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Three Essays on the Short-term, Long-term, and Inter-generational Effects of Environmental Changes on Health Outcomes

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Three Essays on the Short-term, Long-term, and Inter-generational Effects of Environmental Changes on Health Outcomes

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Abstract

This dissertation studies the short-term, long-term, and inter-generational effects of environmental changes on health outcomes. In the first chapter, I use China's "Home Appliances Going to the Country-side" policy to study the effects of the spread of household electronic appliances in rural areas on body weight outcomes and on behaviors associated with caloric intake and caloric expenditure. The analysis is based on the China Health and Nutrition Survey (CHNS). Using data from waves 2004, 2006, and 2009 and difference-in-differences and instrumental-variable approaches, I find that household technology increases the likelihood of obesity among female adults, due to more caloric intake and less caloric expenditure.

In the second chapter, I use unique and comprehensive data from Taiwan to extend the existing literature to the long-term effects of malaria exposure on health outcomes. Malaria prevalence in Taiwan dropped sharply in the 1950s due to the DDT-based eradication campaign from 1952 to 1957. Given that areas with greater changes in malaria prevalence should benefit more from the eradication campaign, I use a difference-in-differences approach to estimate the impact of malaria eradication on future health, education, and labor outcomes in areas with high pre-eradication malaria prevalence. In this study, I find significant long-term effects of early life malaria exposure on future outcomes: less early life malaria exposure improves educational attainment, and lowers the likelihood of getting heart disease, stroke, and renal failure for women, and lowers the likelihood of getting stroke and cerebral palsy for men.

In the third chapter, I use the Great Chinese Famine of 1959--1961 to examine the

effects of *in utero* exposure to famine on the health outcomes of offspring. In particular, I focus on the weight and height outcomes of children whose mothers were exposed to famine while *in utero*. The results indicate that daughters of famine survivors have a lower z-score of body mass index (BMI) than daughters of parents who were not exposed to famine. I do not find any impact on BMI for sons.

1. The Effect of Household Technology on Obesity and Weight Gain among Chinese Female Adults: Evidence from China's "Home Appliances Going to the Countryside" Policy

1.1 Introduction and Literature Review

For the first half of the twentieth century, many societies were fighting malnutrition, food scarcity, and infectious diseases. Increasing one's body weight was thus important for survival and productivity (Fogel, 1994). However, the new pandemic of obesity and its accompanying problem of non-communicable diseases originated in the U.S. have spread to many other rich countries at an alarming rate in the last thirty years (WHO, 2000). The pandemic is also spreading to many developing countries, such as Mexico, China, and Thailand (Popkin and Gordon-Larsen, 2004). What is even more worrisome is that, while the communicable diseases are not yet eradicated, the increase in obesity that is linked to many non-communicable diseases is rising at a faster rate in those developing countries than in developed countries. For example, a recent meta-analysis of nationally representative data by Wang et al. (2007) estimated that the proportion of the population that is overweight or obsess (BMI $\geq$ 24) in China increased by 49.5%, from 20.0% to 29.9%, between 1992 and 2002.[1]

At the simplest level, weight gain is caused by more energy intake than energy

[1] A growing body of evidence suggests that Asian people have a higher risk for obesity-related diseases for any given BMI (e.g. Tuan et al., 2008). Thus, on the basis of a meta-analysis of associations of BMI with risk factors for cardiovascular disease among 240,000 Chinese adults, and from the longitudinal relationships of BMI to cardiovascular events in 76,000 participants, the Working Group on Obesity in China has recommended that a BMI of 18.5 to 23.9 should be considered as optimal, 24.0 to 27.9 as overweight, and 28.0 and above as obese (Cooperative Meta-analysis Group of the Working Group on Obesity in China, 2002).

expenditure. Obesity is viewed as the physiological response to an individual's interaction with his or her physical, economic, political, and socio-cultural environment (Sallis et al., 2006). These environments collectively reinforce ways of living to either promote or inhibit energy balance at the individual level. China, while enjoying a same fast-growing economy in the past two decades that was experienced by developed countries decades ago, now faces the concurrent technological advancements and globalization that were not faced by developed countries when they were in the same economic transition decades ago. These developments create for China a very unique obesogenic environment. In this paper, we examine the effects of the spread of household electronic appliances, byproducts of economic growth, technological development, and globalization, on weight outcomes in rural areas in China.

Our study contributes to two streams of the obesity literature. First, a large body of literature by economists has studied the effects of various environmental changes, predominantly in developed countries, that may affect energy intake or energy expenditure. The factors that lead to increase in energy intake include, for example, the prevalence of fast food restaurants (e.g., Cutler et al., 2003; Chou et al., 2004; Currie et al., 2010; Anderson and Matsa, 2011) and the decline in food prices (e.g., Chou et al., 2004). Other factors that affect energy expenditure include increasingly sedentary employment (e.g., Philipson and Posner, 2003; Lakdawalla and Philipson, 2007) and urban sprawl (e.g., Zhao and Kaestner, 2010). However, the growing use of energy-saving devices that profoundly change people's daily routines has not received sufficient attention in the obesity literature. For example, house cleaning has been automated through the use of dishwashers, clothes-washing machines, and vacuum cleaners. Food preparation has become less labor-intensive through the use of

microwaves and refrigerators. However, there is very little evidence showing the impact of such household mechanization on obesity.

Second, it has been documented that the timing and the scale of economic and ecological changes have created a highly obesogenic environment in developing countries (Popkin, 2001). These powerful changes have sped the "nutrition transition" experienced by developing countries. As industrialization and economic growth occur, the accompanying environmental changes tend to lower the cost of energy intake but raise the cost of energy expenditure (Philipson and Posner, 2003). These changes lead to a shift in dietary patterns that encourages high energy intake as well as types of physical activity that discourage energy expenditure. For example, the low cost of hydrogenated fat and refined carbohydrates promote the consumption of palatable, yet calorie-rich, food. The growing use of motorized transport (e.g., Bell et al., 2002) and energy-saving devices (e.g., Lanningham-Foster et al., 2003), increasingly sedentary employment, and the seduction of television, computers, and video games all discourage energy expenditure. While this line of research has laid a solid framework to study the obesity pandemic in developing countries, most studies so far focus on establishing association rather than causality. We seek to contribute to the literature by providing causal evidence on the nutrition transition.

The importance of understanding the causal effect of household mechanization on obesity is further illuminated by the fact that women have experienced greater increases in obesity rates than men (Wang et al., 2007; Flegal et al., 2010). It is also suggested that overweight propensity is more prevalent than underweight propensity in young women aged 20-49 in most developing countries, regardless of the level of urbanization (Mendez et al.,

2005). Since household work is performed mostly by women, the infiltration of energy-saving and task-simplifying household devices is certainly capable of affecting energy imbalance among women. The growth in the amount of their spare time also allows more women to enter the labor force and participate in increasingly sedentary employment, or to undertake more sedentary activities, such as watching TV or using a computer. Thus, in this paper, we focus particularly on adult women in China.[2]

It is the modernization and industrialization of China over such a short period of time that makes our research possible. In most developed countries, it took a long time -- several decades usually -- for such appliances as televisions and washing machines to become adopted in most households. This slow penetration rate made it almost impossible to study the effects of the spread of such appliances on weight outcomes, which have experienced drastic changes over a much briefer time frame. On the other hand, China underwent a dramatic transition from an economy merely struggling to survive to an economy that has become highly Westernized in a short period of time. Since the "reform and opening-up" policy in 1979, penetration rates of household appliances in China have been rising far more rapidly than in developed countries. For example, TV ownership increased from 3.8 sets per hundred persons in 1985 to 15.5 in 1990 and to 27.0 in 1997. This represents almost a nine-fold increase in just twelve years (World Bank, 2001). Moreover, refrigerator ownership in urban areas increased from 42.3 per hundred households in 1990 to 87.4 in 2002; and cellphone ownership grew from 7.1 per hundred households in 1999 to 62.9 in 2002. [3] Because of the rapid growth in penetration of home appliances, researchers are therefore able

[2] The results on males will be briefly discussed in Section 7.

[3] Source: http://www.chinability.com/Durables.htm (accessed January 2013).

to use available data in China to study the effect of technological progress on weight outcomes.

Identifying the causal effect of household appliance ownership on women's weight outcomes is challenging, however. First, the simple correlation between home appliance ownership and weight may reflect causality in two directions: having more home appliances may cause energy imbalance, or more sedentary and heavier housewives may tend to adopt more home appliances to ease their housework tasks. Second, omitted variables, such as preferences or an innate tendency toward sedentary behaviors, are likely to be correlated with both home appliance ownership and heavier weight. In this paper, we attempt to overcome these challenges by making use of a unique policy change in China that promoted the purchase of home appliances in rural areas.

