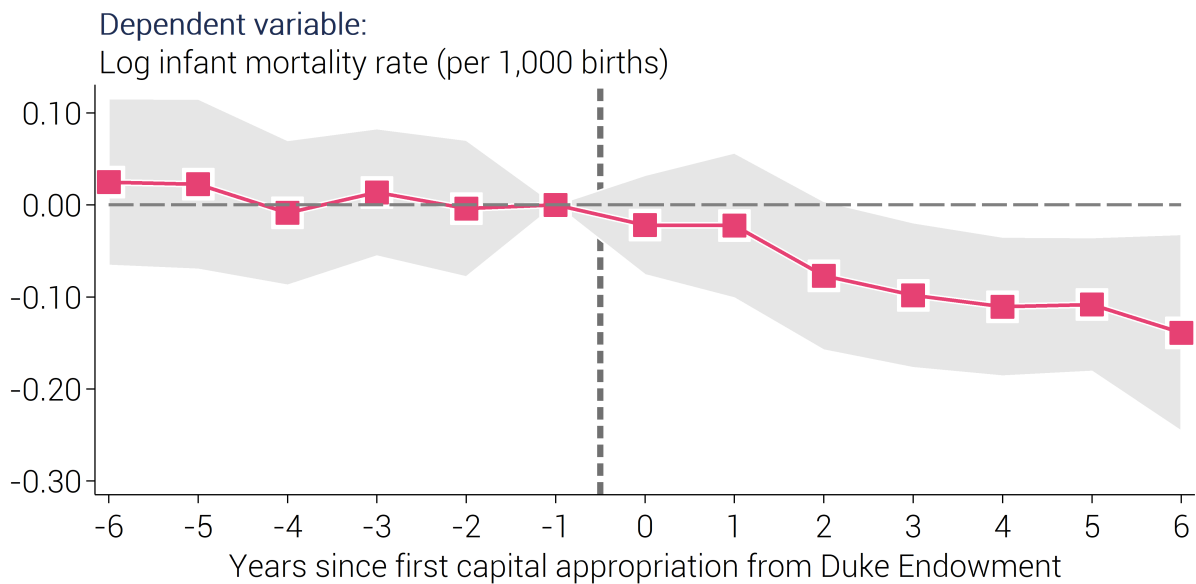
(a) DiD roll-out design



(b) Hospital beds

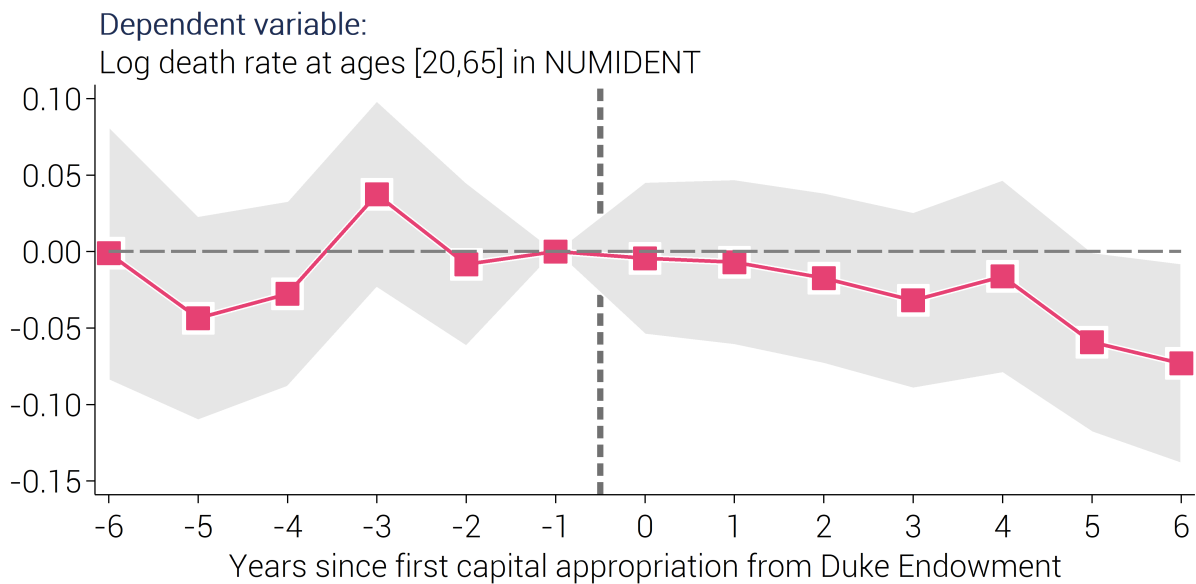
Notes: Figure 1a plots the fraction of ever-treated counties in North Carolina by calendar year. Treatment is measured by appropriations for capital expenditures from the Duke Endowment. Treatment timing is based on the *Annual Report of the Hospital Section* for the years 1927 to 1942, published by the Duke Endowment. Figure 1b plots the total number of beds in general hospitals per 100,000 population from 1927 to 1942. Data are not available prior to 1927. Hospital bed counts are based on the annual reports of the *American Medical Association* and annual population figures are linearly interpolated from decennial census data (Manson et al. 2019). The vertical dashed lines denote the first year of Duke Endowment treatment in 1927.

