Mass Vaccination and Educational Attainment*

EVIDENCE FROM THE 1967–68 MEASLES ERADICATION CAMPAIGN

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October 28, 2022

We show that the first nationwide mass vaccination campaign against measles increased educational attainment in the United States. Our empirical strategy exploits the variation in exposure to the childhood disease across states right before the Measles Eradication Campaign of 1967–68. The campaign reduced reported measles incidence by 90 percent within two years. Our results suggest that mass vaccination against measles increased the years of education on average by about 0.1 years for males in the affected cohorts. Their college graduation rate increased by approximately two percentage points.

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*Contact: maarit.olkkola@aalto.fi. We are very grateful for comments from Matti Sarvimäki, Miri Stryjan, Aino Kalmbach, Claudia Troccoli, Juuso Mäkinen, Maurice Forget, Kristiina Huttunen, Otto Toivanen, and Libertad González, as well as for valuable feedback from seminar and conference participants at the Helsinki Graduate School of Economics, the Norwegian School of Economics, the World Economic History Congress 2018, and the Essen Health Meeting 2017. We are also grateful for invaluable help from Guy Hall at the National Archives at Atlanta. Maarit Olkkola gratefully acknowledges funding from the Yrjö Jahnsson Foundation, the Finnish Cultural Foundation, the OP Group Research Foundation, the Alfred Kordelin Foundation, the Otto A. Malm Foundation, and the HSE Support Foundation. Sonja Dobkowitz gratefully acknowledges financial support by the Deutsche Forschungsgemeinschaft (German Research Foundation) via the RTG-2281 "The macroeconomics of inequality". This paper supersedes a working paper circulated as "Vaccine-preventable Childhood Disease and Adult Human Capital: Evidence from the 1967 Measles Eradication Campaign in the United States".

1 Introduction

Infectious disease epidemics disrupt communities temporarily but may also lead to long-term changes in children's human capital accumulation (Almond 2006; Beach et al. 2022; Meyers and Thomasson 2021). Measles is the world's leading childhood cause of vaccine-preventable illness and was until the 1960s one of the most common sources of epidemics in the United States. Before the measles vaccine, measles infected 80 to 95 percent of American children by age ten and resulted in 48,000 hospitalizations, 1,600 cases of severe brain damage, and 400 child deaths annually (Centers for Disease Control and Prevention 2020; National Communicable Disease Center 1966b,a, 1967a). These most severe health costs of measles are well documented, but less evidence exists about the long-term effects of measles control for educational outcomes.

This paper shows that the first mass vaccination campaign against measles in 1967–1968 resulted in a modest but statistically significant increase in the educational attainment of men in the affected cohorts. To identify these effects, we take advantage of *the Measles Eradication Campaign* launched in 1967 that reduced reported measles cases by 90 percent within two years. The immunization campaign intended to increase the public provision of the measles vaccine and to achieve universal measles immunization coverage (Sencer et al. 1967). The vaccine was more expensive than immunizations available for other diseases (Colgrove 2007), and surveys conducted before the campaign revealed that vaccination rates were mainly increasing in wealthy suburban neighbourhoods (National Communicable Disease Center 1966b; Cliff et al. 1993; Pendergrast 2010). Consequently, the mass vaccination campaign became part of President Johnson's War on Poverty and the first nationwide federally subsidized immunization campaign, after the federal government created formal immunization assistance in 1962 (Johnson et al. 2000; Hinman et al. 2011).

Publicly provided preventive health inputs may affect human capital accumulation in several ways (Bärnighausen et al. 2014; Lleras-Muney and Cutler 2014; Almond et al.

2018; Dupas 2014; Baird et al. 2016). Among them, at least two mechanisms suggest a potential causal link between mass vaccination against measles and educational attainment. First, due to immunization, some children never contracted measles or its complications. They also did not suffer from measles-induced immune system suppression (Mina et al. 2015, 2019) and may have, therefore, been less ill during their childhood. Healthier children, in turn, may have been less absent from school or learned better while at school (Miguel and Kremer 2004). Second, after mass vaccination reduced measles outbreaks, primary school children regardless of immunization or disease status were less exposed to measles control efforts, such as quarantines.

To identify the educational impacts of mass vaccination against measles, we use a difference-in-differences strategy that employs variation by cohort and geographic area. First, we take advantage of the sudden reduction in measles cases due to the Measles Eradication Campaign of 1967–68. The reduced measles exposure was more likely to benefit younger cohorts because they were less likely to have contracted measles by the time of the immunization campaign. Measles can only be acquired once, and either a measles infection or immunization after age one confers immunity. According to survey evidence, vaccination rates increased for the cohorts targeted by the campaign in line with the regular age profile of measles infections. Thus, different proportions in each cohort of children were vaccinated or infected with measles.

Second, we exploit variation in treatment intensity by geographic location. We compare the evolution of educational attainment for children born in states that experienced higher reported measles incidence right before the campaign to those cohorts born in states that experienced lower measles incidence. Reported measles incidence declined more in the high baseline measles incidence states. Therefore, measles exposure changed more for cohorts of children born in the states that experienced a higher measles burden prior to the campaign. Thus, younger cohorts were likely to benefit more from the campaign in the states with high measles exposure, compared to older cohorts.

Before the cohorts affected by the campaign, the average educational attainment was lower in the states with a higher measles burden but developing similarly to the states with less measles exposure. Subsequently, the average educational attainment improved for the cohorts affected by the campaign and, notably, the gap in educational attainment narrowed between high and low measles exposure areas.

Our main estimates suggest that mass vaccination against measles increased the educational attainment of men. Moving from the 5th percentile (baseline measles incidence 45 reported cases per 100,000) to the 95th percentile of our treatment intensity variable (baseline measles incidence 2,327 reported cases per 100,000), years of education increased on average by 0.12 for the male cohorts targeted by the vaccination campaign or born after the large decline in measles incidence. Our estimate can be interpreted as the effect of reducing measles incidence from a state with a high measles burden right before the campaign to close to zero, which is in line with the rapid reduction of measles incidence for these cohorts. For women, our point estimates are smaller and most of them are not statistically significant. For men, the college graduation rate also increased by approximately two percentage points on average. Our results are robust to a different definition of our treatment intensity measure, to controlling for several baseline characteristics of states, and to a placebo analysis with a different disease.

Our paper is the first to identify impacts of reduced childhood measles exposure on educational attainment in adulthood. The most closely connected literature explores the educational impacts of immunization policies (for tuberculosis Bütikofer and Salvanes (2020), for polio Serratos-Sotelo et al. (2019), for tetanus Canning et al. (2011), for vaccines against multiple diseases Alsan (2017) and Lee Luca (2016)). Compared to this research, the contribution of our analysis is that we are able to isolate the effect of a mass vaccination campaign against one of the most common childhood diseases on completed education in adulthood. Measles continues to pose a major health risk to millions of children in many low-income countries but has also resurged in places that had previously eliminated endemic infection, such as the United States and many European countries (World Health Organization 2019).[1] Compared to prior research on measles and educational outcomes (Anekwe et al. 2015; Nandi et al. 2019), we are able to take advantage of a policy-driven reduction in measles exposure, a large representative sample on a national level, and follow-up until adulthood. In terms of institutional context, the paper closest to our analysis is Atwood (2022), who examines the introduction of the measles vaccine in the United States and finds that it increased earnings and employment in adulthood for the cohorts born after the licensing of the vaccine. Instead, we focus specifically on the measles eradication campaign, a federal policy initiative to achieve widespread coverage with the vaccine. Reported measles cases first reached unprecedentedly low levels in 1967, the first year of the campaign. Prior to this, the vaccine had failed to reach most poor families. Thus, our analysis is consistent with the hypothesis that the public provision of the measles vaccine was a significant element in reaching the educational benefits we estimate.

Our analysis also contributes to the literature that uses historical disease eradication campaigns and medical innovations as quasi-experimental variation to estimate the causal impacts of reduced disease exposure on human capital (Acemoglu and Johnson 2007; Bleakley 2007, 2010; Cutler et al. 2010; Bhalotra and Venkataramani 2015; Beach et al. 2016; Lazuka 2020; Battaglia and Kisat 2021). Our empirical set-up takes advantage of a federal policy response to a medical advance that leads to a sudden and sizeable reduction in exposure to measles soon after the licensing of the measles vaccine. A strength of our analysis is that we are able to employ a direct measure of reported incidence rather than mortality rates used as a proxy for disease incidence in most previous studies. Furthermore, the age profile specific to measles allows us to specify the potential beneficiaries of the eradication campaign in a precise manner.

Additionally, we contribute to the literature on the long-term economic effects of infec-

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[1] 2019 witnessed the highest number of measles cases and deaths globally in 23 years and a record number of reported measles cases for a decade both in the United States (1,282) and in Europe (104,248) (Centers for Disease Control and Prevention 2021; World Health Organization Regional Office for Europe 2020). The World Health Organization and the Centers of Disease Control warn of a measles crisis following the COVID-19 pandemic because 26 countries have paused their vaccination programs, leaving more than 94 million children at risk (World Health Organization 2020).

tious disease (Almond 2006; Parman 2015; Beach et al. 2022; Meyers and Thomasson 2021; Beach et al. 2022; Ager et al. 2022) and childhood health shocks.[2] Most of this literature has focused on in utero or infant exposure. In contrast, we contribute to the scarce literature on health shocks of school-age children. Our analysis highlights that disease exposure in the first grades of school may also affect longer-term educational attainment. Finally, we also connect to the literature on public policy and preventive health inputs (Kremer and Glennerster 2011; Dupas 2014; Baird et al. 2016; Banerjee et al. 2021).

2 Measles in the United States

Measles and education in the 1960s

Before the measles vaccine was licensed in 1963, virtually everyone in the United States contracted measles in childhood. More than half of American children acquired the virus by age six and more than 80 percent by age ten (Figure 1a).[3] As a disease, measles is clinically straightforward to diagnose and not often confused with other diseases (Cliff et al. 1993; National Communicable Disease Center 1966b). The first sign of infection is usually a high fever, which lasts for four to seven days. Other symptoms include a runny nose, a cough, red and watery eyes, and a characteristic rash.