Specifically, we exploit the variation in households' home appliance adoption generated by China's "Home Appliances Going to the Countryside" (hereafter, HAGC) policy to identify the effect of household technology on weight outcomes. The HAGC policy gave rural purchasers of selected home appliances a 13% subsidy. The policy was implemented in three provinces and one city in 2007, in another ten provinces in 2008, and then in the remaining provinces in 2009. As a result, the increase in home appliance ownership in rural areas was much larger during the policy period. Taking advantage of the emergence of this policy, we are able to apply difference-in-differences (DID) and instrumental variable (IV) techniques to isolate the true effect of home appliances on people's weight outcomes. More specifically, for the DID approach we use rural areas as the treatment group and urban areas as the control group. We use the data from China Health and Nutrition

Survey 2004, 2006, and 2009. The 2004 and 2006 waves encompass our pre-policy periods and the 2009 wave corresponds to our post-policy period. For the IV approach, we use subsidy eligibility to instrument home appliance ownership, since subsidy eligibility is highly correlated with the purchase of the subsidized home appliances and will affect people's weight outcomes only through its impact on home appliance ownership. Both the DID and IV estimates suggest a significant impact of household technology on people's weight gains.

1.2 Background

1.2.1 The HAGC Policy

The Home Appliances Going to the Countryside (HAGC) policy was a government-funded four-year project aimed at stimulating consumption of home appliances in rural areas and improving the living quality of the rural population. In December 2007 the policy was first introduced in Shandong, Henan, Sicuan provinces, and Qingdao city. Households that purchased the subsidized products (color TVs, refrigerators [including freezers], washing machines, and cell phones) received 13% discounts. One year later (December 2008) the program was extended to Inner Mongolia, Liaoning, Dalian, Heilongjiang, Anhui, Hubei, Hunan, Guangxi, Chongqin, and Shanxi. In February 2009 the policy was extended to the whole country, and the number of subsidized products was increased to include motorcycles, computers, water heaters, and air conditioners. Each province could choose two of the four products to promote. The price ceilings for these products were RMB3500 for color TVs, RMB2500 for refrigerators (including freezers), RMB1000 for cell phones, RMB2000 for washing machines, RMB3500 for computers, RMB2500 for wall-mounted air-conditioners,

and RMB4000 for floor-stand air-conditioners.[4] Rural consumers could not take advantage of the subsidy policy if they purchased products that exceed the price ceilings.

We focus on the four home appliances that were subsidized at the inception of the HAGC policy (color TVs, washing machines, refrigerators, and cell phones), and do not include the additional four home appliances that were introduced in 2009 (motorcycles, computers, water heaters, and air conditioners). There are two reasons for our doing so. First, since each province could decide to choose two from these additional four home appliances to promote, the subsidies to additional appliances were not the same across the country. Second, the CHNS data do not contain information on ownership of water heaters.

The HAGC policy clearly had a large impact on the sales of the first four subsidized home appliances. In Figure 1-1 we compare the changes in the ownership of subsidized home appliances in rural and urban areas before and after the HAGC policy. The gaps in home appliance ownership between rural and urban areas became much narrower after the HAGC policy. Households with none of these four home appliances decreased from 10.3% to 2.3% in rural areas, and from 2.8% to 0.9% in urban areas (Panel A of Figure 1-1). The ownership of each home appliance also shows a large increase in rural areas after the policy relative to the increase in urban areas (Panels C-F). Using the variation generated by this natural experiment, we are able to examine changes in weight outcomes for women in rural areas that are affected by the policy (the treatment group) relative to women in urban areas that are not affected by the policy (the control group).

1.2.2 Conceptual Framework

[4] The exchange rate of Chinese yuans to 1 USD was 6.2185 on March 15, 2013.

Earlier obesity interventions were based on the notion that obesity is an individual biological and psychological problem. In contrast, the socio-ecological model, becoming widely used as an underlying theoretical construct for studies on obesity prevention, conceptualizes obesity as recursive interplay between individuals and surrounding environments at both micro and macro levels (Sallis et al., 2006; Rimer and Glanz, 1997). Broadly speaking, micro-environments are individuals' day-to-day environments, such as home, school, workplace, and community. Macro-environments encompass institutional settings and regulations, social and cultural forces, venues to connect micro-environments, and so forth.

In our context, the adoption of home appliances in rural China can be viewed as reshaping the micro-environments. Taking color TV as an example, past research has established a strong association between TV watching and obesity. TV watching is likely to promote an obesogenic environment through several avenues. First, television-viewing time will displace time involved in physical activities. The sedentary nature of watching TV further encourages unhealthy dietary behaviors, such as snacking. Moreover, unhealthy dietary habits may further develop due to the exposure to food advertisements on TV (e.g., Chou et al., 2008). The change in such a micro-environment has occurred in conjunction with the change in macro-environments. For example, the number of Chinese TV stations increased from 12 stations in 1965 to 3,240 stations in 1997 (Hazelbarth, 1997). TV programming is also more Westernized, and advertising content has shifted to more modern marketing.[5] The micro- and macro-environments thus together contribute to a greater obesogenic environment.

[5] According to one estimate, media advertising in China increased 35-fold between 1981 and 1992 (Hazelbarth, 1997).

To cite another example, while having a refrigerator allows households to preserve and store more perishable fresh fruits and vegetables, it also has the same capacity for meat and processed food. A household response to such a change in micro-environments will depend on the macro-environments. Since the early 1980s and particularly in the 1990s, the agro-food industry has been expanding greatly in China. Food processing companies (such as Danone, Coca-cola, Kraft, MasterFoods, Nestle, PepsiCo and Tyson's), foreign super-market chains (such as Carrefour and Wal-Mart), and fast-food chains (such as McDonald's and KFC) have invested in joint ventures in China. Those micro- and macro- changes have coincided with the nutrition transition in China: people are eating more meat, fish, vegetable oils, and processed foods, and fewer basic grains such as rice (Du et al., 2004).

To summarize, a greater obesogenic environment at both the micro and macro levels has been created in tandem with globalization and modernization. In this paper, we are thus interested in how this change in the micro-environment in the form of having more modern home appliances has affected individuals' weight outcomes.

1.3 Data

1.3.1 Data and Sample

The China Health and Nutrition Survey (CHNS) data are well-suited to an analysis of the impact of home appliances on individual weight gain and calorie balance. The data were collected in the years 1989, 1991, 1993, 1997, 2000, 2004, 2006, and 2009, and were gathered over a three-day period using a multistage, random cluster process to draw a sample from nine provinces (Guangxi, Guizhou, Heilongjiang, Henan, Hubei, Hunan, Jiangsu,

Liaoning, and Shandong).[6] Counties in the nine provinces were stratified by income, and a weighted sampling scheme was used to randomly select four counties in each province. The same households were interviewed over time, if possible. There are rich data for physical activity, time allocation, dietary habits, and calorie intake at the individual level as well as detailed information about home appliance ownership at the household level.

In this paper we use observations from waves 2004, 2006, and 2009. We do not use earlier data for two reasons. First and foremost, rural electrification in China was not complete until 2002 (Peng and Pan, 2006). While the electricity access rate was nearly universal (98%) in rural areas in 2002, the electricity consumption rate in rural areas was much lower relative to urban areas. One important reason is that most power-generating facilities (predominantly hydropower stations) were small in capacity, unreliable because of seasonality, had no connection to major grids, and suffered inefficiency with high rates of line losses. After 2002, the rural electricity system was merged with the urban system, providing a more reliable electricity supply with higher efficiency. The second reason that we do not use earlier data is that cell phone survey started in 2004. Cell phones were one of the four subsidized products at the beginning of the HAGC policy.

Our preliminary analysis suggests that there was less significant impact of home appliances on male weight outcomes. Thus, we focus on female adults (older than 20 years old) in this paper. We will briefly discuss the results on males in Section 7. Pooling three waves of data together, we have 13,633 individual-wave observations. We exclude pregnant,

[6] Out of these nine provinces, the HAGC policy was introduced in rural areas in Henan and Shandong in December 2007, in Guangxi, Heilongjiang, Hubei, Hunan and Liaoning in December 2008, and Guizhou and Jiangsu in February 2009. The CHNS was conducted between June and August in the survey year. Thus, for residents in Guizhou and Jiangsu, there were only four to six months in the post period.

breastfeeding women and also those without arms or legs (N=182). In order to include individual fixed effects to control for time-invariant personal characteristics, we delete 2,320 subjects who were interviewed in only one wave. Our final sample for analysis consists of 11,131 individual-wave observations: 7,487 are in the treatment group (rural areas) while 3,644 are in the control group (urban areas).[7]

1.3.2 Outcome Variables

Our weight outcome measures include continuous body mass index (BMI) and four dichotomous variables indicating whether a person is underweight, of normal weight, overweight, or obese. BMI is defined as weight in kilograms divided by height in meters squared. In the CHNS data, height was measured without shoes to the nearest 0.2 cm with a portable stadiometer, and weight was measured in light indoor clothing without shoes to the nearest 0.1 kg with a balance beam scale. We then classify people's weight status based on their BMIs according to the World Health Organization's Asian standard (Appendix Table 1-A1). Compared to the international standard, the cutoff values for the classifications "overweight" and "obese" are much lower for the Asian standard.