Figure 2: Event study estimates for infant mortality



Notes: Figure 2 plots OLS estimates of coefficient values and 95% confidence intervals for the lead and lag indicator variables for time periods from $t = -20$ to $t = 15$ around the first year that a county received an appropriation for capital expenditures from the Duke Endowment. The plot omits binned indicators for the time periods $t = -20$ to $t = -7$ and $t = 7$ to $t = 15$, respectively. The omitted category is 1 year before initial treatment. An observational unit in the regression is a county-by-year birth cohort. The dependent variable is the natural log of the number of infant (age 0 to 1) deaths per 1,000 live births in birth county c and year t . Control variables include county fixed effects and year fixed effects. Regressions are weighted by birth cohort size in county c and year t . Standard errors are clustered by county of birth.

Figure 3: Event study estimates for long-run mortality



Notes: Figure 3 plots OLS estimates of coefficient values and 95% confidence intervals for the lead and lag indicator variables for time periods from $t = -20$ to $t = 15$ around the first year that a county received an appropriation for capital expenditures from the Duke Endowment. The plot omits binned indicators for the time periods $t = -20$ to $t = -7$ and $t = 7$ to $t = 15$, respectively. The omitted category is 1 year before initial treatment. An observational unit in the regression is a county-by-year birth cohort. The dependent variable is the natural log of the number of deaths at ages 20 to 65 per 1,000 live births in birth county c and year t . Control variables include county fixed effects and year fixed effects. Regressions are weighted by birth cohort size in county c and year t . Standard errors are clustered by county of birth.

A Appendix Tables

Table A1: Two-way fixed effects DiD estimates in logs and levels

	Logs		Levels			
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Effects on infant mortality						
=1 if Duke exposure	-0.092*** (0.031)	-0.088*** (0.031)	-6.570*** (2.368)	-5.094** (2.409)	-6.475*** (2.250)	-5.574** (2.272)
Panel B: Effects on mortality at ages 20 to 65.						
=1 if Duke exposure	-0.025 (0.018)	-0.019 (0.018)	-2.269 (1.451)	-2.150 (1.451)	-1.854 (1.518)	-1.937 (1.426)
N	2,300	2,300	2,300	2,300	2,300	2,300
Controls	No	Yes	No	Yes	No	Yes
Weights	No	No	Yes	No	Yes	No

Notes: Each cell represents an estimate from a separate OLS regression. The dependent variables are the natural log of the number of deaths (columns 1 and 2) and the number of deaths (columns 3 to 6) per 1,000 live births of individuals born in county c and year t . Mortality is measured by infant deaths in panel A and deaths at ages 20 to 65 in panel B. All regressions control for county fixed effects and year fixed effects. Regressions in columns 3 and 4 are weighted by the number of live births in county c and year t . Regressions in columns 2, 4, and 6 include controls for the percent of the population illiterate, black, other (non-white) race, and urban, as well as retail sales and manufacturing wages per capita. Standard errors are clustered at the county level.

Point estimates marked ***, **, and * are statistically significant at the 1, 5, and 10 percent levels, respectively.

Table A2: [Goodman-Bacon \(2019\)](#) decomposition diagnostic

Type	Average Estimate	Number of 2x2 Comparisons	Total Weight
Earlier Treated vs. Later Treated Controls	-0.044	66	0.099
Later Treated vs. Earlier Treated Controls	-0.044	66	0.077
Treated vs. Untreated Controls	-0.102	12	0.824
DD coefficient	-0.092	144	1.000

Notes: The table decomposes the TWFE DiD ATT estimate from panel A and column 1 of Table A1 into the average estimate and total weight contributed by earlier vs. later treated comparisons, later vs. earlier treated comparisons, and treated vs. untreated comparisons, as well as the number of unique 2x2 comparisons found in each category.

Table A3: Stacked regression DiD estimates in logs

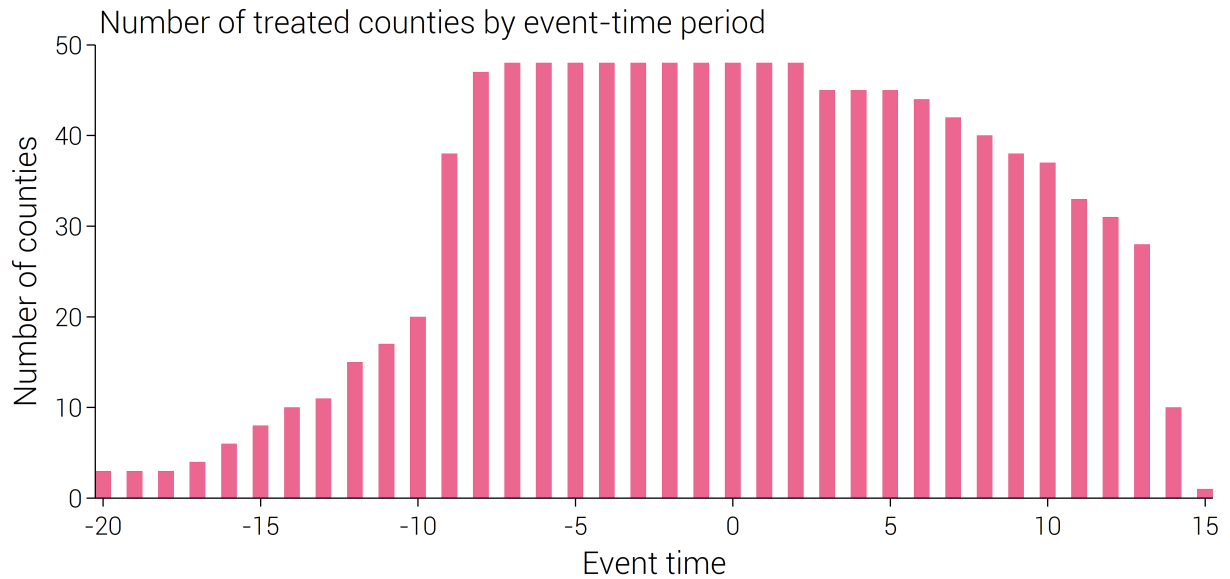
	Ln(infant mortality)		Ln(mortality at 20 to 65)	
	(1)	(2)	(3)	(4)
=1 if Duke exposure	-0.084*** (0.027)	-0.086*** (0.030)	-0.036*** (0.014)	-0.036** (0.015)
N	9,152	9,152	7,813	7,813
Controls	No	Yes	No	Yes