Before mass vaccination, measles may have affected educational attainment through at least two potential mechanisms. First, children who contracted measles were more susceptible to serious complications directly after infection and to other infections later in childhood, which may have affected their human capital acquisition. Most patients with measles recover within three weeks, but complications occur in a sixth to a third of reported cases (National Communicable Disease Center 1966a; Centers for Disease Control and Prevention 2015). Common complications include diarrhea, ear infection,

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[2] Reviewed by Currie (2009); Almond and Currie (2011); Currie and Vogl (2013); Almond et al. (2018)).

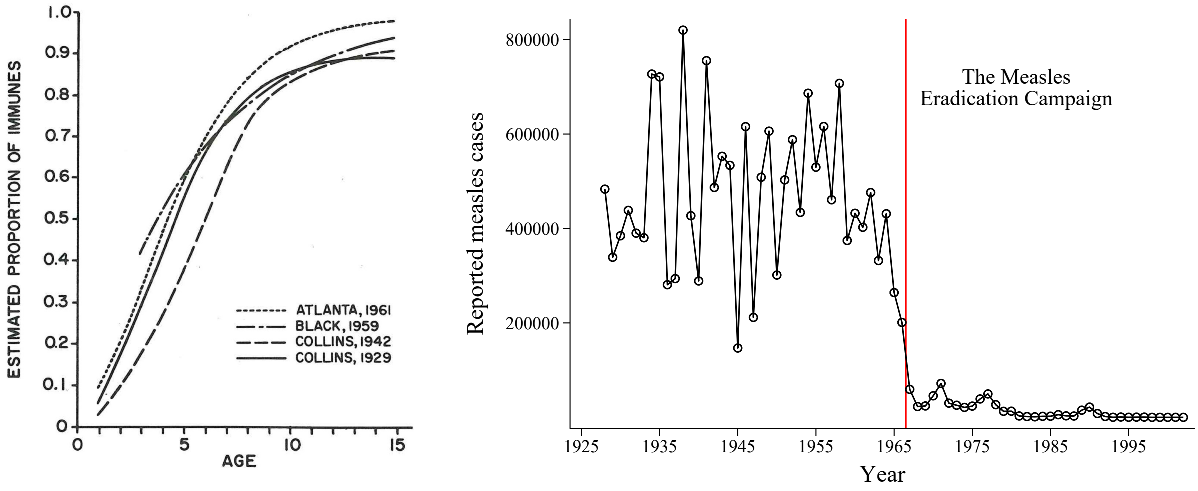
[3] See also Centers for Disease Control and Prevention (2015), National Communicable Disease Center (1967a).

and pneumonia. Severe complications may cause deafness or blindness, and about one in 500 to 1000 cases results in permanent brain damage from inflammation of the brain (World Health Organization 2019; Cliff et al. 1993; National Communicable Disease Center 1967a). No specific treatment for the measles virus exists, and after a measles infection, one is immune to the virus for the rest of one's life. However, the measles virus may suppress the immune system for up to three years and, therefore, increase the likelihood of contracting other infections (Mina et al. 2015, 2019).

Second, measles spread most effectively in schools and these outbreaks led to school absences. Children in the first few grades of school had the highest infection rates (National Communicable Disease Center 1966a), and outbreaks were estimated to have resulted in six million missed school days annually (National Communicable Disease Center 1967c). These absences were due to both illness and quarantine; thus, the outbreaks affected both those who became ill and those who were saved from infection. The number of lost school days due to measles is also likely to be an underestimate because children may also miss school due to measles-induced immune system suppression.

The first grades of school were central to the spread of the virus because school entry was often the first time that large numbers of children congregated indoors. Children transmit the virus both through direct contact and through the air, infecting between 3.7 and up to 203 susceptibles (Guerra et al. 2017). Therefore, measles spreads fast in an unimmunized population but outbreaks also die away quickly when all susceptible hosts are exhausted. For a new outbreak to emerge, an adequate number of susceptibles has to gather, usually resulting in a cycle of larger epidemics about every two to three years (Langmuir et al. 1962; Fine and Clarkson 1982). In the early 1960s, most of the new susceptibles were children below school-age, who would catch the virus upon school entry. Some younger children, however, would catch the virus earlier from their school-age siblings (National Communicable Disease Center 1966b, 1967a).

Figure 1: Measles as a prevalent childhood health problem before the vaccine.



(a) The age profile of measles infections before the vaccine.

(b) The reported annual cases of measles in the United States.

Note: Figure 1a, first published in Immunization Against Disease 1966–1967 (National Communicable Disease Center 1967a), presents the age profiles of measles infection as estimated in four surveys conducted before the vaccine (Collins 1929; Collins et al. 1942; Black 1959; Epidemic Intelligence Service 1961). Figure 1b presents the reported cases of measles in the United States with data from Project Tycho (van Panhuis et al. 2013).

The Measles Eradication Campaign of 1967–68

After the licensing of the measles vaccine in 1963, public health authorities noticed that immunization coverage was not increasing as rapidly as they had anticipated. The reported annual cases were between about 200,000 and 500,000 until 1966 (Figure 1b).[4] Surveys also revealed that take-up of the measles vaccine was not proportional across population groups but concentrated among wealthy suburban families (National Communicable Disease Center 1966b; Cliff et al. 1993; Pendergrast 2010).

The reasons for the slow progress were varied. First, many parents did not consider measles a dangerous disease. Second, the measles vaccine could only be administered

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[4] Reported cases are considered to represent only about ten percent of the estimated three to four million annual cases (Langmuir et al. 1962; National Communicable Disease Center 1967a). Reported cases are likely to reflect especially the cases with complications requiring medical attention. Since measles complications are more common for younger children, a possibility is also that the reported cases mostly reflect cases for younger children, whereas the majority of cases occurs for school-aged children. Reporting of cases without complications was unlikely, because measles was a common disease not considered dangerous and there were no treatment measures available (Harpaz 2004).

after age one, when contacts with child health care were less regular. Third, vaccinating a child against measles in 1963 cost parents ten dollars (about 90 dollars in 2021 USD), which corresponded to approximately ten times the federal minimum hourly wage—considerably more than many of the vaccines available against other diseases (Colgrove 2007).

Consequently, the federal government began providing subsidies for measles vaccination in 1965 in order to act against the exclusion of low-income families (Colgrove 2006). By 1966, however, only Rhode Island had staged a state-wide measles immunization campaign (National Communicable Disease Center 1966c). Given the situation, federal public health officials began advocating for a campaign targeting the entire susceptible population in order to benefit the poor, to increase awareness of the dangers of measles, and to achieve the herd immunity level required for interrupting disease transmission.[5] They received support from President Lyndon B. Johnson, who had outlined the federal administration's "War on Poverty" initiative in his State of the Union address in January 1964 (Colgrove 2006).

The directors of the National Communicable Disease Center[6] announced the national Measles Eradication Campaign in November 1966 (Sencer et al. 1967; Colgrove 2006). They argued that by expanding the coverage of the new vaccine, it would be possible to eradicate measles from the United States within a year. According to campaign plans, measles eradication would require a concerted effort on four fronts: immunizing all infants at one year of age, immunizing all schoolchildren who had not yet had the disease, surveilling the situation through better reporting systems, and promptly controlling any remaining epidemics (Sencer et al. 1967; National Communicable Disease Center 1966b). The office of President Johnson summarized the objectives of the campaign in March 1967: "Our goal is to eliminate measles from the United States in 1967. The

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[5] A coverage of 93–95 percent is required to ensure herd immunity, i.e., to prevent measles epidemics (World Health Organization 2009). In 1967, the estimate of the herd immunity level was lower, at around 55 percent (Sencer et al. 1967), with considerable uncertainty.

[6] The National Communicable Disease Center was a predecessor to the Centers for Disease Control and Prevention.

Surgeon General's target for this year is the vaccination of between 8 and 10 million children—all susceptible children between the ages of one and seven. Thereafter, with the vaccination of the 4 million children born each year, measles will join the ranks of the conquered diseases of childhood" (National Communicable Disease Center 1967c).[7]

The Measles Eradication Campaign was the first major federal immunization campaign after the Vaccination Assistance Act of 1962, which created the first formal nationwide immunization assistance structure (Johnson et al. 2000; Hinman et al. 2011). During the campaign, The National Communicable Disease Center supported states with vaccines, funding, and help with planning. It encouraged state health authorities to contact all private physicians to recommend the vaccine to all susceptible children. In addition, schools organized vaccination drives to make access to the vaccine as easy as possible (National Communicable Disease Center 1966a). States were also advised to ensure that the measles vaccination would be introduced into the routine of clinics and child health conferences (National Communicable Disease Center 1966b).

The campaign distributed 11.7 million doses of the measles vaccine in 1967–1968 (Hendriks and Blume 2013), and campaign records indicate that all states participated (National Communicable Disease Center 1967b). In addition, the campaign acted as a push for states to pass laws requiring immunizations for children entering school (Colgrove 2007). From 1966 to 1968, reported measles cases fell by 90 percent (Figure 1b) and remained low thereafter (Orenstein et al. 2004). Some smaller irregular epidemics continued to occur, because immunizations for subsequent birth cohorts declined (Bloch et al. 1985). Measles vaccination lost all federal support in 1969, when funds were reduced and targeted to the new vaccine against rubella (Cliff et al. 1993).

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[7] Smallpox, diphtheria and polio are examples of childhood diseases that had previously diminished in importance.

3 Measuring the long-term impacts of a mass vaccination campaign

Variation in measles exposure

Our empirical strategy uses geographical and cohort variation in exposure to measles to estimate the causal effect of mass vaccination on educational outcomes. First, we exploit state-level measles incidence rates right before the Measles Eradication Campaign of 1967–68 as a measure of treatment intensity of the campaign. Second, we take advantage of cohort variation in measles exposure induced by the campaign.

We use a difference-in-differences identification strategy and measure the treatment intensity of the immunization campaign, $Exposure_s$, with cumulative measles incidence rates (reported cases per 100,000 population) in the birth state s of an individual in 1963–1966, right before the campaign (Figure 2). The measure is constructed with data from Project Tycho at the University of Pittsburgh (van Panhuis et al. 2013) and it approximates the cohort burden of measles for school-age children before the campaign.[8] We calculate the cumulative incidence in a four-year window for each state in order to capture at least one epidemic cycle of the disease, which has a period of two to three years. Thus, we avoid the risk of mismeasuring the cohort burden of measles by catching only a trough of the cycle. From now on, we use the expression *measles exposure* or *exposure* to refer to our measure of $Exposure_s$.

