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 eligible subgroups based on income or clinical outcomes. Universal insurance thus represents a larger investment shock and may have broader impacts on inequalities than narrowly targeted interventions. 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.

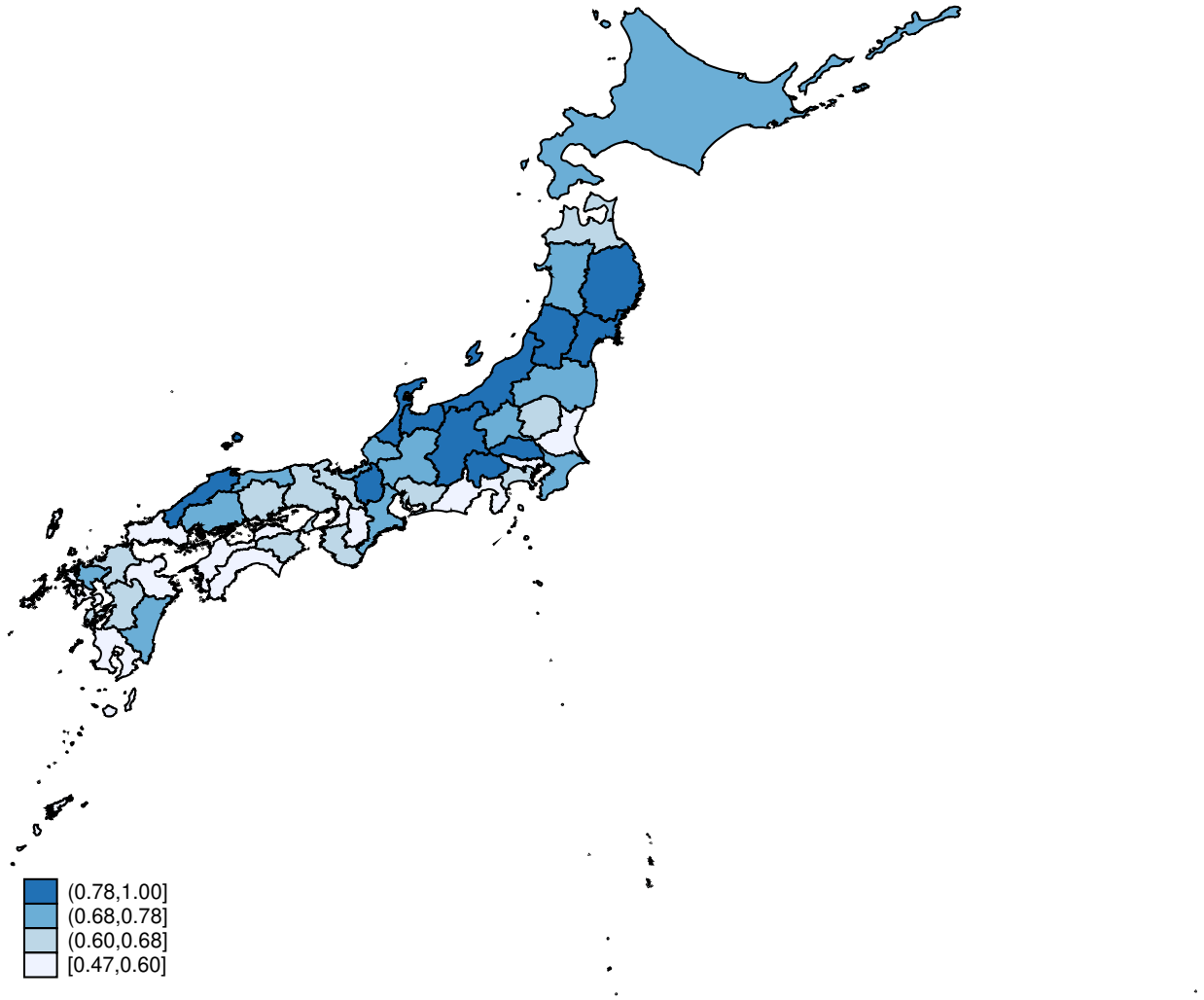
Figure 1 shows the pre-reform insurance rate across prefectures in 1955. The median prefecture had an insurance rate of 68%. In the least insured prefectures such as Kagoshima and Kochi, less than 50% of the population had insurance. In Osaka, Shizuoka, Yamaguchi, and Tokyo, less than 60% had insurance. 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.

