Extended Commentary on Atwood (2022)

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1 Introduction

This document presents additional results from my reanalysis of Atwood (2022). Section 2 performs a formal test for a trend break in the event study in Wiebe (2024); there is no post-vaccine trend break, contradicting a treatment effect of the vaccine. Section 3 tests for heterogeneity by race, sex, and region; I find null or opposite effects for Black men and women, and puzzling differences by region. Section 4 calculates regression weights; cohorts with medium vaccine exposure have the least weight. Section 5 plots the raw data. I find that people born in states with above-median measles incidence have higher incomes as adults, for all cohorts. This contradicts the story where poor states have high measles incidence. Section 6 reruns the event study for contemporary disease incidence. I find large effects corresponding to the rubella epidemic and the rubella and mumps vaccines. Section 7 runs a placebo test using incidence of other diseases as the treatment variable. While Atwood emphasizes immune amnesia as an explanation, the results from these two sections are consistent with reporting capacity, initial health levels, or immune amnesia.

2 Event study: testing for trend breaks

In Wiebe (2024) I run an event study on the economic outcomes and find post-vaccine trends that are inconsistent with a treatment effect of the vaccine. This involves eyeballing the event study coefficients and seeing a trend when cohorts have equal access to the vaccine. Following Roodman (2018), Fig. 6, here I formally test for a trend break using a piecewise regression with kink points at 1949 and 1964, corresponding to the 1949-1964 cohorts with increasing exposure to the vaccine. This creates three line segments corresponding to three time windows, representing cohorts with no, partial, and full access to the vaccine.

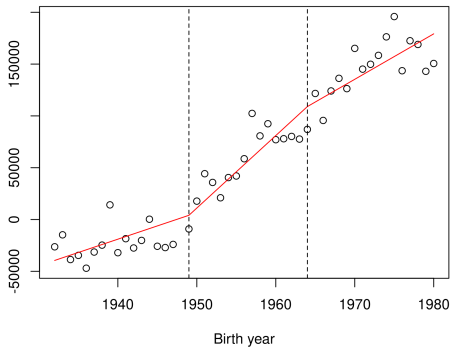
Specifically, this piecewise regression replaces $Measles_s \times Exposure_c$ with three terms:

$$Measles_s \times c, Measles_s \times \max(0, c - 1949), \text{and } Measles_s \times \max(0, c - 1964)$$

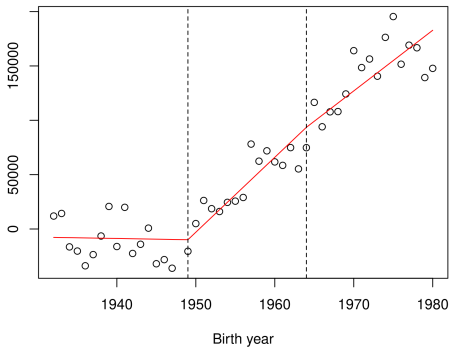
where c is birth year. The coefficients on the second and third terms represent the change in slope at the kink points, so we can interpret the p-values directly as tests of a trend break. Note that this is not the same as testing whether the slope of the trends is 0 in the before and after periods, as predicted by a true vaccine effect. I restrict the sample to 1932-1980; note that birthyear 1948 is omitted in the event study.

I overlay the piecewise regression on top of the event study coefficients, scaling the piecewise fit to be centered vertically at the average of the coefficients (by adding the average difference between the piecewise fit and the coefficients). The results are plotted in Figure 1. We can see that the trends follow the same path after 1964 (apart from hours worked). The p-values for the 1949 trend break are 0.00, 0.00, 0.00, 0.00, 0.08, and 0.03. The p-values for the 1964 trend break are 0.10, 0.46, 0.10, 0.06, 0.17, and 0.00. So except for hours worked, we do not reject the null hypothesis of no change in trend in 1964 (at the 5% level). Note that a true vaccine effect requires an even stronger result than a trend break, namely, a slope of 0 over 1964-1980.

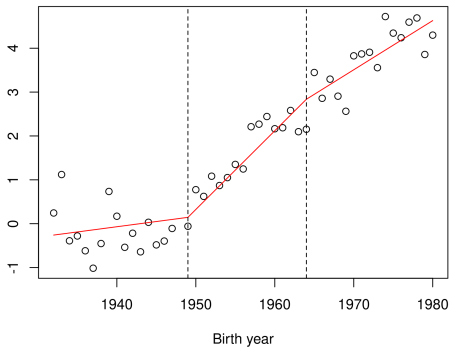
Figure 1: Piecewise regressions



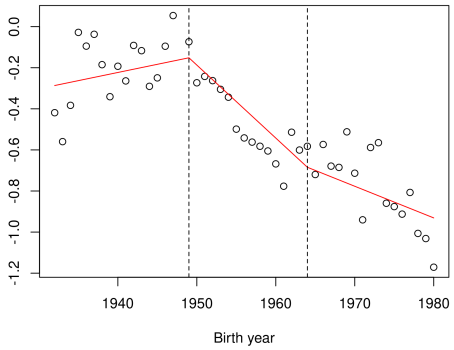
(a) Income



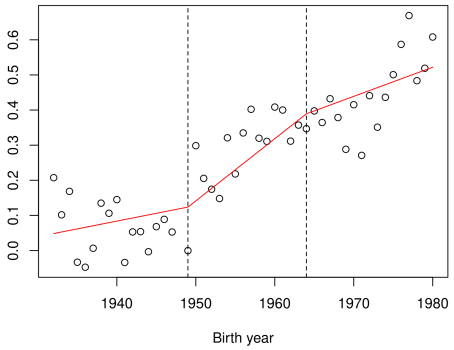
(b) Income (nonzero)



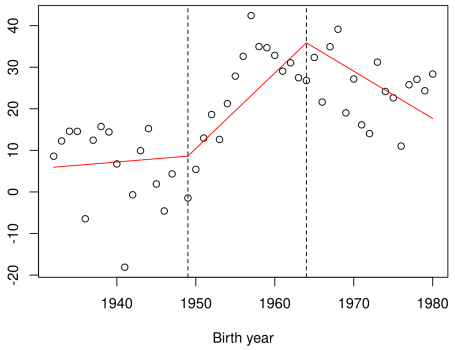
(c) Log income



(d) Poverty



(e) Employed



(f) Hours worked

Note: Data from 1960-1990 censuses and 2000-2017 ACS. The sample is restricted to native-born whites and Blacks aged 25-60, and birthyears 1932-1980. *Measles* is the state-level average measles rate over 1952-1962. Fixed effects: survey year, age×Black×female, state-of-birth×Black×female, and division × birthyear; note that the interaction terms contain all component terms. Income is in 2018 dollars. Standard errors are clustered by birthyear × state-of-birth.

3 Heterogeneity

Atwood does not test for heterogeneity in treatment effects along important dimensions, such as race, sex, and region. I run subsample regressions for race-sex groups (Black men, Black women, white men, white women) and census regions (Northeast, Midwest, South, West). Note that a subsample regression is equivalent to interacting each variable with the group indicator using the full sample.