Energy intake is based on three consecutive 24-hour recalls. The three consecutive days were randomly allocated from Monday to Sunday and are almost equally balanced

[7] In the CHNS data, there are two variables that can be used for defining the area. One is the actual inhabited area recorded by the CHNS interviewers; the other is the self-reported registration area (hukou). In this paper, we use the former area to determine the group to which people belong. One reason is that the interview-based inhabited area is more reliable than the self-reported registration area. More importantly, people living in an urban area with a rural registration must go back to their rural registration area to redeem the subsidy; and people living in a rural area with an urban registration are still eligible for the subsidy if their household is recorded in their local community. In our robustness check, we exclude those whose self-reported area are different from their current residence. Our results are similar.

across the 7 days of the week for each sampling unit. During the survey, the interviewers asked individuals each day to report all food consumed away from home on a 24-hour recall basis, and the same daily interview was used to collect at-home individual consumption. The Food Composition Table (FCT) for China was utilized to calculate nutrient values, such as total calorie intake, fat consumption, carbohydrate consumption, and protein consumption, as well as consumption in each food category (see Table 1-4 for the list of 21 food categories). Information about the total number of breakfasts, lunches, dinners, and snacks during the three-day period is also available.

To examine the effect of household technology on energy expenditure, we follow Cawley (2004) and assume that each day people need to spend time on sleep, leisure, occupation, transportation, and home production (the SLOTH model). "Sleep" is measured by daily sleep time. "Leisure" activity can be further divided into sedentary leisure activity and physical leisure activity. In the CHNS data, sedentary leisure activities include watching TV and DVDs, playing video games, surfing the internet, participating in chat rooms, playing computer games, and reading. The sedentary leisure activity level is measured by the time spent on these activities. To measure the intensity of each daily physical activity, we construct the metabolic equivalent of task (MET) hours by multiplying the time spent on each activity by the specific MET values based on the Compendium of Physical Activities (Ainsworth et al., 2000). A unit of MET is defined as the ratio of a person's working metabolic rate relative to his/her resting (basal) metabolic rate. We calculate MET hours for physical leisure activity,

transportation, occupational activity and home production.[8]

The descriptive statistics of the dependent variables are presented in Table 1-1. While the BMIs of rural and urban residents are very similar before and after the HAGC policy, the probability of being overweight (BMI > 23.0) or obese (BMI > 30.0) increased in rural areas after the policy but remained constant in urban areas. Notably, rural residents also increased their 3-day average fat intake and hours spent on sedentary activities, while urban residents experienced slight decreases.

1.3.3 Ownership of Home Appliances

To measure household technology adoption, we first aggregate over the four home appliances that were subsidized when the HAGC policy was initiated in order to formulate a summated rating scale, which we call a technology index. The index is defined as:

$$Technology\ Index = Color\ TV + Washing\ Machine + Refrigerator + Cell\ Phone \quad (1)$$

The four variables on the right hand side are dummies for the ownership of the four home appliances, and the technology index is a discrete variable that ranges from 0 to 4. Before the policy, urban households had a higher technology index than did rural households (2.97

8 The number in the parentheses after each set of activities discussed below indicates their MET value. In the CHNS, physical leisure activities include martial arts (4.5), gymnastics, dancing, acrobatics (5), track and field, swimming (7.5), soccer, basketball, tennis (6), badminton, volleyball (5), and other (ping pong, Tai Chi, etc.) (5). The time spent on each physical activity is self-reported. Occupational activities are based on self-reported activity levels: heavy physical activities (e.g., farmer, athlete, dancer, steel worker, lumber worker, and mason) (6), moderate physical activities (e.g., driver, electrician) (4), and light physical activities (e.g., sedentary job, job with some standing and sitting, office work, counter salesperson, and lab technician) (1.5). There are three types of transportation to and from work or school: motorized vehicle (1.5), bicycle (4), and walking (3). Note that the MET hours for transportation only capture the energy spent on commuting to work or school. Home production includes buying food (2.3), preparing and cooking food (2.25), washing and ironing clothes (2.15), and cleaning the house (3.0).

versus 2.27, see Table 1-1). However, the difference becomes smaller after the policy change.

This summated measure is based on two assumptions: first, all home appliances have homogenous effects on weight outcomes, and second, the number of home appliances does not matter. We later loosen these two assumptions by using alternative measures of household technology in Sections 6 and 7.

1.4 Empirical Strategy

1.4.1 Difference-in-differences Estimation

The introduction of the HAGC policy allows us to exploit the variation in home appliance adoption to identify the effect of household technology on weight outcomes and related nutritional behaviors. The most straightforward approach is to use the difference-in-differences (DID) framework. The treatment group consists of rural population -- those who were eligible to receive the 13% subsidy for the four home appliances after the HAGC policy was implemented. And the control group consists those who reside in urban areas. The basic equation for the DID model is:

$$\boldsymbol{Outcome_{ipt} = \alpha_0 + \alpha_1 Rural_{ipt} \times Post_t + \alpha_2 X_{ipt} + \tau_t + \eta_i + \tau_t \times \zeta_p + \varepsilon_{ipt}.} \quad (2)$$

In this model, *i* indexes individuals, *p* indexes provinces, and *t* indexes survey waves. We control for time-varying characteristics $\boldsymbol{X_{ipt}}$ that include age, age-squared, household income, household size, the interaction term of household income and household size, work status, insurance status, and education dummies. Individual fixed effects (η_i) and wave fixed effects (τ_t) are also included. To capture the province-specific common trend, we also include an interaction between wave and province dummies ($\tau_t \times \zeta_p$). Finally, ε_{ipt} represents individual

idiosyncratic errors.

Our outcome measures include the technology index, weight outcomes, energy intake, and energy expenditures. Since the errors within each province may be correlated and the impact of policy varies between the rural and urban areas, all regressions are clustered by 18 province-rural cells.[9] The main variable of interest is the interaction between rural and post dummies. *Rural* is equal to one for rural residents, and *Post* is equal to one for wave 2009. The parameter α_1 captures the DID effect. For example, if the dependent variable is the technology index, a positive sign for α_1 indicates that after the HAGC policy the average technology index in rural areas increased compared to that in urban areas.

There are two important underlying assumptions for a valid DID estimate. First, there is no other contemporary shock (other than the HAGC policy) that might differentially affect weight outcomes of the treatment and control groups. This assumption will be verified later in Section 7. Second, there should be no differences in the underlying trends in weight outcomes and nutrition behaviors between the control and treatment groups. We discuss the extent to which violations of these assumptions may affect our results in more detail below.

1.4.2 Parameterized Model and Two-Stage Least Squares Estimation

While coefficient α_1 in equation 2 identifies the causal effects of home appliances on weight outcomes, it measures the Intent-to-Treat (ITT) effect, because we estimate the reduced-form effects on all rural residents rather than just on those families that purchase the appliances. However, our rich individual-level data, together with the policy change, allow us to estimate the treatment effect on the treated (TOT) using the following equation:

[9] There are nine provinces, and each province is divided into rural and urban areas.

$$\boldsymbol{Outcome_{ipt} = \beta_0 + \beta_1 Tech_{ipt} + \beta_2 X_{ipt} + \tau_t + \eta_i + \tau_t \times \zeta_p + \varepsilon_{ipt}.} \quad (3)$$

Our main variable of interest is *Tech* in equation 3, which measures the impact of household appliances on weight outcomes. If equation 3 is estimated using OLS, the coefficient β_1 will be biased due to reverse causality or omitted variables such as preferences. To resolve those potential problems, we estimate equation 3 using instrumental variables estimation. The HAGC policy provides a source of instrumental variables. Specifically, we estimate the following equation as our first-stage equation:

$$\boldsymbol{Tech_{ipt} = \alpha_0 + \alpha_1 Rural_{ipt} \times Post_t + \alpha_2 X_{ipt} + \tau_t + \eta_i + \tau_t \times \zeta_p + \varepsilon_{ipt}.} \quad (4)$$

Thus, the model can be identified by the variation in technology adoption after HAGC in the treatment group relative to the control group ($Rural \times Post$).

We have a very strong first stage result (see column 1 of Appendix Table 1-A2). The estimated coefficient α_1 in equation 4 of our instrumental variable (*Rural × Post*) is 0.313 with a standard error of 0.066. The F-ratio is 22.28, which is above the critical value of 10 (Cameron and Trivedi, 2005). The first stage result is important in its own right. It suggests that the purchase of home appliances increased by 14% in rural areas after the HAGC policy was enacted (=0.31/2.27). [10]

1.5 Empirical Results

1.5.1 Weight Outcomes

In Table 1-2 we present the estimates on weight outcomes. DID results are reported in Panel A. In column 1, without controlling for individual fixed effects, the DID estimate suggests

[10] All of the percentage changes in the following sections use means listed in Table 1-1.

that the BMI increased significantly in rural areas after the HAGC policy. Relative to the average BMI (23.19) in rural areas prior to the policy, the HAGC increased rural residents' BMI by 1% (=0.33/23.19). Adding individual fixed effects in column 2 does not change the coefficient to any notable degree.

