Reducing Inequality While Improving Health: Long-Run Impacts from the Onset of Universal Health Insurance in Japan*

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Abstract

Exploiting the onset of universal insurance in Japan, this paper estimates the long-run impacts of universal insurance on health, human capital, and economic outcomes. Between 1956 and 1961, prefectures in Japan expanded community-based insurance and implemented universal insurance in 1961. The expansions resulted in large variations in the exposure to universal insurance across birth cohorts and prefectures. Exploiting the variations, I show that exposure led to substantial health benefits in prime age, reducing cancer mortality for men and the prevalence of chronic conditions for women. Exposure further increased high school graduation rates for both gender and increased college graduation specifically for women. Consistent with the education gains, full-time employment increased for women whereas home production shifted to men. Thus, in addition to health gains, universal insurance led to greater gender equality with increases in women's education and economic opportunities.

Keywords: universal insurance, long-run impacts, health, gender inequality, human capital, employment, earnings, Japan

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1 Introduction

In the second half of the twentieth century, a growing number of countries expanded public health insurance programs to achieve universal insurance for citizens. UK, Japan, and several Nordic countries such as Sweden and Norway were among the first to implement universal insurance in the 1950s-1960s, and expansion later gained momentum in Latin America, Africa, and Asia-Pacific countries in the 1990s and the 2000s (Light 2003; Savedoff *et al.* 2012; Wagstaff *et al.* 2016). In 2015, universal health coverage was included in the United Nation's Sustainable Development Goals for 2030 (Desa *et al.*, 2016). While the short-term impacts of insurance have been well measured and documented (Escobar *et al.* 2011; Sommers *et al.* 2017), the long-run impacts of universal insurance are not directly observable until decades after the original reform.

This paper examines the long-run impacts of Japan's onset of universal insurance in 1961. Announced in 1956, the reform required all prefectures to expand public, community-based insurance and implement universal insurance by April 1961. Prior to the reform, the primary source of insurance was industry union insurance for workers and families, and uninsurance was high among non-union workers, the unemployed, and the elderly. Following the onset of universal insurance, the national insurance rate increased from 71% in 1955 to 100% in 1961, and greater coverage gains occurred in prefectures with initially low insurance rates.

I exploit the rapid expansion during the reform period to study the impacts of universal insurance on health and economic outcomes over the long run. I focus on the 1956-1965 cohorts and measure their exposure to universal insurance using the average insurance rate from in-utero to age 5. Of these cohorts, exposure was greater for those born closer to 1961, the onset year of universal insurance, and increased more in prefectures with lower pre-reform insurance rates. I then obtain the health and economic outcomes of these cohorts in prime age (41-50) from administrative records and current population surveys. Consistent with the literature on early-childhood investments, for more exposed cohorts,

one would expect greater increases in health and economic self-sufficiency in prime age.

While the national onset of universal insurance was set for 1961, several prefectures implemented universal insurance ahead of the national timeline in 1959. To address the concern that early-expansion prefectures could also have favorable fiscal, industrial, or demographic conditions that affect long-run outcomes, I construct a simulated exposure measure using a linear interpolation of insurance rates from the 1955 level to universal insurance during the 1956-1961 expansion. Unlike the endogenous exposure, simulated exposure is determined solely from two variations induced by policy: the national timeline to achieve universal insurance by 1961, and the size of expansion due to prefectures' pre-reform insurance rates. To the extent that confounding factors do not follow the same variations across cohorts and prefectures as induced by the insurance reform, instrumenting endogenous exposure with the simulated measure can address biases from alternative drivers of expansion.

To measure long-run outcomes, I obtain administrative survey records from the Ministry of Health, Labour and Welfare in Japan. For mortality, I use the universe of death certificates to construct all-cause and cause-specific mortality rates by year, age, and prefecture, and I regress the mortality rates on simulated exposure across cohorts and prefectures in the empirical analysis. To measure the disease conditions of individuals, I use the Comprehensive Survey of Living Conditions and specifically the health questionnaire which reports a range of disease conditions diagnosed in hospital visits. For education, employment, and earnings, I use the Employment Status Survey and link records of individuals to those of the spouse's to understand marital sorting and the division of labor in households.

I find that exposure to universal insurance had significant impacts on health in prime age. For men, gaining a ten percentage point exposure reduced mortality by 1.1 per 100,000 individuals, and over 90% of the reduction was from cancer-related deaths, the leading cause of death in prime age. For women, gaining a ten percentage point exposure

reduced the prevalence of having any chronic condition by 0.36 percentage points, or by 6.3% below the mean. This effect was driven by lower prevalence rates of diabetes conditions. However, exposure did not significantly impact women's mortality in prime age.

In addition to health, universal insurance further impacted education attainment and labor force participation, with differential effects by gender. Gaining a ten percentage point exposure increased high school graduation rates by 1.3 percentage points for both gender and further increased college graduation by 1.1 percentage points specifically for women. This effect reduced the gender gap in college education by 4.5%. Consistent with the education gains, exposure increased women's probability of marrying a college-educated spouse by 4.3% and increased her full-time employment by 3.6%. Although labor supply and earnings did not increase with exposure at the household level, the increase in women's employment shifted home production to men and reduced the earnings gap by 8.3% between spouses. These results are robust to including prefecture- and cohort-level trends capturing long-run shifts in the societal norm towards gender.

Taken together, universal insurance not only improved health in prime age, but further improved human capital and the gender equality in economic participation and earnings. The differential increase in women's college education, in particular, had further impacts on marital sorting and the division of labor in households, ultimately increasing women's economic resources relative to men. These results suggest that universal insurance could contribute to an inclusive and equitable society with increased investments in less advantaged populations.

The long-run impacts in Japan relate to a large literature showing the health and human capital impacts of insurance. In high-income countries such as the US, childhood exposure to public insurance has been linked to a variety of outcomes including lower disease burdens (Boudreaux *et al.* 2016; Wherry *et al.* 2018; Miller and Wherry 2019;), lower crime rates (Hendrix and Stock, 2022), and higher college enrollment and earnings

as adults (Cohodes *et al.* 2016; Brown *et al.* 2020). In developing countries, health interventions such as de-worming and neonatal care to at-risk newborns have lasting impacts on health, education, and labor market outcomes (Miguel and Kremer 2004; Baird *et al.* 2016; Bharadwaj *et al.* 2013). This paper contributes to the evidence exploiting the onset of universal insurance which affected the entire uninsured population rather than specific subgroups based on means-tested criteria or health outcomes. Universal insurance thus represents a larger investment shock than targeted interventions with potentially broader implications for inequalities between population groups. The increase in women's education and economic inclusion, in particular, supports the notion that universal insurance could advance gender equality in health and economic prosperity across the developing world (Remme *et al.*, 2020).

The remainder of the paper is organized as follows. Section 2 introduces the historic onset of universal insurance in Japan and data measuring health, human capital, and employment outcomes in prime age. Section 3 introduces the simulated exposure measure to isolate exogenous variations of the reform and to identify the long-run impacts of exposure. Section 4 shows the estimation results and conducts robustness checks. Section 5 discusses the findings and potential mechanisms. Section 6 concludes.

2 Background and Data

2.1 Japan's Universal Insurance Reform

In the 1950s, the primary source of health insurance in Japan was industry union insurance provided to workers and dependent families. Those without union insurance may obtain community-based insurance from municipality governments, but eligibility differed across municipalities with many municipalities lacking coverage for the elderly, the unemployed, and non-union workers. These individuals would have to borrow from family members to cover the medical expenses in the event of illness. As the plight and inequality facing the

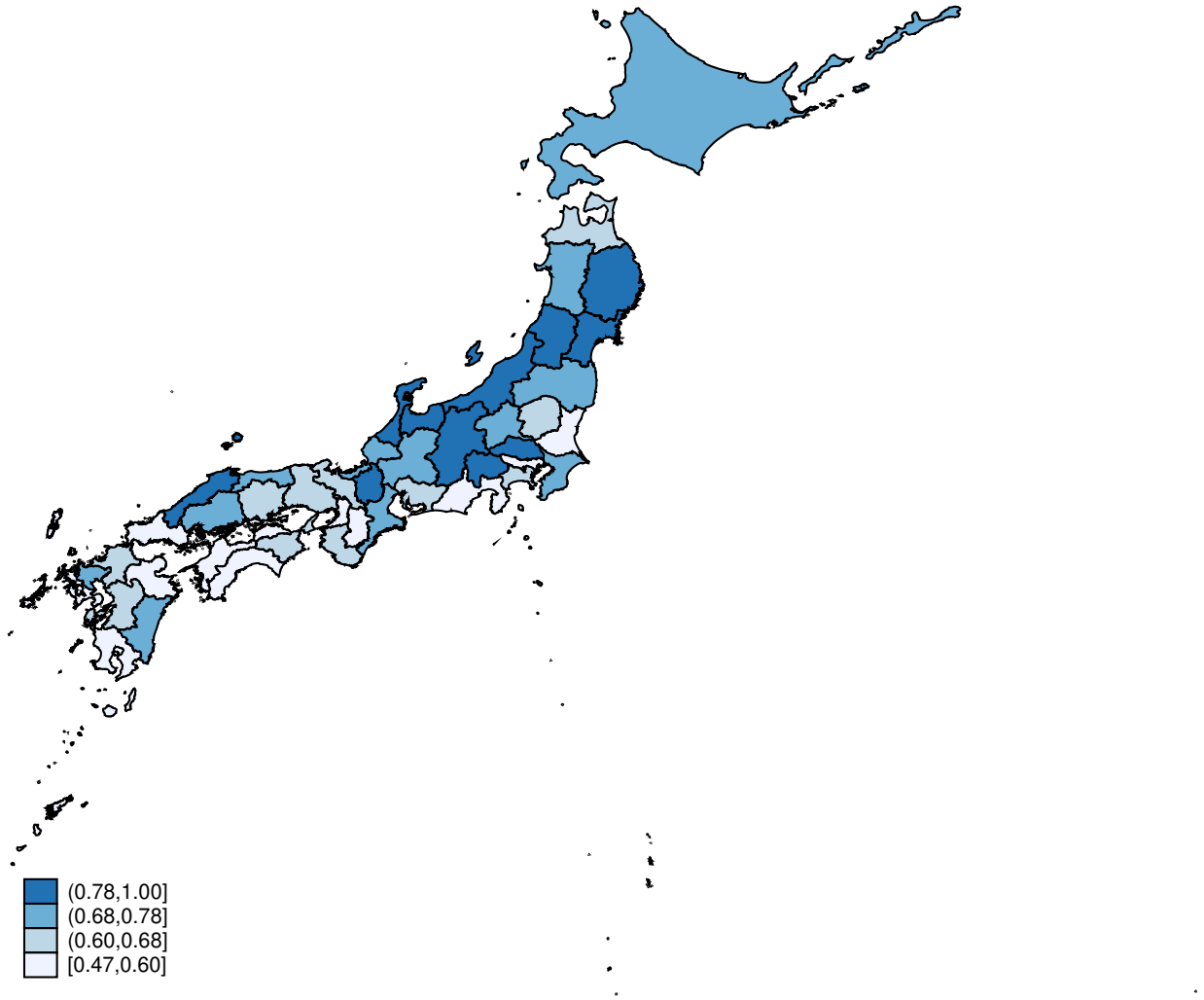
uninsured became a social issue in the early 1950s, reforming health insurance to achieve universal insurance gained popularity in the policy realm (Shimazaki, 2013).

In January 1956, Prime Minister Ichiro Hatoyama announced the plan to cover the entire Japanese population with universal insurance. Towards this goal, community-based insurance was expanded to enroll all residents without private insurance. In 1957, the Ministry of Health and Welfare launched a four-year plan and the timeline to achieve universal insurance by April 1961. The expanded community-based insurance would cover the same set of services as private insurance, and the central government would subsidize municipalities for 20% of the benefit payments.

Figure 1 shows the pre-reform insurance rate across prefectures in 1955. The median prefecture had an insurance rate of 68%. In the least insured prefectures such as Kagoshima and Kochi, less than 50% of the population had insurance. In Osaka, Shizuoka, Yamaguchi, and Tokyo, less than 60% had insurance. These prefectures saw substantial coverage expansions during the reform period from 1956 to 1961. In contrast, prefectures such as Niigata, Shiga, Iwate, and Yamagata already had near universal insurance in 1955, and expansions were smaller in these prefectures.

Prefectures further differed in the speed of expansion towards universal insurance. Supplementary Figure A1 plots the growth of insurance rates across prefectures in 1955-1961. In Miyagi, Akita, and Tokushima, for instance, expansions picked up speed shortly after 1955 and universal insurance was already achieved by 1959. In contrast, expansion was initially slow in Kanagawa, Kyoto, and Osaka and accelerated only a few years before 1961. In Ehime, Kochi, and Fukuoka, insurance instead followed a linear increase over time. Thus, both the initial insurance rate and the growth rate over time contributed to the large variations in the expansion paths in 1956-1961.

Figure 1: Pre-reform insurance rates in prefectures in 1955



Notes: Figure plots the 1955 insurance rates across prefectures in Japan. Different color scales correspond to the inter-quartile ranges of insurance rates.

2.2 Data

I measure the long-run outcomes of the 1956-1965 cohorts using administrative records obtained from the Ministry of Health, Labour and Welfare in Japan. For mortality, I use the universe of death certificate records to calculate the mortality rate of disease conditions. Specifically, I count the number of deaths due to a disease condition by prefecture, year, and age, and divide the death count by the population in the prefecture-year-age to calculate the cause-specific mortality rate, expressed in the number of deaths per 10 thousand individuals.[1] Panel A of Table 1 summarizes the mortality statistics for the 1956-1965 cohorts in prime age. For both gender, cancer is the leading cause of death accounting for 24%-51% of the overall mortality in prime age. Cardiovascular disease, another major cause of death after cancer, is more concentrated in men with a much lower mortality rate.