Table 1 shows the subsample regressions by race and sex. The effects on income are negative for Black men, and noisy for Black women. Poverty increases for Black men, with no effect for Black women. The effects for white women are larger than for white men. Figures 2 - 6 show the corresponding event studies. The pattern for Blacks is not very clear, while the coefficients for whites resemble the full sample graphs; this is consistent with Blacks being a smaller subgroup.

Table 2 runs subsample regressions by census region. The income results are somewhat inconsistent. For example, the effect in levels is larger in the Northeast compared to the Midwest, but for log income the order is reversed. In the South, level income increases but log income decreases. We also see that employment decreases in the Northeast and poverty increases in the South. This could be explained by limited variation in measles incidence within region, especially with division-birthyear fixed effects (there are 9 divisions and 4 regions).

3.1 Race and sex

Table 1: Subsample regressions: race and sex

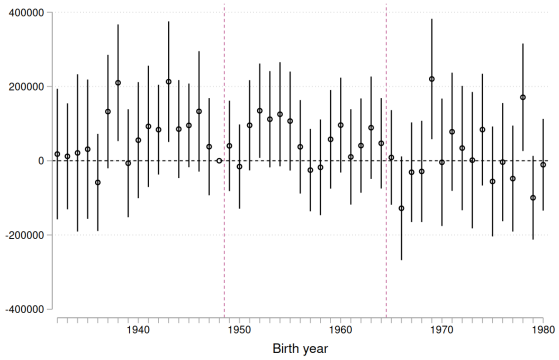
	(1) Income	(2) Income (>0)	(3) Log income	(4) Poverty	(5) Employed	(6) Hours worked
Panel A: Black men						
Measles × Exposure	-863.1	1286.9	-0.102***	0.0593***	-0.00515	-3.075***
	(989.9)	(1168.3)	(0.0368)	(0.0124)	(0.0101)	(0.674)
Observations	1558984	1125184	1125184	1409706	1156122	1333349
R^2	0.040	0.072	0.060	0.044	0.018	0.041
Panel B: Black women						
Measles × Exposure	5887.5***	7763.5***	0.00763	0.00724	-0.0178**	-4.259***
	(748.3)	(824.2)	(0.0330)	(0.0124)	(0.00862)	(0.519)
Observations	1841024	1277048	1277048	1820434	1289488	1576160
R^2	0.076	0.103	0.135	0.047	0.014	0.037
Panel C: White men						
Measles × Exposure	8769.9***	7107.5***	0.141***	-0.0504***	0.0157***	0.0294
	(880.8)	(956.2)	(0.0133)	(0.00343)	(0.00246)	(0.237)
Observations	13557609	11235661	11235661	13322155	12104275	11408711
R^2	0.042	0.066	0.064	0.015	0.006	0.035
Panel D: White women						
Measles × Exposure	7532.5***	6482.5***	0.273***	-0.0553***	0.0189***	2.291***
	(467.9)	(522.7)	(0.0167)	(0.00394)	(0.00228)	(0.295)
Observations	14007396	9439507	9439507	13934029	9448144	11769477
R^2	0.073	0.065	0.061	0.016	0.003	0.034

Standard errors in parentheses

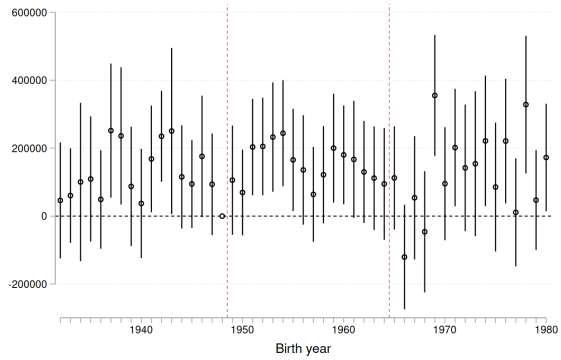
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Note: Data from 1960-1990 censuses and 2000-2017 ACS. The sample is restricted to native-born whites and Blacks aged 25-60. Each panel restricts the sample to a race-sex category. *Measles* is the average measles rate over 1952-1962. *Exposure* is the number of years that the vaccine is available to a cohort. Fixed effects: survey year, age, state-of-birth, and division × birthyear; note that the interaction terms contain all component terms. State-level averages are matched to individuals by state-of-birth. Income is in 2018 dollars. Standard errors are clustered by birthyear × state-of-birth.

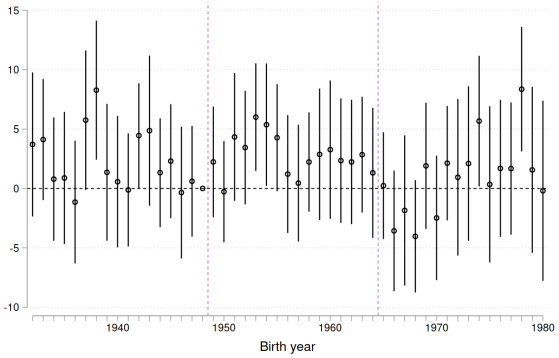
Figure 2: Event study: Black men



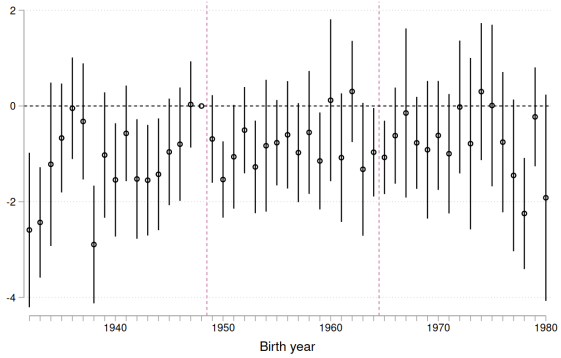
(a) Income



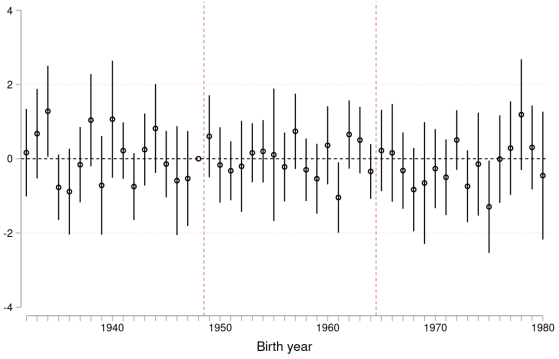
(b) Income (nonzero)