Columns 3 to 10 report the changes for the various weight categories including underweight, normal weight, overweight, and obese. The estimates of α_1 are positive and statistically significant for the overweight and obese categories. They imply that the probabilities of being overweight and obese increased significantly in rural areas after the policy change; in other words, the increase in BMI occurred in the right tail of the BMI distribution. The increases are not small: based on columns 8 and 10, the probabilities of being overweight and obese increased by 8% (=0.0386/0.48) and 11% (=0.0296/0.27), respectively. In the category of obese, controlling for individual fixed effects yields a much lower estimate of α_1. It implies that individual time-invariant unobserved characteristics are positively correlated with the probability of being obese and with the purchase of home appliances. For example, a more sedentary person may rely more on modern technology and is also more likely to be obese. As a result, failing to control for these unobserved characteristics tends to overestimate the impact of technological change on an individual's weight gain.

Panel B of Table 1-2 reports estimates of equation 4 using IV. Controlling for individual fixed effects, in general, lowers the estimates in the IV regressions, and our discussion below focuses on the results after controlling for individual fixed effects. In Table 1-2, consistent with the DID results, the IV results indicate that technology adoption due to

the policy change significantly increases BMI and the probabilities of being overweight and obese. The coefficient of the technology index (the estimate of β_1 in equation 3) is 1.037 in column 2. This suggests that if the household purchases one new type of home appliance, the female's BMI increases by 1.037 on average. That is, the weight of a woman with a height of 1.6 m will increase by 2.65 kg. Moreover, given that the average change in the technology index before and after the HAGC policy is 0.7 (=2.97-2.27 in Table 1-1), this estimate implies that the policy is associated with an increase in BMI of about 0.726 (=0.7×1.037). This in turn corresponds to a 3 percent (=0.726/23.19) increase in BMI in rural areas.

More importantly, the increase in BMI has a pronounced effect on individual weight status, producing significant increases in the probabilities of being overweight and obese but a significant decrease in the probability of being normal weight. This implies that the adoption of household technology has had a larger impact on the right tail of the BMI distribution: shifting normal weight individuals to the overweight or obese categories, but not shifting underweight individuals to the normal weight category. In columns 8 and 10 of Table 1-2, there are precisely estimated β_1 values of 0.128 and 0.095. Using a similar calculation to that above, these estimates imply increases of 19% and 25% in the probabilities of being overweight and obese, respectively, as a result of the HAGC policy.

1.5.2 Energy Intake

Obesity is the result of an imbalance between energy intake and energy expenditure. Table 1-3 presents the impact of home appliances on calorie intake and on the consumption of fat, carbohydrates, and protein. Only the results with individual fixed effects are presented.

Overall, the DID estimates indicate positive associations between the HAGC policy

and energy intake (Panel A). Estimates of α_1 are statistically significant only when calorie and fat intakes are outcomes (columns 1 and 2). The estimates of α_1 (83.25 and 7.138) imply that after the policy, calorie consumption per day rose by 83.25 kilocalories (kcal) and fat consumption by 7.138 grams (g) in rural areas. The IV regressions (Panel B) show similar patterns. Estimates in columns 1 and 2 suggest that, with each additional new type of home appliance, daily calorie and fat consumption rose by 273.5 kcal and 23.13 g respectively in rural areas. Taken together, our estimates suggest that a 3% to 9% increase in calorie intake and an 11% to 25% increase in fat consumption resulted from the HAGC policy.[11] However, our results show no significant change in the consumption of carbohydrates and protein.

To find out the source of calorie and fat intakes, we further examine the impact of technological change on specific types of food consumption. The food items in the CHNS are based on the Chinese Food Composition Table (FCT) from the National Institute of Nutrition and Food Safety, Chinese Center for Disease Control and Prevention. Using the FCT, we examine the impact of home appliances on 21 different categories of food (Table 1-4). We find significant increases in the consumption of meats, poultry, and eggs, as well as significant decreases in the consumption of cereals, bacteria and algae, drinks, and candies. These findings confirm that the increase in energy intake is mainly due to the change in dietary patterns -- especially a shift away from traditional cereal products to meat and poultry that contain high calories. From the DID and IV estimates, the policy-induced acquisition of home appliances led to a 9.8% to 22% decrease in cereals consumption, and increased the consumption of meats and poultry by 10.8% to 24.6% and 31.1 to 74.0% respectively. These

[11] Guo et al. (2000) suggest that a large increase in fat consumption has taken place among Chinese adults and that this increase is not related to income status.

results are not surprising, since one of the four subsidized appliances (refrigerators) would be expected to have an impact on such foods as meats, poultry, and eggs, all of which have a greater need for cold storage.

A related question is whether the increase in energy intake that we have observed could be due to an increase in the number of meals eaten rather than an increase in the energy intake of each meal. Cutler et al. (2003) suggest the former is responsible for weight gain, because the improvement in household technology reduces the fixed costs of preparing food. Here we examine the total number of breakfasts, lunches, dinners, and snacks during the 3-day survey period. However, our results (Table 1-5) do not support Cutler's contention. We find no significant impact of household technology on the number of meals. Instead, to our surprise, a one-unit increase in the technology index (i.e., one new type of home appliance) is associated with a decrease of 0.6 snack over the three-day period (column 4). Our results therefore suggest that it is the increase in energy intake of each meal, rather than an increase in the number of meals, that leads to the additional energy intake.

1.5.3 Energy Expenditure

Next we turn to analyze the impact of household technology on energy expenditure. As we noted earlier, the first component in the SLOTH model is sleep. From column 1 of Table 1-6, we see that both the DID and IV estimates indicate there is no significant impact on total time spent sleeping.[12]

The second component of the SLOTH model is leisure activity. Leisure activity can

[12] However, modern technology may have an impact on the quality of sleep (Suganuma et al., 2007), but such information is not collected in the CHNS. We therefore cannot establish the association between poor sleep quality and weight gain, as suggested in Lyytikäinen et al. (2011).

be divided into sedentary leisure activity and physical leisure activity. The effect of household technology on sedentary leisure activity is presented in the next two columns of Table 1-6. The dependent variables here are the natural log of daily minutes spent on sedentary activities during weekdays (column 2) and weekends (column 3). As Table 1-6 shows, the estimates of α_1 in equation 2 imply that the HAGC policy significantly increased sedentary activity time in rural areas. Sedentary activity time increased by 11.6% (=exp(0.11)-1) on weekdays and by 9.7% (=exp(0.0925)-1) on weekends after the policy was enacted in rural areas. The IV estimates in Panel B of Table 1-6 also suggest that household technology has made people more sedentary. Here the estimates of β_1 indicate that one new type of home appliance will lead to a 43.6% (=exp(0.362)-1) increase in sedentary activity time on weekdays and a 35% (=exp(0.3)-1) increase on weekends. Given that the average change in the technology index in rural areas is 0.7 and the average sedentary time in rural areas before the policy was about two hours per day, the IV estimates indicate that household technology has increased the time spent on sedentary activity daily by 30.5% (37 minutes) and 24.5% (29 minutes) on weekdays and weekends, respectively.

For examining the impact of household technology on physical leisure activity and on the other SLOTH model components (occupational activity, transportation-related activity and home production), we use metabolic equivalent of task (MET) hours and the probability of participating in a physical activity as dependent variables (Table 1-7). Overall, we only find significant estimates on physical leisure activity. In Panel I column 1, the HAGC policy significantly reduced MET hours of physical leisure activity by 50% (=1.103/2.17), relative to mean MET hours prior to the policy in rural areas. The IV results suggest that one new

type of home appliance is associated with a 3.74 MET-hour reduction in physical leisure activity. In Panel II column 1, the impact on the probability of engaging in physical leisure activity is significant, and the estimates suggest that the HAGC-subsidized items have lowered the probability of undertaking physical leisure activity by over 55% (0.0219/0.4).

To summarize, our results from Tables 1-6 and 1-7 suggest that household technology has in fact had a significant impact on people's choices of leisure activity. Because of household technology, they have tended to engage in more sedentary activity but less physical activity during their spare time.