Notes: Each cell represents an estimate from a separate OLS regression. The dependent variables are natural logs of deaths per 1,000 live births of individuals born in county c and year t : infant deaths in columns 1 and 2 and deaths at ages 20 to 65 in columns 3 and 4. All regressions control for county fixed effects and year fixed effects. All regressions are weighted by average the number of live births in county c . Regressions in columns 2 and 4 include controls for the percent of the population illiterate, black, other (non-white) race, and urban, as well as retail sales and manufacturing wages per capita. Standard errors are clustered at the county level.

Point estimates marked ***, **, and * are statistically significant at the 1, 5, and 10 percent levels, respectively.

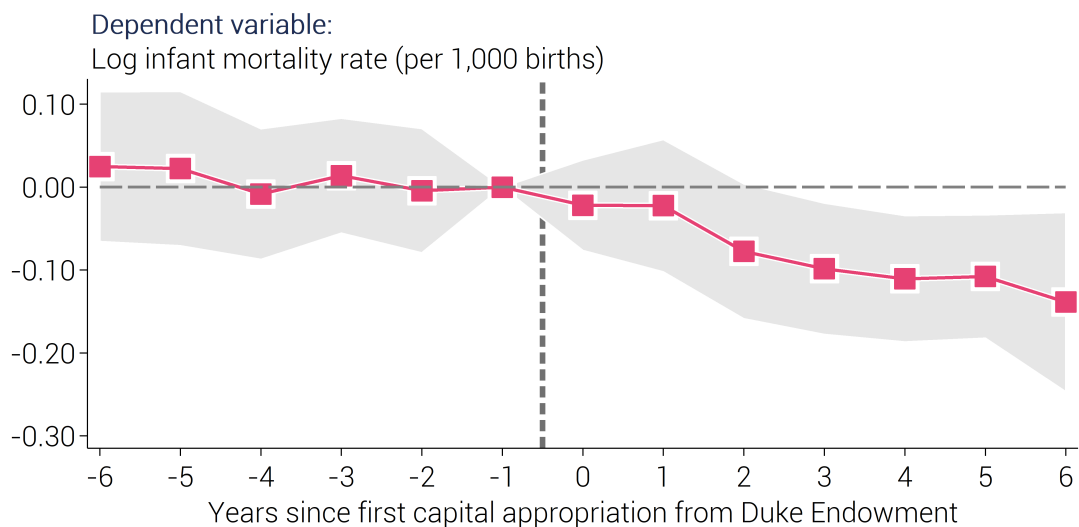
B Appendix Figures

Figure A1: Number of treated counties by event-time period

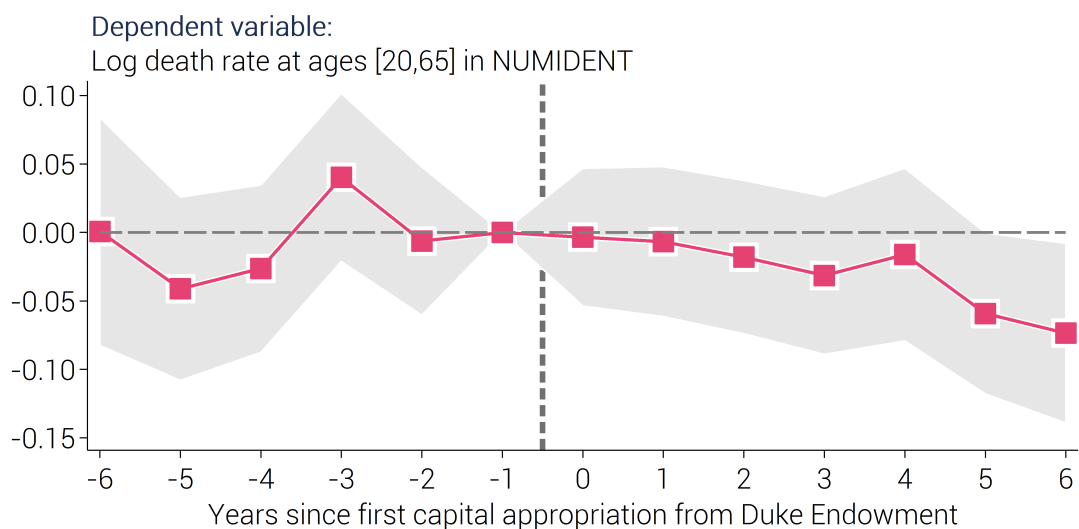


Notes: Figure A1 plots the number of treated North Carolina counties in each event-time period in the birth county-by-year panel used in the regressions reported in Table 1 and the event studies plotted in Figures 2 and 3.