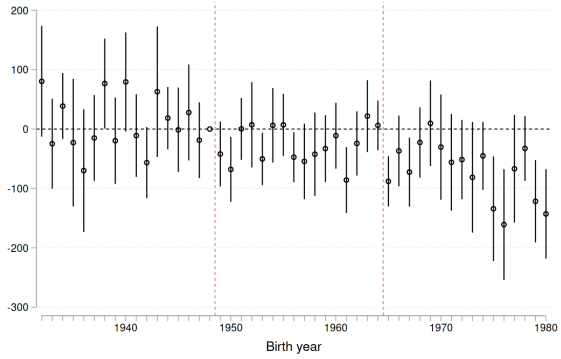
(c) Log income



(d) Poverty



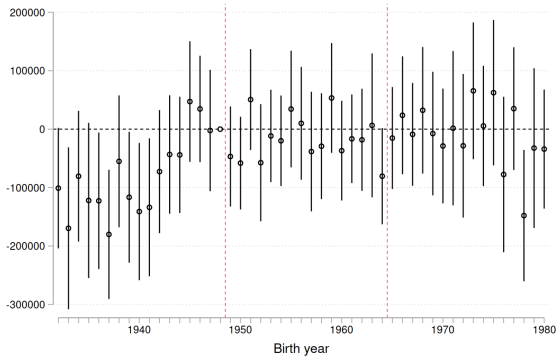
(e) Employed



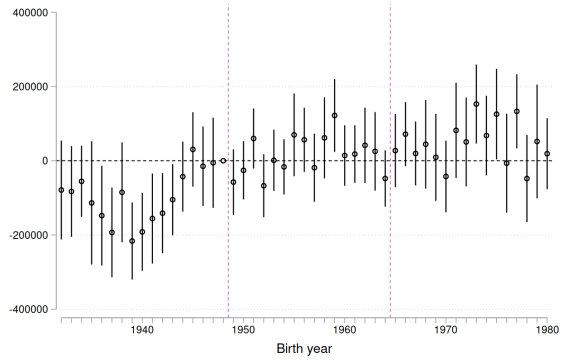
(f) Hours worked

Note: Data from 1960-1990 censuses and 2000-2017 ACS. The sample is restricted to native-born whites and Blacks aged 25-60, and birthyears 1932-1980. Birthyear 1948 is omitted. The red dashed lines indicate the 1949-1964 cohorts with increasing exposure to the vaccine. *Measles* is the state-level average measles rate over 1952-1962. Fixed effects: survey year, age, state-of-birth, and division × birthyear; note that the interaction terms contain all component terms. Income is in 2018 dollars. Standard errors are clustered by birthyear × state-of-birth.

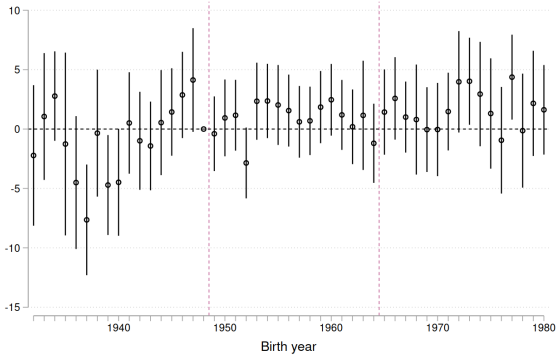
Figure 3: Event study: Black women



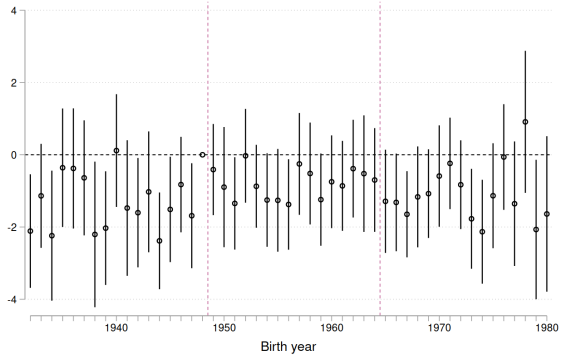
(a) Income



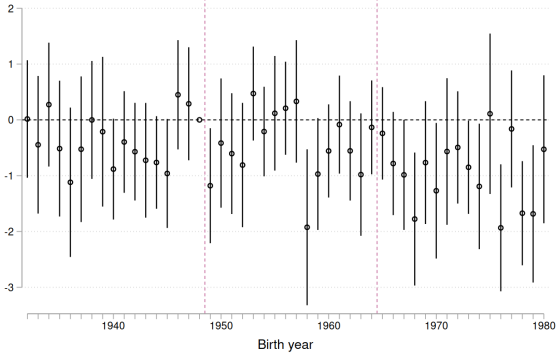
(b) Income (nonzero)



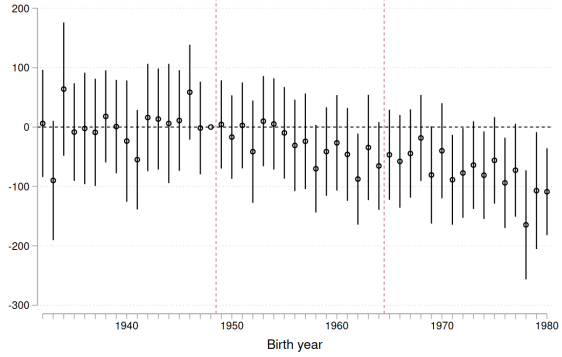
(c) Log income



(d) Poverty



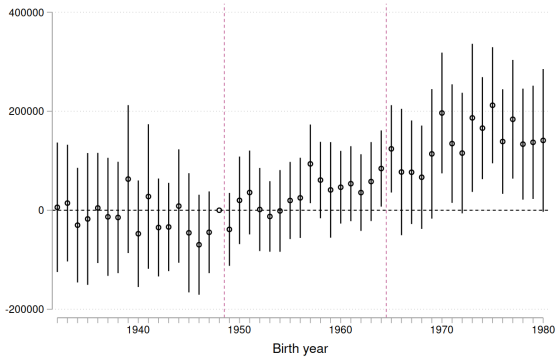
(e) Employed



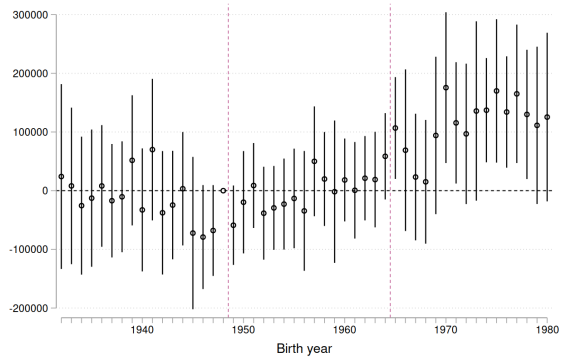
(f) Hours worked

Note: Data from 1960-1990 censuses and 2000-2017 ACS. The sample is restricted to native-born whites and Blacks aged 25-60, and birthyears 1932-1980. Birthyear 1948 is omitted. The red dashed lines indicate the 1949-1964 cohorts with increasing exposure to the vaccine. *Measles* is the state-level average measles rate over 1952-1962. Fixed effects: survey year, age, state-of-birth, and division × birthyear; note that the interaction terms contain all component terms. Income is in 2018 dollars. Standard errors are clustered by birthyear × state-of-birth.