I next use the Comprehensive Survey of Living Conditions and specifically the health questionnaire to measure the prevalence of disease conditions in prime age. The questionnaire is administered every three years and includes a detailed list of disease conditions diagnosed in hospital visits. I focus on common chronic conditions (diabetes, hypertension, obesity, and cardiovascular diseases) as well as more severe conditions such as stroke and cancer. Panel A of Table 1 summarizes the prevalence rates for the 1956-1965 cohorts. Hypertension and diabetes are by far the leading chronic conditions, affecting 1.7%-4.1% of the population in prime age, whereas obesity and cardiovascular diseases have much lower prevalence rates (around 0.3%). Due to the low prevalence rates, I examine metabolic conditions due to either diabetes or obesity, and examine circulatory conditions due to either hypertension or cardiovascular diseases in the empirical analysis, as in Miller and Wherry (2019). Severe conditions such as cancer and stroke also have fairly low prevalence rates (0.2%-0.5%) in prime age.

[1] Population counts come from official statistics published by the Statistics Bureau of Japan, available at https://www.e-stat.go.jp/stat-search/database?page=1&toukei=00200524&tstat=000000090001. More detailed statistics by prefecture-year-age-gender are used to calculate gender-specific mortality rates.

Table 1: Summary Statistics

	Full Sample			Men			Women		
	N	mean	s.e.	*N*	mean	s.e.	*N*	mean	s.e.
				Panel A: Health Outcomes					
Mortality (per 10,000 individuals)									
All-Cause	9,188	3.32	0.050	4,600	4.35	0.09	4,588	2.28	0.02
Cancer	9,188	1.11	0.013	4,600	1.05	0.02	4,588	1.17	0.01
Cardiovascular	9,188	0.40	0.013	4,600	0.63	0.02	4,588	0.17	0.01
Prevalence of Disease Conditions									
Hypertension (%)	262,033	4.05	0.064	128,197	4.99	0.080	133,836	3.13	0.12
Diabetes (%)	262,033	1.67	0.049	128,197	2.41	0.061	133,836	0.96	0.054
Obesity (%)	262,033	0.34	0.012	128,197	0.46	0.025	133,836	0.23	0.013
Cardiovascular (%)	262,033	0.33	0.016	128,197	0.54	0.022	133,836	0.14	0.016
Cancer (%)	262,033	0.50	0.020	128,197	0.17	0.010	133,836	0.81	0.041
Stroke (%)	262,033	0.24	0.016	128,197	0.33	0.025	133,836	0.16	0.017
				Panel B: Human Capital and Employment					
High School (%)	329,502	94.32	0.35	160,598	93.04	0.41	168,904	95.61	0.31
College Degree (%)	329,502	26.01	1.90	160,598	38.30	2.27	168,904	13.61	1.42
College-Educated Spouse (%)	331,397	18.75	1.23	161,294	9.39	0.91	170,103	28.16	1.68
Full-Time Employed (%)	332,157	69.26	0.69	161,766	93.80	0.20	170,391	44.56	1.45
Home Production (%)	332,157	12.33	0.42	161,766	0.65	0.02	170,391	24.08	0.87
Log Personal Earnings	332,449	4.72	0.03	161,899	5.77	0.03	170,550	3.67	0.05

Notes: Table summarizes the health and economic outcomes of the 1956-1965 cohorts in prime age (age 41-50). Panel A summarizes the mortality and prevalence rates of disease conditions. Mortality rates are derived from death certificate records and calculated as the number of deaths per 10 thousand individuals. Prevalence rates calculate the share of population with a diagnosis of the disease condition during hospital visits, as reported in the health questionnaire of the Comprehensive Survey of Living Conditions. Panel B summarizes education, employment, and earnings for individuals and spouses using data from the Employment Status Survey. Details of the sample construction are provided in the main text.

I examine the education, employment, and earnings of individuals using the Employment Status Survey. Conducted every five years, the survey includes detailed information on labor force participation, employment status, earnings, and family care activities. I focus on prime-age individuals from the 1956-1965 cohorts sampled in the 1992-2017 waves of the survey. For each individual, I collect education and employment variables as well as those of the spouse (if any) using the household relationship pointer.

Panel B of Table 1 summarizes education and economic outcomes. While high school graduation rates were comparable across gender, the share with a college degree was substantially higher for men (38.3%) than for women (13.6%). A larger fraction of women (28.2%) were married to college-educated spouses than having college degrees themselves (13.6%). Less than half of all women were full-time employed compared to 93.8% of men, and 24.1% of women specialized in home production compared to less than 1% of men. On average, earnings generated by men were over twice as high as earnings by women.

2.3 Migration

One concern with the current population surveys is that individuals' prefecture at birth is not known and is assumed to be the same as the prefecture in prime age. This introduces measurement error in the insurance exposure and introduces selection bias if migration responded endogenously to exposure. To investigate, I estimate the exposure impact on migration using the Mobility Survey, a survey on the migration history of individuals from birth to the current age. As I show in Section 2.3, exposure to universal insurance had no significant impact on migration or selective migration across prefecture characteristics such as infant mortality, insurance coverage, or income. Furthermore, I show in robustness tests that the long-run impacts on health and employment are robust to dropping prefectures with the highest shares of migrants from the analysis. These results indicate that endogenous migration responses to exposure are unlikely to be a major source of bias in this context.

3 Empirical Strategy

3.1 Insurance Exposure

I measure the exposure to universal insurance in the 1956-1965 cohorts using the average insurance rate from in-utero (age 0) to age 5. Formally, let $b(i) = t - a(i)$ be the birth year of individual i of age a in year t. The exposure of individual i in prefecture p is given by

$$exposure_{iatp} = \frac{1}{6} \sum_{\tau=-1}^{4} insr_{b(i)+\tau,p}, \tag{1}$$

where insurance rate $insr$ is averaged across the year before birth ($\tau = -1$), birth year $b(i)$, and up to four years after birth ($\tau = 4$). I focus on age 0-5 since the first cohort impacted by the reform, the 1956 cohort, turned age 5 in 1961, so that exposure would not further differ above age 6, where insurance was universal. In the empirical analysis, I also consider alternative exposure measures across smaller age bands in early childhood (for instance, age 0-1 covering in-utero and the birth year). However, consistent with the literature on critical periods of investment (**?**; Currie and Almond 2011), I generally find larger exposure impacts when exposure covers longer duration of early childhood in age 0-5.

Appendix Figure A2 illustrates the insurance exposure across cohorts and prefectures. In the 1956-1958 cohorts, exposure was less than below 80% in the least exposed prefectures and varied greatly across prefectures. In the 1959-1961 cohorts, the mean exposure increased while the variance across prefectures decreased substantially. After 1961, cohorts in all prefectures gained full exposure to universal insurance.

To study the long-run impacts of exposure, I estimate the following equation,

$$y_{iatp} = \beta_0 + \beta_1 \cdot exposure_{iatp} + \theta_a + \mu_t + \delta_p + X_p \cdot \psi_t + \epsilon_{iatp}, \tag{2}$$

where the long-run outcome, y_{iatp}, is regressed on $exposure_{iatp}$ with fixed effects of individual age θ_a, survey year μ_t, and prefecture δ_p. These controls account for differences in

outcomes by age and year as well as pre-existing differences across prefectures. To further account for alternative prefecture characteristics that may affect long-run outcomes, I interact the 1955 values of per capita GDP, community insurance rate, and demographic composition with survey year indicators in $X_p \cdot \psi_t$. With these controls, β_1 captures the differential impact of exposure across cohorts and prefectures during the insurance reform.

In addition to the basic controls in equation 2, I further account for cohort differences that may lead to long-term trending in outcomes. For instance, if later cohorts have access to better health technology or are born healthier, the cohort differences could bias upward the exposure impacts on health. In one specification, I control for cohort differences with prefecture-specific trends in birth year, $\phi_p \cdot b(i)$. Alternatively, I construct 5-year cohort indicators and estimate the following specification

$$y_{iatp} = \beta_0 + \beta_1 \cdot exposure_{iatp} + \theta_a + \mu_t + \delta_p + \zeta_{1961p} + \eta_{pt} + \epsilon_{iatp}, \tag{3}$$

where indicator ζ_{1961p} captures the differential impact of the post-1961 cohorts in prefecture p. η_{pt} further accounts for unobserved determinants of long-run outcomes across prefecture-year.

Despite the large number of controls, OLS estimates of β_1 may still be biased if omitted factors are correlated with exposure and affect outcomes in the long run. For instance, the supply of health workers and the capacity of health facilities could impact the speed of expansion and in turn affect care quality and health outcomes. In early expansion states, support for universal insurance may be correlated with support for alternative policies that impact growth in the long run. To the extent that these alternative factors are not fully captured in equation 2 and 3, I instrument exposure with a simulated measure exploiting variations specific to the reform.

3.2 Simulated Exposure

I simulate insurance exposure drawing on two variations specific to the reform. First, the timeline to achieve universal insurance by 1961 was set by policy and imposed uniformly across prefectures. Second, prefectures differed in the size of expansion due to differences in the pre-reform insurance rate. These variations allow for the construction of simulated exposure that does not depend on prefectures' endogenous responses to policy. Specifically, interpolating between the 1955 and 1961 insurance rate, simulated insurance rate in year τ and prefecture p is

$$insr^{simu}_{\tau p} = insr_{1955p} + \frac{\tau - 1955}{6}(1 - insr_{1955p}), \tag{4}$$

where the gap from universal insurance, $1 - insr_{1955p}$, is divided evenly over the reform period to construct insurance rates under a linear growth path. The resulting simulated exposure is

$$exposure^{simu}_{iatp} = \frac{1}{6}\sum_{\tau=-1}^{4} insr^{simu}_{b(i)+\tau,p}, \tag{5}$$

which is the average insurance rate from in-utero to age 5, with the birth year given by $b(i) = t - a(i)$. Different from the exposure in equation 1, the simulated measure does not depend on the observed, endogenous growth path of insurance. While alternative factors could affect the expansion speed and correlate with endogenous exposure, they are not captured in the simulated exposure.

For the simulated exposure to be a valid instrument, it should strongly predict endogenous exposure but should not correlate with unobserved determinants of outcomes. The exclusion restriction is likely satisfied if omitted factors do not exhibit the same variations across cohorts and prefectures as induced by the insurance reform. This is plausible since no other reform was implemented across prefectures during the timeline of universal insurance. On the other hand, simulated exposure strongly predicts endogenous exposure with an F-statistic well above 1,000 in the first stage (Appendix Table A1). Thus,

instrumenting exposure with the simulated measure may further address omitted variable biases in the OLS estimates.

Applying the instrument, I obtain the two-stage-least-squares (TSLS) estimates of exposure from the following equation

$$y_{iatp} = \gamma_0 + \gamma_1 \cdot \widehat{exposure}_{iatp} + \rho_a + \omega_t + \phi_p + X_p \cdot \nu_t + \epsilon_{iatp}, \tag{6}$$

where $\widehat{exposure}_{iatp}$ is the predicted exposure from the first stage, and γ_1 estimates the causal impact of exposure on outcomes. I show OLS estimates from equation 2 and the TSLS estimates from equation 6 in the main results. In robustness checks, I consider specifications that more flexibly control for cohort differences and long-run trending across prefectures and find similar results compared to the main specification. To further understand the heterogeneous impacts across prefectures, I also estimate the following reduced form equation where the simulated instrument is interacted with a full set of prefecture indicators

$$y_{iatp} = \gamma_0 + \sum_{j=1,2,..,46} \gamma_j \cdot 1\{pref_p = j\} \cdot exposure^{simu}_{iatj} + \rho_a + \omega_t + \phi_p + X_p \cdot \nu_t + \epsilon_{iatp}, \tag{7}$$

where γ_j indicates the impact of insurance exposure specific to prefecture j.

4 Results

4.1 Mortality

I first examine the impact of exposure on prime-age mortality rates in Table 2. In Panel A, OLS estimates suggest that a ten percentage point higher exposure in early childhood reduced all-cause mortality by 0.06 (per 10 thousand individuals) in prime age. gaining a ten percentage point exposure reduced all-cause mortality by 0.06 per 10 thousand

individuals. This effect is very similar to the TSLS estimates based on simulated exposure in Panel B. In column (2), the mortality reduction was mainly concentrated in men, with a ten percentage point exposure reducing male mortality by 0.11 per 10 thousand individuals compared to an insignificant reduction by 0.037 for women. Relative to the mean mortality rates (cf Table 1), these effects correspond to a 26% reduction for men and a 1.6% reduction for women.

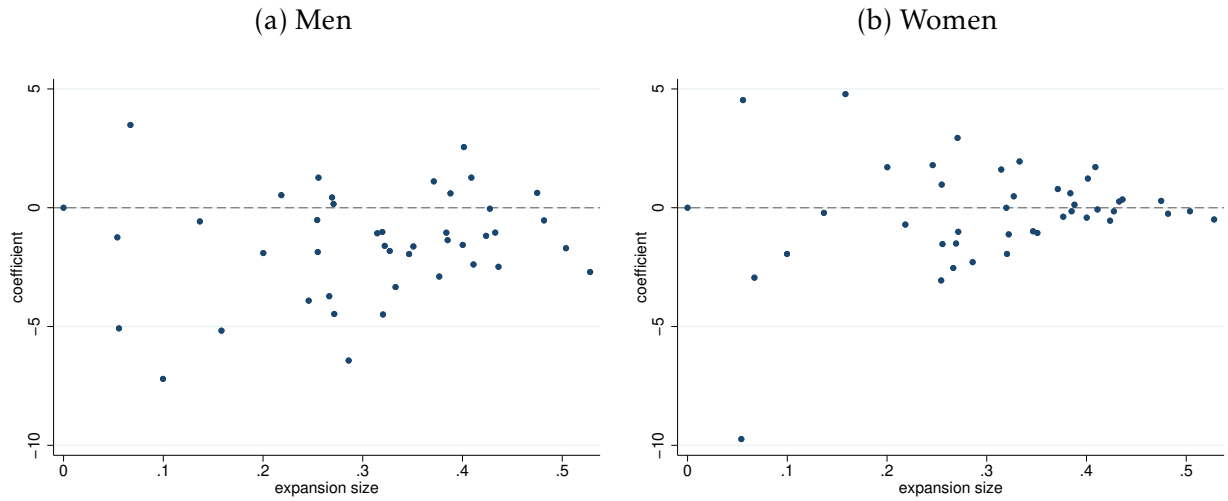
Several factors may contribute to the overall similarity between OLS and TSLS estimates for all-cause mortality. First, in the first stage, the simulated exposure strongly predicts endogenous exposure, with the F-statistic exceeding 1,500 in pooled regressions (Table A1). This suggests that a substantial share of variations in the observed exposure could be explained by differences in predicted exposure across cohorts and prefectures during the reform onset. Moreover, the similarity would also indicate that, conditional on controls of prefecture covariates and time trends, observed exposure rates are not substantially correlated with alternative, unobserved drivers of mortality in the error term, leading to similar OLS estimates in the absence of instrument. This is akin to specification tests that compare estimate differences to test for the null that an endogenous regressor is in fact exogenous (Hausman, 1978). p-values from specification tests are shown in the last row of Table 2. Consistent with small differences in the estimated effects, I cannot reject the null that exposure is uncorrelated with omitted factors of all-cause mortality.