Due to the age profile of measles infections, our measure of exposure is predominantly driven by cohorts born in the 1950s, who are in school before the campaign. Thus, children born in the 1960s are less likely to have contracted measles by 1966 and more likely to benefit from the vaccination campaign than previous cohorts. Survey evidence also documents that vaccination coverage increases and measles infections decline more for the younger cohorts (Figure 3).[9] Notably, the cohort burden of measles is higher

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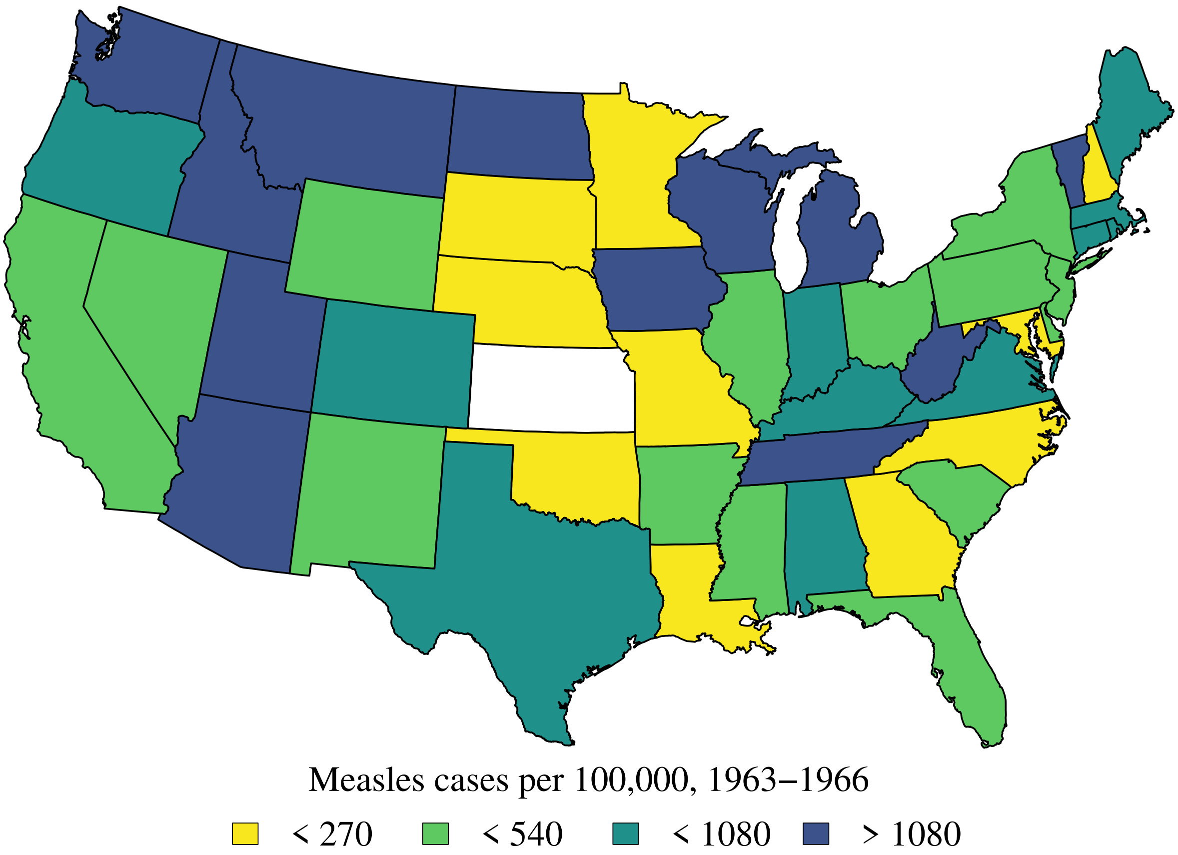
[8] Data for Kansas is not available for this period. Therefore, Kansas is not included in our analysis.

[9] The surveys only report results by age, and since they were conducted in September, years of

Figure 2: Baseline measles exposure by state.



Notes: Authors' calculations using data from Project Tycho (van Panhuis et al. 2013). 540 is the median number of cases per 100,000 across states in 1963–1966. Alaska and Hawaii are included in the analysis but not displayed on the illustrative map. Kansas is excluded from the analysis, because it has no measles incidence data available for 1963–1966.

for children born in the 1950s in the states that experienced higher measles exposure in 1963–1966 than for children in states where baseline exposure was lower. During the vaccination campaign, reported measles incidence declined almost one-to-one with our exposure measure (Figure 4).[10]

Before the cohorts affected by the campaign, our sample data suggests that individuals born in states below and above median exposure follow parallel trends in average years of education (Figure 5a). For the cohorts targeted by the immunization campaign, the gap in educational attainment narrows. The parallel trends prior to the cohorts affected

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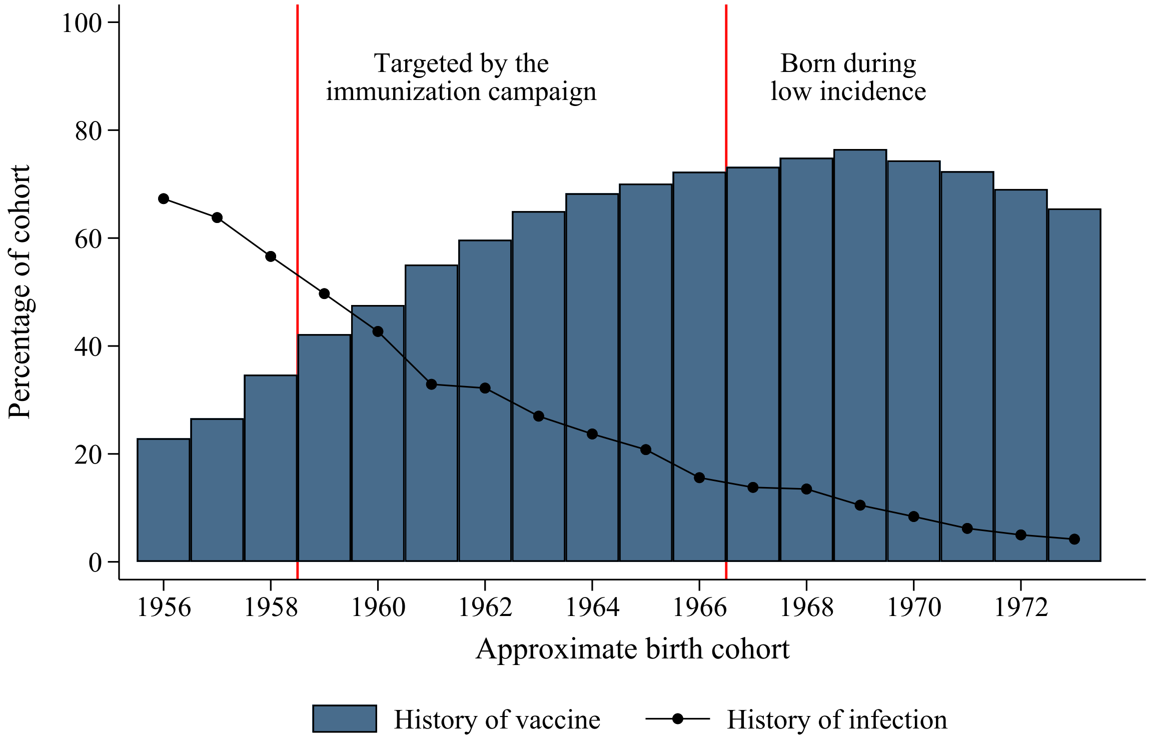
birth inferred from the survey data are not exact. Thus, we assign about a fourth of the children incorrectly a birth year that is one year later than their true birth year. Assigning the correct birth year would be unlikely to substantially alter the trends observed in the figure.

[10] Appendix B details how the exposure measure is related to measles incidence over a longer time span and gives more information on the interpretation of the measure.

Figure 3: The increase in measles vaccination by cohort.

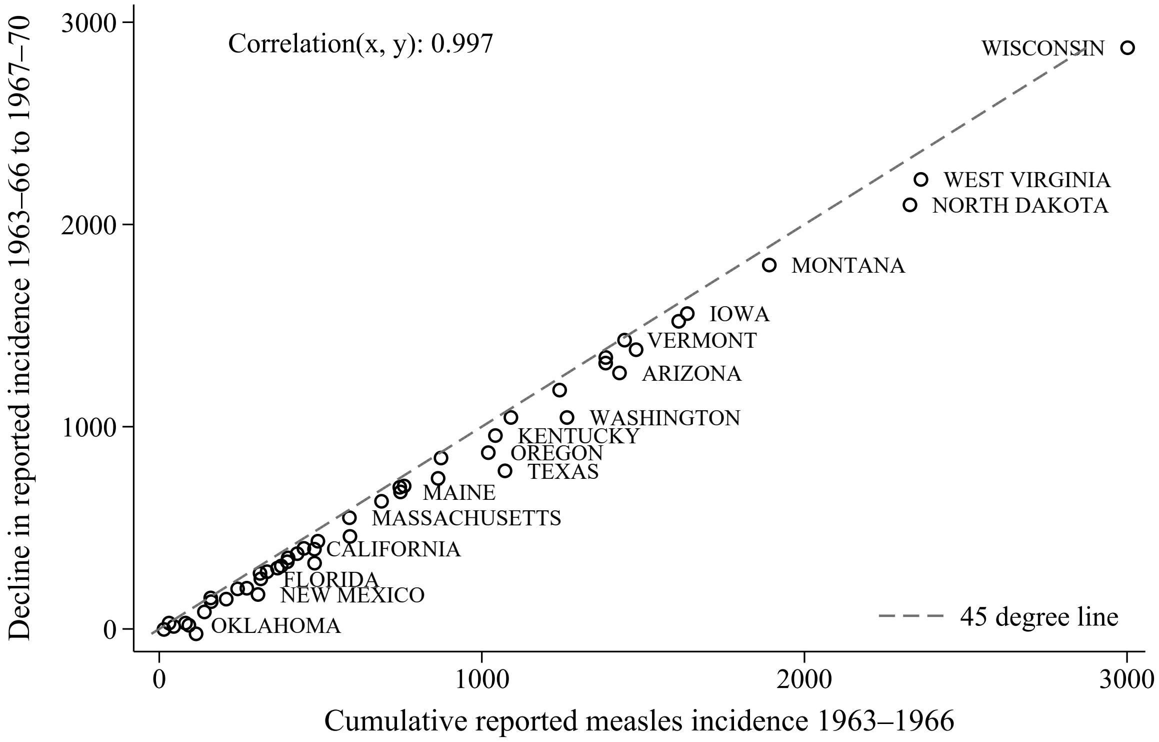


Notes: Figure 3 presents the evolution of measles vaccinations and infections by approximate birth cohort in the United States. The survey only reports results based on age. Therefore, birth years have to be inferred from the data and are partly incorrectly assigned, since the survey was conducted in September. Measles infection data for the cohorts born 1956–1961 comes from the 1966 national immunization survey (National Communicable Disease Center 1966d). Measles vaccination data for the cohorts born 1956–1964 comes from the 1969 national immunization survey (National Communicable Disease Center 1970). Measles infection data for the cohorts born 1962–1973 and measles vaccination data for the cohorts born 1965–1973 come from the 1975 national immunization survey (Center for Disease Control 1976).

by the campaign also suggest that the convergence in educational trends is unlikely to be merely due to some mean-reverting shock.