1.6 Robustness Checks

In this section, we provide two sets of robustness checks. First, we examine the robustness of our findings to alternative definitions of the technology index. In equation 1, we constructed the technology index as the sum of the four ownership dummies. As a result, this index can only capture the effect of getting a new *type* of home appliance, but it ignores the impact of an increase in the *number* of home appliances of the same type. For example, if a household already had one TV, the impact of buying an additional TV would not be captured by our technology index. To analyze the possible effect of an increase in number of home appliances, we use the sum of the total number of these home appliances to construct the new technology index. The IV estimates based on the new index are shown in Panel I of Table 1-8.[13] (We only show results of those statistically significant and economically important

[13] The first stage result is very strong (Table 1-A2, column 2). The F-ratio is 17.81, which is above the critical value of 10 (Cameron and Trivedi, 2005).

outcome variables.[14]) All coefficients remain significant but suggest a larger impact of the policy-induced acquisition of home appliances. Given that the average change in the total number of these four home appliances is 1.34 (not shown in the table) in rural areas, our results indicate that household technology increased average BMI and the probability of being obese by 4.8% and 38% respectively. Relative to the estimates based on the old technology index (3% and 25%), the larger IV estimates based on the new technology index indicate that an increase in the number of home appliances can also contribute to weight gain.

Our second robustness check is related to the classification of our treatment and control groups. Instead of using current residence to define rural population, we use a more stringent criterion to classify the treatment and control groups: the treatment group now only includes those who live in a rural area with a rural hukou and the control group only includes those who live in an urban area with an urban hukou. Our results are similar. As can be seen in Panel II of Table 1-8, the DID estimate implies that the HAGC policy is associated with a 1.6% increase in BMI (0.381/23.19), while the IV estimate indicates a 2.7% (0.88×0.7/23.19) increase. The coefficient on total calorie intake is not precisely estimated, probably due to the decrease in our sample size.

1.7 Discussion

1.7.1 Alternative Explanations

The identification strategy that we have used above requires several assumptions. For example, if the long-run trends in individual weight outcomes differ between control and treatment groups, then we may risk interpreting pre-existing trends as treatment effects. One

[14] All other results are available upon request.

potential candidate that could result in differential long-run development is exposure to an adverse event, such as the 1959-1961 China Famine, *in utero*. Based on the fetal origins hypothesis (Barker and Osmond, 1986), a fetus faced with undernutrition would not only experience a slower growth rate in response to a compromised intrauterine environment, but the result might also be a permanent modification of the structure and function of organs and systems involved metabolism and physiology. These physiological alterations, together with the rapid change in the environment, put the "programmed" cohorts at risk of chronic diseases and metabolic syndromes, such as obesity. In other words, people born during the famine may have a different response to current environment. Previous studies have shown a positive relationship between intrauterine exposure to famine and being overweight/obese in adulthood (Yang et al., 2007). In our context, since the China Famine affected the rural areas more than urban areas, we may risk interpreting the underlying change of developmental biology resulting from the intrauterine exposure to famine as treatment effects. We take two approaches to examine this potential alternative explanation.

First, we apply a DID approach to examine changes from 2004 to 2006 (both waves are before the HAGC policy). We still use rural areas as the treatment group and urban areas as the control group; but in this specification, the post-period refers to wave 2006. If the differences that we observed were due to pre-existing trends, the changes in rural areas from 2004 to 2006 should be significantly different from the changes in urban areas, even though the HAGC policy happened after 2006. As shown in Panel I of Table 1-9, however, only the coefficient of fat consumption is significant, but with a sign opposite to our main result. These results suggest that there is no pre-existing trend between treatment and control groups.

Second, since the Chinese great famine happened between 1959 and 1961, we drop those who were born during the famine or who were very young at that time (born between 1956 and 1958), and re-estimate the models. Our results (in Panel II) are very similar to our main estimations. These findings lead us to reject the hypothesis that the significant differences that we observed between rural and urban areas are the result of a pre-existing trend, rather than of the HAGC policy.

Another alternative explanation for our findings is that our treatment effect might be capturing other contemporaneous changes that affect weight outcomes in the control and treatment groups differently. We also take two approaches to examine this possibility. First, we conduct a falsification test on home appliances that were not subsidized by the HAGC policy. Second, we directly control for the differential responses to the possible contemporaneous change.

To conduct a falsification test, we reconstruct the technology index using ten household electrical appliances in the CHNS that were not subsidized by the HAGC policy. If the improvement of household technology was due to other shocks in rural areas between 2006 and 2009, it is highly unlikely that only the sales of the four HAGC-policy-subsidized home appliances were affected, but not other home appliances. The other home appliances we use here are VCRs, sewing machines, electric fans, cameras, microwave ovens, electric rice cookers, pressure cookers, telephones, VCDs or DVDs, and satellite dishes.[15] The first stage is not significant (column 5 of Table 1-A2), which indicates that the differences between rural

[15] There are 16 household electrical appliances included in the CHNS. Besides these 10 unsubsidized home appliances we list here, color TVs, washing machines, refrigerators, and cell phones were subsidized at the beginning of the policy, and computers and air conditioners were subsidized from 2009 in provinces that selected them to subsidize.

and urban areas after the policy only arose for the subsidized home appliances, but not for other home appliances.

We consider three possible contemporaneous shocks that may have contaminated our treatment effects: the insurance expansion under National Health Care Reform, the rapid economic development in rural areas, and the different trends of price change between rural and urban areas during our study period. For the first possible shock, while both rural and urban areas experienced some insurance coverage expansion during our study period, it is the former that experienced a striking increase.[16] Given that previous studies (e.g., Kelly and Markowitz, 2009) have established a positive relationship between insurance coverage and body mass index, it is possible that we are capturing the differential effect of insurance coverage. To address this possibility, we include the interaction between the rural dummy and household insurance status. Similarly, to control for possible differential responses to economic growth, we include the interaction between the rural dummy and household income (in logarithms). Our results remain similar after including these two interaction terms (Panel I of Table 1-10). The effect on obesity cannot be precisely estimated, but the magnitude is very similar to our previous finding.[17]

For the third possible contemporaneous shock, we control for prices at the community

[16] In 2003, the New Rural Cooperative Medical Scheme was introduced to rural areas in China. Insurance coverage among rural residents increased from 13% in 2003 to 93% in 2008. The Urban Employees Basic Medical Insurance, established in 1998, covers urban employees. The Urban Residents Basic Medical Insurance, piloted in 79 cities in 2007 covers children, students, elderly, disabled, and other non-working urban residents. Source: Barber and Yao (2011).

[17] Since this is the only specification among many in which we find an insignificant coefficient of technology on obese, we interpret this as partially driven by multicolinearity, in particular when two new interaction terms are also not statistically significant.

level.[18] Food prices are based on free market prices. In order to keep as many observations as possible, we control for the prices of those food items with relatively few missing values, and these are usually the most common food items for Chinese consumers. In Panel II of Table 1-10, we control for the prices of rice, unbleached flour, unbleached noodles, white sugar, eggs, soy sauce, vinegar, green vegetables, cabbage, pork (fatty and lean), pork (lean only), chicken (live), beef, common carp, and fish. In Panel III, in addition to food prices, we also control for the prices of electricity, gasoline, and natural gas. Our results are robust and remain significant. These results suggest that our main findings are not driven by contemporaneous shocks.

1.7.2 The Effect of Each Home Appliance

In this subsection, we loosen the assumption that all home appliances have homogenous effects on weight outcomes by including four indicators of appliance ownership in our estimation. We view this specification as a robustness check rather than an attempt to isolate the effect of each home appliance. While our technology index used in our main specification may contain measurement errors, estimations based on four ownership indicators are also plagued by the problem of multicolinearity. Nevertheless, if we also find consistent evidence in this subsection, we will be more confident in our conclusion that the adoption of home appliances has had a significant impact on weight outcomes and related behaviors.

We estimate the equation as follows:

[18] The community questionnaire (filled out for each of the primary sampling units) collected information from a knowledgeable respondent on community infrastructure (water, transport, electricity, communications, and so on), services (family planning, health facilities, retail outlets), population, prevailing wages, and related variables.

$$Outcome_{ipt} = \beta_0 + \sum_k \beta_{1k} Appliance_{k_{ipt}} + \beta_2 X_{ipt} + \tau_t + \eta_i + \tau_t \times \zeta_p + \varepsilon_{ipt},$$

$$\boldsymbol{k} \in \{\text{TV, Washing Machine, Refrigerator, Cell Phone}\} \quad (5)$$

To deal with four endogenous variables in equation 5, we need IVs that are correlated with each home appliance. The impact of the HAGC policy varies across areas due to different pre-policy penetration rates of home appliances. Generally speaking, the policy should have had a large impact on areas with lower pre-policy penetration rates. On the basis of this reasoning, we construct *low penetration* dummy variables for each home appliance, which are equal to one when the local penetration rates are lower than average in rural areas before the HAGC policy. In order to capture enough variability, we calculate the penetration rates at the community level. As a result we have four IVs -- *Low Penetration_k* × *Post* × *Rural, k* ∈ {TV, Washing Machine, Refrigerator, Cell Phone}-- to instrument the four endogenous home appliance ownership variables.

The first stage results (in Table 1-A3) are consistent with our expectation that the number of each of the home appliances increased more in rural communities that had lower penetration rates prior to the HAGC policy. The triple interaction term is statistically significant at the 1% level for each respective home appliance. The F-tests of joint significance of four interaction terms are greater than 10 for all the four home appliances.