Across causes of death, the reduction in male mortality was mainly due to reductions in cancer-related deaths. In columns 3-4, gaining a ten percentage point exposure decreased cancer mortality by 0.1 per 10,000 individuals, or by 2.3% of the sample average for men. This effect accounts for 88% of the overall mortality reduction for men in prime age. For women, despite similarly high mortality rates from cancer, the impact of insurance exposure on mortality was small and statistically insignificant. Columns 5-8 examine mortality from cardiovascular diseases as well as chronic conditions such as diabetes and hypertension. For both gender, results indicate fairly small and insignificant impacts of

exposure on non-cancer mortality rates in prime age.

Figure 2 plots heterogeneous impacts of exposure across prefectures, where each dot represents an estimated coefficient γ_j from equation 7. Prefectures are sorted based on the expansion size, or the difference between unity and the pre-reform insurance rate in 1955, on the horizontal axis. In most prefectures, all-cause mortality for men showed sizable reductions following the reform (Panel A), with the median prefectures showing a 0.1-0.4 reduction from a ten percentage point exposure in childhood. For women, the mortality effects were small and averaged around zero (Panel B). For cancer mortality (Appendix Figure A4), exposure led to substantial reductions for men across prefectures but had very small mortality impacts for women. Several prefectures with small expansion sizes showed large reductions in mortality. In Appendix Table A2, I show that results are robust to removing the small expansion prefectures with near universal insurance rates (above 91%) prior to the reform.

Figure 2: Prefecture-specific impacts of exposure on all-cause mortality



Notes: Figure shows prefecture-specific impacts of exposure on all-cause mortality by gender. Each dot represents an estimated γ_j coefficient in equation 7. Prefectures are sorted based on expansion size on the horizontal axis, with expansion size the gap between unity and pre-reform insurance rate in 1955.

Table 3 explores heterogeneous impacts across age groups in childhood. Deviating from the main specification where exposure is averaged between in-utero and age 5, I estimate alternative specifications where exposure is measured over earlier life years in age 0-1 and age 0-3. In column 1, I find that increasing exposure in age 0-1 substantially reduced all-cause mortality in prime age, and the magnitude is comparable to 91% of the cumulative exposure impact by age 5 (column 3). This is consistent with evidence that in-utero and neonatal investments play outsized roles in the life-cycle production of health **??**. Similarly for cancer mortality, exposure in early life years (age 0-1 and 0-3) substantially reduced mortality in prime age, and the magnitudes could account for nearly 80% of the exposure impact by age 5 (columns 4-5).

Robustness. I next examine whether the long-run impacts are robust to more flexible controls of cohort differences and prefecture-level confounds that might lead to differential trending in the long run. In Appendix Table A3, I show estimates from the main specification in column 1 (equation 6) and control for cohort differences using linear cohort trends in column 2. In column 3, I control for prefecture-by-cohort effects constructing

Table 2: Long-run impacts of exposure on mortality (per 10 thousand individuals)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Deaths		Cancer		Cardiovascular		Diabetes/Hypertension	
				Panel A: OLS				
exposure	-0.58***		-0.54***		0.040		-0.010	
	(0.16)		(0.11)		(0.076)		(0.022)	
exposure · men		-1.08***		-0.94***		0.080		-0.008
		(0.19)		(0.12)		(0.089)		(0.029)
exposure · women		-0.054		-0.13		0.002		-0.011
		(0.16)		(0.11)		(0.073)		(0.022)
				Panel B: TSLS				
exposure	-0.59***		-0.56***		0.050		-0.003	
	(0.17)		(0.11)		(0.076)		(0.025)	
exposure · men		-1.13***		-0.99***		0.10		-0.006
		(0.21)		(0.12)		(0.090)		(0.033)
exposure · women		-0.037		-0.12		-0.003		0
		(0.16)		(0.11)		(0.074)		(0.99)
y mean	3.32		1.11		0.40		0.065	
N	9,188		9,188		9,188		9,188	
F-statistic	1,572.3		1,572.3		1,572.3		1,572.3	
men		778.4		778.4		778.4		778.4
women		808.2		808.2		808.2		808.2
p-value	0.87	0.39	0.52	0.043	0.56	0.026	0.37	0.073

Notes: Table estimates the impact of exposure to universal insurance on mortality in prime age, where mortality is measured per 10 thousand individuals within age-year-prefecture cells and by gender. Panel A shows the OLS estimates. Panel B shows two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. Results stratifying by gender also control for prefecture-by-female fixed effects to account for gender differences in outcomes. The last rows show F-statistics from the first stage and p-values from specification tests under the null of exogenous exposure. Standard errors clustered at the level of prefectures in the parentheses.

Table 3: Long-run impacts of exposure on mortality, heterogeneity by exposure age

	(1)	(2)	(3)	(4)	(5)	(6)
	All-Cause			Cancer		
exposure · men	-1.03***	-0.96***	-1.13***	-0.72***	-0.79***	-0.99***
	(0.16)	(0.17)	(0.21)	(0.097)	(0.098)	(0.12)
exposure · women	-0.23	-0.097	-0.037	-0.16	-0.13	-0.12
	(0.14)	(0.14)	(0.16)	(0.097)	(0.092)	(0.11)
exposure age	0-1	0-3	0-5	0-1	0-3	0-5
y mean	3.32	3.32	3.32	1.11	1.11	1.11
N	9,188	9,188	9,188	9,188	9,188	9,188

Notes: Table estimates the long-run impacts of exposure on mortality when exposure is measured over different age bands in 0-5. Mortality is measured per 10 thousand individuals within age-year-prefecture cells and additionally stratified by gender. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

5-year cohort dummies and interacting them with prefecture indicators. I further absorb covariate-by-year effects in the main specification with a full set of prefecture-by-year effects to flexibly account for differential trending across space. Both specifications show fairly similar if somewhat larger impacts on mortality compared to the main estimates in column 1. Across causes of death, nearly 90% of the reduction in male mortality was driven by reductions in cancer-related deaths (columns 4-6), whereas the exposure impacts on mortality from cardiovascular diseases and chronic conditions were generally small and insignificant (Appendix Table A4).

4.2 Migration

One concern with the empirical strategy is that migration might lead to measurement errors in the exposure measure and further introduce selection biases with endogenous migration responses to exposure. This would be the case if exposure affected the migration propensity of individuals or induced differential migration based on prefecture characteristics such as health or income. For instance, if universal insurance reduced the

out-migration of sicker individuals from prefectures with historically high mortality rates, then the impact of exposure on health could be under-estimated due to changes in the health stock of prefectures. On the other hand, if exposure allowed more individuals to migrate to high-income prefectures in prime age, then the exposure impact on earnings and employment could be over-estimated due to positive selection on outcomes.

To empirically examine the migration responses to exposure, I use data from the Mobility Survey, a representative survey containing the migration history of individuals from birth to the survey year. Based on the data, I construct a binary indicator of migration equal to one if the respondent's current prefecture differs from her prefecture at birth. I then estimate the migration responses to exposure in Appendix Table A5. Across specifications, I find no significant impact of exposure on migration by prime age. Specifically, TSLS estimates show that a ten percentage point higher exposure led to a small decrease in migration rates by 0.1 percentage point (column 1), or by 3.3% of the sample average, and the null effect is robust to additional controls of cohort differences and prefecture-specific trending over time (columns 3 and 5). Across gender, women showed larger reductions in migration, although the magnitude remains small and statistically insignificant across specifications.

To explore whether individuals selectively migrated across prefectures, I stratify the sample based on prefecture characteristics and estimate heterogeneous impacts above and below the median prefecture in Appendix Table A6. In columns 1-2, I stratify by the pre-reform insurance rate in 1955 and find that exposure had no significant impact on migration across prefectures with initially high and low insurance rates. In columns 3-4, stratifying by the infant mortality in 1955 indicates a small but statistically insignificant reduction in migration across prefectures, with larger reductions among prefectures above the median mortality rate. This suggests that the estimated exposure impact on health might be biased downward due to composition changes in the low-health prefectures, where ex ante one might expect greater health gains from exposure to universal insurance.

Columns 5-6 stratify by the per capita income in the current prefecture and similarly find that migration was also unrelated to exposure across the income of prefectures in prime age.

To more directly assess the confounding impact of migration, I re-estimate the exposure impact on mortality dropping prefectures with the highest migrant shares from the analysis. In the event that long-run impacts operated mainly through migration across space, excluding the high-migration prefectures would lead to material changes in the estimated impacts of exposure.[2] In Appendix Table A7, I find that dropping the top 10% prefectures with the highest migrant shares leads to very similar reductions in all-cause mortality and cancer mortality compared to the main results in Table 2. Moreover, the mortality reduction in cardiovascular diseases and chronic conditions remains small and insignificant (Appendix Table A8). Taken together, results suggest that migration is not a major response margin of exposure and is unlikely to substantially bias estimates of long-run impacts in this context.

4.3 Disease Conditions

In addition to reducing mortality, exposure to universal insurance could also lower the prevalence of disease conditions in prime age. Table 4 examines several common diseases such as metabolic conditions (diabetes or obesity), circulatory conditions (hypertension or cardiovascular), stroke, and cancer. According to TSLS estimates in Panel B, exposure significantly reduced metabolic conditions in prime age, and this effect is mainly concentrated in women (column 2). Specifically, gaining a ten percentage point exposure reduced diabetes/obesity prevalence rates by 0.24 percentage points for women, or by 21.8% below the mean. For men, the exposure impact is small and indistinguishable from zero. The gender-specific estimates also indicate reductions in women's hypertension/cardiovascular

[2] As shown in Appendix Figure A3, migrant share ranges from less than 5% in the lowest 10% prefectures to over 43% in the top 10%. The median prefecture has 17% migrants, similar to the average (20%).

conditions in column 4, although the effect is only marginally significant. Compared to the large reduction in chronic conditions, exposure had no significant impact on the prevalence of more severe diseases such as stroke or cancer in prime age (columns 5-8).

Table 4: Long-run impacts of exposure on disease conditions

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Diabetes/Obesity		Hypertension/ Cardiovascular		Cancer		Stroke	
				Panel A: OLS				
exposure	-0.013*		0		0.002		0	
	(0.007)		(0.008)		(0.004)		(0.003)	
exposure · men		-0.004		0.016		0		0
		(0.009)		(0.011)		(0.004)		(0.004)
exposure · women		-0.022***		-0.017		0.004		0
		(0.006)		(0.010)		(0.006)		(0.003)
				Panel B: TSLS				
exposure	-0.014*		-0.005		0.002		0	
	(0.008)		(0.009)		(0.004)		(0.004)	
exposure · men		-0.003		0.010		-0.001		-0.001
		(0.010)		(0.012)		(0.003)		(0.004)
exposure · women		-0.024***		-0.021*		0.005		0
		(0.008)		(0.011)		(0.006)		(0.003)
y mean	0.019		0.043		0.005		0.002	
N	262,033		262,033		262,033		262,033	
F-statistic	1,575.8		1,575.8		1,575.8		1,575.8	
men		815.2		815.2		815.2		815.2
women		846.5		846.5		846.5		846.5
p-value	0.94	0.15	0.095	0.22	0.77	0.48	0.12	0.29

Notes: Table estimates the long-run impacts of exposure on the prevalence of disease conditions in prime age. Panel A shows the OLS estimates. Panel B shows two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. Results stratifying by gender also control for prefecture-by-female fixed effects to account for gender differences in outcomes. The last rows show F-statistics from the first stage and p-values from specification tests under the null of exogenous exposure. Standard errors clustered at the level of prefectures in the parentheses.

I next examine the exposure impact on two composite measures of chronic conditions. The first measure indicates whether the individual has any of the four chronic conditions (diabetes, obesity, hypertension, or cardiovascular). The second measure standardizes the prevalence rate of each condition using z-scores and uses the average z-score as a composite

index of diseases (Anderson 2008; Boudreaux *et al.* 2016; Miller and Wherry 2019). In Appendix Table A9, both measures indicate significant reductions in chronic conditions for women. In detail, a ten percentage point exposure reduced the prevalence of having any condition by 0.36 percentage points, an effect about 50% larger than the reduction in metabolic conditions alone (column 1, Table 4). The exposure also reduced the average prevalence rate across conditions by a small but significant 0.009 standard deviations. This effect is comparable to those found in previous studies using the index. In particular, Boudreaux *et al.* (2016) finds that an additional year of insurance exposure in age 0-5 reduced chronic conditions by 0.011 standard deviations in adulthood. In my context, an additional year of exposure would reduce the composite index by 0.039/6 = 0.007 standard deviations on average and by 0.086/6 = 0.014 standard deviations for women.

Figure A5 plots heterogeneous impacts of exposure across prefectures. Consistent with the gender differences in Table A9, in most prefectures, women saw decreased rates of chronic conditions and lower z-scores in the composite index. The exposure impacts for men were much smaller and averaged around zero. The reduction in chronic conditions from a marginal increase in exposure is fairly similar across prefectures with median to large expansion sizes, but increased among small expansion prefectures with near universal insurance in 1955. In a robustness check, I show that the exposure impact on chronic conditions is not sensitive to excluding small expansion prefectures (over 91% insured in 1955) from the analysis (Appendix Table A10).