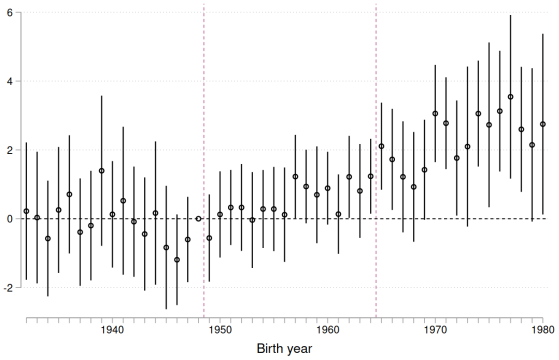
Figure 4: Event study: White men



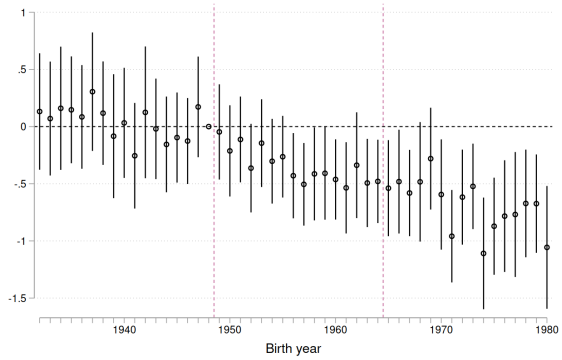
(a) Income



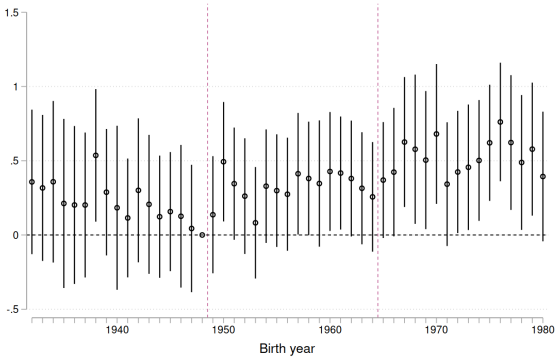
(b) Income (nonzero)



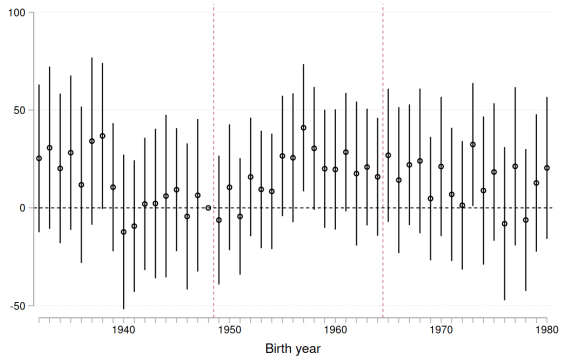
(c) Log income



(d) Poverty



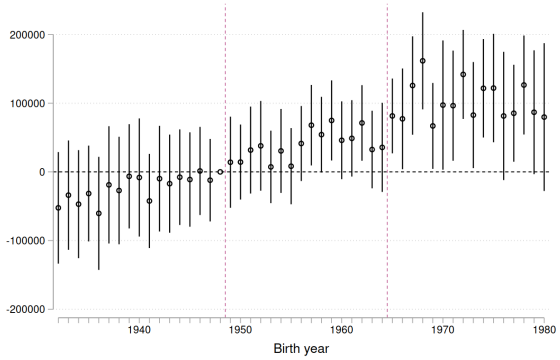
(e) Employed



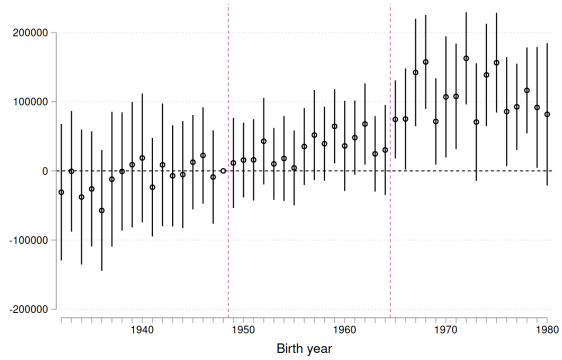
(f) Hours worked

Note: Data from 1960-1990 censuses and 2000-2017 ACS. The sample is restricted to native-born whites and Blacks aged 25-60, and birthyears 1932-1980. Birthyear 1948 is omitted. The red dashed lines indicate the 1949-1964 cohorts with increasing exposure to the vaccine. *Measles* is the state-level average measles rate over 1952-1962. Fixed effects: survey year, age, state-of-birth, and division × birthyear; note that the interaction terms contain all component terms. Income is in 2018 dollars. Standard errors are clustered by birthyear × state-of-birth.

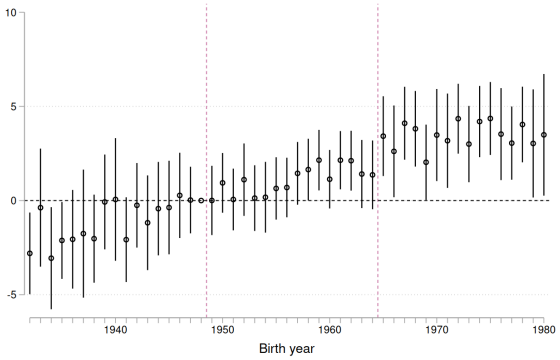
Figure 5: Event study: White women



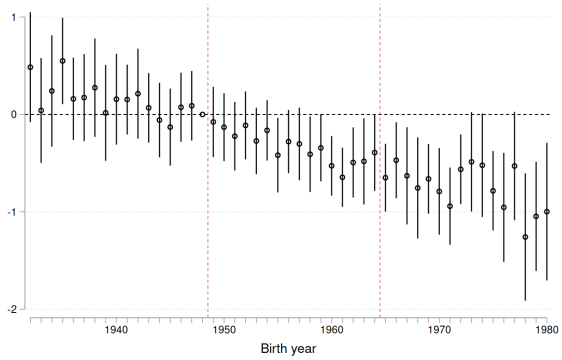
(a) Income



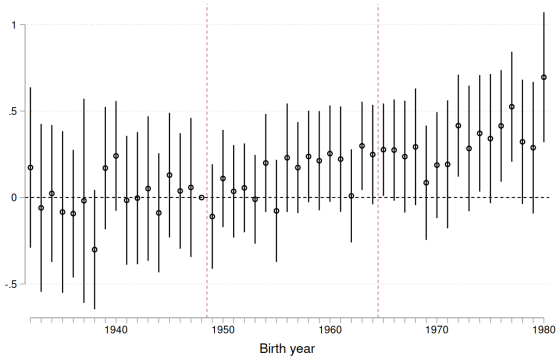
(b) Income (nonzero)