The IV estimates are shown in Table 1-11. These estimates give us a better understanding about the impact of each home appliance. Our results suggest that color TVs have the most significant impact on people's BMIs. Besides color TVs, refrigerators and cell phones also significantly increase the probability of being overweight. As we expected, the increase in calorie intake is due to refrigerators, and the increase in sedentary activity is

caused by color TVs. Finally, both refrigerators and cell phones lower the MET hours of physical leisure activity. Overall, these results confirm our main finding that home appliances have significantly affected individuals' weight outcomes and related behaviors.

1.7.3 The Impact of Home Appliances on Male Adults

The results for males based on two-stage least squares are shown in Table 1-12. We do not find any significant impact of home appliances on male weight outcomes. However, the impact of home appliances on nutrition intake is similar to the impact on females -- the adoption of home appliances leads to more nutrition intake, and it is mainly due to a higher level of nutrition intake at each meal, not the number of meal. From Panel II of Table 1-12, the consumptions of fat, carbohydrates, and protein all increase, and these estimates are larger and more significant compared to results for female in Table 1-3. The coefficients of 488.3, 25.34, 49.88, and 9.84 imply 19.93%, 34.47%, 13.76%, and 12.45% increases in the consumptions of total calorie, fat, carbohydrate, and protein, respectively. There is no evidence on the impact of home appliances on sleep time or energy expenditure for male.

These results shed important light on our findings on female adults. First, the different impact of home appliances on caloric expenditure can be explained by the fact that women are responsible for most of the housework. Because couples usually eat together, it is very likely that the impacts of home appliances on caloric intake are similar for men and women. Secondly, the increase in caloric intake alone may not have significant impact on weight outcomes. It could be the joint effect of an increase in caloric intake and a decrease in caloric expenditure that leads to weight gain for females. Lastly, there could be a gender-specific genotype-environment interaction effect that leads to differential response to the environment

(e.g. caloric intake) between male and female. While understanding the mechanisms of gender-specific differences is important, it is beyond the scope of this paper.

1.8 Conclusion

Using the "home appliances going to the countryside" policy in China as a natural experiment, we estimate the effect of household technology on weight outcomes and related behaviors among female adults in rural areas. China, one of the fast growing countries, has provided us with a unique setting to examine such a hard-to-study question in developed countries. We exploit the variation in households' adoption of home appliances across rural and urban areas due to the HAGC policy, and employ the instrumental variables to identify the causal effects of household technology. Our main results are that household appliances have increased the weight status of Chinese female adults significantly. The HAGC policy-induced household technology has increased the probability of being obese by 11% to 25% in rural areas.

Two underlying mechanisms stand out. The first is an increase in energy intake. Our results show a 3% to 9% increase in calorie intake and an 11% to 25% increase in fat consumption after the policy came into effect in rural areas. These increases are due to the change of dietary patterns in each meal -- a shift away from traditional cereal products to meat and poultry that contain high calories. The second is a decrease in energy expenditure. Our results show that the household technology has lowered the probability of undertaking physical leisure activity and reduced MET hours of physical leisure activity significantly. Moreover, the average time spent on sedentary activities (such as watching TV and reading) increased by 11.6% to 30.5% on weekdays and by 9.7% to 24.5% on weekends.

This study provides empirical support for the causal links between technology advancement and dietary and activity patterns and weight outcomes. China's experiences provide an important lesson for other countries that are undergoing rapid economic and nutritional transitions. At the same time, how to design public policies to avert many of the potentially harmful consequences associated with these transitions should be on the top of the public health agenda.

1.9 Reference

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Figure 1-1 Changes in the Ownership of Subsidized Home Appliances after the HAGC Policy

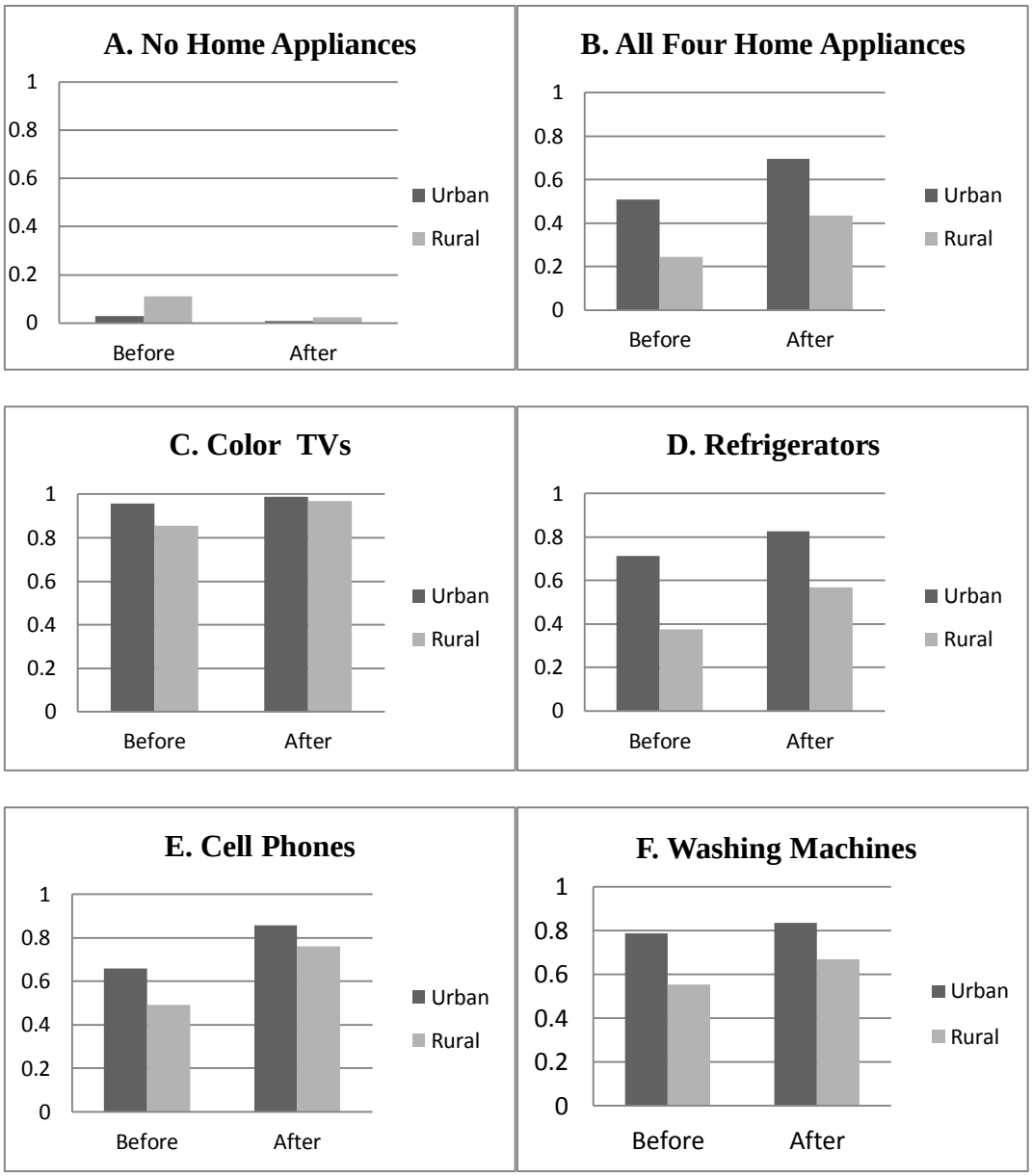

Table 1-1 Descriptive Statistics

	Before[1]		After[2]	
Variable	Rural	Urban	Rural	Urban
BMI	23.19	23.59	23.51	23.59
Underweight[3]	0.06	0.05	0.06	0.06
Normal Weight[3]	0.46	0.41	0.42	0.40
Overweight[3]	0.48	0.54	0.52	0.54
Obese[3]	0.27	0.32	0.31	0.31
3-Day Average Calorie Intake (cal)	2105	2031	2047	1913
3-Day Average Fat Intake (cal)	64.87	77.38	69.22	76.24
3-Day Average Carbohydrate Intake (cal)	318.15	267.83	295.03	242.27
3-Day Average Protein Intake (cal)	61.25	64.83	60.39	62.85
Sedentary Activity (Weekdays) (minutes/day)	126.63	159.69	135.37	157.23
Sedentary Activity (Weekends) (minutes/day)	125.83	171.91	134.10	166.87
Sleep Time (hours/ day)	8.19	7.97	7.98	7.75
MET hour of Physical Leisure Activity	2.17	8.39	1.84	8.71
MET hour of Transportation	5.88	4.16	6.05	3.80
MET hour of Occupational Activity	88.02	49.68	83.07	39.06
MET hour of Home Production	42.56	47.78	43.31	49.51
Number of Breakfasts (in three days)	2.68	2.45	2.60	2.36
Number of Lunches (in three days)	2.73	2.56	2.71	2.52
Number of Dinners (in three days)	2.91	2.82	2.91	2.81
Number of Snacks (in three days)	0.33	0.76	0.66	1.26
Technology Index	2.27	3.12	2.97	3.51
Age	50.06	51.00	53.61	54.63
Total Household Income (RMB)	21334	32148	34169	45713
Household Size	3.76	3.34	3.74	3.33
Insurance Status	0.35	0.46	0.93	0.90
Current Work Status	0.59	0.40	0.58	0.34
Did Not Attend School	0.37	0.26	0.40	0.29
Primary School	0.24	0.17	0.22	0.17
Lower Middle School Degree	0.26	0.25	0.26	0.26
Upper Middle School Degree	0.08	0.15	0.07	0.13
Technical or Vocational Degree	0.04	0.10	0.04	0.09
University or College Degree	0.01	0.07	0.01	0.07
Master's Degree	0.00	0.00	0.00	0.00
Sample Size	5094	2566	2393	1078

[1] Before the policy change (waves 2004 and 2006 in CHNS data)

[2] After the policy change. (wave 2009 in CHNS data)

[3] Classification of weight status is shown in Appendix Table 1-A1.