Table 5 estimates heterogeneous impacts of exposure across age groups in childhood. Exposure in utero and the first year of life (age 0-1) significantly reduces chronic conditions in prime age. In terms of magnitude, exposure in age 0-1 could account for 71% of the reduction in metabolic conditions and 76% of the reduction in circulatory conditions implied by cumulative exposure by age 5. For composite measures, similar comparisons suggest that roughly 75% of the reduction in prevalence rates and 73% of the reduction in the standardized index could be attributed to insurance exposure in age 0-1 (Appendix

Table A11).

Table 5: Long-run impacts of exposure on disease conditions, heterogeneity by exposure age

	(1)	(2)	(3)	(4)	(5)	(6)
	Diabetes/Obesity			Hypertension/ Cardiovascular		
exposure · men	-0.002	-0.002	-0.003	0.007	0.009	0.010
	(0.008)	(0.008)	(0.010)	(0.007)	(0.009)	(0.012)
exposure · women	-0.017***	-0.019***	-0.024***	-0.016**	-0.016**	-0.021*
	(0.006)	(0.006)	(0.008)	(0.007)	(0.008)	(0.011)
exposure age	0-1	0-3	0-5	0-1	0-3	0-5
y mean	0.019	0.019	0.019	0.043	0.043	0.043
N	262,033	262,033	262,033	262,033	262,033	262,033

Notes: Table estimates the long-run impacts of exposure on the prevalence of disease conditions when exposure is measured over different age bands in 0-5. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Robustness. I next examine the robustness of results to alternative cohort and prefecture controls in the regression. In Appendix Table A12, controlling for prefecture-year effects in addition to prefecture differences across 5-year cohort groups (column 3) gives very similar reductions in women's metabolic conditions compared to the main estimate (column 1). Under linear cohort trends, however, effects for both gender are shifted upward and the reduction for women is no longer significant (column 2). This is likely due to multicollinearity under the linear trend assumption. For circulatory conditions (columns 4-6), flexibly controlling for prefecture-by-year and prefecture-by-cohort-group effects gives significant reductions in women's conditions (column 6) whereas estimates under linear cohort trends instead show large *relative* reductions in women's conditions compared to men (column 5). Similar patterns apply to composite measures in Appendix Table A13, where estimates controlling for prefecture-by-cohort-group effects are very similar to the

main result but shifted upward under linear cohort trends.

To assess the potential bias from migration, Appendix Table A14 estimates the exposure impact on chronic conditions excluding the top 10% prefectures with the highest migrant shares from the analysis. In low-migration prefectures, gaining a ten percentage point exposure decreased women's metabolic conditions by 0.28 percentage points under the main specification in column 1 and decreased conditions by 0.30 percentage points under flexible controls of cohort and prefecture differences in column 3. These effects are slightly larger but comparable to the main results in Table 4. Similarly, for the composite measures, Appendix Table A15 estimates that a ten percentage point exposure decreased the prevalence of any condition by 0.36-0.41 percentage points and decreased the composite index by 0.008-0.009 standard deviations, comparable to the full sample estimates in Appendix Table A9.

4.4 Education

I next examine whether insurance exposure could also impact human capital formation and economic success in the long run. Table 6 estimates the exposure impact on education attainment in columns 1-4. According to TSLS estimates in Panel B, gaining a ten percentage point exposure increased the probability of graduating high school by 1.3 percentage points and further increased the probability of graduating college by 0.47 percentage points. Across gender, exposure increased high school as well as college graduation rates for women, but had no significant impact on men's college graduation rates beyond high school (columns 2 and 4).

Since women had substantially lower college graduation rates than men (13.6% compared to 38.3% for men, cf Table 1), exposure to universal insurance could have sizeable impacts on the gender education gap in the long run. In particular, gaining a ten percentage point exposure increased women's college graduation by 1.1 percentage points (column 4), or by 8.1% above the mean. Compared to the 24.7% gap in college graduation

rates, the differential increase reduced the gender gap by 4.5%. This effect is somewhat smaller according to OLS estimates in Panel A, where the exposure impact on education is overall smaller. Applying OLS estimates, gaining a ten percentage point exposure reduced the gender gap in college graduation by 3.5%.

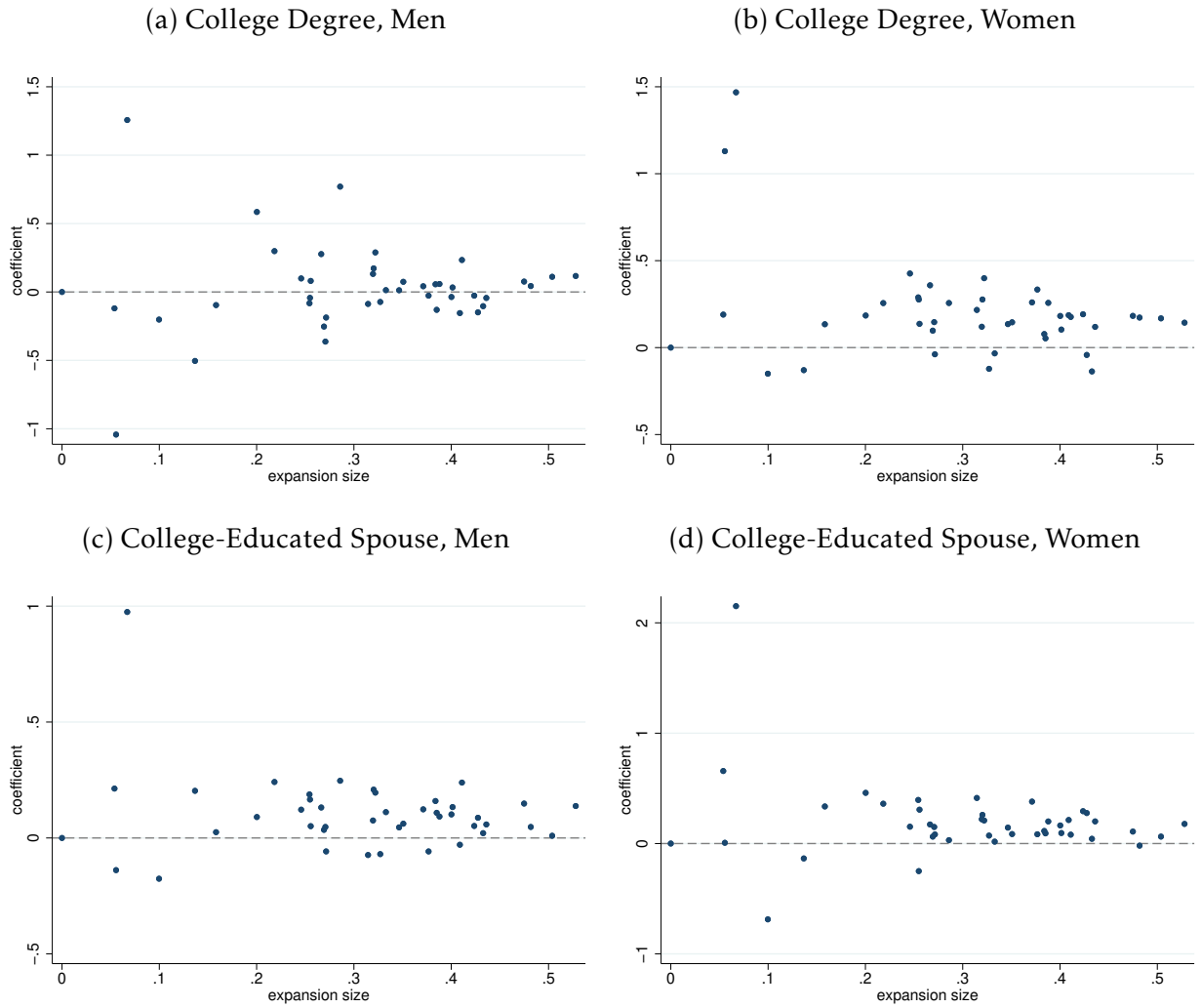
In columns 5-8, I examine whether exposure further impacted the marital sorting between education groups. For women, exposure increased the probability of marrying a college-educated spouse by 1.2 percentage points (column 6) without affecting the marriage rate on the extensive margin (column 8). For men, by contrast, exposure substantially increased his marriage rate by 0.95 percentage points but had smaller impacts on marrying a college-educated spouse (0.66 percentage point increase) compared to the extensive margin response. While suggestive, these results are consistent with exposure improving woman's match quality in marriages through assortative sorting on education.

Table 6: Long-run impacts of exposure on education and marital outcomes

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	High School		College Degree		Married to College-Educated Spouse		Married	
					Panel A: OLS			
exposure	0.088***		0.028		0.072***		0.049	
	(0.025)		(0.021)		(0.017)		(0.030)	
exposure · men		0.089***		-0.029		0.051***		0.082***
		(0.026)		(0.018)		(0.019)		(0.030)
exposure · women		0.087***		0.086***		0.095***		0.016
		(0.027)		(0.032)		(0.021)		(0.035)
					Panel B: TSLS			
exposure	0.13***		0.047**		0.092***		0.063**	
	(0.032)		(0.021)		(0.015)		(0.029)	
exposure · men		0.13***		-0.012		0.066***		0.095***
		(0.032)		(0.025)		(0.018)		(0.030)
exposure · women		0.12***		0.11***		0.12***		0.030
		(0.033)		(0.026)		(0.020)		(0.033)
y mean	0.94		0.26		0.19		0.78	
N	329,502		329,502		331,397		331,397	
F-statistic	1,265.0		1,265.0		1,266.1		1,266.1	
men		660.5		660.5		660.6		660.6
women		746.7		746.7		749.2		749.2
p-value	0.002	<0.001	0.25	0.52	0.023	0.077	0.10	0.27

Notes: Table estimates the long-run impacts of exposure on education and marital outcomes by prime age. Panel A shows the OLS estimates. Panel B shows two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. Results stratifying by gender also control for prefecture-by-female fixed effects to account for gender differences in outcomes. The last rows show F-statistics from the first stage and p-values from specification tests under the null of exogenous exposure. Standard errors clustered at the level of prefectures in the parentheses.

Figure 3: Prefecture-specific impacts of exposure on education and marital outcomes



Notes: Figure shows prefecture-specific impacts of exposure on college graduate rates and the probability of marrying a college-educated spouse, with estimates shown separately by gender. Each dot represents an estimated γ_j coefficients in equation 7. Prefectures are sorted based on expansion size on the horizontal axis, with expansion size the gap between unity and pre-reform insurance rate in 1955.

Figure 3 plots heterogeneous impacts of exposure across prefectures. In panel (a)-(b), exposure led to large increases in women's college graduation rates whereas the impacts on men were small and averaged around zero. In panel (c)-(d), both gender saw increased probability of marrying a college-educated spouse, with the effect sizes nearly twice as large for women than for men. For both education and marital sorting, the impact of a marginal increase in exposure was roughly constant in prefectures with median to large

expansion sizes but showed larger variations in prefectures with small expansion sizes. In a robustness check, I show that the main results in Table 6 are not sensitive to excluding small expansion prefectures (over 91% insured in 1955) from the analysis (Appendix Table A16).

Appendix Table A17 explores heterogeneous impacts across age groups in childhood. In column 1, gaining a ten percentage point exposure in utero and the first year of life (age 0-1) increased women's high school graduation rates by 0.82 percentage points, or by 68% of the exposure impact by age 5. In column 4, exposure in age 0-1 further increased women's college graduation by 0.7 percentage points, or by 65% of the exposure impact by age 5. For men, exposure had no significant impact on college graduation rates but increased high school graduation at similar rates compared to women in early childhood. Appendix Table A18 estimates the impact of early life exposure on marital sorting. Specifically, gaining a ten percentage point exposure in age 0-1 increased women's probability of marrying a college-educated spouse by 0.83 percentage points, or by 69% of the exposure impact by age 5.

Robustness. I next examine the robustness of results to alternative cohort and prefecture controls in the regression. In Appendix Table A19, including prefecture-by-year and prefecture-by-cohort-group effects results in very similar increases in college graduation rates (column 6), whereas including linear cohort trends tends to increase the exposure impact especially for high school (column 2). In Appendix Table A20, both sets of estimates indicate similar exposure impacts on marrying a college-educated spouse, with slightly larger estimates under linear cohort trends.

Appendix Table A21 estimates the exposure impact in the low-migration prefectures. Across specifications, gaining a ten percentage point exposure increased high school graduation by 1.4-1.9 percentage points and increased college graduation for women by 0.7-0.9 percentage points. While the effect magnitude is smaller in low-migration prefectures, the differential increase in women's college graduation remains sizeable and

significant across specifications. In Appendix Table A22, exposure increased women's probability of marrying a college-educated spouse by 1.3-1.5 percentage points but had no significant impact on the marriage rate of women, comparable to the main results in Table 6.

4.5 Employment and Earnings

Table 7 estimates the exposure impact on employment outcomes and earnings in prime age. Columns 1-4 first examine whether the individual was employed or exclusively engaged in home production activities such as caregiving and housekeeping. In column 1, exposure on average had very small impacts on employment, with a ten percentage point exposure increasing the employment rate by an insignificant 0.19 percentage points, or by 2.8% above the mean. The average effect, however, masks substantial heterogeneity by gender. For women, gaining a ten percentage point exposure increased employment rate by 1.6 percentage points while reducing the share in home production by 0.8 percentage points. In contrast, exposure reduced employment rate for men by 1.2 percentage points while increasing the share in home production by 0.95 percentage points. Taken together, the differential responses suggest that exposure increased labor market participation for women and shifted home production from women to men in households.