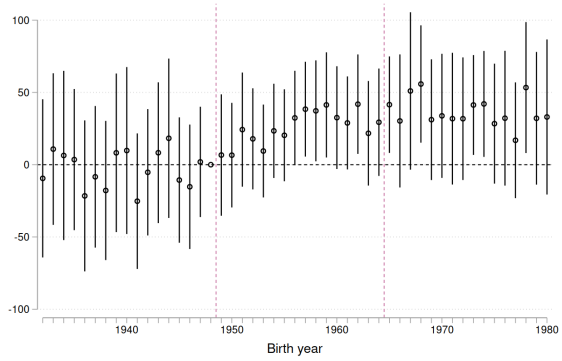
(c) Log income



(d) Poverty



(e) Employed



(f) Hours worked

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3.2 Region

Table 2: Subsample regressions: region

	(1) Income	(2) Income (>0)	(3) Log income	(4) Poverty	(5) Employed	(6) Hours worked
Panel A: Northeast						
Measles × Exposure	25365.5***	28936.8***	0.220***	-0.00309	-0.0676***	-4.022***
	(2012.4)	(2499.6)	(0.0295)	(0.00808)	(0.00453)	(0.427)
Observations	7378346	5602516	5602516	7284434	5809782	6092134
R^2	0.123	0.126	0.143	0.023	0.008	0.122
Panel B: Midwest						
Measles × Exposure	10710.6***	7483.3***	0.358***	-0.0837***	0.0421***	3.137***
	(630.3)	(781.4)	(0.0159)	(0.00371)	(0.00227)	(0.212)
Observations	8964574	6782105	6782105	8854610	7085950	7524545
R^2	0.119	0.120	0.135	0.027	0.014	0.123
Panel C: South						
Measles × Exposure	5277.8***	3633.4***	-0.0601***	0.0349***	0.0195***	0.191
	(730.5)	(773.3)	(0.0206)	(0.00767)	(0.00336)	(0.428)
Observations	10566753	7658141	7658141	10360776	7935666	8787374
R^2	0.121	0.136	0.146	0.065	0.014	0.103
Panel D: West						
Measles × Exposure	1013.8	215.7	0.148***	0.0137	0.0855***	0.981
	(1700.8)	(2120.9)	(0.0398)	(0.0107)	(0.00749)	(0.734)
Observations	4055341	3034644	3034644	3986505	3166636	3683644
R^2	0.100	0.118	0.121	0.015	0.009	0.102

Standard errors in parentheses

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Note: Data from 1960-1990 censuses and 2000-2017 ACS. The sample is restricted to native-born whites and Blacks aged 25-60. Each panel restricts the sample to a census region. *Measles* is the average measles rate over 1952-1962. *Exposure* is the number of years that the vaccine is available to a cohort. Fixed effects: survey year, birthyear, age×Black×female, state-of-birth×Black×female, and census division × birthyear; note that the interaction terms contain all component terms. State-level averages are matched to individuals by state-of-birth. Income is in 2018 dollars. Standard errors are clustered by birthyear × state-of-birth.

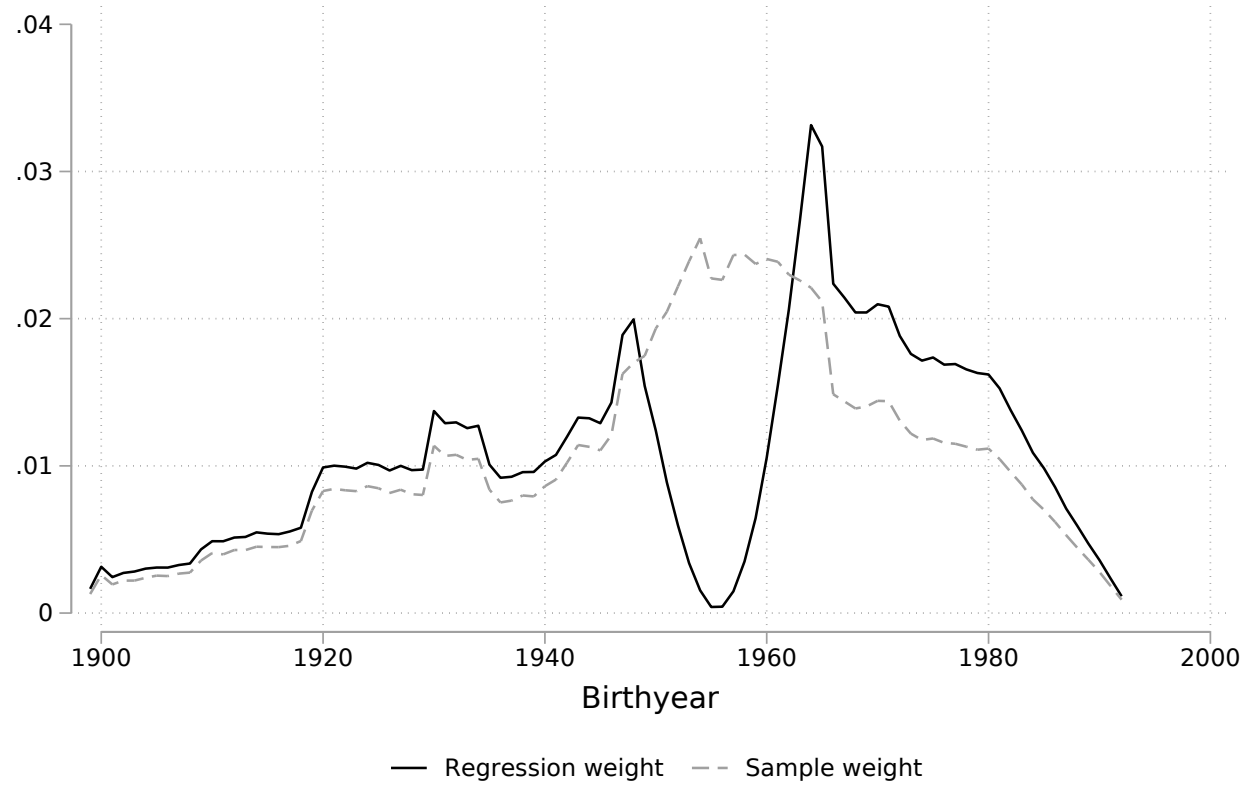
4 Regression weights

I calculate regression weights by regressing the treatment variable on the fixed effects, saving the residuals, squaring them, and normalizing by the total.

Wisconsin has 0.36 of the weight and Lousiana has 0.09, which matches those states having the largest and smallest reported measles incidence (following the intuition that more extreme values get more weight). For comparison, the state-level average is 0.02.

Below I plot the regression weight by birthyear. We see that the weight drops between 1949 to 1964, with the smallest weight in the middle of the treatment window (1955). This again seems to be due to extreme values getting more weight (recall that the vaccine exposure variable is defined to increase from 0 to 16 from 1948 to 1964). This also explains why Atwood finds similar results when dropping birthyears 1949-1963 (Table 3, no and full exposure only), because those observations contribute little to the estimate.