Figure A2: Event study without binned endpoints



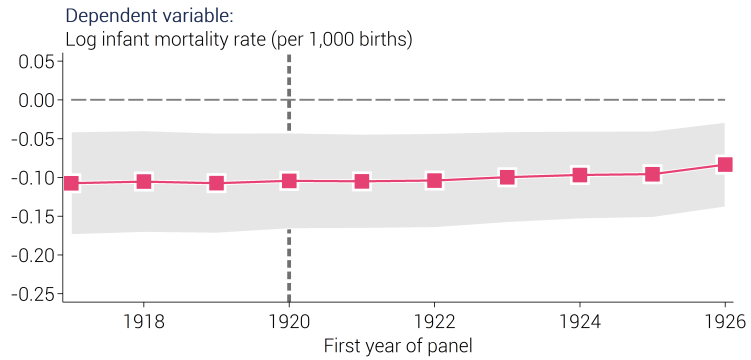
(a) Log infant mortality rate



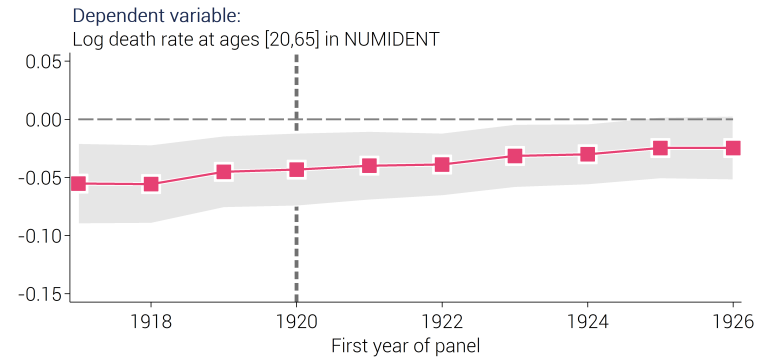
(b) Log mortality rate age 20 to 65

Notes: Figure A2 modifies the event study specifications in Figures 2 and 3 to estimate separate coefficients for each event study period for the time periods $t = -20$ to $t = -7$ and $t = 7$ to $t = 15$ instead of replacing them with binned indicators. Figure A2 plots the coefficients for time periods $t = -6$ to $t = 6$ to correspond to the unbinned coefficients shown in Figures 2 and 3.

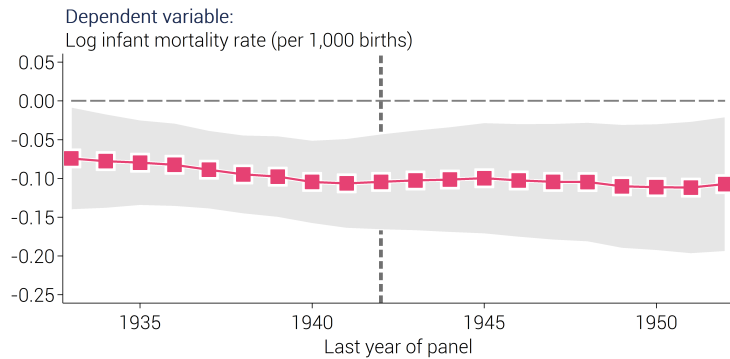
Figure A3: Robustness of TWFE DiD estimates to changing panel length



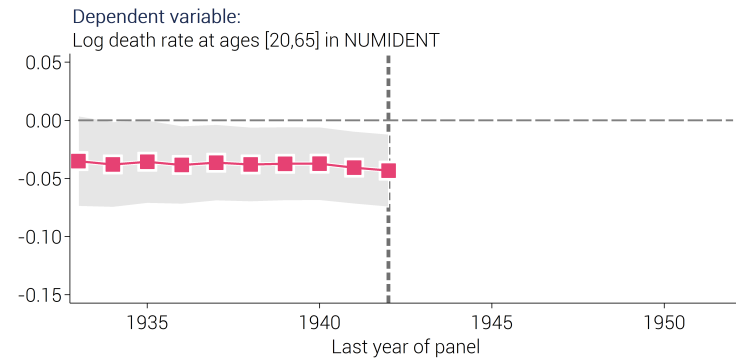
(a) Changing first year of panel (log IMR)



(b) Changing first year of panel (log mortality age 20-65)