I next examine the exposure impact on personal and household earnings in columns 5-8. Consistent with the employment responses, personal earnings increased by 8.4% for women following a ten percentage point exposure but decreased for men by 9.0% (column 6). These effects roughly offset each other and the average exposure impact was not statistically distinguishable from zero (column 5). Combining earnings for married couples, I then examine earnings responses at the household level in columns 7-8. Contrasting the large shifts in personal earnings across gender, exposure had no significant impact on the joint earnings of households. For men, a ten percentage point exposure decreased his household earnings by less than 1% in column 8 despite the 9% drop in personal earnings

Table 7: Long-run impacts of exposure on employment and earnings

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Employment		Home Production		Log Earning, Personal		Log Earning, Household	
				Panel A: OLS				
exposure	0.026		0.005		-0.051		0.043	
	(0.022)		(0.026)		(0.14)		(0.088)	
exposure · men		-0.11***		0.093***		-0.90***		-0.051
		(0.024)		(0.016)		(0.12)		(0.096)
exposure · women		0.17***		-0.086**		0.82***		0.14
		(0.024)		(0.038)		(0.19)		(0.11)
				Panel B: TSLS				
exposure	0.019		0.008		-0.040		0.069	
	(0.024)		(0.026)		(0.16)		(0.085)	
exposure · men		-0.12***		0.095***		-0.90***		-0.022
		(0.025)		(0.016)		(0.13)		(0.093)
exposure · women		0.16***		-0.083**		0.84***		0.16
		(0.028)		(0.038)		(0.22)		(0.11)
y mean	0.69		0.12		4.72		5.82	
N	332,157		332,157		332,449		332,449	
F-statistic	1,265.8		1,265.8		1,265.1		1,265.1	
men		660.4		660.4		661.1		661.1
women		748.5		748.5		747.4		747.4
p-value	0.45	0.57	0.55	0.81	0.74	0.73	0.37	0.44

Notes: Table estimates the long-run impacts of exposure on employment and earnings in prime age. Panel A shows the OLS estimates. Panel B shows two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. Results stratifying by gender also control for prefecture-by-female fixed effects to account for gender differences in outcomes. The last rows show F-statistics from the first stage and p-values from specification tests under the null of exogenous exposure. Standard errors clustered at the level of prefectures in the parentheses.

in column 6. For women, exposure increased her household earnings by a modest 1.6% in column 8 despite the 8.4% increase in personal earnings in column 6. The weaker responses in household earnings suggest that exposure decreased employment for men while increasing women's earnings in households, and the substitution is consistent with the average null effect on earnings and employment across individuals.

These results show that exposure to universal insurance significantly increased women's economic participation and resources in the long run. In column 2, gaining a ten percentage point exposure increased women's employment by 1.2 percentage points and by 2.8 percentage points relative to men. Compared to the gender differences in employment rate (93.8% for men and 44.6% for women, cf Table 1), the differential increase in women's employment reduced the gender gap by 5.7%. For personal earnings, similarly, the ten percentage point exposure differentially increased women's earnings by 17.4% relative to men, reducing the gender gap in earnings by 11.4%. These effects are larger than the 4.5% reduction in the gender gap in college graduation, suggesting persistent impacts of insurance exposure on economic outcomes well after the initial increase in education.

Figure 4 plots heterogeneous impacts of exposure across prefectures. Consistent with the gender differences in Table 7, in most prefectures, exposure substantially increased women's employment while decreasing it for men. Specifically, among prefectures with medium to large expansion sizes, a ten percentage point exposure increased women's employment by roughly 1.5 percentage points and decreased men's employment by roughly one percentage point. The exposure impacts in the smallest expansion prefectures are larger by comparison. Similarly, for personal earnings, estimates show large exposure impacts increasing earnings for women while decreasing it for men, with larger effect sizes among the small expansion prefectures. In Appendix Table A23, I show that the estimated exposure impacts are robust to excluding the smallest expansion prefectures (over 91% insured in 1955) from the analysis.

Figure 4: Prefecture-specific impacts of exposure on employment and earnings

Notes: Figure shows prefecture-specific impacts of exposure on employment and personal earnings by gender. Each dot represents an estimated γ_j coefficients in equation 7. Prefectures are sorted based on expansion size on the horizontal axis, with expansion size the gap between unity and pre-reform insurance rate in 1955.

Appendix Table A24 explores heterogeneous impacts across age groups in childhood. In column 1, gaining a ten percentage point exposure in utero and the first year of life (age 0-1) increased women's employment by 0.95 percentage points, or by 59% of the exposure impact by age 5. Compared to men, the differential increase in women's employment (1.79 percentage points) reduced the gender gap in employment by 3.6%, or by 64% of the cumulative reduction from exposure by age 5. Appendix Table A25 finds similarly large

impacts of early life exposure on personal earnings. In detail, gaining a ten percentage point exposure in age 0-1 differentially increased women's earnings by 4.8%, or by 57% of the exposure impact by age 5. The differential increase relative to men (11%) further reduced the gender earnings gap by 7.7%, or by 68% of the cumulative reduction from exposure by age 5.

Robustness. I next examine the robustness of results to alternative controls in the regression. In Appendix Table A26, introducing additional cohort and prefecture controls slightly increased the exposure impact on women's employment. In column 3, for instance, controlling for prefecture-by-year and prefecture-by-cohort-group effects indicates a 1.8 percentage point increase in women's employment, slightly larger than the 1.6 increase in column 1. Compared to men, exposure differentially increased women's employment by 2.8 percentage points, and the magnitude was comparable across specifications. These patterns also apply to personal earnings in Appendix Table A27, where additional controls of cohort and prefecture differences led to larger increases in women's earnings but very similar increases in relative earnings (17.4% relative to men) across specifications.

In the low-migration prefectures, Appendix Table A28 estimates that a ten percentage point exposure increased women's employment by 1.4-1.5 percentage points and decreased men's employment by 1.1-1.3 percentage points. The differential increase in women's employment (2.6-2.7 percentage points) was comparable to the main result estimates in Table 7. Turning to personal earnings, in Appendix Table A29, gaining a ten percentage point exposure increased women's earnings by 7.1%-7.8% and decreased men's earnings by 8.8%-9.5%. The differential increase for women (16.6%-16.7% relative to men) was slightly lower but comparable to the main result estimate (17.4%) in Table 7.

5 Discussion

5.1 The Exposure Impact on Health

The long-run impacts of insurance exposure on health are consistent with the fetal origin hypothesis that a stressful intrauterine and early childhood environment could predispose individuals for diseases later in life (Barker, 1992). In particular, chronic diseases such as diabetes, obesity, hypertension, and cardiovascular diseases have long been shown to have fetal and early life origins whereby infections or insults in the perinatal period activate adaptive regulations of immune and metabolic processes that are pre-markers of future diseases (Hales *et al.* 1991, Barker *et al.* 1993, McMillen and Robinson 2005). Moreover, epigenetic responses could influence individuals' sensitivity to environmental stressors such as carcinogens through modified gene expression, suggesting a potential pathway from perinatal conditions to cancer in adulthood (Walker and Ho 2012, Goyal *et al.* 2019).

In the economics literature, studies of policy interventions on health have generally found large impacts of early-life exposure on chronic diseases later in life. Focusing on the onset of Medicaid program in the US in the 1960s, Boudreaux *et al.* (2016) finds that Medicaid exposure in age 0-5 significantly reduced adult chronic diseases captured using an index of diabetes, obesity, cardiovascular, and hypertension prevalence rates, and the magnitudes were comparable to results applying the same index in Japan. Apart from health insurance, providing information on proper infant care to mothers has also been shown to substantially lower cardiac and metabolic risks and mortality above age 40 (Hjort *et al.* 2017, Bütikofer *et al.* 2019), with some reduction in cancer mortality in older ages (Bhalotra *et al.*, 2017).

While one has yet to observe mortality in older ages, current estimates in the literature indicate relatively small impacts of universal insurance on mortality in Japan. For instance, Kondo and Shigeoka (2013) shows that the insurance reform did not immediately affect age-specific mortality in the short run, and over a longer horizon, early-life exposure

mainly reduced cancer mortality for men without affecting mortality from cardiac or metabolic diseases in prime age. This contrasts with studies showing both short and long run mortality effects of insurance (Goodman-Bacon 2018, Goodman-Bacon 2021). The delayed mortality effects might suggest that some of the benefits of early-life interventions are initially latent and become amplified over the life course (Dalgaard *et al.* 2021, Lleras-Muney and Moreau 2022). Across gender, to the extent that women suffer from severe illnesses at older ages than men (Case and Paxson 2005, Schünemann *et al.* 2017), exposure could improve different health outcomes for men and women at younger ages, although over time, both gender should see stronger mortality effects in old age. Future studies on elderly outcomes are thus necessary to fully understand the long-run impacts of exposure on the longevity and morbidity of individuals.

5.2 Human Capital Gains

In addition to health, universal insurance also improved human capital and economic outcomes in prime age. These impacts are highly gendered and primarily concentrated in women. While exposure had no significant impact on men's education beyond high school, it increased women's college education and her probability of marrying a college -educated spouse. The human capital gains led to further increases in women's economic opportunities, reducing the gender gap in employment and earnings in households.

The impact on human capital is consistent with several investment responses to insurance. First, the reduction in the financial cost of medical services may increase parents' health investments in children. In the long run, health investments could lead to better school performance and education attainment especially for girls (Field *et al.* 2009; Baird *et al.* 2016). Second, by relaxing the liquidity constraint of households, insurance could also increase the consumption of non-health services and help families maintain investments in children during health shocks (Sheu and Lu 2014; Liu 2016). Furthermore, the ability to obtain health insurance regardless of the husband's coverage may have increased

the bargaining power of women, who may invest more in the health of girls than boys (Thomas 1994; Rangel 2006). While the exact mechanism is difficult to tease out, the reform impacts suggest that gender differences in the responses to policy could mitigate the inequalities in the long-run development of children.

6 Conclusion

This paper documents the substantial long-run benefits of universal insurance in Japan, one of the first countries to achieve universal insurance through accelerated expansions starting in the 1950s. Exploiting differences in the exposure to universal insurance across cohorts and prefectures, I show that universal insurance led to reductions in chronic conditions and cancer mortality in prime age. Furthermore, exposure increased college education especially for women, increased her full-time employment, and reduced the gender gap in employment and earnings in households. The impact on women's education and economic opportunities suggests that universal insurance can contribute to an inclusive and equitable society with increased investments in less advantaged populations.

The long horizon of the health and economic benefits has implications for countries which recently rolled out universal insurance, such as Mexico, Thailand, Vietnam, and China. In these countries, while current estimates already indicate reduced infant mortality and improved education outcomes in school age (Celhay *et al.* 2019; Gruber *et al.* 2014; Alcaraz *et al.* 2017; Khiem and Kuo 2021; Huang and Liu 2023), the life-cycle impacts of insurance are likely to be substantial but not yet observable in the short run. Moreover, minority groups may experience greater increases in investments that impact their health, human capital, and economic success in the long run. The tendency for the return to health and human capital investments to accumulate over the life cycle implies that short-term evaluations of universal insurance could be missing out important benefits on health and economic wellbeing that materialize only decades after the initial reform.

Data Availability

The data used in this study were made available to the author through data-sharing agreements facilitated by the Government Statistics Anonymized Data Usage Promotion Program in Japan. The agreements restrict the use of data to the proposed research only and prohibit lending or transfers of data to third parties or entities. Interested readers can access the raw survey data following the application instructions available at https://www.soumu.go.jp/english/dgpp_ss/seido/2jiriyou.htm.

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Competing Interests

The author declares no competing interests.

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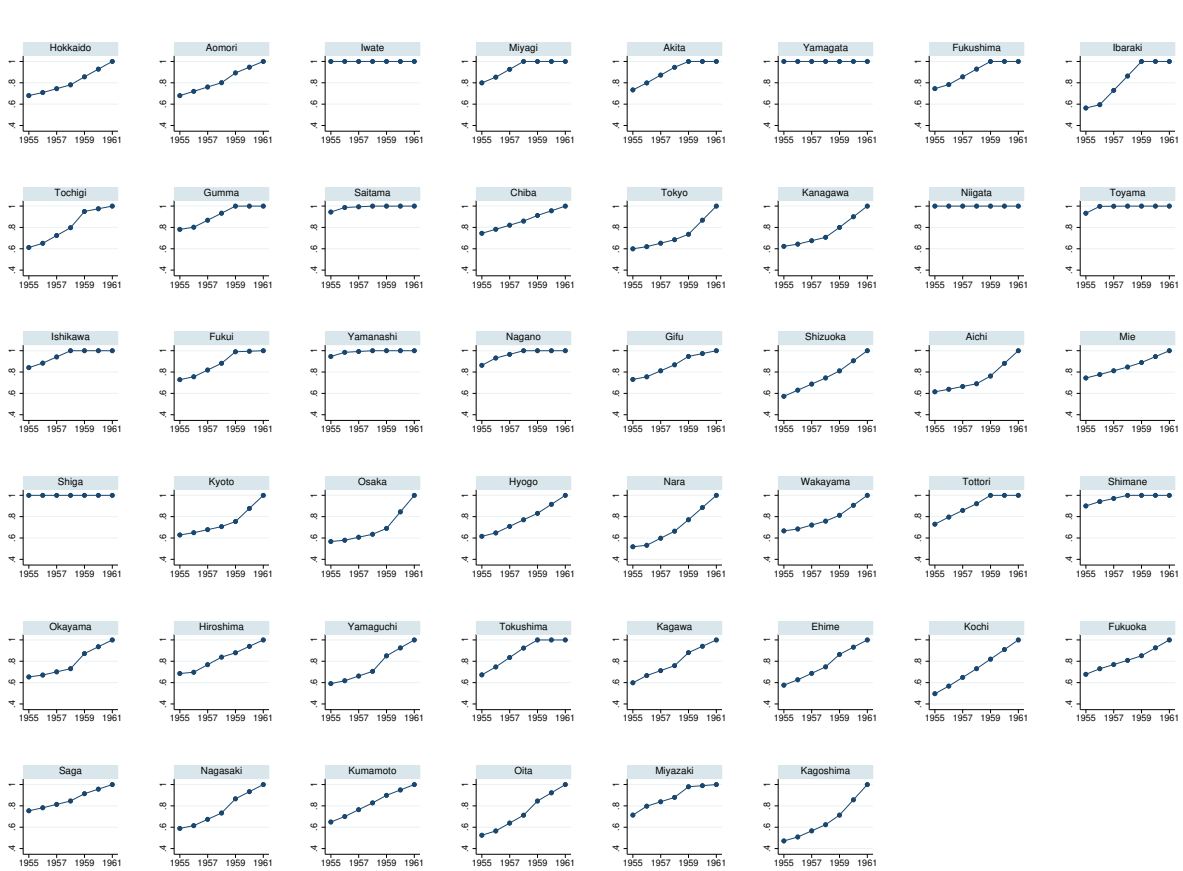
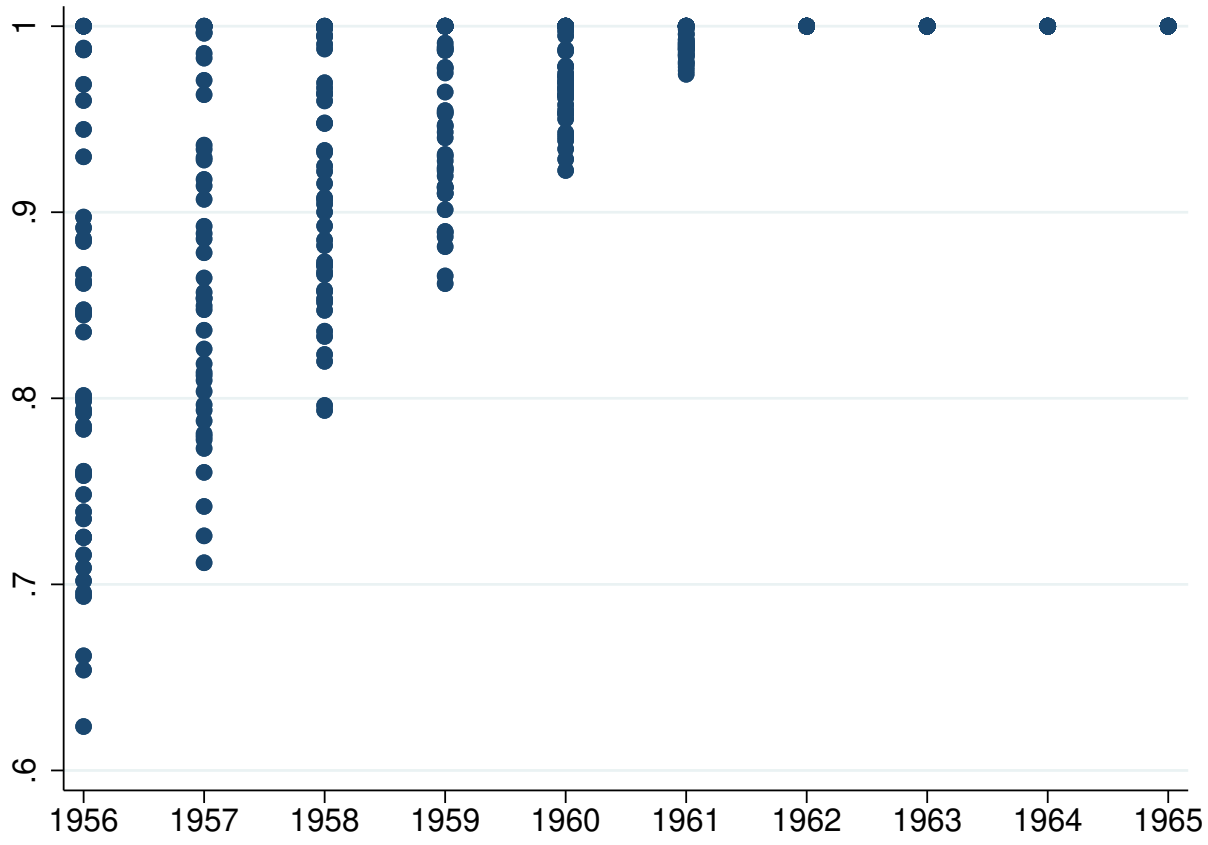
A Appendix Figures