Figure 6: Regression weight: birthyear



5 Plotting raw data

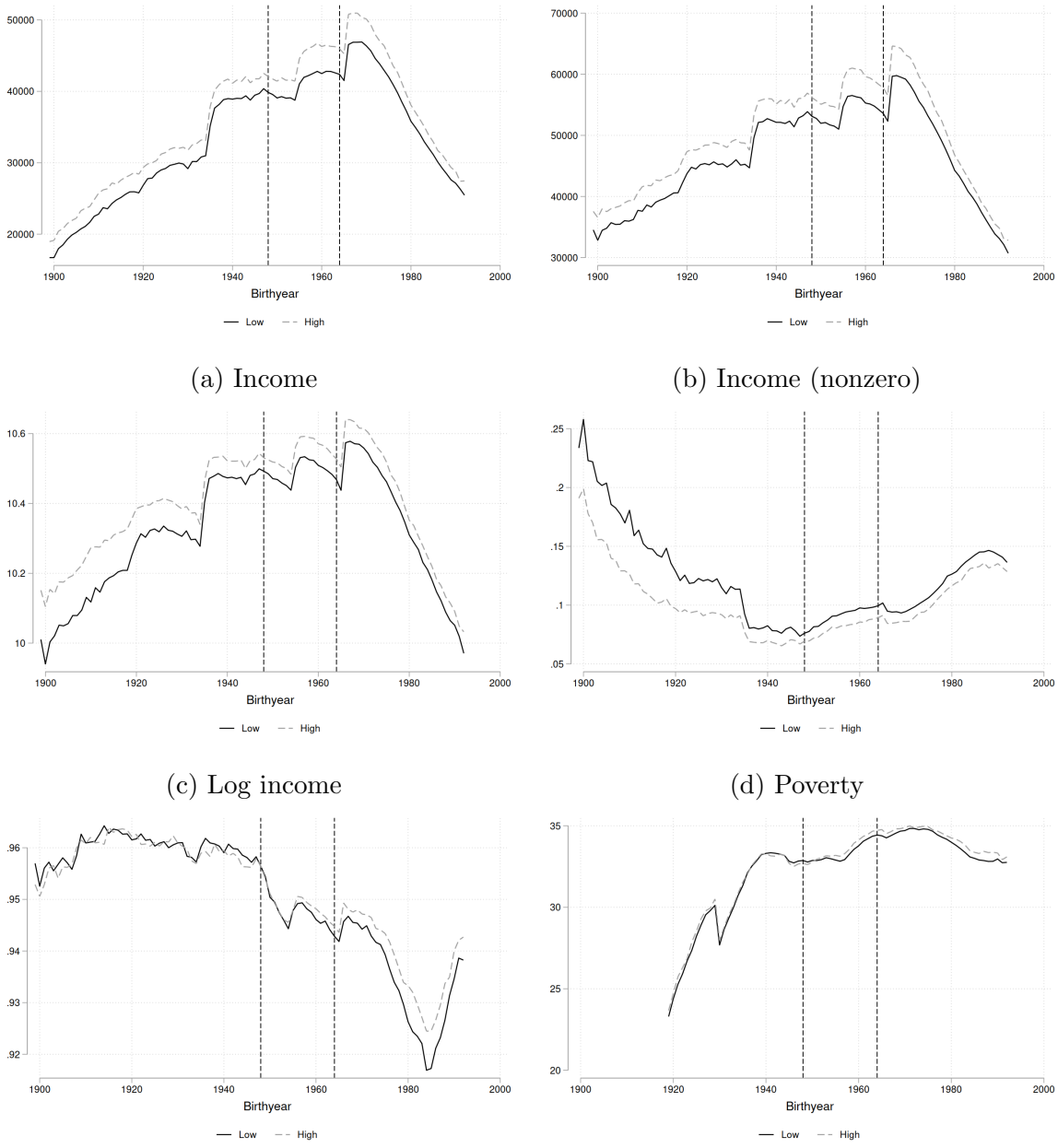
I compare average economic outcomes by birthyear, for above- vs below-median measles states. In Figure 7 we see that people born in high-measles states have higher incomes as adults than people born in low-measles states. This is surprising, since we'd expect the high-measles states to be poorer.

In Figure 8 I plot the difference. The gap between High and Low states is constant for income, nonzero income, and employment. But the gap is shrinking for log income, poverty, and hours worked, indicating differential trends before the vaccine (in the raw data, not conditioning on fixed effects). For income, the gap increases rapidly during the vaccine exposure years, then shrinks to be smaller than the initial gap. This could be explained by the income distribution being compressed for younger workers.

5.1 Age composition

As shown in Figure 9, there are spikes in average age, due to the sample being restricted to ages [25,60] and composition effects from combining different census years. For example, the youngest age in birthyear 1965 is 25 (from the 1990 census), while the youngest age in birthyear 1966 is 34 (from the 2000 census), since age 24 (from the 1990 census) is excluded from the sample. The average age is decreasing with birthyear because age = survey year - birthyear.

Figure 7: High vs Low measles



(a) Income

(b) Income (nonzero)

(c) Log income

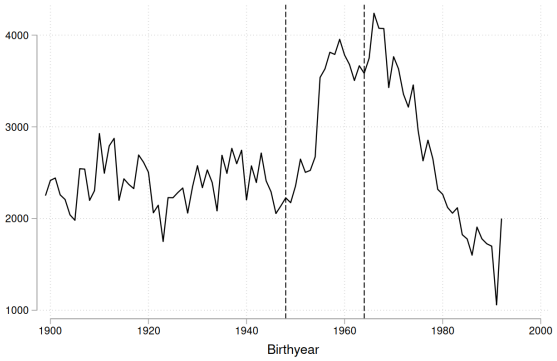
(d) Poverty

(e) Employed

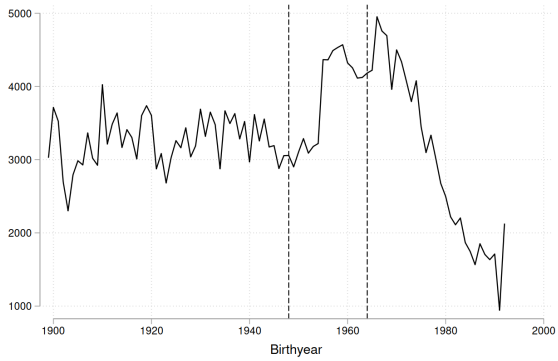
(f) Hours worked

Note: Average outcome by birthyear, for states with above- and below-median pre-vaccine average reported measles rates. The spikes are caused by average age jumping when different census years are combined.

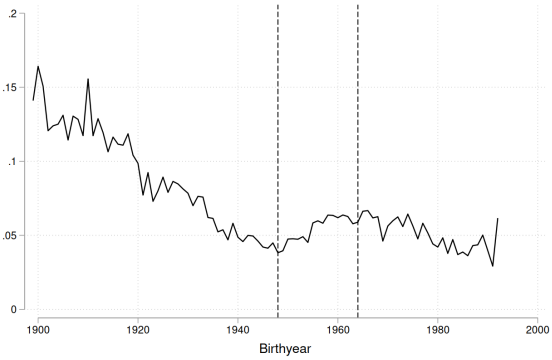
Figure 8: High vs Low measles: difference



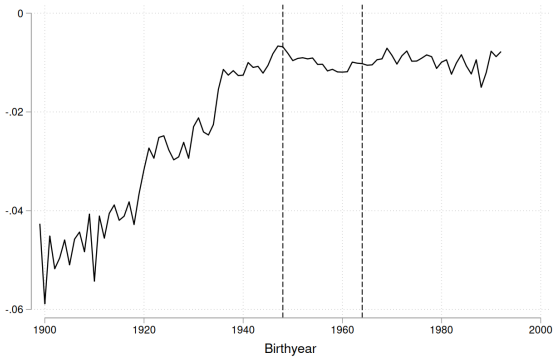
(a) Income



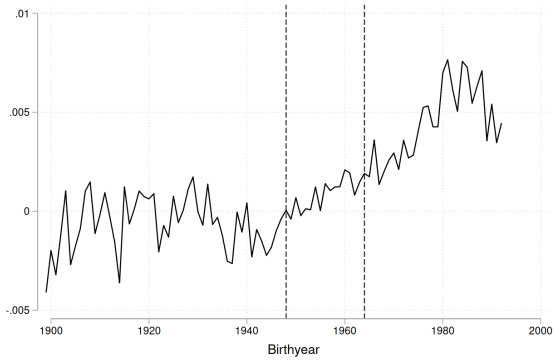
(b) Income (nonzero)