A causal interpretation of these differences in educational trends relies on an assumption about parallel trends in the absence of mass vaccination against measles. In the dynamic difference-in-differences framework of our main analysis, the measure of exposure should, therefore, not be correlated with any other determinants of educational outcomes among states. This assumption would be violated if some unobserved determinants of educational trends explained the differential evolution of educational attainment among states with different levels of measles exposure. In our main analysis, birth

Figure 4: Decline in reported measles incidence by exposure.



Notes: Authors' calculations using data from Project Tycho (van Panhuis et al. 2013). 540 is the median number of cases per 100,000 across states in 1963–1966. Alaska and Hawaii are included in the analysis but not displayed on the illustrative map. Kansas is excluded from the analysis, because it has no measles incidence data available for 1963–1966.

cohort fixed effects control for shocks common to a birth cohort nationwide and state fixed effects control for time-invariant differences in educational outcomes among states. In another specification, we include other observed characteristics of states.

The exact mechanisms driving the variation in the spread of measles epidemics are imperfectly understood. However, differences in local measles exposure are considered to be affected at least by four factors: climate, population density, prior vaccination coverage, and environmental shocks to the conditions in which the virus thrives (Cliff et al. 1993; Moss and Griffin 2012).[11] In most environments, larger measles epidemics occur every two to three years. Sometimes the waves of measles epidemics are temporarily larger whereas smaller communities may miss waves entirely. Our measles exposure

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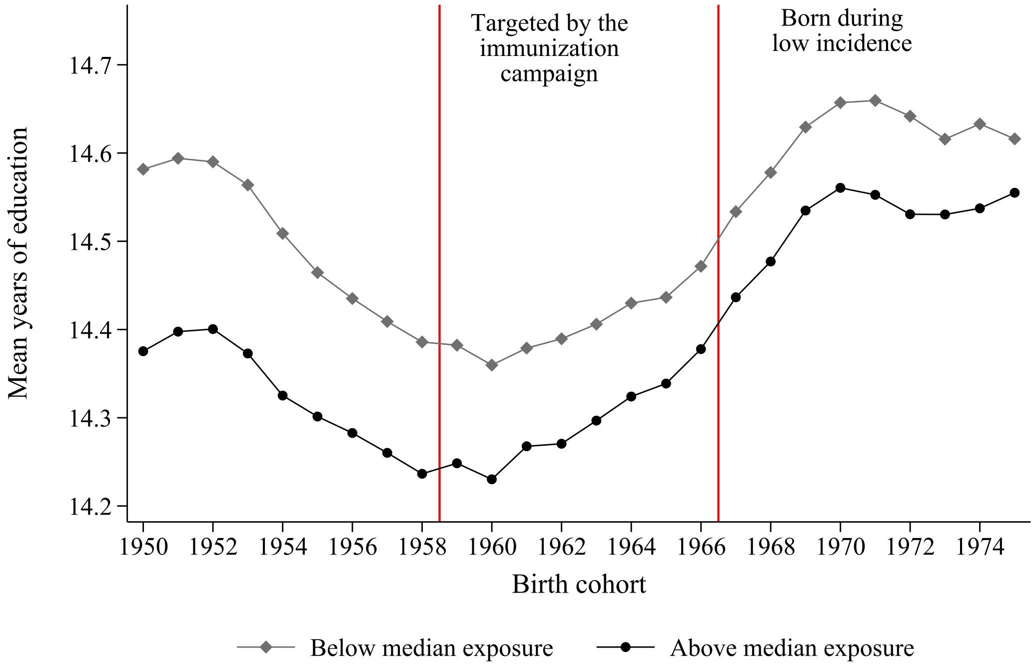
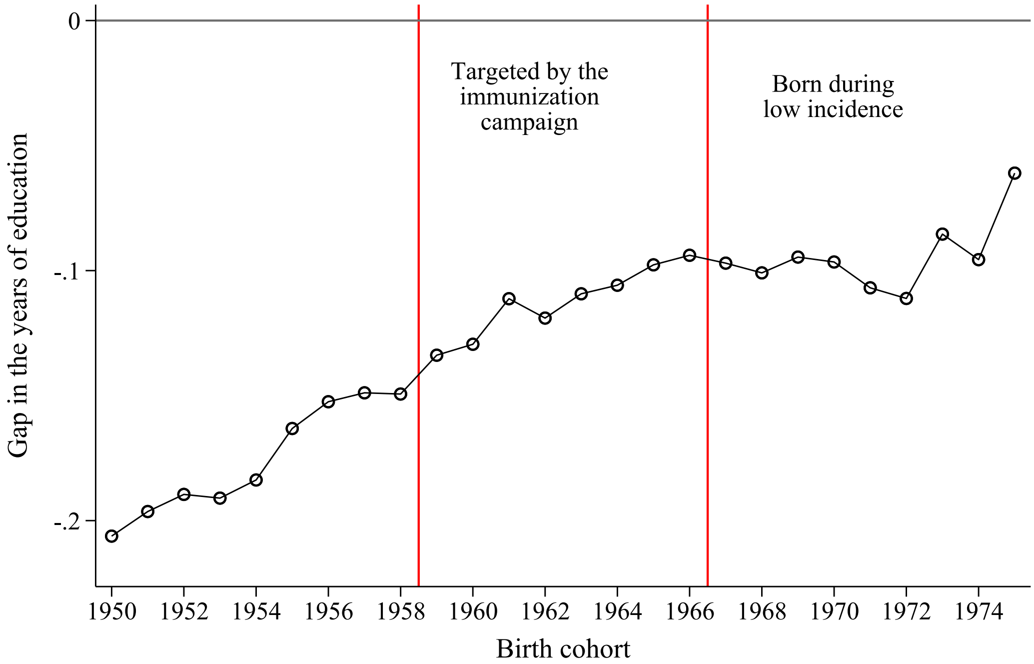
[11] For example, epidemics may occur annually in densely populated cities, while communities with less than 250,000 inhabitants may have a larger lag between epidemics and communities with less than 10,000 inhabitants may miss waves of measles epidemics entirely (Cliff et al. 1993).

Figure 5: Cohort trends in educational attainment by $Exposure_s$.

(a) The mean years of education for below and above median exposure.



(b) The gap in the mean years of education between below and above median exposure.



Notes: Figure 5a presents the evolution of the mean years of education by birth cohort for individuals born in below median versus above median measles exposure states, based on data from IPUMS and Project Tycho. Figure 5b presents the difference of the two series in Figure 5a.

measure aggregates this local variation during 1963–1966 to the state level.

While there remains a possibility that the variation in reported measles incidence may be correlated with some unobserved determinants of educational attainment, our identifying assumption requires the effect of these other determinants not to vary between our treated and untreated cohorts. We consider the variation we are exploiting unlikely to be related to other factors strongly affecting educational attainment. First, only one out of sixteen baseline demographic, socioeconomic, education and health characteristics of above and below median incidence states is statistically different from each other (Table A.1 in the appendix A). Second, from the known determinants of measles incidence, only pre-existing vaccination coverage is a potential sign of investment related to human capital.

Unfortunately, no data about state-level measles vaccination coverage exists for the 1960s. However, vaccination coverage is unlikely to be solely driving the differences in our exposure measure because measles requires more than 90 percent immunization coverage for herd immunity. In the age group driving the incidence rate, i.e. five- to nine-year-olds, vaccination coverage was 19.3 percent in 1965 and 28 percent in 1966. In contrast, in this age group 54.3 percent in 1965 and 49 percent in 1966 reported a history of measles illness (National Communicable Disease Center 1967a).[12] If differences in reported measles incidence were partly driven by vaccination coverage, states with higher coverage could also be investing more in some other factors that increase educational attainment. If anything, this would work against finding effects of reduced measles exposure, since states with lower measles exposure would improve their educational outcomes more than the high measles exposure states due to these other investments, regardless of them benefiting less from reduced measles incidence. In this case, our estimates could be interpreted as a lower bound of the impact on educational attainment.

Differences in measles exposure are unlikely to cause either selective survival or mi-

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[12] The survey does not report vaccination or infection rates by state.

gration. First, measles has a low fatality rate in this context.[13] Second, we assign the measure of exposure based on place of birth. Thus, it is not affected by migration after birth. Third, since measles was a common disease and its severe complications were relatively rare, most parents did not think of measles as a dangerous disease (Hendriks and Blume 2013; National Communicable Disease Center 1966c). In fact, one of the aims of the Measles Eradication Campaign was to change this view in order to induce all parents to vaccinate their children. Consequently, it is unlikely that parents would have reacted to differences in measles incidence rates in a systematic way, such as moving to a different state.

Outcome variables and estimation equations

We measure educational attainment for cohorts born 1950–1975 with individual-level data as recorded in the 2000 US census five percent sample and the 2001–2018 American Community Survey one percent samples from the Minnesota Population Center Integrated Public Use Microdata Series (IPUMS) (Ruggles et al. 2020). The data includes cross-sectional demographic and socioeconomic information on individuals. Our outcomes, y_{isc}, are years of education and the probabilities of graduating from high school and college defined for an individual i, born in state s, and belonging to the cohort born in year c.[14] We additionally use the individual's birth state to assign them the measure of measles exposure.