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

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.

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.

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

[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.

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 (Barker 1990; 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,

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

where the long-run outcome, y_{iatp}, is regressed on $exposure_{iatp}$ with fixed effects of indi-

vidual 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 $\psi_t \cdot X_p$. 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

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

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 show that similar results hold in specifications with more flexible controls of cohort differences and long-run trending across prefectures.

4 Results

4.1 Mortality

Table 2 examines the impact of exposure on mortality in prime age. According to OLS estimates in Panel A, gaining a ten percentage point exposure reduced all-cause mortality by 0.06 per 10 thousand individuals. This effect is nearly fully driven by the mortality reduction in cancer, the leading cause of death in prime age. In Panel B, TSLS estimates from simulated exposure show very similar effects. Since the prediction coefficient in the first stage is fairly close to one, the similarity between OLS and TSLS estimates suggests that exposure is mainly induced by the insurance reform and is plausibly exogenous to alternative determinants of outcome.

Across gender, expansion had larger impacts on men's mortality in prime age, with a ten percentage point exposure reducing mortality by 0.1 per 10,000 individuals, or by 2.3% of the sample average for men. By comparison, the exposure impact on women's

mortality was small and insignificant. Across disease conditions, around 88% (0.99/1.13) of men's mortality reduction was from cancer-related deaths. Apart from cancer, however, exposure had no significant impact on alternative causes of death such as cardiovascular diseases or chronic conditions in prime age.

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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	All-Cause		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)
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
y mean	3.32		1.11		0.40		0.065	
N	9,188		9,188		9,188		9,188	

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 additionally stratified 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. Standard errors clustered at the level of prefectures in the parentheses.

I next explore effect heterogeneity across exposure age in Table 3. This is meaningful because long-run outcomes may be more responsive to exposure in the first few years of life, with smaller impacts from additional exposure at older ages. Across age bands, I find that exposure in age 0-1 already led to substantial reductions in all-cause mortality in prime age, and the effect could account for 91% of the exposure impact by age 5. For

cancer-related deaths, while additional exposure above age 3 led to further mortality reductions for men, exposure from utero till age 3 could account for nearly 80% of the exposure impact by age 5.

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 while varying the exposure index across 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.

Robustness. For robustness checks, I estimate alternative specifications that more flexibly control for cohort differences and prefecture-level trending over the long run. In Appendix Table A2, I introduce linear cohort trends in column 2 and further control for prefecture-year effects in addition to cohort differences in column 3. Both specifications indicate fairly similar if somewhat larger impacts of exposure on mortality, with nearly 90% of the reduction driven by cancer-related deaths (column 4-6). By contrast, exposure had consistently small and insignificant impacts on mortality from cardiovascular diseases and chronic conditions in prime age (Appendix Table A3).

4.2 Migration

One concern with the long-run estimates is that migration could result in measurement error in exposure and introduce selection biases to estimates. This might occur if exposure

affected the overall migration rates 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 in less covered prefectures, then the long-run impact on health could be under-estimated due to changes in the health stock of individuals. Moreover, if exposure increased the migration to high-income prefectures in the long run, then the economic impact of exposure could be over-estimated due to positive selection on outcomes.

To empirically assess the potential bias from migration, I use migration data from the Mobility Survey to construct a binary outcome variable equal to one if the individual's current prefecture differs from the prefecture at birth. Across specifications, I find that exposure had no significant impact on migration by prime age (Appendix Table A4). To explore differential migration, in Appendix Table A5, I estimate exposure impacts across the 1955 insurance rate and infant mortality rate in the birth prefecture and continue to find insignificant migration responses along these characteristics (column 1-4). Moreover, in the long run, migration was also unrelated to the per capita income in the prefecture in prime age. (column 5-6).

In a more direct test, I examine the robustness of results after excluding prefectures with the highest share of migrants from the analysis. In the event that long-run impacts mainly operated through migration, dropping the high-migration prefectures would lead to material changes in the estimated impacts of exposure.[2] In Appendix Table A6, I show that dropping the top 10% prefectures with the highest migrant share indicates very similar reductions in all-cause mortality and cancer mortality in prime age. The mortality reduction in cardiovascular diseases and chronic conditions remains small and insignificant (Appendix Table A7). These patterns suggest that migration is unlikely to substantially bias the long-run impacts in this context.