Figure A1: Insurance expansion across prefectures in 1955-1961



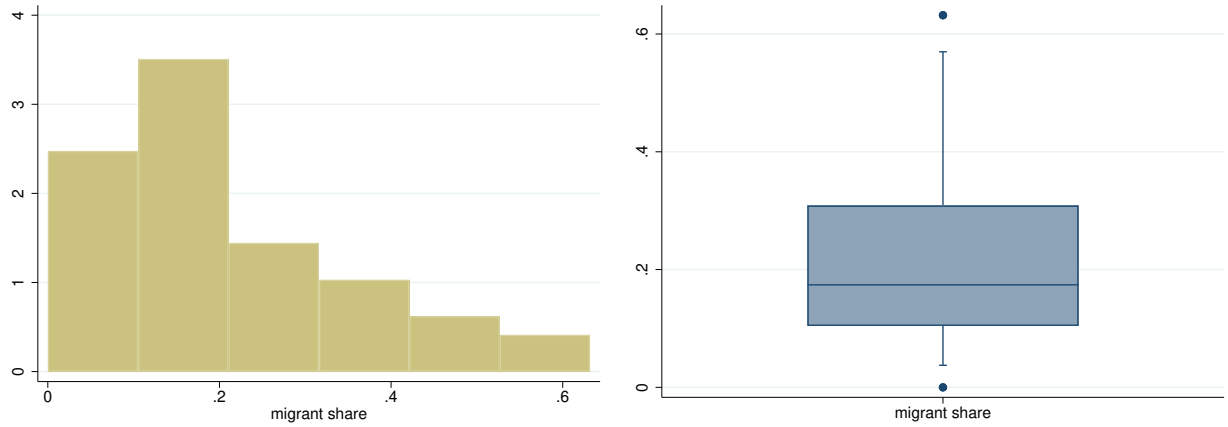
Notes: Figure plots the trend of insurance rates in 1955-1961 in each of the 47 prefectures in Japan. Prefectures differed in the pre-reform insurance rate in 1955 and the incremental expansion each year during the reform period in 1956-1961. The variations in the expansions over time are illustrated in the Figure.

Figure A2: Insurance exposure by birth cohorts and prefectures



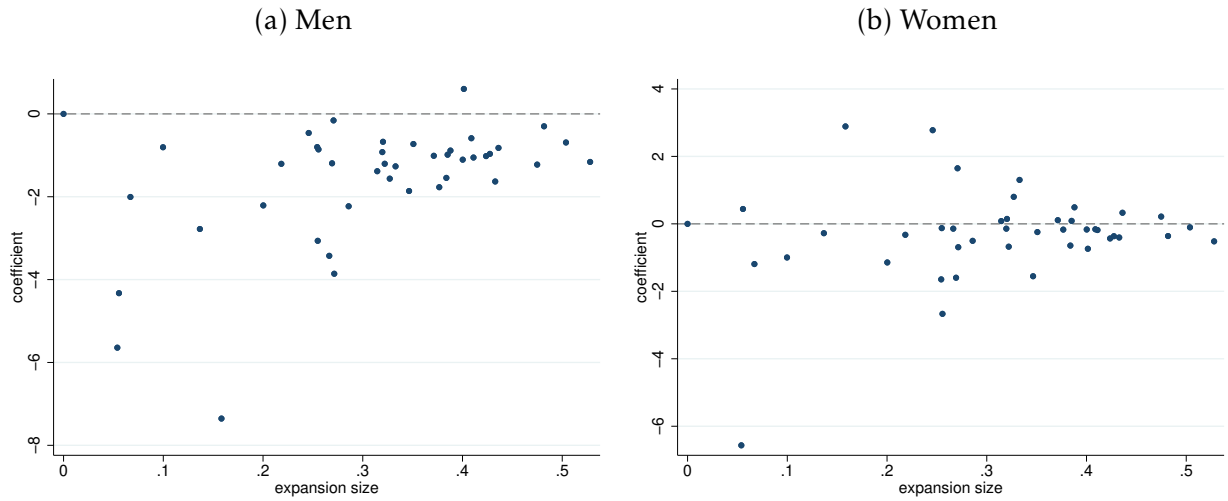
Notes: Figure plots the insurance exposure from in-utero to age 5 for the 1956-1965 birth cohorts. Each dot indicates exposure in a given prefecture and cohort. Overall, exposure increased and the variance across prefectures decreased in cohorts born closer to 1961, the implementation year of universal insurance.

Figure A3: Distribution of migrant shares across prefectures



Notes: Figure shows the histogram of prefecture-level migrant shares on the left panel and a box plot of the distribution on the right panel. The median prefecture has a migrant share of 17% (average 20%).

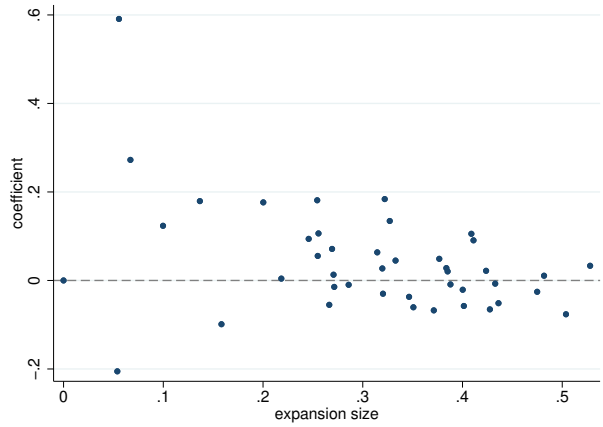
Figure A4: Prefecture-specific impacts of exposure on cancer mortality



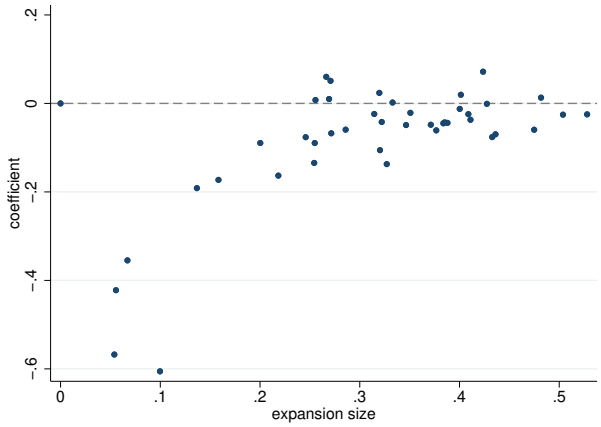
Notes: Figure shows prefecture-specific impacts of exposure on cancer mortality by gender. Each dot represents an estimated γ_j coefficients in equation 7. Prefectures are sorted based on expansion size on the horizontal axis, with expansion size the gap between unity and pre-reform insurance rate in 1955.

Figure A5: Prefecture-specific impacts of exposure on chronic conditions

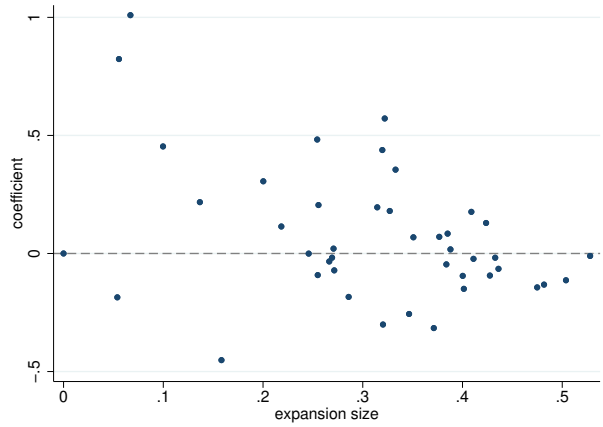
(a) Any Condition, Men



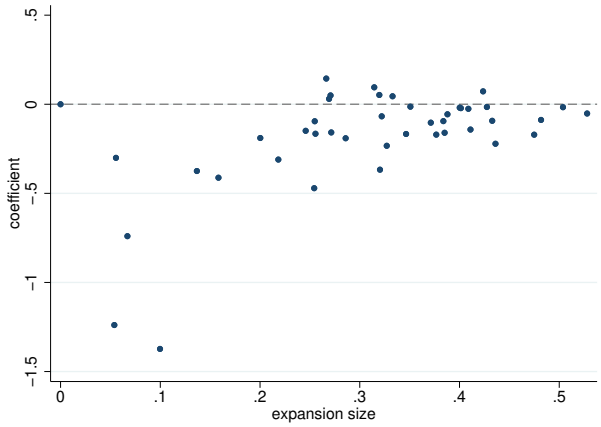
(b) Any Condition, Women



(c) Composite Index, Men



(d) Composite Index, Women



Notes: Figure shows prefecture-specific impacts of exposure on the prevalence of chronic conditions by gender. Each dot represents an estimated γ_j coefficients in equation 7. Prefectures are sorted based on expansion size on the horizontal axis, with expansion size the gap between unity and pre-reform insurance rate in 1955.

B Appendix Tables

Table A1: First-stage prediction of exposure from the simulated instrument

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
exposure	1.18***	1.10***	1.12***	1.18***	1.10***	1.12***	1.19***	1.13***	1.13***
	(0.030)	(0.014)	(0.017)	(0.030)	(0.013)	(0.016)	(0.034)	(0.021)	(0.020)
dataset	Comprehensive Survey of Living Conditions			Death Certificates			Employment Status Survey		
covariate-year prefecture *	Y	Y		Y	Y		Y	Y	
year FE			Y			Y			Y
linear cohort trend		Y			Y			Y	
5-year cohort dummy			Y			Y			Y
F-statistic	1,575.9	6,325.6	4,206.2	1,572.3	6,639.2	5,169.0	1,265.0	2,852.8	3,359.8
N		262,033			9,188			329,502	

Notes: Table estimates the first-stage prediction of exposure from the simulated instrument, constructed under a linear growth path of insurance from the 1955 level to universal insurance in 1961. Results are shown separately for the three survey samples included in the study. Standard errors clustered at the level of prefectures in the parentheses.

Table A2: Long-run impacts of exposure on all-cause and cancer mortality, excluding small expansion prefectures

	(1)	(2)	(3)	(4)
	Deaths		Cancer	
	Panel A: OLS			
exposure	-0.59***		-0.63***	
	(0.19)		(0.12)	
exposure · men		-1.10***		-1.04***
		(0.21)		(0.12)
exposure · women		-0.072		-0.23*
		(0.19)		(0.13)
	Panel B: TSLS			
exposure	-0.65***		-0.56***	
	(0.12)		(0.11)	
exposure · men		-1.15***		-1.08***
		(0.22)		(0.13)
exposure · women		-0.064		-0.21*
		(0.17)		(0.12)
y mean	3.32		1.11	
N	7,790		7,790	
F-statistic	1,843.2		1,843.2	
men		914.3		914.3
women		928.7		928.7
p-value	0.85	0.51	0.78	0.079

Notes: Table estimates the long-run impacts of exposure on all-cause mortality and cancer mortality. Different from the main results in Table 2, the estimation sample excludes small expansion prefectures with near univeral insurance (91% and above) prior to the reform in 1955. Panel B shows two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. Results stratifying by gender also control for prefecture-by-female fixed effects to account for gender differences in outcomes. The last rows show F-statistics from the first stage and p-values from specification tests under the null of exogenous exposure. Standard errors clustered at the level of prefectures in the parentheses.