Our main dynamic difference-in-differences estimation equation, 1, includes birth state fixed effects, γ_s, and birth cohort fixed effects, ζ_c:

$$y_{isc} = \alpha + \sum_{\substack{k=1950 \\ k \neq 1958}}^{1975} \beta_k [Exposure_s \times \mathbb{1}(c = k)] + \gamma_s + \zeta_c + \epsilon_{isc} \quad (1)$$

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[13] Around 500 children died each year due to measles in the United States in the decade before vaccination (Bloch et al. 1985).

[14] High school graduation includes the General Educational Development Certificate, because the IPUMS data does not differentiate between the regular high school diploma and the GED certificate before 2008. College graduation is defined as Associate degree or higher.

We exclude the cohort of 1958 as a normalization, because this cohort turned ten years old during the campaign. Thus, children in this cohort were unlikely to benefit from either the immunizations given to those up to age seven or from less exposure to school disruptions, because they mostly completed elementary school by 1968. Following Bleakley (2010), we rescale the $Exposure_s$ measure to equal zero for the 5th percentile state (baseline measles incidence 45 reported cases per 100,000) and one for the 95th percentile state (baseline measles incidence 2,327 reported cases per 100,000). Thus, the estimate of β can be interpreted as the effect of reducing measles incidence from a state with a high measles burden right before the campaign to close to zero, which is in line with the rapid reduction of measles incidence for these cohorts.

Our additional estimation equation, 2, incorporates the age profile of measles infections and summarizes the estimated impact of the mass vaccination campaign on education into one point estimate:

$$y_{isc} = \alpha + \beta(Exposure_s \times \omega_c) + \gamma_s + \zeta_c + \epsilon_{isc} \tag{2}$$

We assign a *cohort weight*, ω_c, for each cohort, corresponding to the proportion of children in the cohort who are unlikely to have contracted measles by the time of the campaign and, therefore, could have benefited from the vaccine that protects from infection. We interact this cohort weight with our measure of exposure, essentially scaling the treatment intensity variable by the proportion of each cohort that was likely to benefit from the campaign. This specification pools the estimates for different cohorts together and assumes that the age profile of measles infections is fixed across states, because data on the age profile is not available by state.

We approximate the cohort weights ω_c with two different data sources. The weights are presented in Table A.2 in the appendix. In our second specification, we use an average of the age profiles of measles infections from four studies conducted before the measles vaccine (Figure 1a) for the same cohorts as in our main specification (1950–1975). For example, the 1950 and 1951 cohorts receive a weight of zero, since according to the age profile of infections virtually all individuals in these cohorts have contracted measles by 1967. Conversely, cohorts born 1967 onwards receive a weight of one because none of them have contracted measles before the campaign. The rest of the cohorts receive a weight between zero and one. In the third specification, we use the age profile reported in the September 1966 Immunization Survey (National Communicable Disease Center 1966d), recording what proportion of each approximate cohort had not contracted measles by September 1966, prior to the campaign.[15] The survey only lists infection rates for children up to age ten. Therefore, the estimates from this specification only use data for cohorts born 1956 onward.

4 Long-term educational impacts of measles control

Main results on educational attainment

Figure 6 reports our main results from equation 1 on educational attainment for individuals born in the United States between 1950 and 1975. The left panel displays outcomes of the difference-in-differences analysis for males and the right panel for females.[16] For men, the average years of education increase among the cohorts affected by the Measles Eradication Campaign but not among the earlier cohorts. Moving from the 5th percentile exposure state (baseline measles incidence 45 reported cases per 100,000) to the 95th percentile treatment intensity state (baseline measles incidence 2,327 reported cases per 100,000), educational attainment increases by 0.12 years for the male cohorts targeted by the vaccination campaign or born after the large decline in measles incidence.[17] These men also increase their college graduation rates by about two percentage points on average.

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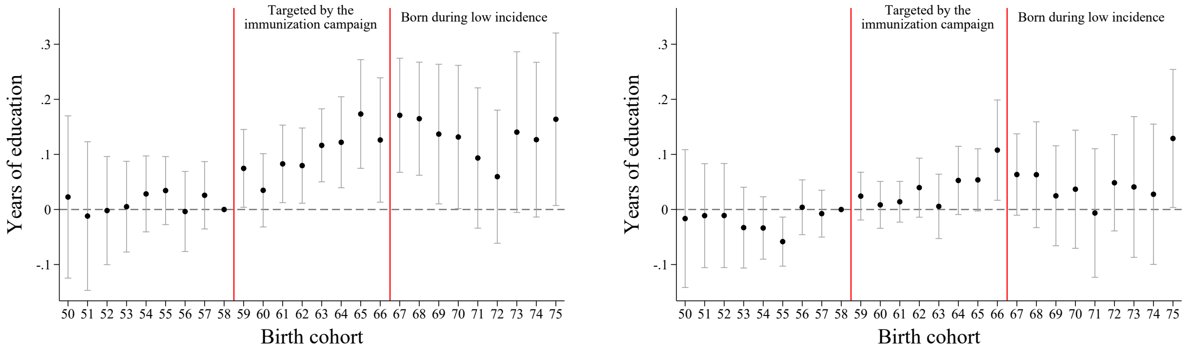
[15] The survey only reports results based on age. Therefore, birth years have to be inferred from the data and are partly incorrectly assigned, since the survey was conducted in September.

[16] Figure A.1 in the appendix A presents the pooled results for men and women.

[17] Since our measure of measles exposure takes on multiple values, the units of the regressor have been scaled to correspond to an increase in baseline reported measles incidence 1963–1966 from the 5th percentile to the the 95th percentile state, following Bleakley (2010).

Figure 6: Educational impacts of mass vaccination against measles.

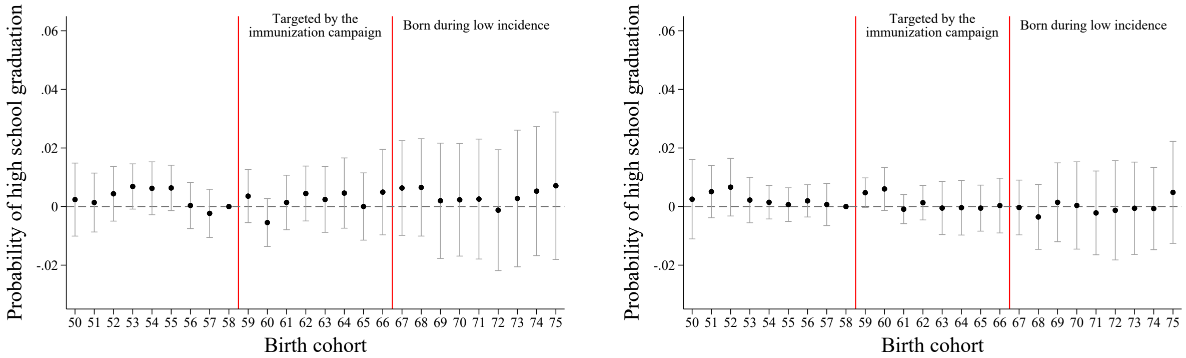


(a) Years of education for males

Mean of the coefficients 1959–1975: 0.118 Dep. var. mean for the cohort 1958: 14.222

(b) Years of education for females

Mean of the coefficients 1959–1975: 0.043 Dep. var. mean for the cohort 1958: 14.383

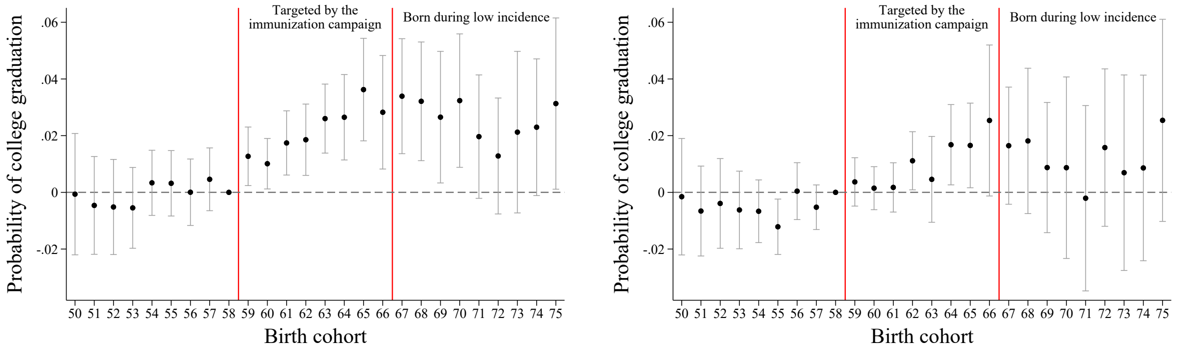


(c) High school graduation for males

Mean of the coefficients 1959–1975: 0.003 Dep. var. mean for the cohort 1958: 0.894

(d) High school graduation for females

Mean of the coefficients 1959–1975: 0.000 Dep. var. mean for the cohort 1958: 0.922



(e) College graduation for males

Mean of the coefficients 1959–1975: 0.024 Dep. var. mean for the cohort 1958: 0.342

(f) College graduation for females

Mean of the coefficients 1959–1975: 0.011 Dep. var. mean for the cohort 1958: 0.380

Notes: Difference-in-differences estimates from equation 1 using data from IPUMS and Project Tycho. The estimates are scaled for an increase in $Exposure_s$ from the 5th percentile to the 95th percentile. Vertical bars denote 95 percent confidence intervals. Standard errors are clustered at the state level. The regressions do not include data for Kansas, because Kansas has no measles incidence data available for 1963–1966. High school graduation includes the General Educational Development Certificate, because the IPUMS data does not differentiate between the regular high school diploma and the GED certificate before 2008. College graduation is defined as Associate degree or higher. The number of observations is 9,015,194 for males and 9,485,761 for females.

For women, our point estimates are positive but approximately half the size of those for men. However, the estimates are relatively imprecise and, therefore, we are unable to rule out the possibility of economically meaningful effects for women. For the high-school graduation rate, our point estimates are close to zero for both sexes but we are only able to rule out effects larger than approximately two percentage points.

For white men and women, our estimates are similar to those of the population overall but more precise (Figure A.2 in the appendix A).[18] The statistical power of detecting effects for other ethnicities is not sufficient, because, for many states, the census sample data includes very few individuals of other ethnicities than white per cohort. As the last analysis with our main specification, we conduct a robustness check of our treatment intensity variable by restricting it to data only from 1966, the year before the campaign (Figure A.3 in the appendix A). The point estimates display a similar pattern and the estimates for males are statistically significant but smaller in magnitude.