[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%).

4.3 Disease Conditions

Table 4 estimates the exposure impact on the prevalence of disease conditions in prime age. According to OLS estimates in Panel A, insurance exposure reduced metabolic conditions (indicated by diabetes or obesity) but had no significant impact on circulatory conditions (hypertension or cardiovascular), stroke, or cancer. TSLS estimates show fairly similar effects in Panel B. In particular, gaining a ten percentage point exposure reduced diabetes/obesity prevalence rates by 0.14 percentage points, or by 7.4% below the mean. This effect is more concentrated in women whereas the effect on men was smaller and not statistically significant (column 2, Panel B). The gender-specific effects also indicate a marginally significant reduction in women's hypertension/cardiovascular conditions (column 4, Panel B). Compared to chronic conditions, exposure had no significant impact on the prevalence of more severe conditions such as stroke or cancer in prime age.

Focusing on chronic conditions, I estimate the exposure impact on having any chronic condition (diabetes, obesity, hypertension, or cardiovascular) in Appendix Table A8. I also examine a summary index based on the standardized z-score of prevalence rates averaged across conditions (Anderson 2008; Boudreaux *et al.* 2016; Miller and Wherry 2019). Both measures indicate substantial reductions in chronic conditions for women, with a ten percentage point exposure reducing the prevalence of any condition by 0.36 percentage points and the summary index by roughly 0.01 standard deviation. By comparison, the exposure impacts for men were small and indistinguishable from zero.

Table 5 estimates heterogeneous effects across exposure age. While additional exposure led to further reductions in chronic conditions, a large portion of the benefit was driven by exposure in utero and the first year of life (age 0-1). For diabetes and obesity, in particular, exposure in age 0-1 accounted for roughly 70% of the exposure impact by age 5. Similar patterns apply to the summary index in Appendix Table A9, with exposure in age 0-1 accounting for 73% of the reduction in the standardized z-score by age 5. These effects were fully concentrated in women with small and insignificant effects on men.

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)
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
y mean	0.019		0.043		0.005		0.002	
N	262,033		262,033		262,033		262,033	

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. Standard errors clustered at the level of prefectures in the parentheses.

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 while varying the exposure index across 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 examine the robustness of results to alternative specifications in Appendix Table A10. Controlling for cohort differences as well as prefecture-year effects, for instance, indicates similar reductions in prevalence rates as the main estimate, whereas results appear attenuated under linear cohort trends in column 2. In the low-migration sample, the exposure impact on diabetes/obesity increased slightly and the impact on hypertension/cardiovascular conditions was insignificant (Appendix Table A11). These patterns also hold for summary measures of chronic conditions, where the exposure impact was comparable if larger under additional controls of cohort and prefecture-year effects but was attenuated under linear cohort trends in the regression (Appendix Table A12 and A13).

4.4 Education

I next examine the exposure impacts on education attainment in Table 6. Compared to OLS estimates, TSLS estimates are larger in magnitude and indicate substantial differences by gender. In column 1-2, gaining a ten percentage point exposure increased high school graduation rates for both gender by roughly 1.2 percentage points. The education gains led to further increases in college graduation for women, whereas any additional impact on men's education beyond high school was small and insignificant (column 2 and 4). Compared to the sample average (13.6% for women vs 38.3% for men, cf Table 1), the differential impact of exposure reduced the gender gap in college education by 4.5%.

Column 5-8 examines the marital outcome between gender and education groups. For women, exposure increased her probability of marrying a college-educated spouse by 1.2 percentage points without affecting the overall marriage rate of women (column 6 and 8). For men, by contrast, exposure significantly increased marriage rate on the extensive margin but had smaller impact on the spouse's college education. These differences are consistent with increases in own and spousal education resulting in greater match quality for women's marital outcomes.

Across exposure age, increasing exposure in age 0-1 by ten percentage points can already increase high school graduation rates by 0.8 percentage points, or by two-thirds of the effect by age 5 (Appendix Table A14). Further exposure ultimately increased women's college graduation but did not affect men's education beyond high school. Moreover, exposure in age 0-1 substantially increased spousal education for women and the marriage rate of men, and both effects strengthen with additional exposure in older ages (Appendix Table A15).