Table A3: Long-run impacts of exposure on all-cause and cancer mortality, alternative specification

	(1)	(2)	(3)	(4)	(5)	(6)
	All-Cause			Cancer		
exposure · men	-1.13***	-1.28***	-1.31***	-0.99***	-1.19***	-1.08***
	(0.21)	(0.23)	(0.22)	(0.12)	(0.16)	(0.15)
exposure · women	-0.037	-0.19	-0.22	-0.12	-0.32	-0.21
	(0.16)	(0.18)	(0.16)	(0.11)	(0.13)	(0.12)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	3.32	3.32	3.32	1.11	1.11	1.11
N	9,188	9,188	9,188	9,188	9,188	9,188

Notes: Table estimates the long-run impacts of exposure on all-cause mortality and cancer mortality. I show two-stage-least-squares (TSLS) estimates of exposure where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A4: Long-run impacts of exposure on mortality from cardiovascular diseases and chronic conditions, alternative specification

	(1)	(2)	(3)	(4)	(5)	(6)
	Cardiovascular			Diabetes/Hypertension		
exposure · men	0.10	0.078	0.065	-0.006	-0.003	-0.010
	(0.090)	(0.089)	(0.084)	(0.033)	(0.034)	(0.034)
exposure · women	-0.003	-0.028	-0.042	0	0.002	-0.005
	(0.074)	(0.066)	(0.064)	(0.99)	(0.024)	(0.023)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	0.40	0.40	0.40	0.065	0.065	0.065
N	9,188	9,188	9,188	9,188	9,188	9,188

Notes: Table estimates the long-run impacts of exposure on mortality due to cardiovascular diseases or chronic conditions. I show two-stage-least-squares (TSLS) estimates of exposure where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A5: Long-run responses of migration to exposure

	(1)	(2)	(3)	(4)	(5)	(6)
			Panel A: OLS			
exposure	0.003		-0.17		-0.056	
	(0.12)		(0.16)		(0.15)	
exposure · men		0.12		-0.04		0.068
		(0.12)		(0.15)		(0.14)
exposure · women		-0.081		-0.25		-0.14
		(0.16)		(0.20)		(0.19)
			Panel B: TSLS			
exposure	-0.039		-0.14		-0.082	
	(0.13)		(0.16)		(0.14)	
exposure · men		0.098		-0.006		0.054
		(0.12)		(0.15)		(0.14)
exposure · women		-0.13		-0.23		-0.18
		(0.16)		(0.19)		(0.18)
covariate-year	Y	Y	Y	Y		
prefecture *						
year FE					Y	Y
linear cohort trend			Y	Y		
5-year cohort dummy					Y	Y
y mean	0.30		0.30		0.30	
N	7,295		7,295		7,295	
F-statistic	1,980.4		4,532.0		4,872.0	
men		1,347.0		2,973.1		4,643.3
women		799.4		2,218.0		2,332.0
p-value	0.40	0.63	0.34	0.48	0.32	0.49

Notes: Table estimates the long-run responses of migration to exposure using data from the Mobility Survey. Migration is a binary outcome variable equal to one if the individual's current prefecture in prime age differs from her birth prefecture. I show OLS estimates from equation 2 in Panel A and two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in columns 1-2, I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in columns 3-4 and as a discrete change in levels for the 1961-1965 cohorts in columns 5-6. I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in columns 5-6. Results stratifying by gender also include prefecture-by-female fixed effects to account for gender differences in outcomes. The last rows show F-statistics from the first stage and p-values from specification tests under the null of exogenous exposure. Standard errors clustered at the level of prefectures in the parentheses.

Table A6: Long-run responses of migration across prefecture characteristics

	(1)	(2)	(3)	(4)	(5)	(6)
exposure · men	-0.011	-0.27	-0.014	-0.22	0.090	0.007
	(0.14)	(0.34)	(0.13)	(0.29)	(0.17)	(0.14)
exposure · women	-0.28	0.10	-0.16	-0.42	0.004	0.022
	(0.20)	(0.32)	(0.17)	(0.30)	(0.17)	(0.16)
birth prefecture						
1955 insurance rate	below 50%	above 50%				
1955 infant mortality			below 50%	above 50%		
current prefecture income					below 50%	above 50%
y mean	0.32	0.26	0.31	0.27	0.16	0.37
N	4,342	2,953	4,865	2,430	2,440	4,854

Notes: Table estimates the long-run migration responses across different prefecture characteristics using data from the Mobility Survey. Migration is a binary outcome variable equal to one if the individual's current prefecture in prime age differs from her birth prefecture. I estimate separate effects across birth prefectures with different insurance (column 1-2) and infant mortality rate (column 3-4) in 1955, and show similar heterogeneity based on the per capita GDP in the current prefecture in prime age (column 5-6). In each column, I focus on prefectures below or above the median prefecture for a given characteristic. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A7: Long-run impacts of exposure on all-cause and cancer mortality, low-migration sample

	(1)	(2)	(3)	(4)	(5)	(6)
	All-Cause			Cancer		
exposure · men	-0.98***	-1.09***	-1.13***	-0.97***	-1.09***	-1.06***
	(0.24)	(0.22)	(0.21)	(0.14)	(0.17)	(0.15)
exposure · women	-0.027	-0.13	-0.17	-0.18	-0.30	-0.27
	(0.19)	(0.19)	(0.17)	(0.13)	(0.15)	(0.14)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	3.36	3.36	3.36	1.12	1.12	1.12
N	8,388	8,388	8,388	8,388	8,388	8,388

Notes: Table estimates the long-run impacts of exposure on all-cause mortality and cancer mortality, excluding the top 10% prefectures with the highest migrant share (above 40%). I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A8: Long-run impacts of exposure on mortality from cardiovascular diseases and chronic conditions, low-migration sample

	(1)	(2)	(3)	(4)	(5)	(6)
	Cardiovascular			Diabetes/Hypertension		
exposure · men	0.064	0.090	0.063	0.019	0.009	0.012
	(0.092)	(0.095)	(0.091)	(0.034)	(0.034)	(0.034)
exposure · women	-0.025	0.002	-0.026	0.004	-0.005	-0.003
	(0.077)	(0.069)	(0.070)	(0.024)	(0.027)	(0.026)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	0.39	0.39	0.39	0.067	0.067	0.067
N	8,388	8,388	8,388	8,388	8,388	8,388

Notes: Table estimates the long-run impacts of exposure on mortality from cardiovascular diseases and chronic conditions, excluding the top 10% prefectures with the highest migrant share (above 43%) from the analysis. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A9: Long-run impacts of exposure on chronic conditions

	(1)	(2)	(3)	(4)
	Any Chronic Condition		Composite Index	
	Panel A: OLS			
exposure	-0.009		-0.028	
	(0.011)		(0.025)	
exposure · men		0.014		0.016
		(0.024)		(0.036)
exposure · women		-0.031***		-0.072***
		(0.011)		(0.024)
	Panel B: TSLS			
exposure	-0.013		-0.039	
	(0.013)		(0.029)	
exposure · men		0.010		0.009
		(0.016)		(0.040)
exposure · women		-0.036***		-0.086***
		(0.012)		(0.025)
y mean	0.057		0	
N	262,033		262,033	
F-statistic	1,575.8		1,575.8	
men		815.2		815.2
women		846.5		846.5
p-value	0.27	0.39	0.31	0.21

Notes: Table estimates the long-run impacts of exposure on the prevalence of chronic conditions (diabetes, obesity, hypertension, and cardiovascular diseases) in prime age. The composite index is the average of standardized prevalence rates (z-scores) across conditions. Panel A shows the OLS estimates from equation 2. Panel B shows two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. Results stratifying by gender also controls for prefecture-by-female fixed effects to account for gender differences in outcomes. The last rows show F-statistics from the first stage and p-values from specification tests under the null of exogenous exposure. Standard errors clustered at the level of prefectures in the parentheses.

Table A10: Long-run impacts of exposure on chronic conditions, excluding small expansion prefectures

	(1)	(2)	(3)	(4)
	Any Chronic Condition		Composite Index	
	Panel A: OLS			
exposure	-0.012		-0.027	
	(0.012)		(0.028)	
exposure · men		0.011		0.017
		(0.015)		(0.038)
exposure · women		-0.034***		-0.070***
		(0.012)		(0.025)
	Panel B: TSLS			
exposure	-0.018		-0.043	
	(0.014)		(0.031)	
exposure · men		0.005		0.003
		(0.017)		(0.043)
exposure · women		-0.040***		-0.089***
		(0.012)		(0.027)
y mean	0.057		0	
N	221,224		221,224	
F-statistic	1,840.5		1,840.5	
men		943.7		943.7
women		979.0		979.0
p-value	0.27	0.50	0.20	0.22

Notes: Table estimates the long-run impacts of exposure on the prevalence of chronic conditions (diabetes, obesity, hypertension, and cardiovascular diseases) in prime age. The composite index is the average of standardized prevalence rates (z-scores) across conditions. Different from the results in Table A9, the estimation sample excludes small expansion prefectures with near universal insurance (91% and above) prior to the reform in 1955. P Panel A shows the OLS estimates from equation 2. Panel B shows two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. Results stratifying by gender also controls for prefecture-by-female fixed effects to account for gender differences in outcomes. The last rows show F-statistics from the first stage and p-values from specification tests under the null of exogenous exposure. Standard errors clustered at the level of prefectures in the parentheses.

Table A11: Long-run impacts of exposure on chronic conditions, heterogeneity by exposure age

	(1)	(2)	(3)	(4)	(5)	(6)
	Any Chronic Condition			Composite Index		
exposure · men	0.006	0.008	0.010	0.002	0.006	0.009
	(0.011)	(0.012)	(0.016)	(0.026)	(0.031)	(0.040)
exposure · women	-0.027***	-0.028***	-0.036**	-0.063***	-0.068***	-0.086***
	(0.009)	(0.009)	(0.012)	(0.019)	(0.019)	(0.025)
exposure age	0-1	0-3	0-5	0-1	0-3	0-5
y mean	0.057	0.057	0.057	0	0	0
N	262,033	262,033	262,033	262,033	262,033	262,033

Notes: Table estimates the long-run impacts of exposure on the prevalence of chronic conditions when exposure is measured over different age bands in 0-5. The composite index is the average of standardized prevalence rates (z-scores) across conditions. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A12: Long-run impacts of exposure on diabetes and hypertension prevalence rates, alternative specification

	(1)	(2)	(3)	(4)	(5)	(6)
	Diabetes/Obesity			Hypertension/ Cardiovascular		
exposure · men	-0.003	0.012	-0.002	0.010	0.035**	0.003
	(0.010)	(0.010)	(0.010)	(0.012)	(0.015)	(0.013)
exposure · women	-0.024***	-0.01	-0.024***	-0.021*	0.004	-0.028**
	(0.008)	(0.009)	(0.009)	(0.011)	(0.016)	(0.012)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	0.019	0.019	0.019	0.043	0.043	0.043
N	262,033	262,033	262,033	262,033	262,033	262,033

Notes: Table estimates the long-run impacts of exposure on the prevalence of diabetes and hypertension conditions in prime age. I show two-stage-least-squares (TSLS) estimates of exposure where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A13: Long-run impacts of exposure on the prevalence of chronic conditions, alternative specification

	(1)	(2)	(3)	(4)	(5)	(6)
	Any Chronic Condition			Composite Index		
exposure · men	0.010	0.045**	0.005	0.009	0.078	-0.006
	(0.016)	(0.020)	(0.018)	(0.040)	(0.056)	(0.046)
exposure · women	-0.036***	-0.002	-0.041***	-0.086***	-0.019	-0.10***
	(0.012)	(0.017)	(0.013)	(0.025)	(0.040)	(0.027)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	0.057	0.057	0.057	0	0	0
N	262,033	262,033	262,033	262,033	262,033	262,033

Notes: Table estimates the long-run impacts of exposure on the prevalence of chronic conditions in prime age. The composite index is the average of standardized prevalence rates (z-scores) across conditions. I show two-stage-least-squares (TSLS) estimates of exposure where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A14: Long-run impacts of exposure on diabetes and hypertension prevalence rates, low-migration sample

	(1)	(2)	(3)	(4)	(5)	(6)
	Diabetes/Obesity			Hypertension/ Cardiovascular		
exposure · men	-0.003	0.009	-0.004	0.005	0.030*	0
	(0.011)	(0.011)	(0.011)	(0.012)	(0.015)	(0.012)
exposure · women	-0.028***	-0.017*	-0.030***	-0.014	0.01	-0.019
	(0.007)	(0.008)	(0.008)	(0.011)	(0.016)	(0.012)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	0.019	0.019	0.019	0.044	0.044	0.044
N	237,427	237,427	237,427	237,427	237,427	237,427

Notes: Table estimates the long-run impacts of exposure on the prevalence of diabetes and hypertension conditions excluding the top 10% prefectures with the highest migrant share (above 43%) from the analysis. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A15: Long-run impacts of exposure on the prevalence of chronic conditions, low-migration sample

	(1)	(2)	(3)	(4)	(5)	(6)
	Any Chronic Condition			Composite Index		
exposure · men	0.003	0.035*	-0.002	0.007	0.072	-0.002
	(0.017)	(0.019)	(0.018)	((0.045)	(0.062)	(0.051)
exposure · women	-0.036***	-0.005	-0.041***	-0.082***	-0.020	-0.091***
	(0.013)	(0.017)	(0.013)	(0.026)	(0.042)	(0.028)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	0.058	0.058	0.058	0	0	0
N	237,427	237,427	237,427	237,427	237,427	237,427

Notes: Table estimates the long-run impacts of exposure on the prevalence of chronic conditions, excluding the top 10% prefectures with the highest migrant share (above 43%) from the analysis. The composite index is the average of standardized prevalence rates (z-scores) across conditions. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A16: Long-run impacts of exposure on education and marital outcomes, excluding small expansion prefectures

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	High School		College Degree		Married to College-Educated Spouse		Married	
					Panel A: OLS			
exposure	0.11***		0.030		0.082***		0.061	
	(0.027)		(0.022)		(0.020)		(0.034)	
exposure · men		0.11***		-0.027		0.060***		0.093***
		(0.027)		(0.020)		(0.022)		(0.033)
exposure · women		0.11***		0.088***		0.10***		0.028
		(0.028)		(0.032)		(0.022)		(0.039)
					Panel B: TSLS			
exposure	0.16***		0.048*		0.099***		0.072**	
	(0.033)		(0.025)		(0.018)		(0.033)	
exposure · men		0.16***		-0.010		0.073***		0.10***
		(0.034)		(0.029)		(0.021)		(0.033)
exposure · women		0.16***		0.11***		0.13***		0.040
		(0.035)		(0.028)		(0.021)		(0.037)
y mean	0.94		0.26		0.19		0.77	
N	279,726		279,726		281,389		281,389	
F-statistic	1,396.7		1,396.7		1,400.6		1,400.6	
men		734.4		734.4		735.7		735.7
women		829.5		829.5		837.0		837.0
p-value	0.001	<0.001	0.34	0.64	0.052	0.14	0.19	0.42

Notes: Table estimates the long-run impacts of exposure on education and marital outcomes in prime age. The composite index is the average of standardized prevalence rates (z-scores) across conditions. Different from the results in Table 6, the estimation sample excludes small expansion prefectures with near universal insurance (91% and above) prior to the reform in 1955. P Panel A shows the OLS estimates from equation 2. Panel B shows two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. Results stratifying by gender also controls for prefecture-by-female fixed effects to account for gender differences in outcomes. The last rows show F-statistics from the first stage and p-values from specification tests under the null of exogenous exposure. Standard errors clustered at the level of prefectures in the parentheses.