To place these estimates into context, the effect on men's years of education is relatively modest in magnitude – 0.7 percent of the baseline years of education, which exceed 14 years. However, in our sample, the overall average years of education increased from 14.22 for the male cohort born 1958 to 14.36 for the male cohort born 1975 – an increase of 0.14 years over 17 years. Therefore, the estimated effect is considerable compared to the overall trend. Another way of assessing the magnitude of the effect is to consider the costs of the immunization campaign. In 1967, the National Communicable Disease Center estimated vaccinating one child during the campaign to cost two dollars (17 dollars in 2022 USD) (National Communicable Disease Center 1967c). Consequently, the educational benefits from the mass vaccination campaign against measles are likely to exceed the costs with any sensible estimates of returns to schooling for these cohorts (Acemoglu and Autor 2011; Heckman et al. 2006), even without taking in to account all other benefits of measles vaccination.

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[18] The estimates only include non-Hispanic white men and women, but they are similar if Hispanics are included.

Our estimated effects on college graduation are larger than those for high school graduation. While we are unable to rule out relatively large increases or decreases of the high-school graduation rate, the clearer pattern for college graduation is not entirely surprising given the overall trends in educational attainment for these cohorts. The cohorts in our data sample have high-school graduation rates (including the GED) of approximately 90 percent and college graduation rates of about 35 percent. In general, high school graduation rates had already peaked for those cohorts born in the 1950s and remained roughly constant for the rest of the twentieth century (Goldin and Katz 2008). While the reasons for this stagnation remain somewhat elusive, any potential increase in human capital due to reduced measles exposure may not have been enough to offset other factors affecting high-school dropout in this period.

Additionally, our point estimates for males are larger than for females. This finding is consistent with earlier literature suggesting that boys tend to be more vulnerable to health insults in early childhood and that the immune system functions differently in boys and girls (Klein and Flanagan 2016; Takahashi and Iwasaki 2021). Boys also seem to be more likely to be hospitalized for respiratory infections in the primary school ages (Jensen-Fangel et al. 2004; Falagas et al. 2007) and there is some evidence that measles incidence and its complications are more common among boys (Muenchhoff and Goulder 2014). Consequently, boys may be more vulnerable to measles and to the immune system suppression caused by the measles virus. Our results are also in line with evidence from Bangladesh, where boys but not girls increased their school enrollment as a result of a measles vaccination (Driessen et al. 2015).

Our findings contribute to a large literature that estimates the effect of health and educational interventions on children's later life outcomes. Below, we compare our estimated effects to other historical papers that examine reductions in exposure to infectious diseases. These papers usually consider settings with substantially lower baseline educational attainment than our context. In addition, we compare our estimates to the effects of class size reductions and teacher value added to provide a comparison with non-health related interventions targeting educational outcomes.

For disease control campaigns, Bütikofer and Salvanes (2020) find that a tuberculosis testing and vaccination campaign in 1940s Norway increased schooling by 0.5 years and the likelihood to graduate high school by 5.5 percentage points for a municipality with pre-campaign infection rate of one person per 100 inhabitants. These estimates are much larger than the effects we estimate for measles. Bleakley (2007, 2010) finds mixed results on educational attainment from the eradication of hookworm in the early 20th century United States and the eradication of malaria in the 1950s Brazil, Mexico and Colombia. However, he finds large effects on literacy in these contexts. Battaglia and Kisat (2021) find that the malaria eradication campaign in the 1920s United States increased schooling by 0.1 years for individuals exposed to the program in highly malarious areas (with a mortality rate of 1 death per 1,000). They also experienced a two percentage point increase in their likelihood of middle school completion. Lazuka (2020) finds that access to antibiotics to treat pneumonia in infancy increased schooling by 0.15 years for children born in 1930s Sweden. The estimates from Battaglia and Kisat (2021) and Lazuka (2020) are of similar magnitude to our estimates for males.

For educational interventions, Chetty et al. (2011) find that students assigned to small classes in Tennessee in the 1980s are 1.8 percentage points more likely to be enrolled in college at age 20. This estimate is of similar magnitude to our estimate for the effect of reduced measles exposure on college graduation of males. Chetty et al. (2014) find that a 1 SD increase in teacher value-added in a single grade in primary school in the school years 1988–2009 raises the probability of college attendance at age 20 by 0.82 percentage points from a sample mean of 37%. Our sample mean for college graduation is comparable (36%), and taking both estimates at face-value, this means not being exposed to measles has an effect comparable to having a 1 SD better teacher for two to three years in primary school.

Additional results including the age profile of measles infections

The results from equation 1 are intent-to-treat estimates, because they do not take into account who took up the vaccine. Although we do not know exactly who received the vaccine, the increasing pattern of educational attainment for the affected male cohorts documented in our estimates is in line with the proportion of children that were likely to benefit from the protection offered by the vaccine. Thus, our second specification takes into account that parts of the cohorts born before the campaign could not have benefited from the vaccine itself, because they had already contracted measles before 1967. This specification scales the treatment intensity variable with the proportion of each cohort that had not yet been infected with measles.

Table 1 reports results from equation 2 on the years of education using two alternative sources of data to determine the proportion of susceptible children that could be protected from measles infection. *Column 1* presents estimates using cohort weights approximated from Figure 1a. The estimated 0.13 years of increase in educational attainment for males is in line with the results from our main specification, but the estimate is only statistically significant at the ten percent level. The estimate for females is positive but not statistically significant. In *column 2*, we control for baseline characteristics of states, interacted with the birth cohort fixed effects. The controls include demographic, socioeconomic, education and health characteristics in 1959–1962 from the 1962 and 1967 County Data Books and the 1960 vital statistics (Haines and Inter-university Consortium for Political and Social Research 2010; National Center for Health Statistics 1963; Manson et al. 2018). Our estimated effects for males are slightly lower than in column 1 but statistically significant at the five percent level.

Column 3 changes the specification from the previous columns by replacing the cohort weights with data from the 1966 Immunization Survey documenting the proportion of children that had not been infected with measles by 1966. For males, the estimate from this specification is larger in magnitude than in the previous columns and statistically

Table 1: Additional results on the years of education, pooling cohorts

Panel A: Years of education for males

	Equation 2 estimates of β				Placebo
	(1)	(2)	(3)	(4)	(5)
$Exposure_s \times \omega_t$	0.133*	0.115**	0.196**	0.159**	−0.154
	(0.076)	(0.056)	(0.094)	(0.072)	(0.102)
Dep. variable mean	14.22	14.22	14.22	14.22	14.22
State controls	No	Yes	No	Yes	No
Cohort weight	Age profile	Age profile	Survey	Survey	Age profile
Cohorts	1950–1975	1950–1975	1956–1975	1956–1975	1950–1975
Observations	9015194	9015194	6928380	6928380	9015194
Clusters	50	50	50	50	50

Panel B: Years of education for females

	Equation 2 estimates of β				Placebo
	(1)	(2)	(3)	(4)	(5)
$Exposure_s \times \omega_t$	0.076	0.047	0.086	0.019	−0.134
	(0.061)	(0.054)	(0.080)	(0.073)	(0.086)
Dep. variable mean	14.38	14.38	14.38	14.38	14.38
State controls	No	Yes	No	Yes	No
Cohort weight	Age profile	Age profile	Survey	Survey	Age profile
Cohorts	1950–1975	1950–1975	1956–1975	1956–1975	1950–1975
Observations	9485761	9485761	7251415	7251415	9485761
Clusters	50	50	50	50	50

Notes: Significance levels: ** 5% level, * 10% level. Standard errors in parentheses, clustered at the state level. The estimates of β are difference-in-differences estimates from equation 2 using data from IPUMS, Project Tycho, the 1962 and 1967 County Data Books, NHGIS, and the 1960 vital statistics. The estimates are scaled for an increase in $Exposure_s$ from the 5th percentile to the 95th percentile. In columns 1, 2 and 5, cohort weights ω_c are derived from measles infection age profiles reported in Figure 1a. In columns 3 and 4, cohort weights come from the 1966 immunization survey. The cohort weights approximate the proportion of children who could benefit from protection against measles. Columns 2 and 4 control for four sets of state characteristics, interacted with the birth cohort fixed effects. *Demographic characteristics* include 1960 proportions of urban, rural farm, non-white, foreign born and white-collar worker. *Income distribution characteristics* include the 1959 median household income, the proportions of households with income below 3,000$ and with income above 10,000$, and public welfare expenditure per capita 1962. *Educational characteristics* include the median years of education for adults over 25 and the proportion of adults with more than 12 years of education in 1960, as well as state education expenditure per capita in 1962. *Health characteristics* include state health and hospital expenditure per capita in 1962, the crude death rate 1959, the infant mortality rate 1960, and the mortality rate of one- to four-year-olds 1960. The reported dependent variable mean is calculated for the cohort born 1958. The regressions do not include data for Kansas, because Kansas has no measles incidence data available for 1963–1966.

significant at the five percent level. For females, the estimate is again not statistically significant. In *column 4*, we again control for the same baseline characteristics of states than in column 2. Our estimated effects for males are lower than in column 3 but remain statistically significant at the five percent level.

Finally, *column 5* reproduces the analysis in column 1 but replaces our exposure measure with a placebo measure of hepatitis A reported incidence data instead of measles data, also from Project Tycho (van Panhuis et al. 2013).[19] The point estimates are negative and not statistically significant for either males or females.

5 Conclusion

This paper examines the effects of the first federal mass vaccination campaign against measles on educational attainment in the United States. Our paper is the first to identify impacts of reduced childhood measles exposure on educational attainment in adulthood. We show that the Measles Eradication Campaign conducted in 1967–1968 resulted in a modest but statistically significant increase in the educational attainment of men in the affected cohorts. We conclude that even when considering only the educational benefits of mass vaccination against measles, the benefits are very likely to exceed the costs, given the high returns to increases in educational attainment. These results contribute to the literature on the positive link between publicly provided preventive health care investments and human capital formation. They highlight the importance of taking into account the potential long-term benefits of public health investments.

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[19] Hepatitis A is the only other notifiable disease for which (i) the Porject tycho data is available incidence prior to 1967 (ii) reported incidence is of similar magnitude than measles (iii) no widely used vaccine exists prior to 1975. However, hepatitis A incidence data is only available from 1966 onwards. Thus, we use only data from that year but we scale the exposure measure using the same procedure as for measles.