Robustness. Appendix Table A16 examines the robustness of the education increase to alternative specifications. Controlling for cohort differences as well as prefecture-year effects, for instance, would indicate larger increases in high school graduation rates and a similar effect on college. Outside the top 10% prefectures with the highest migrant share,

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)
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
y mean	0.94		0.26		0.19		0.78	
N	329,502		329,502		331,397		331,397	

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. Standard errors clustered at the level of prefectures in the parentheses.

the exposure impact on high school graduation becomes larger whereas the impact on college is smaller but remains significant at conventional levels (Appendix Table A17). For marital outcomes, the increase in spousal education for women remains comparable across specifications in the full and low-migration sample (Appendix Table A18 and A19).

4.5 Employment and Earnings

Exposure also impacted employment and earnings across gender in Table 7. For women, gaining a ten percentage point exposure increased full-time employment by 1.6 percentage points and reduced the share in home production by 0.8 percentage points. Contrary to the employment gains for women, exposure reduced men's full-time employment while shifting home production from women to men in households (column 2 and 4). Due to the employment change, earnings increased for women by 8.4% but decreased for men (column 6). The relative rise in women's employment, moreover, did not affect the joint earnings of spouses in households (column 8).

These results show that exposure to universal insurance increased women's economic participation and resources relative to men. Specifically, gaining a ten percentage point exposure reduced the employment gap between gender by 5.7% and reduced the earnings gap by 8.3% without reducing the household labor supply or earnings.[3] These effects grew larger with additional exposure at older ages, but exposure in age 0-1 already led to large increases in women's employment relative to men, reducing the gender employment gap by 3.6% (Appendix Table A20) and the earnings gaps by 5.2% (Appendix Table A21).

Robustness. The differential increase in women's employment is robust to controls of long-run trending that may represent shifts in the societal norm towards gender. Controlling for cohort and prefecture-year effects, for instance, would indicate a 5.6% reduction in the gender employment gap from a ten percentage point exposure (column 3, Appendix Table

[3] In detail, estimates from column 2 of Table 7 suggest a smaller employment gap by 1.2+1.6=2.8 percentage points from the exposure, or 5.7% of the baseline differences by gender (49.2% in Table 1). Similar calculation for log earnings follows from estimates in column 6 of Table 7.

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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Full-Time Employed		Home Production		Log Earnings, Personal		Log Earnings, 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)
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
y mean	0.69		0.12		4.72		5.82	
N	332,157		332,157		332,449		332,449	

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. Standard errors clustered at the level of prefectures in the parentheses.

A22) with a corresponding 5.3% reduction in the low-migration sample (Appendix Table A23). Both effects are comparable to the 5.7% reduction from Table 7. For earnings, similar increases in the exposure would reduce the gender gap by 8.3% (column 3, Appendix Table A24) and reduce the gap by 7.9% in the low-migration sample (Appendix Table A25), both comparable to the main result (8.3% reduction) from Table 7.

5 Discussion

5.1 Effect Sizes

To assess the plausibility of the health effects, I compare the health gains in Japan with effects from other countries expanding insurance during the same historic period. One possible comparison is with the onset of Medicaid in the US, a public insurance program that began covering low-income individuals in 1966. While Medicaid offers means-tested rather than universal insurance, the onset of Medicaid in 1966 was only five years after the universal insurance in Japan. Thus, long-run impacts of both programs are based on cohorts gaining exposure in the 1950s-1960s. To the extent that health and health technology did not differ substantially between the two countries, one might expect similar impacts of exposure on mortality and disease conditions in prime age.

Using an index of disease conditions, Boudreaux *et al.* (2016) finds that gaining an additional year of Medicaid exposure in age 0-5 reduced chronic conditions by 0.011 standard deviation in age 25-43. This effect is comparable to the estimate in Japan, where a similar increase in the exposure to universal insurance would lower the index by 0.007 standard deviation on average and by 0.014 for women (1/6*0.086=0.014, Appendix Table A8). For mortality, a one year increase in exposure reduced mortality by 0.1 per 10,000 individuals in Japan, or by 3.0% below the mean (Table 2). This effect is comparable to but less than the 6%-8% mortality reduction for treated Medicaid children in Goodman-Bacon (2021).