Table 1-2 The Effect of Home Appliances on Weight Outcomes

	BMI		Underweight		Normal Weight		Overweight		Obese	
	A: Difference-in-differences Estimates									
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Rural x Post	0.330***	0.316***	-0.0118	-0.0135	-0.0238	-0.0251	0.0356***	0.0386***	0.0416***	0.0296**
	(0.0380)	(0.0552)	(0.00786)	(0.00943)	(0.0149)	(0.0151)	(0.0119)	(0.0112)	(0.0115)	(0.0115)
Rural	-0.422***		0.0120**		0.0529***		-0.0649***		-0.0495***	
	(0.111)		(0.00500)		(0.0113)		(0.0118)		(0.0141)	
	B: Instrumental Variable Estimates									
Technology Index	1.228***	1.037***	-0.0429	-0.0455	-0.0888*	-0.0827*	0.132***	0.128***	0.154***	0.0956***
	(0.278)	(0.257)	(0.0369)	(0.0365)	(0.0485)	(0.0429)	(0.0479)	(0.0415)	(0.0342)	(0.0287)
Including Individual FE	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes

Standard errors, reported in parentheses, are adjusted for clustering by 18 province-rural cells.

All regressions include age, age square, household income, household size, the interaction term of household income and household size, work status, insurance status, education dummies, wave fixed effects, and interaction between wave and province dummies.

*** p<0.01, ** p<0.05, * p<0.1

The sample size for all regressions is 11,131.

Table 1-3 The Effect of Home Appliances on Energy Intake

	Calories	Fat	Carbohydrates	Protein
	A: Difference-in-differences Estimates			
Rural x Post	83.25*	7.138***	3.546	1.503
	(42.41)	(2.312)	(7.628)	(1.504)
	B: Instrumental Variable Estimates			
Technology Index	273.5*	23.13***	12.32	5.012
	(143.1)	(7.470)	(24.57)	(4.761)

Standard errors, reported in parentheses, are adjusted for clustering by 18 province-rural cells.
All regressions include age, age square, household income, household size, the interaction term of household income and household size, work status, insurance status, education dummies, wave fixed effects, individual fixed effects, and interaction between wave and province dummies.
*** p<0.01, ** p<0.05, * p<0.1
The sample size for all regressions is 11,131.

Table 1-4 The Effect of Home Appliances on Food Selection

		Difference-in-differences Estimates		Instrumental Variable Estimates	
(1)	Cereals	-120.9**	(43.62)	-393.8**	(167.2)
(2)	Potatoes and Starch	-13.18	(9.484)	-43.29	(31.46)
(3)	Dried Beans	-0.397	(13.14)	-1.761	(41.49)
(4)	Vegetables	-63.60	(49.02)	-211.8	(170.1)
(5)	Bacteria and Algae	-6.342**	(2.796)	-21.06**	(10.38)
(6)	Fruit	-8.138	(25.72)	-25.87	(84.48)
(7)	Nuts and Seeds	-6.162*	(3.396)	-20.21	(12.87)
(8)	Meats	18.29*	(10.41)	59.70**	(26.89)
(9)	Poultry	8.721	(5.804)	29.63*	(16.50)
(10)	Milk	4.700	(6.696)	13.95	(20.65)
(11)	Eggs	9.277***	(2.897)	30.04**	(12.05)
(12)	Fish and Seafood	-3.795	(7.957)	-13.06	(25.48)
(13)	Foods for Infants and Young Children	-0.0211	(0.224)	-0.0399	(0.712)
(14)	Savory Snack Foods and Biscuits	-7.077	(6.470)	-23.31	(20.79)
(15)	Fast Food	-0.143	(6.686)	-0.567	(21.36)
(16)	Drinks	-12.41	(7.660)	-40.58*	(24.39)
(17)	Alcoholic Drinks	3.982	(3.966)	13.56	(12.49)
(18)	Candies, Dried fruit, and Candied Fruit	-1.448*	(0.732)	-4.753*	(2.828)
(19)	Grease	-0.108	(0.463)	-0.362	(1.430)
(20)	Condiment	-3.483	(4.860)	-11.57	(16.48)
(21)	Other	-0.324	(0.460)	-1.026	(1.477)

Standard errors, reported in brackets, adjust for clustering by 18 province-rural cells.

All regressions include age, age square, household income, household size, the interaction term of household income and household size, work status, insurance status, education dummies, wave fixed effects, individual fixed effects, and interaction between wave and province dummies.

*** p<0.01, ** p<0.05, * p<0.1 The sample size for all regressions is 11,131.

Table 1-5 The Effect of Home Appliances on Number of Meals

	Breakfast	Lunch	Dinner	Snack
	A: Difference-in-differences Estimates			
Rural x Post	0.00695	0.0384	0.0260	-0.180**
	(0.0318)	(0.0269)	(0.0171)	(0.0839)
	B: Instrumental Variable Estimates			
Technology Index	0.0176	0.124	0.0865	-0.596*
	(0.100)	(0.0989)	(0.0556)	(0.354)

Standard errors, reported in parentheses, are adjusted for clustering by 18 province-rural cells.
All regressions include age, age square, household income, household size, the interaction term of household income and household size, work status, insurance status, education dummies, wave fixed effects, individual fixed effects , and interaction between wave and province dummies.
*** p<0.01, ** p<0.05, * p<0.1
The sample size for all regressions is 11,131.

Table 1-6 The Effect of Home Appliances on Sleep Time and Sedentary Activity Time

	Sleep Time (log hours/day)	Weekday Sedentary Activity Time (log minutes/ day)	Weekend Sedentary Activity Time (log minutes/ day)
		A: Difference-in-differences Estimates	
Rural x Post	-0.000323	0.110***	0.0925***
	(0.00970)	(0.0307)	(0.0264)
		B: Instrumental Variable Estimates	
Technology Index	0.000464	0.362***	0.300***
	(0.0316)	(0.0981)	(0.0891)

Standard errors, reported in parentheses, are adjusted for clustering by 18 province-rural cells.
All regressions include age, age square, household income, household size, the interaction term of household income and household size, work status, insurance status, education dummies, wave fixed effects, individual fixed effects, and interaction between wave and province dummies.
*** p<0.01, ** p<0.05, * p<0.1
The sample size for all regressions is 11,131.

Table 1-7 The Effect of Home Appliances on Physical Activity

	Physical Leisure Activity	Occupational Activity	Transportation	Home Production
	(1)	(2)	(3)	(4)
	Panel I: MET Hours of Physical Activity			
	A. Difference-in-differences Estimates			
Rural x Post	-1.103**	-1.505	0.292	-1.084
	(0.511)	(3.006)	(0.555)	(2.171)
	B. Instrumental Variable Estimates			
Technology Index	-3.743*	-4.570	0.922	-3.493
	(2.081)	(9.508)	(1.813)	(6.952)
	Panel II: Probability of Participating in Physical Activity			
	A. Difference-in-differences Estimates			
Rural x Post	-0.0219***	-0.0112	-0.0108	-0.00338
	(0.00663)	(0.00957)	(0.0241)	(0.0195)
	B. Instrumental Variable Estimates			
Technology Index	-0.0735***	-0.0355	-0.0340	-0.0104
	(0.0282)	(0.0338)	(0.0773)	(0.0632)

Standard errors, reported in parentheses, are adjusted for clustering by 18 province-rural cells.
All regressions include age, age square, household income, household size, the interaction term of household income and household size, work status, insurance status, education dummies, wave fixed effects, individual fixed effects, and interaction between wave and province dummies.
*** p<0.01, ** p<0.05, * p<0.1
The sample size for all regressions is 11,131.