(c) Log income



(d) Poverty

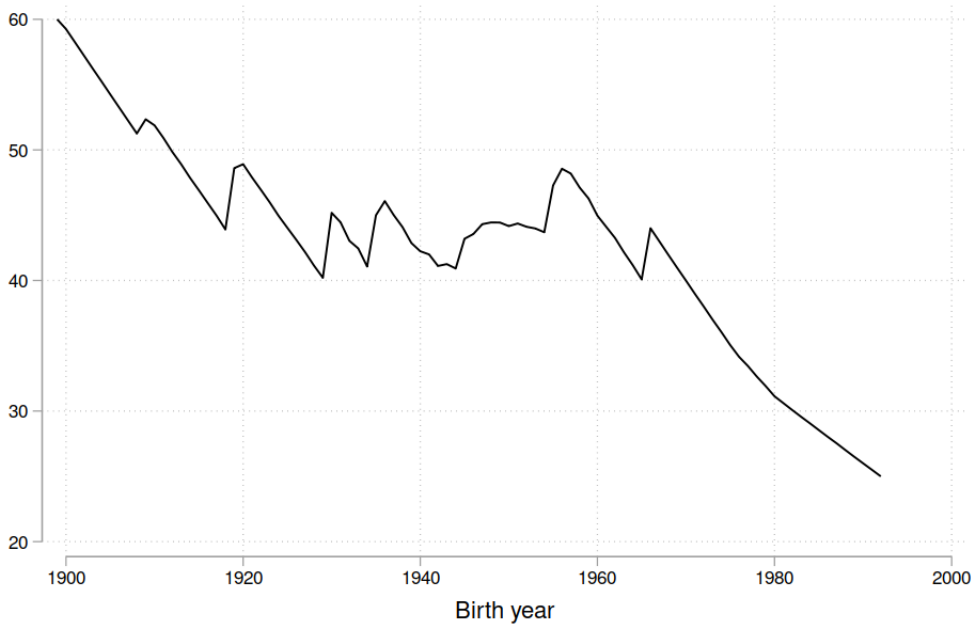


(e) Employed

(f) Hours worked

Note: Difference between above- and below-median measles states in Figure 7

Figure 9: Average age by birthyear



6 Contemporary disease incidence

In Figures 3 and 4, Atwood runs an event study at the state-year level to test for the effect of the measles vaccine on the reported incidence of measles and other diseases (pertussis, chicken pox, mumps, and rubella) that are plausibly worsened by measles' "immune amnesia" effect, where contracting the measles virus wipes an individual's immune system and makes them vulnerable to other diseases. Atwood frames this event study as testing for pretrends, but note that the absence of pretrends in reported disease incidence does not imply the absence of pretrends in economic outcomes.

The regression equation for the disease event study is:

$$y_{st} = \sum_{j=1952, j\neq 1962}^{1975} \beta_j \text{Measles}_s \times \mathbb{1}\{t = j\} + \delta_s + \gamma_t + \theta X_{st} + \varepsilon_{st}. \tag{1}$$

Here y_{st} is the reported disease rate in state s and year t, and $Measles_s$ is the state-level average reported measles rate over 1952-1962, normalized by the under-18 population.[1] Controls include state and year fixed effects, as well as time-varying under-18 population.

I make several changes to Atwood's specification. First, I use calendar time on the x-axis instead of event time, since there is only one event. Second, I use 1962, one year before the vaccine was introduced, as the omitted year; note that Atwood omitted 1963.[2] Third, I run a fully-saturated event study, with one coefficient for each year; note that Atwood estimates coefficients for 1958-1975 (binning 1952-1958 for measles, and 1956-1958 for the other diseases), and plots the coefficients for 1959-1974. I plot the coefficients for the full sample: 1952-1975 (measles) and 1956-1975 (other diseases). Fourth, Atwood's Figure 4 winsorizes the data for pertussis, chicken pox, mumps, and rubella, without providing any justification. I do not winsorize.

Figure 10 shows the results. The measles event study is similar to the original; the post-vaccine coefficient of -1 indicates that the virus was eradicated country-wide.[3] The event studies for pertussis and chicken pox show a decline after 1963. But when extending the sample back to 1956, this decline appears (at least partly) to be following a general trend, instead of representing an effect of the measles vaccine. The mumps event study shows a sustained drop in

[1] Atwood calculates this average over 1952-1963. Since the vaccine was introduced in 1963, I omit this year to avoid post-treatment bias. I also correct a few transcription errors in the population data.

[2] Figures 3 and 4 in Atwood (2022) use 1963 as the omitted year, but incorrectly label 1964 as 0 years relative to measles vaccine availability.

[3] Note that eradication is consistent with a post-vaccine coefficient of -1, regardless of whether reporting rates vary across states. Since actual cases go to zero everywhere, states with reported pre-vaccine rates that are higher by 1 per 100,000 (due to higher reporting rates) will have post-vaccine rates that are lower by 1 (assuming that a rate of zero is measured without error).

reported cases starting in 1968, but this coincides with the introduction of the mumps vaccine in 1967. When using unwinsorized rubella data, we see large spikes on the graph corresponding to the rubella outbreak in 1958 and epidemic in 1964. Moreover, the coefficients become negative and statistically significant in 1969, coinciding with the licensing of the rubella vaccine.