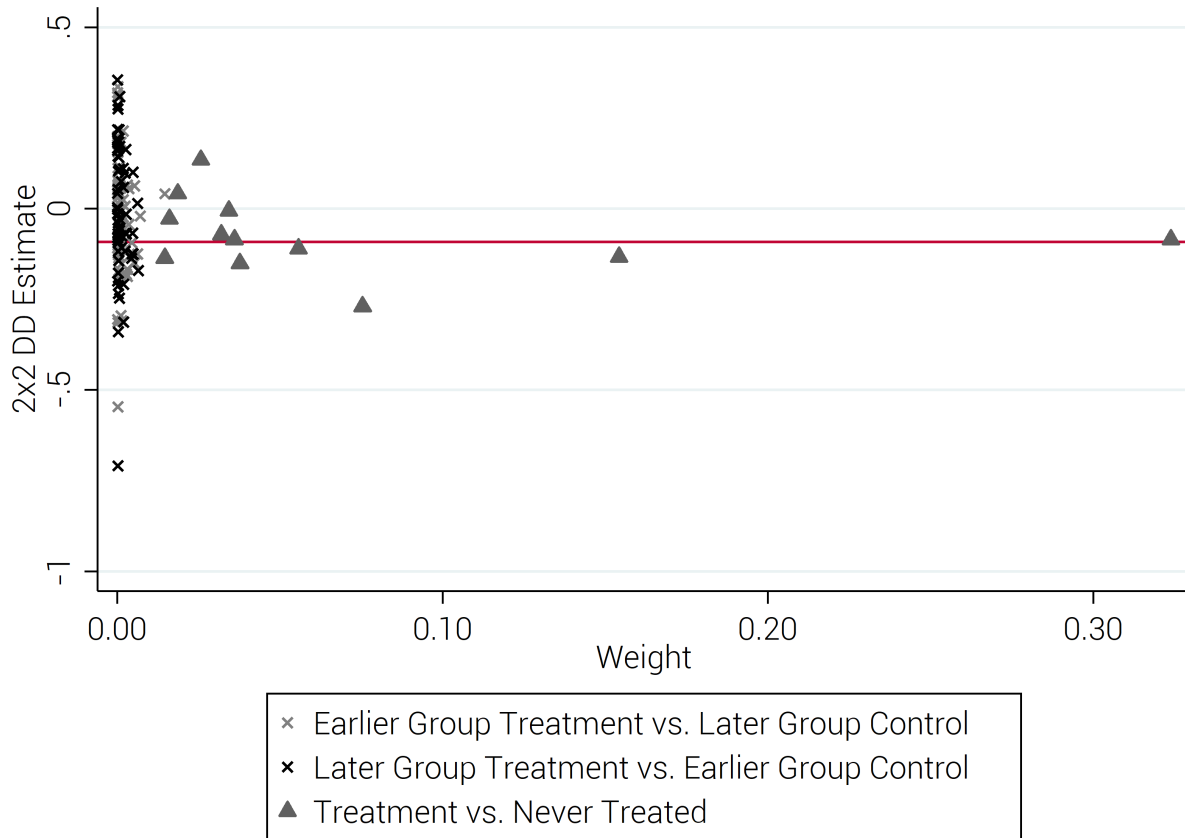
(c) Changing last year of panel (log IMR)



(d) Changing last year of panel (log mortality age 20-65)

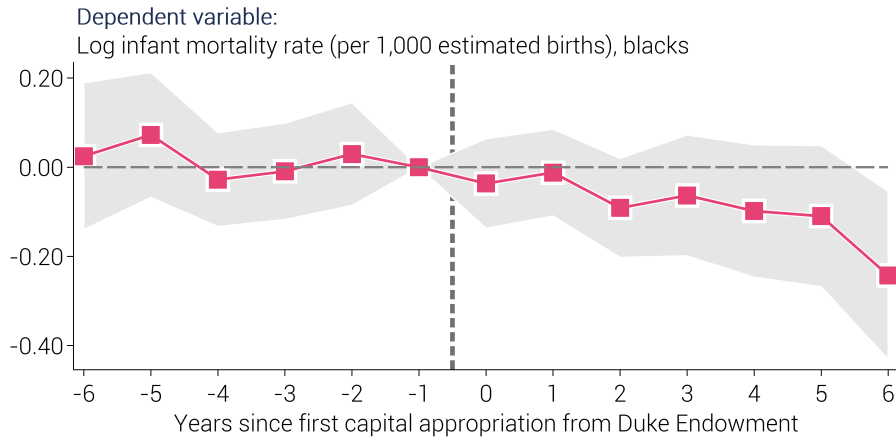
Notes: Figure A3 plots estimates of the TWFE DiD coefficient for exposure to Duke Endowment funding from separate regressions while varying the first (figures A3a and A3b) or the last year (figures A3c and A3d) of the panel. In figures A3a and A3b the last year of the panel is kept unchanged at 1942, while in figures A3c and A3d the first year of the panel is kept unchanged at 1920 to correspond to the end points of the panel in the main sample. Aside from these changes to panel length, the specifications correspond to columns 2 and 4 of Table 1. In each sub-figure, the vertical dashed denotes the first panel year (1920) or the last panel year (1942) from the main sample. Figure A3d only shows estimates up to 1942 since mortality by age 65 is not observed for later birth cohorts.