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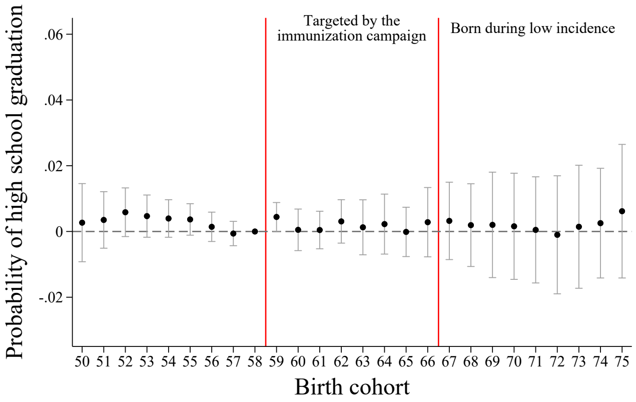
A Appendix: Additional figures and tables

Figure A.1: Educational impacts of mass vaccination against measles, men & women pooled.

(a) Years of education

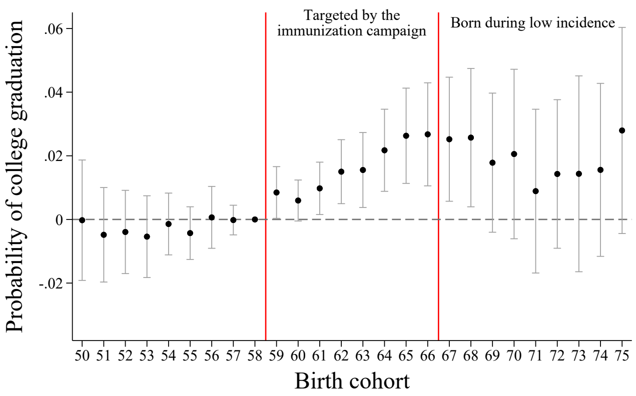
Mean of the coefficients 1959–1975: 0.081 Dep. var. mean for the cohort 1958: 14.305

(b) High school graduation



Mean of the coefficients 1959–1975: 0.002 Dep. var. mean for the cohort 1958: 0.908

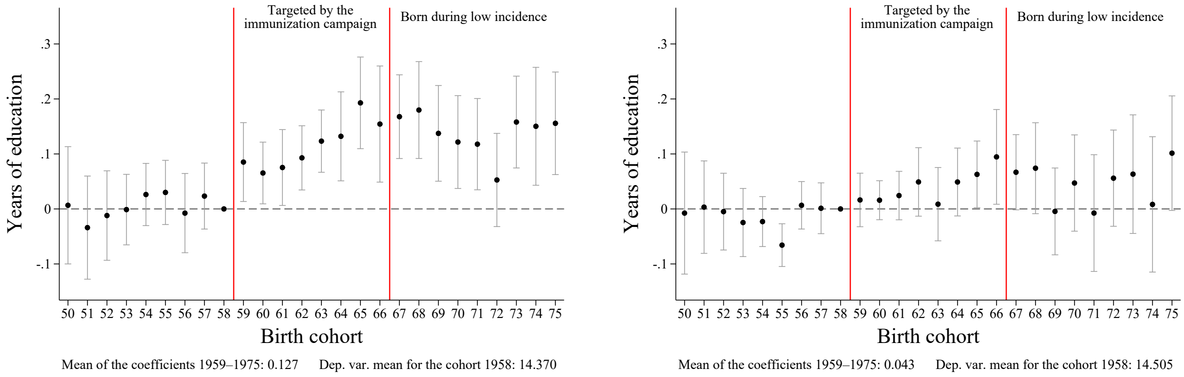
(c) College graduation



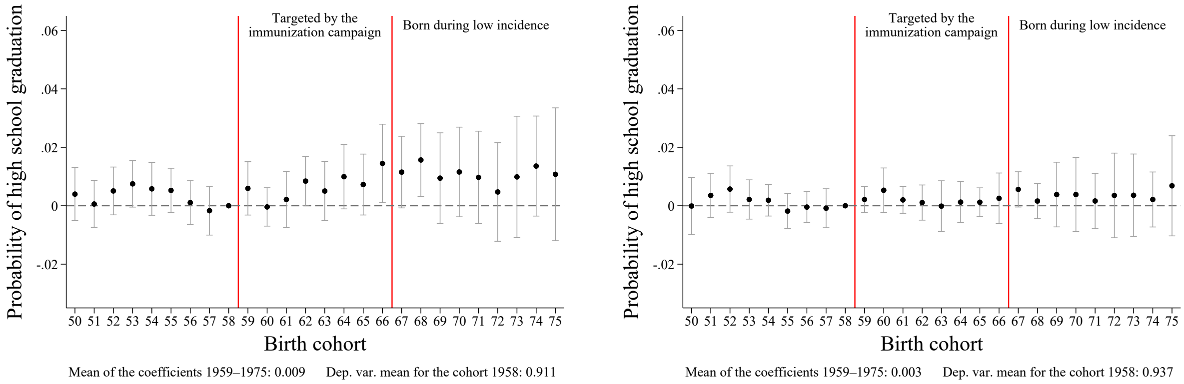
Mean of the coefficients 1959–1975: 0.018 Dep. var. mean for the cohort 1958: 0.361

Notes: Difference-in-differences estimates from equation 1 using data from IPUMS and Project Tycho. The estimates are scaled for an increase in $Exposure_s$ from the 5th percentile to the 95th percentile. Vertical bars denote 95 percent confidence intervals. Standard errors are clustered at the state level. The regressions do not include data for Kansas, because Kansas has no measles incidence data available for 1963–1966. High school graduation includes the General Educational Development Certificate, because the IPUMS data does not differentiate between the regular high school diploma and the GED certificate before 2008. College graduation is defined as Associate degree or higher. The number of observations is 9,015,194 for males and 9,485,761 for females.This is a new graph.

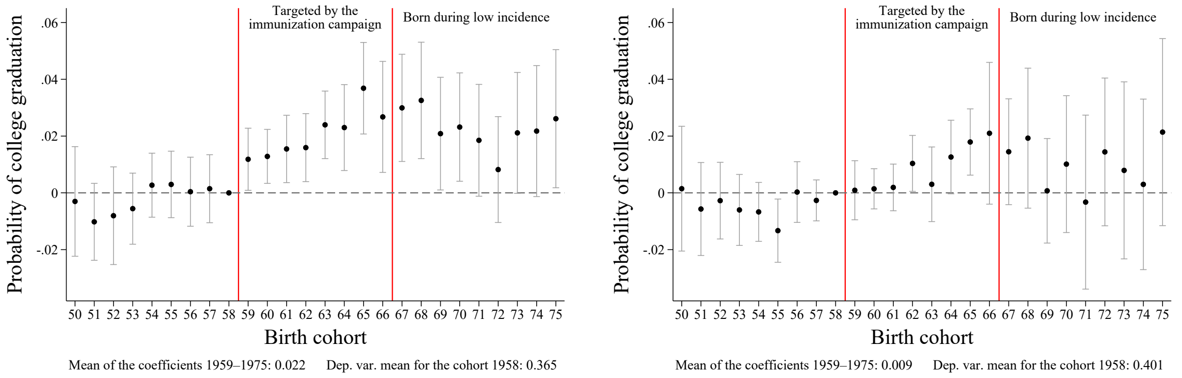
Figure A.2: Educational impacts of mass vaccination against measles, white only.



(a) Years of education for white males **(b)** Years of education for white females



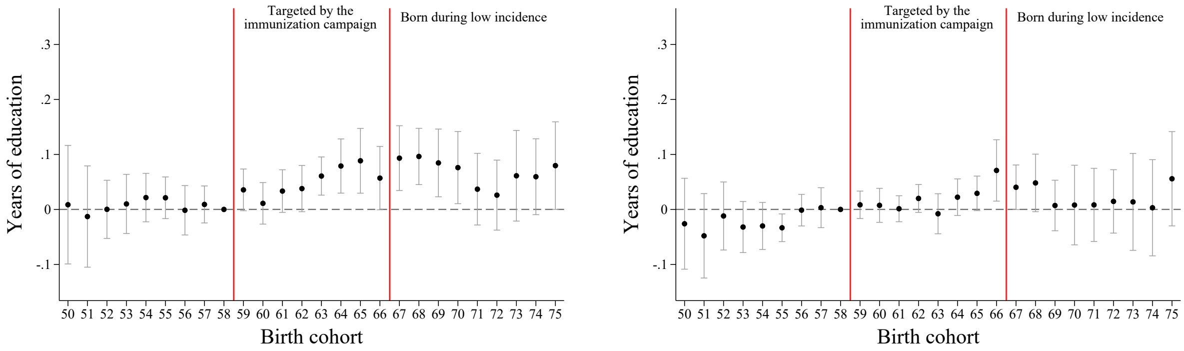
(c) High school graduation for white males **(d)** High school graduation for white females



(e) College graduation for white males **(f)** College graduation for white females

Notes: Difference-in-differences estimates from equation 1 using data from IPUMS and Project Tycho. The estimates are scaled for an increase in $Exposure_s$ from the 5th percentile to the 95th percentile. Vertical bars denote 95 percent confidence intervals. Standard errors are clustered at the state level. The regressions do not include data for Kansas, because Kansas has no measles incidence data available for 1963–1966. High school graduation includes the General Educational Development Certificate, because the IPUMS data does not differentiate between the regular high school diploma and the GED certificate before 2008. College graduation is defined as Associate degree or higher. White refers to white and non-Hispanic as defined by IPUMS USA. The number of observations is 7,380,014 for males and 7,629,261 for females.

Figure A.3: Educational impacts of mass vaccination against measles, 1966 incidence only.