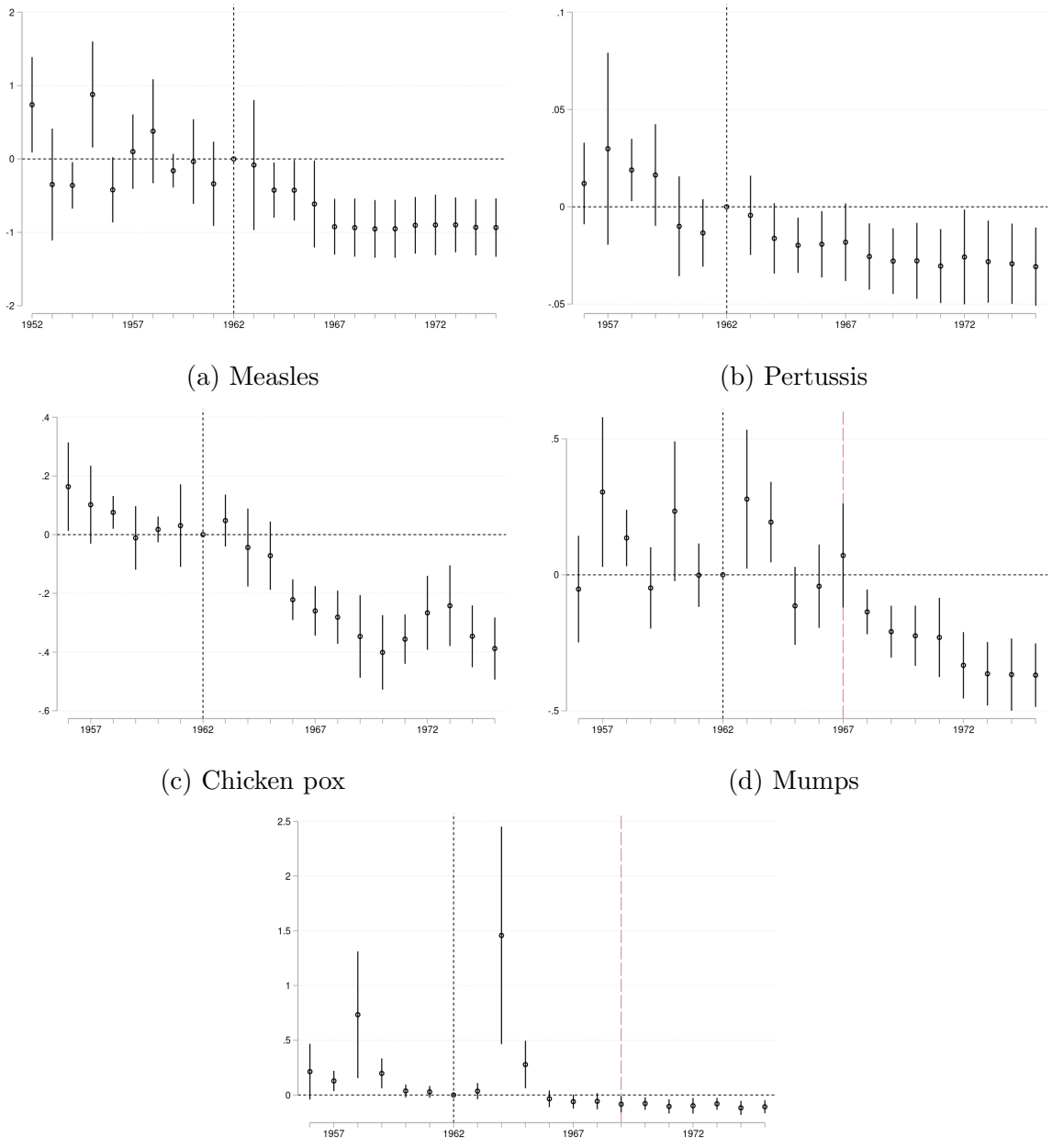
Why would the mumps and rubella vaccines, and the rubella epidemic, have differential effects by pre-vaccine reported measles prevalence? One explanation is that states with more reporting capacity (from, for example, better health infrastructure) report a larger fraction of cases for all diseases. States with higher reported measles incidence also have higher reported rates of mumps and rubella, so the mumps and rubella vaccines decrease measured rates more in those states. Similarly, states with higher reporting capacity would report more cases during an epidemic, leading to higher rubella rates in those states in 1964. Consistent with this explanation, there is a strong cross-sectional correlation between measles incidence and other disease incidence before the vaccine.[4]

Another explanation is that states with worse initial health have more severe cases of disease (even though actual incidence is the same), and more severe cases are more likely to be reported. In this case, low-health states would (1) report more measles cases; (2) report more rubella cases during an epidemic; and (3) see a bigger drop in mumps and rubella cases after the vaccine.

Hence, while Atwood takes Figure 4 to support the immune amnesia hypothesis, the results are also consistent with differences in reporting capacity or initial health levels. So the evidence from the disease event studies does not resolve the question of whether geographic variation in reported measles incidence represents differences in actual disease incidence, reporting capacity, or differential impact of the same disease incidence. The identifying variation used by Atwood remains unclear.

[4]The pre-vaccine (1956-1962) pairwise correlations between average reported measles incidence and average reported rates for the other diseases are: 0.55 (pertussis); 0.89 (mumps); 0.89 (chickenpox); 0.71 (rubella).

Figure 10: Event study: disease prevalence



(a) Measles

(b) Pertussis

(c) Chicken pox

(d) Mumps

(e) Rubella

Note: Event study coefficients from Equation 1. The treatment variable is pre-vaccine average reported measles incidence. The outcome variable is the reported disease rate, normalized by the under-18 population. The vertical line denotes the omitted year (1962). Dates of vaccine introductions: measles 1963; mumps 1967; rubella 1969. There was a rubella outbreak in 1958 and epidemic in 1964. Sample sizes: 1176 (measles); 976 (pertussis); 785 (chicken pox); 820 (mumps); 788 (rubella). Sample: 1952-1975 (measles); 1956-1975 (other diseases). Standard errors are clustered at the state level.

7 Placebo test: other diseases

Given the strong correlation between measles and mumps, chicken pox, and rubella, we should expect to find the same results when calculating pre-vaccine disease incidence using these diseases. Table 3 shows that this is the case. The results for pertussis have the opposite sign for income and poverty; recall that pertussis had the smallest correlation with measles. As discussed above, these results are consistent with multiple hypotheses: reporting capacity, initial health, or immune amnesia.

Table 3: Placebo test: other diseases

	(1) Income	(2) Income (>0)	(3) Log income	(4) Poverty	(5) Employed	(6) Hours worked
Panel A: Pertussis						
Pertussis × Exposure	-7657.9	-42791.2***	-0.462*	0.178**	0.384***	12.90***
	(8824.8)	(10140.8)	(0.253)	(0.0831)	(0.0381)	(4.658)
Observations	31413087	23413673	23413673	30929087	24353429	26455376
R^2	0.123	0.131	0.144	0.049	0.012	0.114
Panel B: Mumps						
Mumps × Exposure	10601.8***	6065.2***	0.444***	-0.105***	0.0734***	5.546***
	(1075.3)	(1320.8)	(0.0230)	(0.00538)	(0.00328)	(0.296)
Observations	23606951	17685064	17685064	23269977	18404650	19937093
R^2	0.121	0.127	0.140	0.040	0.012	0.117
Panel C: Chicken pox						
Chicken pox × Exposure	11816.2***	7942.6***	0.342***	-0.0731***	0.0469***	4.283***
	(622.1)	(790.2)	(0.0164)	(0.00408)	(0.00235)	(0.242)
Observations	22517044	16858148	16858148	22201020	17529345	18829093
R^2	0.125	0.130	0.145	0.045	0.012	0.120
Panel D: Rubella						
Rubella × Exposure	18707.9***	11297.9***	0.745***	-0.186***	0.0979***	8.382***
	(1901.8)	(2323.9)	(0.0391)	(0.00875)	(0.00518)	(0.509)
Observations	22168401	16600567	16600567	21854452	17246630	18561728
R^2	0.125	0.130	0.145	0.046	0.012	0.118

Standard errors in parentheses

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Note: Data from 1960-1990 censuses and 2000-2017 ACS. The sample is restricted to native-born whites and Blacks aged 25-60. Each panel restricts the sample to a race-sex category. Disease incidence is calculated as the average rate over 1956-1962. *Exposure* is the number of years that the vaccine is available to a cohort. Fixed effects: survey year, birthyear, age×Black×female, state-of-birth×Black×female, and census division × birthyear; note that the interaction terms contain all component terms. State-level averages are matched to individuals by state-of-birth. Income is in 2018 dollars. Standard errors are clustered by birthyear × state-of-birth.

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