Goodman-Bacon (2021) also estimates a 2.7% increase in high school graduation rate and a 4.0% increase in college degree for an additional year of Medicaid exposure. These effects are broadly in line with the exposure effects in Japan.[4] While the return to higher education would depend on the labor market conditions specific to a country, similar increases in education have also been found in recent insurance reforms across high- and low-income countries (Brown *et al.* 2020; Huang and Liu 2023).

5.2 Health Impacts Over the Life Cycle

While one has yet to observe mortality outcomes in older ages, the health improvements in prime age already differ in important ways from the short-term impacts of universal insurance. In particular, the reduction in cancer mortality in prime age contrasts with findings from Kondo and Shigeoka (2013) that universal insurance had no immediate impact on age-specific mortality by 1970. Moreover, less acute outcomes such as chronic conditions also improved in prime age and could subsequently affect mortality later in life. Such effects would be consistent with evidence linking early-life investments with old-age mortality (Van den Berg *et al.* 2006; Arpino *et al.* 2018) and the health of future generations (Bhalotra and Rawlings 2013; East *et al.* 2017), but evidence from universal insurance is not yet observable even in countries achieving universal insurance in the 1950s. Thus, continued follow-up of the exposure cohorts is necessary to fully understand the impacts on population health and the spillover from parents to children.

5.3 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

[4] In detail, estimates from Table 6 show that gaining one year of exposure to universal insurance increased high school graduation rates by 2.3% and increased college degree by 3.0%.