Figure A4: Goodman-Bacon (2019) decomposition diagnostic



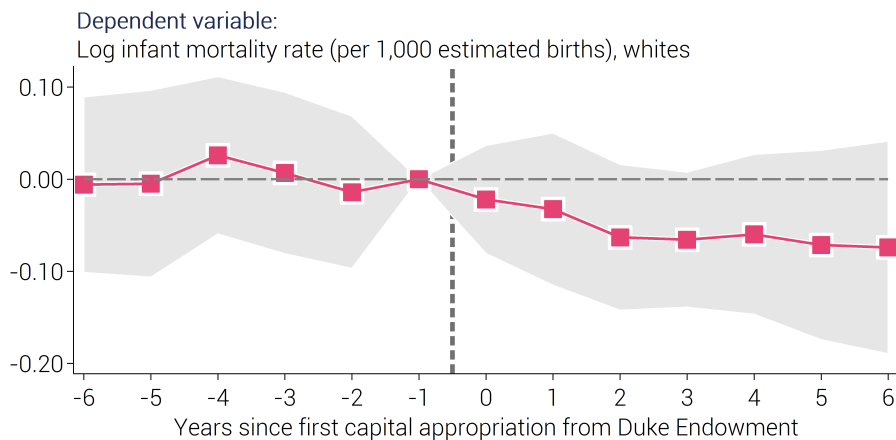
Notes: Figure A4 decomposes the TWFE DiD estimate from column 1 and panel A of Table A1 into separate 2x2 DiD components. The specification does not include controls, except for county and year fixed effects, it not weighted, and has the log of the infant mortality rate as the dependent variable. The figure depicts the distribution of all unique treatment timing comparisons used to identify $\widehat{\delta}^{DD}$. For example, one symbol may represent a comparison between counties treated in 1935 and counties treated in 1937. The horizontal red line displays the overall TWFE DiD estimate.

Figure A5: Event study estimates for black infant mortality



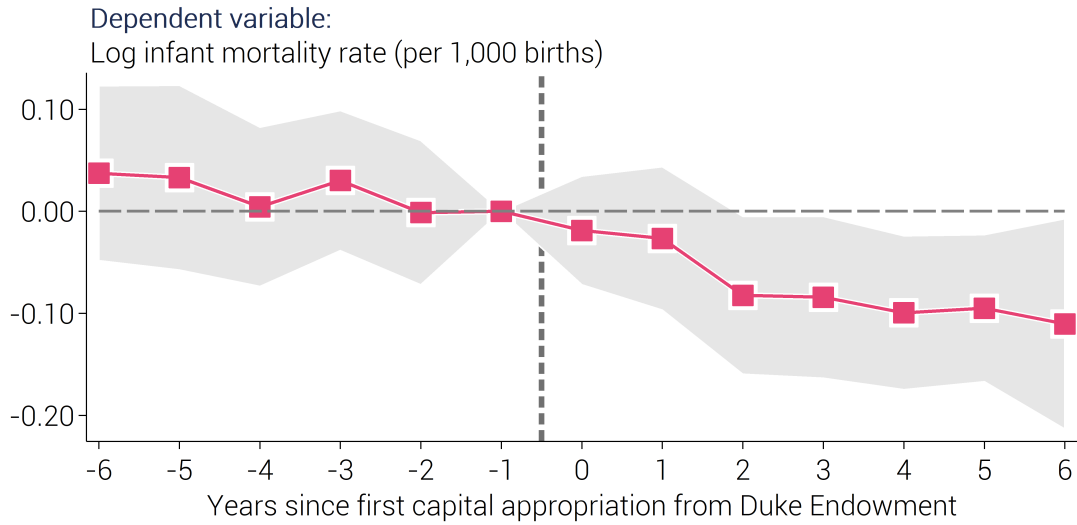
Notes: Figure A5 plots OLS estimates of coefficient values and 95% confidence intervals for the lead and lag indicator variables for time periods from $t = -20$ to $t = 15$ around the first year that a county received an appropriation for capital expenditures from the Duke Endowment. The plot omits binned indicators for the time periods $t = -17$ to $t = -7$ and $t = 7$ to $t = 12$, respectively. The omitted category is 1 year before initial treatment. An observational unit in the regression is a county-by-year birth cohort. The dependent variable is the natural log of the number of infant (age 0 to 1) deaths per 1,000 live births in birth county c and year t . Control variables include county fixed effects and year fixed effects. Regressions are weighted by birth cohort size in county c and year t . Standard errors are clustered by county of birth.