Table A17: Long-run impacts of exposure on education attainment, heterogeneity by exposure age

	(1)	(2)	(3)	(4)	(5)	(6)
	High School			College Degree		
exposure · men	0.085***	0.099***	0.13***	-0.009	-0.011	-0.012
	(0.020)	(0.023)	(0.032)	(0.017)	(0.019)	(0.025)
exposure · women	0.082***	0.095***	0.12***	0.071***	0.082***	0.11***
	(0.021)	(0.024)	(0.033)	(0.020)	(0.021)	(0.026)
exposure age	0-1	0-3	0-5	0-1	0-3	0-5
y mean	0.94	0.94	0.94	0.26	0.26	0.26
N	329,502	329,502	329,502	329,502	329,502	329,502

Notes: Table estimates the long-run impacts of exposure on high school and college graduation rates when exposure is measured over different age bands in 0-5. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A18: Long-run impacts of exposure on marital outcomes, heterogeneity by exposure age

	(1)	(2)	(3)	(4)	(5)	(6)
	Married to College-Educated Spouse			Married		
exposure · men	0.061***	0.055***	0.066***	0.064**	0.073***	0.095***
	(0.016)	(0.015)	(0.018)	(0.023)	(0.024)	(0.030)
exposure · women	0.083***	0.092***	0.12***	0.017	0.023	0.030
	(0.016)	(0.016)	(0.020)	(0.024)	(0.027)	(0.033)
exposure age	0-1	0-3	0-5	0-1	0-3	0-5
y mean	0.19	0.19	0.19	0.78	0.78	0.78
N	331,397	331,397	331,397	331,397	331,397	331,397

Notes: Table estimates the long-run impacts of exposure on marital sorting by education when exposure is measured over different age bands in 0-5. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A19: Long-run impacts of exposure on education attainment, alternative specification

	(1)	(2)	(3)	(4)	(5)	(6)
	High School			College Degree		
exposure · men	0.13***	0.18***	0.16***	-0.012	0.008	-0.008
	(0.032)	(0.035)	(0.033)	(0.025)	(0.039)	(0.033)
exposure · women	0.12***	0.17***	0.15***	0.11***	0.13***	0.11***
	(0.033)	(0.036)	(0.035)	(0.026)	(0.036)	(0.030)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	0.94	0.94	0.94	0.26	0.26	0.26
N	329,502	329,502	329,502	329,502	329,502	329,502

Notes: Table estimates the long-run impacts of exposure on high school and college graduation rates. I show two-stage-least-squares (TSLS) estimates of exposure where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A20: Long-run impacts of exposure on marital outcomes, alternative specification

	(1)	(2)	(3)	(4)	(5)	(6)
	Married to College-Educated Spouse			Married		
exposure · men	0.066***	0.080***	0.063***	0.095***	0.14***	0.11***
	(0.018)	(0.027)	(0.021)	(0.030)	(0.036)	(0.034)
exposure · women	0.12***	0.13***	0.12***	0.030	0.077*	0.042
	(0.020)	(0.029)	(0.021)	(0.033)	(0.038)	(0.036)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	0.19	0.19	0.19	0.78	0.78	0.78
N	331,397	331,397	331,397	331,397	331,397	331,397

Notes: Table estimates the long-run impacts of exposure on marital sorting by education. I show two-stage-least-squares (TSLS) estimates of exposure where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A21: Long-run impacts of exposure on education attainment, low-migration sample

	(1)	(2)	(3)	(4)	(5)	(6)
	High School			College Degree		
exposure · men	0.15***	0.19***	0.18***	-0.024	-0.006	-0.018
	(0.038)	(0.040)	(0.039)	(0.030)	(0.046)	(0.039)
exposure · women	0.14***	0.19***	0.17***	0.071***	0.087**	0.075**
	(0.039)	(0.042)	(0.041)	(0.025)	(0.036)	(0.028)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	0.94	0.94	0.94	0.25	0.25	0.25
N	294,075	294,075	294,075	294,075	294,075	294,075

Notes: Table estimates the long-run impacts of exposure on high school and college graduation rates, excluding the top 10% prefectures with the highest migrant share (above 43%) from the analysis. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A22: Long-run impacts of exposure on marital outcomes, low-migration sample

	(1)	(2)	(3)	(4)	(5)	(6)
	Married to College-Educated Spouse			Married		
exposure · men	0.067***	0.087***	0.067***	0.10***	0.14***	0.11***
	(0.020)	(0.029)	(0.023)	(0.034)	(0.041)	(0.039)
exposure · women	0.13***	0.15***	0.13***	0.018	0.056	0.024
	(0.020)	(0.026)	(0.020)	(0.038)	(0.041)	(0.042)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	0.18	0.18	0.18	0.77	0.77	0.77
N	295,662	295,662	295,662	295,662	295,662	295,662

Notes: Table estimates the long-run impacts of exposure on marital sorting by education, excluding the top 10% prefectures with the highest migrant share (above 43%) from the analysis. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A23: Long-run impacts of exposure on employment, excluding small expansion prefectures

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Employment		Home Production		Log Earning, Personal		Log Earning, Household	
					Panel A: OLS			
exposure	0.024		0.004		-0.044		0.029	
	(0.024)		(0.029)		(0.17)		(0.098)	
exposure · men		-0.11***		0.092***		-0.90***		-0.064
		(0.027)		(0.019)		(0.14)		(0.10)
exposure · women		0.16***		-0.087**		0.83***		0.12
		(0.026)		(0.041)		(0.21)		(0.12)
					Panel B: TSLS			
exposure	0.017		0.006		-0.030		0.044	
	(0.028)		(0.028)		(0.19)		(0.098)	
exposure · men		-0.12***		0.092***		-0.88***		-0.045
		(0.028)		(0.020)		(0.15)		(0.11)
exposure · women		0.15***		-0.083**		0.84***		0.13
		(0.031)		(0.041)		(0.25)		(0.12)
y mean	0.69		0.12		4.71		5.82	
N	282,071		282,071		282,332		282,332	
F-statistic	1,399.8		1,399.8		1,399.3		1,399.3	
men		735.1		735.1		736.3		736.3
women		836.2		836.2		835.1		835.1
p-value	0.55	0.83	0.62	0.56	0.69	0.93	0.64	0.60

Notes: Table estimates the long-run impacts of exposure on employment and earnings in prime age. Different from the results in Table 7, the estimation sample excludes small expansion prefectures with near universal insurance (91% and above) prior to the reform in 1955. P Panel A shows the OLS estimates from equation 2. Panel B shows two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. Results stratifying by gender also controls for prefecture-by-female fixed effects to account for gender differences in outcomes. The last rows show F-statistics from the first stage and p-values from specification tests under the null of exogenous exposure. Standard errors clustered at the level of prefectures in the parentheses.

Table A24: Long-run impacts of exposure on employment, heterogeneity by exposure age

	(1)	(2)	(3)	(4)	(5)	(6)
	Full-Time Employed			Home Production		
exposure · men	-0.084***	-0.091***	-0.12***	0.071***	0.074***	0.095***
	(0.016)	(0.018)	(0.025)	(0.012)	(0.013)	(0.016)
exposure · women	0.095***	0.12***	0.16***	-0.045*	-0.061**	-0.083**
	(0.019)	(0.021)	(0.028)	(0.023)	(0.029)	(0.038)
exposure age	0-1	0-3	0-5	0-1	0-3	0-5
y mean	0.69	0.69	0.69	0.12	0.12	0.12
N	332,157	332,157	332,157	332,157	332,157	332,157

Notes: Table estimates the long-run impacts of exposure on full-time employment and home production when exposure is measured over different age bands in 0-5. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A25: Long-run impacts of exposure on earnings, heterogeneity by exposure age

	(1)	(2)	(3)	(4)	(5)	(6)
	Log Earnings, Personal			Log Earnings, Household		
exposure · men	-0.62***	-0.69***	-0.90***	-0.010	-0.015	-0.022
	(0.088)	(0.097)	(0.13)	(0.070)	(0.072)	(0.093)
exposure · women	0.48***	0.62***	0.84***	0.093	0.12	0.16
	(0.12)	(0.16)	(0.22)	(0.082)	(0.085)	(0.11)
exposure age	0-1	0-3	0-5	0-1	0-3	0-5
y mean	4.72	4.72	4.72	5.82	5.82	5.82
N	332,449	332,449	332,449	332,449	332,449	332,449

Notes: Table estimates the long-run impacts of exposure on personal and household earnings when exposure is measured over different age bands in 0-5. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A26: Long-run impacts of exposure on employment, alternative specification

	(1)	(2)	(3)	(4)	(5)	(6)
	Full-Time Employed			Home Production		
exposure · men	-0.12***	-0.11***	-0.097***	0.095***	0.075***	0.071***
	(0.025)	(0.032)	(0.035)	(0.016)	(0.023)	(0.026)
exposure · women	0.16***	0.17***	0.18***	-0.083**	-0.10**	-0.11**
	(0.028)	(0.033)	(0.035)	(0.038)	(0.045)	(0.048)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	0.69	0.69	0.69	0.12	0.12	0.12
N	332,157	332,157	332,157	332,157	332,157	332,157

Notes: Table estimates the long-run impacts of exposure on employment. I show two-stage-least-squares (TSLS) estimates of exposure where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A27: Long-run impacts of exposure on earnings, alternative specification

	(1)	(2)	(3)	(4)	(5)	(6)
	Log Earnings, Personal			Log Earnings, Household		
exposure · men	-0.90***	-0.83***	-0.77***	-0.022	-0.061	0.003
	(0.13)	(0.17)	(0.19)	(0.093)	(0.10)	(0.093)
exposure · women	0.84***	0.91***	0.98***	0.16	0.12	0.18*
	(0.22)	(0.27)	(0.29)	(0.11)	(0.12)	(0.11)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	4.72	4.72	4.72	5.82	5.82	5.82
N	332,449	332,449	332,449	332,449	332,449	332,449

Notes: Table estimates the long-run impacts of exposure on personal and household earnings. I show two-stage-least-squares (TSLS) estimates of exposure where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A28: Long-run impacts of exposure on employment, low-migration sample

	(1)	(2)	(3)	(4)	(5)	(6)
	Full-Time Employed			Home Production		
exposure · men	-0.13***	-0.11***	-0.11***	0.10***	0.083***	0.086***
	(0.028)	(0.037)	(0.039)	(0.018)	(0.025)	(0.027)
exposure · women	0.14***	0.15***	0.15***	-0.066	-0.084*	-0.082
	(0.030)	(0.035)	(0.036)	(0.042)	(0.049)	(0.052)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	0.70	0.70	0.70	0.12	0.12	0.12
N	296,337	296,338	296,339	296,337	296,337	296,337

Notes: Table estimates the long-run impacts of exposure on employment, excluding the top 10% prefectures with the highest migrant share (above 43%) from the analysis. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.

Table A29: Long-run impacts of exposure on earnings, low-migration sample

	(1)	(2)	(3)	(4)	(5)	(6)
	Log Earnings, Personal			Log Earnings, Household		
exposure · men	-0.95***	-0.94***	-0.88***	-0.074	-0.16	-0.088
	(0.14)	(0.18)	(0.21)	(0.099)	(0.095)	(0.097)
exposure · women	0.71***	0.72***	0.78***	0.092	0.005	0.081
	(0.22)	(0.26)	(0.29)	(0.12)	(0.12)	(0.11)
covariate-year	Y	Y		Y	Y	
prefecture *						
year FE			Y			Y
linear cohort trend		Y			Y	
5-year cohort dummy			Y			Y
y mean	4.72	4.72	4.72	5.79	5.79	5.79
N	296,596	296,596	296,596	296,596	296,596	296,596

Notes: Table estimates the long-run impacts of exposure on personal and household earnings, excluding the top 10% prefectures with the highest migrant share (above 43%) from the analysis. I show two-stage-least-squares (TSLS) estimates from equation 6 where endogenous exposure is instrumented by a simulated measure constructed under a linear growth path of insurance in 1956-1961. In addition to basic controls in column (1), I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column (2) and as a discrete change in levels for the 1961-1965 cohorts in column (3). I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column (3). All specifications control for gender differences in outcomes with prefecture-by-female fixed effects. Standard errors clustered at the level of prefectures in the parentheses.