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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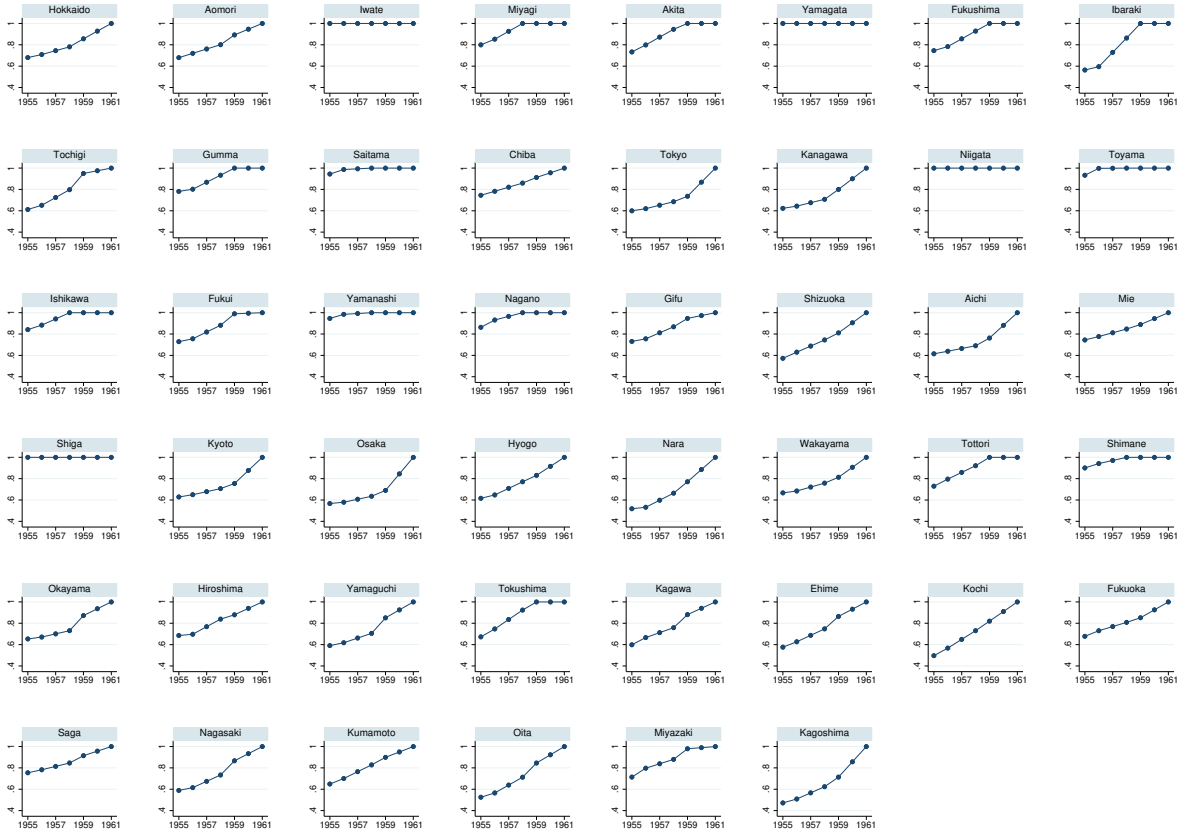
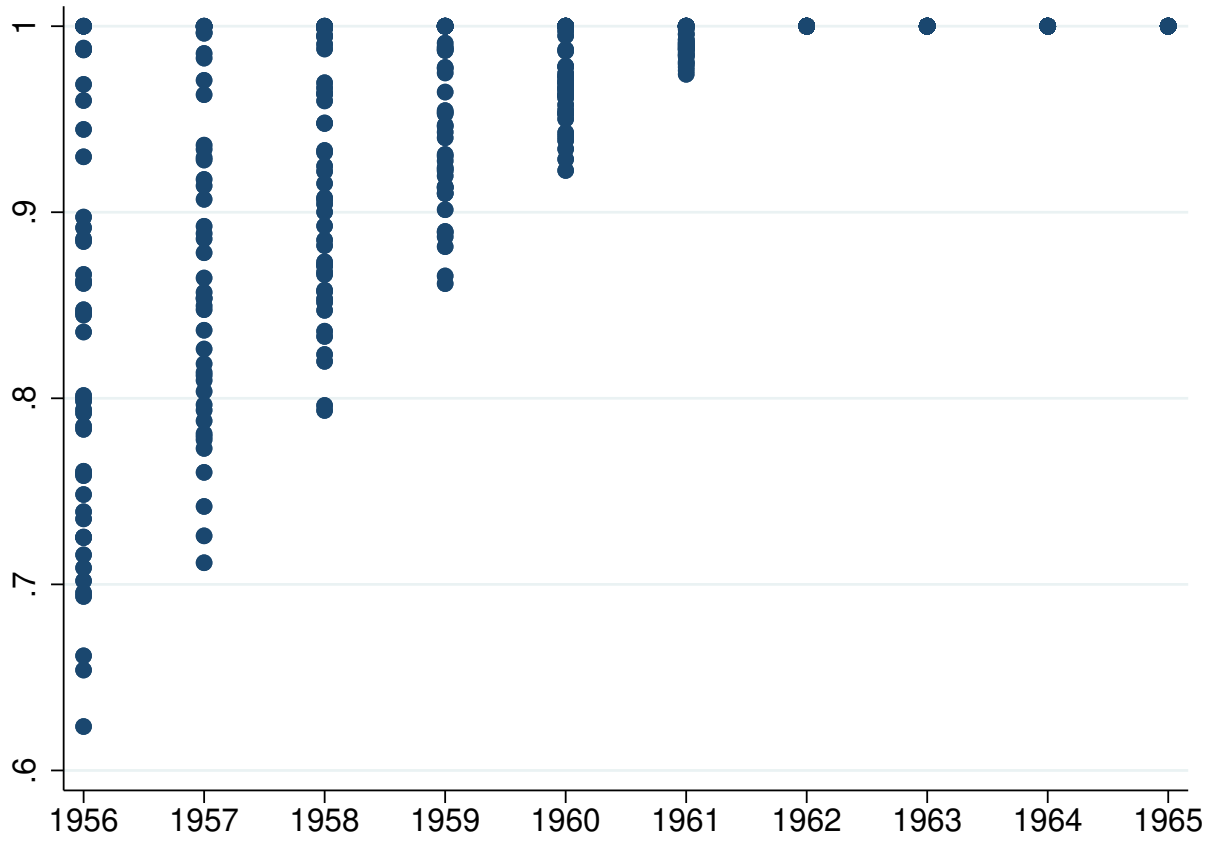
A Appendix Figures