Figure A6: Event study estimates for white infant mortality

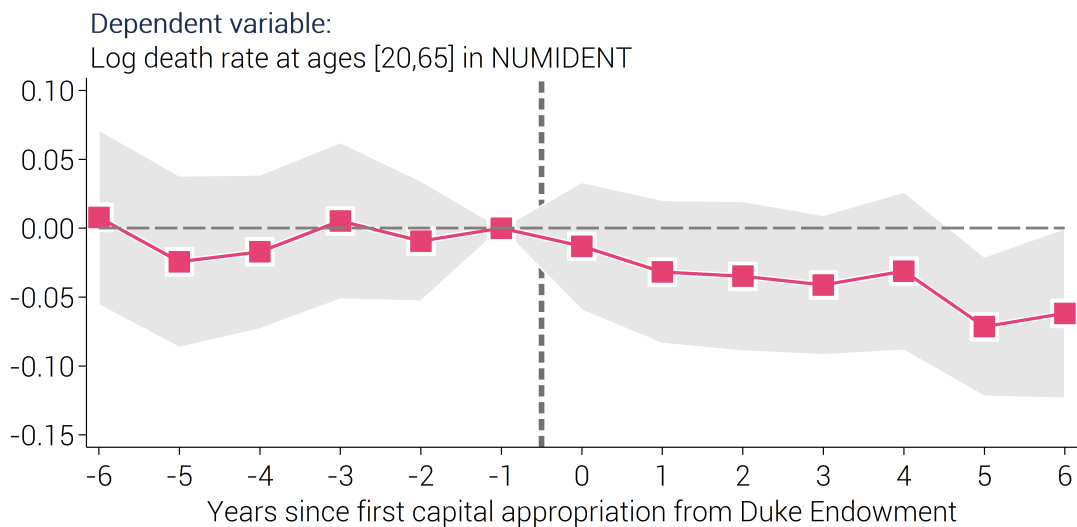


Notes: Figure A6 plots event study estimates for white infant mortality that are otherwise identical in specification to the estimates in Figure A5.

Figure A7: Stacked regression event study



(a) Log infant mortality rate

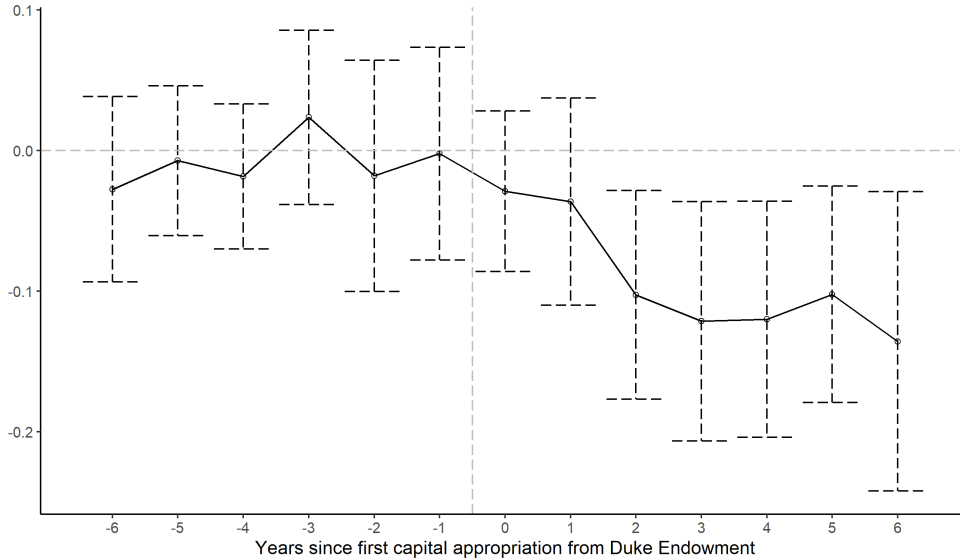


(b) Log mortality rate age 20 to 65

Notes: Figure A7 plots stacked regression results in which the control group for a treatment-timing group (e.g. counties treated in 1927) consist of counties that were not-yet-treated or never-treated in the window from $t - 6$ to $t + 6$.

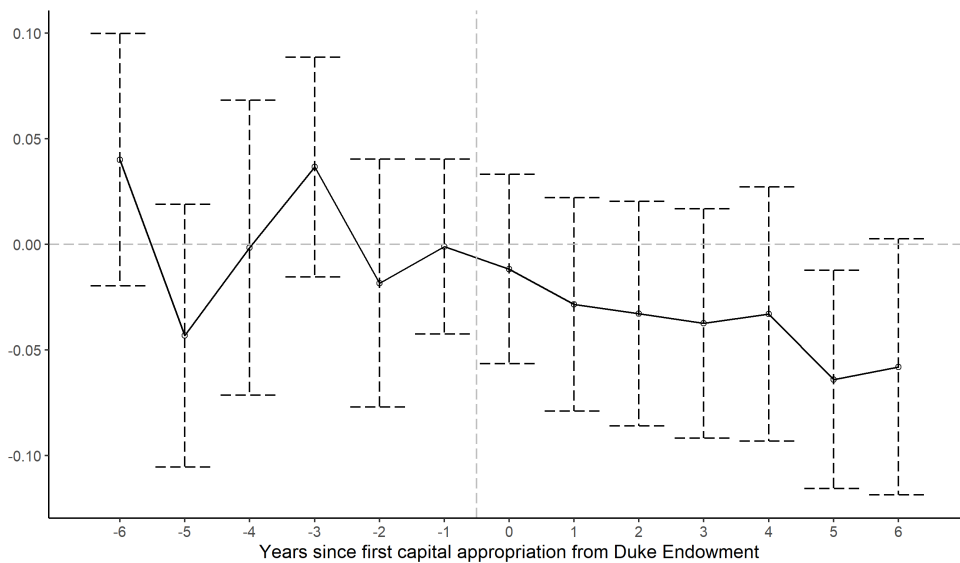
Figure A8: Callaway and Sant'Anna (2021) event study estimates