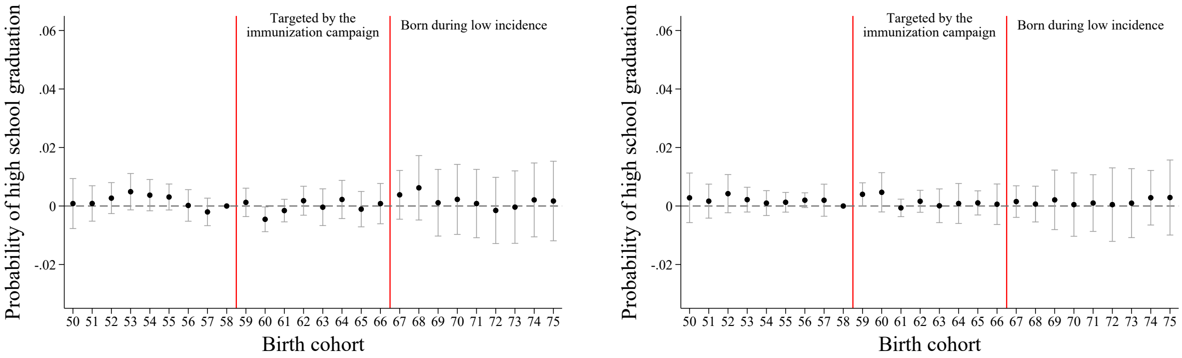


(a) Years of education for males

(b) Years of education for females

Mean of the coefficients 1959–1975: 0.060 Dep. var. mean for the cohort 1958: 14.222

Mean of the coefficients 1959–1975: 0.021 Dep. var. mean for the cohort 1958: 14.383

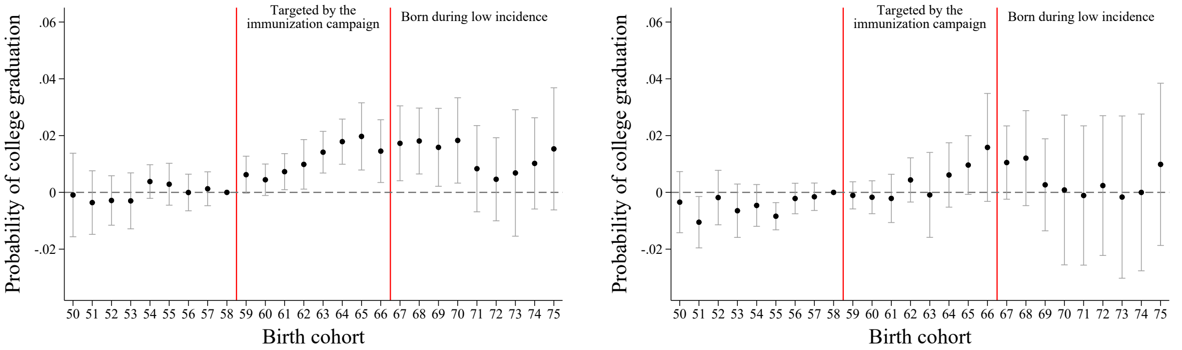


(c) High school graduation for males

(d) High school graduation for females

Mean of the coefficients 1959–1975: 0.001 Dep. var. mean for the cohort 1958: 0.894

Mean of the coefficients 1959–1975: 0.001 Dep. var. mean for the cohort 1958: 0.922



(e) College graduation for males

(f) College graduation for females

Mean of the coefficients 1959–1975: 0.012 Dep. var. mean for the cohort 1958: 0.342

Mean of the coefficients 1959–1975: 0.004 Dep. var. mean for the cohort 1958: 0.380

Notes: Difference-in-differences estimates from equation 1 using data from IPUMS and Project Tycho. $Exposure_s$ consists of reported measles incidence in 1966, as opposed to 1963–1966 in the main analysis. The estimates are scaled for an increase in $Exposure_s$ from the 5th percentile to the 95th percentile. Vertical bars denote 95 percent confidence intervals. Standard errors are clustered at the state level. The regressions do not include data for Kansas, because Kansas has no measles incidence data available for 1966. High school graduation includes the General Educational Development Certificate, because the IPUMS data does not differentiate between the regular high school diploma and the GED certificate before 2008. College graduation is defined as Associate degree or higher. The number of observations is 9,015,194 for males and 9,485,761 for females.

Table A.1: State characteristics by baseline measles exposure

	Below median exposure			*Above median exposure*			*Difference*
State characteristic	Mean	Median	SD	Mean	Median	SD	T-statistic
Measles incidence 1963–1966	268	306	154	1,301	1,241	599	-8.35
Urban	0.65	0.66	0.17	0.60	0.62	0.15	0.91
Rural farm	0.10	0.06	0.08	0.10	0.07	0.08	-0.05
Non-white	0.15	0.09	0.14	0.10	0.04	0.14	1.46
Foreign born	0.04	0.03	0.03	0.05	0.04	0.03	-0.72
White-collar	0.40	0.40	0.06	0.40	0.40	0.04	0.07
Median family income ($) 1959	5,278	5,573	1,145	5,446	5,568	882	-0.58
Family income <3,000 ($) 1959	0.25	0.21	0.12	0.22	0.20	0.08	0.96
Family income >10,000 ($) 1959	0.14	0.13	0.06	0.13	0.13	0.05	0.19
Median years of schooling (25+)	10.46	10.60	1.09	10.76	11.00	1.13	-0.98
12+ years of schooling (25+)	0.41	0.41	0.07	0.43	0.43	0.08	-0.97
Education expenditure per capita 1962	95.98	91.99	23.52	94.30	95.18	23.99	0.25
Expenditure on health & hospitals per capita 1962	13.65	9.35	12.46	8.09	8.02	4.47	2.10
Expenditure on public welfare per capita 1962	11.01	4.05	12.48	7.39	3.14	11.86	1.05
Infant mortality rate (per thousand live births)	27.63	26.25	4.68	25.66	24.19	4.58	1.50
One- to four-year-olds mortality rate (per thousand)	1.19	1.15	0.30	1.12	1.08	0.30	0.76
Crude death rate (per thousand) 1959	9.24	9.25	1.14	8.83	9.23	1.55	1.07

Notes: Data from IPUMS, Project Tycho, the 1962 and 1967 County Data Books, NHGIS, and the 1960 vital statistics. Data for Kansas is excluded, because Kansas does not have measles incidence data available for 1963–1966. Variables are measured for 1960, unless otherwise indicated. *Measles incidence* is measured in cases per 100,000. *White-collar* is proportion of the labor force employed in white-collar occupations. The per capita variables use state total population measured in the 1960 census.

Table A.2: Cohort weights

Birth year	Age in 1967	ω_c, age-specific mean from Figure 1a	ω_c, September 1966 Immunization Survey
1950	17	0	
1951	16	0	
1952	15	0.070	
1953	14	0.092	
1954	13	0.071	
1955	12	0.086	
1956	11	0.126	0.327
1957	10	0.118	0.362
1958	9	0.163	0.434
1959	8	0.208	0.503
1960	7	0.274	0.573
1961	6	0.358	0.671
1962	5	0.458	0.756
1963	4	0.570	0.808
1964	3	0.724	0.868
1965	2	0.837	0.919
1966	1	0.937	0.98
1967	0	1	1
1968	-1	1	1
1969	-2	1	1
1970	-3	1	1
1971	-4	1	1
1972	-5	1	1
1973	-6	1	1
1974	-7	1	1
1975	-8	1	1

Notes: In column 3, cohort weights come from the authors' calculations based on age profiles of measles infection as estimated in four surveys conducted before the measles vaccine (Collins 1929; Collins et al. 1942; Black 1959; Epidemic Intelligence Service 1961). In column 4, cohort weights come from the 1966 immunization survey. The cohort weights approximate the proportion of children who could benefit from protection against measles, i.e., who have not yet been infected with measles by the start of the measles eradication campaign in 1967. This is a new table.

B Appendix: Interpretation of the exposure measure

The variation in treatment intensity across states is the exposure to measles prior to the Measles Eradication Campaign as proxied for by the four-year reported incidence in 1966, i.e. based on the years 1963–1966. As emphasized by figure 4, reported incidence declines almost one-for-one from 1963–1966 to 1967–1970. This means the 1963–1966 exposure measure is not predictive of the same measure in 1967–1970, after the eradication campaign.

We argue that this lack of predictiveness is exactly the change brought about by the campaign. Thus, our estimation should be interpreted as identifying the effect of this decline in reported incidence compared to the counterfactual in which the cumulative reported incidence between 1963 and 1966 had been as predictive for the same measure between 1967 and 1970 as was the case prior to the measles eradication campaign.

We estimate equation 3 to obtain the elasticity of $exposure(s, t-4)$ on $exposure(s, t)$ when including fixed effects for year t (ζ_t) and state s (γ_s) for the period prior to 1966.

$$log(exposure_{st}) = \eta \; log(exposure_{s,t-4}) + \gamma_s + \zeta_t + \epsilon_{st} \tag{3}$$

As shown in table B.1, column 1, prior to the vaccination campaign our exposure measure is highly predicted by exposure four years earlier. We estimate an elasticity of 0.368 with a standard error an order of magnitude smaller.

The additional columns (2 and 3) provide ancillary evidence that $exposure_{t-4}$ was strongly predictive of $exposure_t$ only for $t \leq 1966$. The elasticity of $exposure_t$ with respect to $exposure_{t-4}$ is only positive until 1966. It is estimated to be -0.128 and statistically significant for the period with $t \geq 1967$ (column 2).

We then include data from both the pre- and post-campaign periods, but allow the elas-

Table B.1: Results regarding the interpretation of the exposure measure

	Equation 1 estimates of η		
	(1) Pre-Campaign	(2) Post-Campaign	(3) Both
$log(exposure_{t-4})$	0.368 (0.038)	-0.158 (0.050)	
$log(exposure_{t-4}) \times \mathbb{1}(t \leq 1966)$			0.378 (0.046)
$log(exposure_{t-4}) \times \mathbb{1}(t \geq 1967)$			-0.027 (0.033)
Years $(t-4)$	1950–1962	1963–1975	1950–1975
Years (t)	1954–1966	1967–1979	1954–1979
N	624	624	1248

Notes: Standard errors in parentheses. The table displays estimates of η from equation 1 using measles incidence data from Project Tycho. As both explanatory and dependent variables are in logs, we obtain estimates of elasticities. Column 3 deviates from equation 1 by allowing the effect of exposure to differ before and after the measles vaccination campaign. All regressions include fixed effects for *year* and *state*. Each observation is the log four-year measles incidence in a state and year. The sample for these regressions includes 48 states and the District of Columbia. As in all analyses presented, Kansas is excluded because of missing data. Alaska is also excluded because $exposure_{t-4}$ is missing for 1954-1957.

ticity to differ between the two time periods. The estimates support the other findings. The estimates of the elasticity pre-campaign retain a similar point estimates and only marginally greater standard errors, while the point estimate post-campaign is negative, but small in absolute terms and not statistically distinguishable from 0.