Figure A1: Insurance expansion across prefectures in 1955-1961



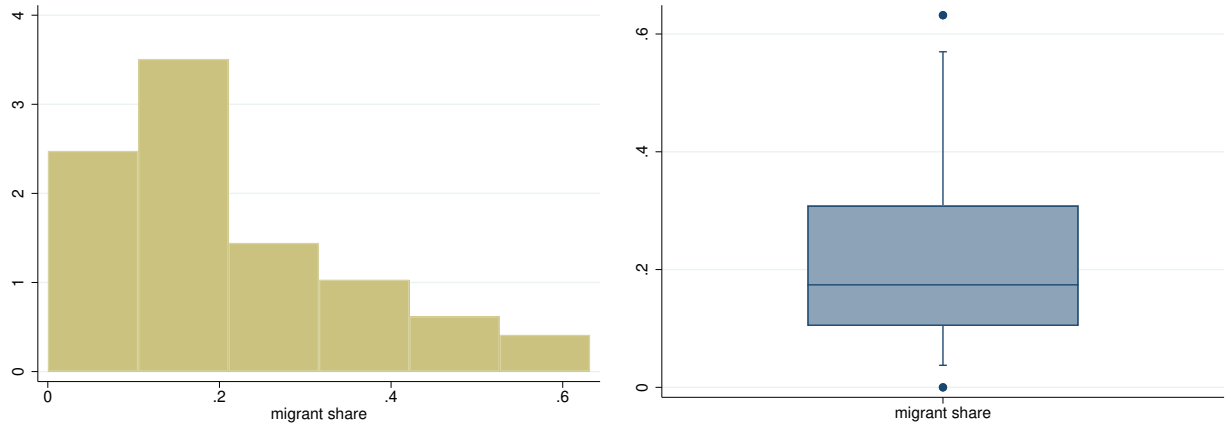
Notes: Figure plots the trend of insurance rates in 1955-1961 in each of the 47 prefectures in Japan. Prefectures differed in the pre-reform insurance rate in 1955 and the incremental expansion each year during the reform period in 1956-1961. The variations in the expansions over time are illustrated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

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, 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 A3: 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 A4: Long-run responses of migration to exposure

	(1)	(2)	(3)	(4)	(5)	(6)
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	

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 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 1-2, I allow prefectures to differ in the birth cohort effects modeled as a linear time trend in birth year in column 3-4 and as a discrete change in levels for the 1961-1965 cohorts in column 5-6. I further include a full set of prefecture-year effects to absorb unobserved determinants of outcome over time in column 5-6. Results stratifying by gender also include prefecture-by-female fixed effects to account for gender differences in outcomes. Standard errors clustered at the level of prefectures in the parentheses.

Table A5: 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 A6: 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 A7: 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 A8: Long-run impacts of exposure on chronic conditions

	(1)	(2)	(3)	(4)
	Any Chronic Condition		Summary 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)
F-statistic	1,575.8		1,575.8	
men		815.2		815.2
women		846.5		846.5
y mean	0.057		0	
N	262,033		262,033	

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 summary index is the average of standardized prevalence rates (z-scores) across conditions. 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 controls for prefecture-by-female fixed effects to account for gender differences in outcomes. Standard errors clustered at the level of prefectures in the parentheses.

Table A9: Long-run impacts of exposure on chronic conditions, heterogeneity by exposure age

	(1)	(2)	(3)	(4)	(5)	(6)
	Any Chronic Condition			Summary 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 while varying the exposure index across age bands in 0-5. The summary 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 A10: 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 A11: 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 A12: Long-run impacts of exposure on the prevalence of chronic conditions, alternative specification

	(1)	(2)	(3)	(4)	(5)	(6)
	Any Chronic Condition			Summary 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 summary 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 A13: Long-run impacts of exposure on the prevalence of chronic conditions, low-migration sample

	(1)	(2)	(3)	(4)	(5)	(6)
	Any Chronic Condition			Summary 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 summary 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 A14: Long-run impacts of exposure on high school and college graduation rates, 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 while varying the exposure index across 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 A15: Long-run impacts of exposure on marital outcomes by education, 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 outcomes between gender and education groups while varying the exposure index across 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 A16: Long-run impacts of exposure on high school and college graduation rates, 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 A17: Long-run impacts of exposure on high school and college graduation rates, 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 A18: Long-run impacts of exposure on marital outcomes by education, 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 outcomes between gender and education groups. 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 A19: Long-run impacts of exposure on marital outcomes by education, 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 outcomes by education groups, 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 A20: 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 while varying the exposure index across 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 A21: 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 while varying the exposure index across 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 A22: 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 A23: 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 A24: 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 A25: 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.