Dependent variable: Log infant mortality rate (per 1,000 births)
 Treatment variable: First capital appropriation - any purpose
 Overall ATT = -0.114
 Overall SE = 0.037



(a) Log infant mortality rate

Dependent variable: Log death rate at ages [20,65] in NUMIDENT
 Treatment variable: First capital appropriation - any purpose
 Overall ATT = -0.053
 Overall SE = 0.025

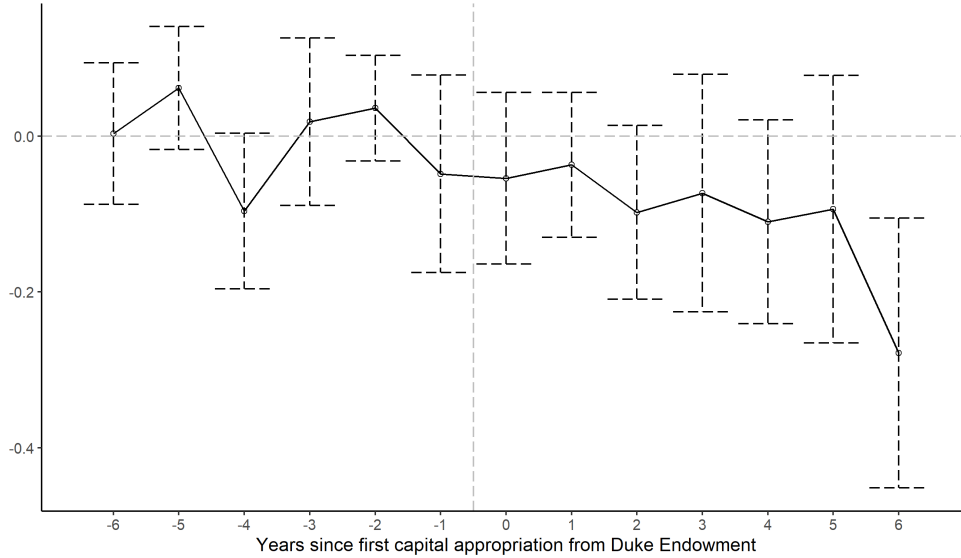


(b) Log mortality rate age 20 to 65

Notes: Figure A8 plots event study results from the Callaway and Sant'Anna (2021) estimator using never-treated units as controls. Estimates for time periods $t = -20$ to $t = -7$ and $t = 7$ to $t = 15$ are not shown.

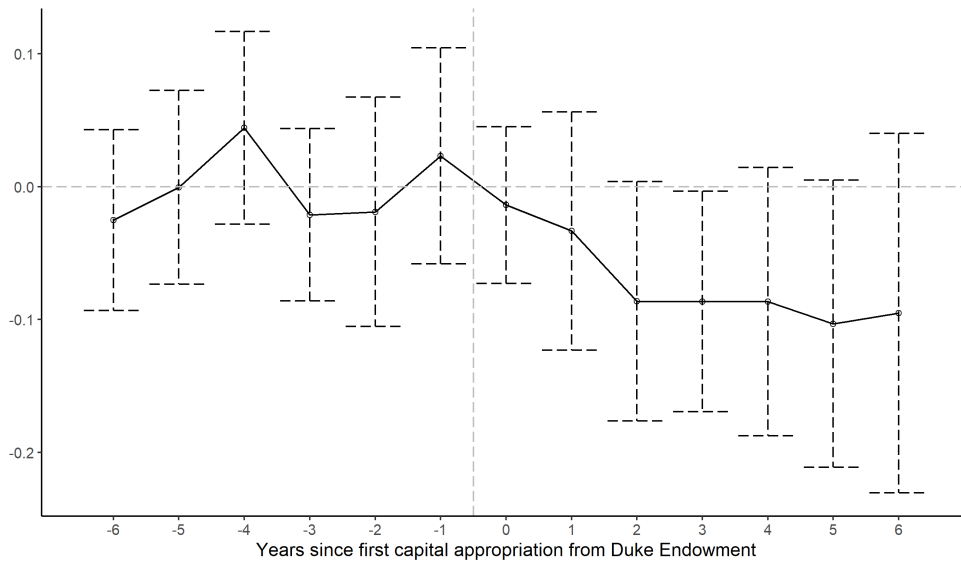
Figure A9: Callaway and Sant'Anna (2021) event study estimates by race

Dependent variable: Log infant mortality rate (per 1,000 estimated births), blacks
 Treatment variable: First capital appropriation - any purpose
 Overall ATT = -0.129
 Overall SE = 0.05



(a) Log infant mortality rate, blacks

Dependent variable: Log infant mortality rate (per 1,000 estimated births), whites
 Treatment variable: First capital appropriation - any purpose
 Overall ATT = -0.071
 Overall SE = 0.039



(b) Log infant mortality rate, whites

Notes: Figure A9 plots event study results by race from the Callaway and Sant'Anna (2021) estimator using never-treated units as controls. Estimates for time periods $t = -17$ to $t = -7$ and $t = 7$ to $t = 12$ are not shown.