Table 1-8 Robustness Checks

	BMI (1)	Obese (2)	Calorie Intake (3)	Fat (4)	Weekday Sedentary Activity Time (5)	Weekend Sedentary Activity Time (6)	MET hours of Physical Leisure Activity (7)
	I. Technology Index (Total Number of Subsidized Products)						
	Instrumental Variable Estimates						
Technology Index	0.831***	0.0766***	219.2*	18.54***	0.303***	0.240***	-3.000*
	(0.202)	(0.0239)	(120.8)	(7.059)	(0.108)	(0.0909)	(1.673)
Sample Size	11,131	11,131	11,131	11,131	11,131	11,131	11,131
	II. Subsidy Eligibility (Excluding People Whose Hukou Are Different than Current Residence)						
	A. Difference-in-differences Estimates						
Rural x Post	0.410***	0.0343*	21.72	10.13***	0.149***	0.117***	-1.775*
	(0.0579)	(0.0168)	(61.07)	(2.818)	(0.0269)	(0.0177)	(0.863)
	B. Instrumental Variable Estimates						
Technology Index	0.703***	0.0575**	45.88	17.58***	0.262***	0.206***	-3.177**
	(0.0834)	(0.0262)	(104.4)	(5.002)	(0.0411)	(0.0321)	(1.529)
Sample Size	7,877	7,877	7,877	7,877	7,877	7,877	7,877

Standard errors, reported in parentheses, are adjusted for clustering by 18 province-rural cells.
All regressions include age, age square, household income, household size, the interaction term of household income and household size, work status, insurance status, education dummies, wave fixed effects, individual fixed effects , and interaction between wave and province dummies.
*** p<0.01, ** p<0.05, * p<0.1

Table 1-9 Alternative Explanations -- Pre-Trend

	BMI	Obese	Calorie Intake	Fat	Weekday Sedentary Activity Time	Weekend Sedentary Activity Time	MET hours of Physical Leisure Activity
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	I. Pre-trend (DID w/ FE)						
Rural x Post	0.0414	0.0116	-3.838	-3.390*	0.0117	0.00971	-1.586
	(0.0496)	(0.00705)	(36.12)	(1.804)	(0.0431)	(0.0421)	(1.461)
Sample Size	6,662	6,662	6,662	6,662	6,662	6,662	6,662
	II. Dropping Those Who Were Born or Very Young During the China Famine (Born from 1956-1961)						
	A. Difference-in-differences Estimates						
Rural x Post	0.321***	0.0350**	106.1**	7.643***	0.111***	0.0952***	-1.427**
	(0.0568)	(0.0133)	(41.04)	(2.418)	(0.0324)	(0.0277)	(0.511)
	B. Instrumental Variable Estimates						
Technology Index	1.090***	0.117***	356.8**	25.75***	0.379***	0.322***	-4.805**
	(0.233)	(0.0346)	(152.8)	(8.422)	(0.0966)	(0.0911)	(2.152)
Sample Size	9,648	9,648	9,648	9,648	9,648	9,648	9,648

Standard errors, reported in parentheses, are adjusted for clustering by 18 province-rural cells.
All regressions include age, age square, household income, household size, the interaction term of household income and household size, work status, insurance status, education dummies, wave fixed effects, individual fixed effects, and interaction between wave and province dummies.
*** p<0.01, ** p<0.05, * p<0.1

Table 1-10 Alternative Explanations -- Contemporary Shocks

	BMI (1)	Obese (2)	Calorie Intake (3)	Fat (4)	Weekday Sedentary Activity Time (5)	Weekend Sedentary Activity Time (6)	MET hours of Physical Leisure Activity (7)
	I. Include Rural × Insurance & Rural × logHHincome						
	A. Difference-in-differences Estimates						
Rural x Post	0.248***	0.0207	35.48	6.023*	0.0839***	0.0686***	-1.010
	(0.0726)	(0.0212)	(48.95)	(3.213)	(0.0248)	(0.0212)	(0.919)
Rural x Insurance	0.112	0.0196	125.1**	3.989	0.0363	0.0424	-0.166
	(0.0951)	(0.0187)	(58.16)	(4.716)	(0.0333)	(0.0353)	(1.285)
Rural x logHHincome	0.0398	-0.000199	-33.87	-2.279	0.0226	0.00663	-0.168
	(0.0626)	(0.0121)	(27.46)	(2.063)	(0.0167)	(0.0164)	(0.455)
	B. Instrumental Variable Estimates						
Technology Index	0.846***	0.0718	121.2	20.63**	0.290***	0.233***	-3.459
	(0.214)	(0.0608)	(153.6)	(9.387)	(0.0798)	(0.0765)	(3.335)
Rural x Insurance	0.113	0.0196	125.2**	4.010	0.0351	0.0412	-0.170
	(0.108)	(0.0191)	(57.30)	(4.706)	(0.0301)	(0.0363)	(1.228)
Rural x logHHincome	0.00914	-0.00449	-38.88	-3.218*	0.0138	0.00229	-0.0115
	(0.0709)	(0.0132)	(26.46)	(1.782)	(0.0191)	(0.0183)	(0.541)
Sample Size	11,131	11,131	11,131	11,131	11,131	11,131	11,131

Table 1-10 Alternative Explanations -- Contemporary Shocks (Continued)

	BMI	Obese	Calorie Intake	Fat	Weekday Sedentary Activity Time	Weekend Sedentary Activity Time	MET hours of Physical Leisure Activity
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	II. Control for Food Prices						
	A. Difference-in-differences Estimates						
Rural x Post	0.200**	0.0271*	89.97*	5.980	0.117**	0.123**	-1.139
	(0.0820)	(0.0138)	(50.81)	(4.156)	(0.0422)	(0.0434)	(0.749)
	B. Instrumental Variable Estimates						
Technology Index	0.659**	0.0865**	291.2**	18.47	0.389**	0.390**	-3.780
	(0.267)	(0.0413)	(147.4)	(12.04)	(0.167)	(0.172)	(2.694)
Sample Size	7,460	7,460	7,460	7,460	7,460	7,460	7,460
	III. Control for Food Prices & Prices for Electricity, Gasoline, and Natural Gas						
	A. Difference-in-differences Estimates						
Rural x Post	0.189*	0.0322**	78.10	5.203	0.107**	0.108**	-1.475
	(0.0968)	(0.0129)	(48.58)	(4.530)	(0.0415)	(0.0436)	(0.924)
	B. Instrumental Variable Estimates						
Technology Index	0.634**	0.105***	257.9*	16.39	0.357**	0.341**	-5.054
	(0.299)	(0.0404)	(142.0)	(13.35)	(0.158)	(0.161)	(3.307)
Sample Size	6,954	6,954	6,954	6,954	6,954	6,954	6,954

The food prices are based on free market price. We included prices of rice, unbleached flour, unbleached noodle, white sugar, eggs, soy sauce, vinegar, green vegetable, cabbage, pork (fatty & lean), pork (lean), chicken (live), beef, common carp, and fish.
Standard errors, reported in parentheses, are adjusted for clustering by 18 province-rural cells.
All regressions include age, age square, household income, household size, the interaction term of household income and household size, work status, insurance status, education dummies, wave fixed effects, individual fixed effects, and interaction between wave and province dummies.
*** p<0.01, ** p<0.05, * p<0.1

Table 1-11 Individual Effect of Each Home Appliance

	BMI (1)	Overweight (2)	Obese (3)	Calorie Intake (4)	Fat (5)	Weekday Sedentary Activity Time (8)	Weekend Sedentary Activity Time (9)	Probability of Physical Leisure Activity (10)	MET Hours of Physical. Leisure Activity (11)
Color TV	1.384**	0.184*	0.119*	302.8	35.53*	1.218**	1.117**	-0.0848	-4.143
	(0.540)	(0.106)	(0.0643)	(366.4)	(18.70)	(0.509)	(0.539)	(0.0986)	(5.814)
Washing Machine	1.180	-0.0155	0.112	-288.8	8.044	0.779	1.117	0.197	9.518
	(1.000)	(0.172)	(0.117)	(490.6)	(24.53)	(0.797)	(0.858)	(0.140)	(8.951)
Refrigerator	0.266	0.0840**	0.0497*	244.4**	9.002	-0.233	-0.313**	-0.0693**	-2.822
	(0.228)	(0.0330)	(0.0267)	(94.89)	(6.045)	(0.151)	(0.132)	(0.0323)	(1.988)
Cell Phone	0.121	0.0642*	0.0237	-12.29	-1.534	-0.180	-0.206	-0.0691***	-3.186**
	(0.202)	(0.0328)	(0.0318)	(121.5)	(5.631)	(0.280)	(0.307)	(0.0231)	(1.335)

Standard errors, reported in parentheses, are adjusted for clustering by 18 province-rural cells.
All regressions include age, age square, household income, household size, the interaction term of household income and household size, work status, insurance status, education dummies, wave fixed effects, individual fixed effects, and interaction between wave and province dummies.
*** p<0.01, ** p<0.05, * p<0.1
The sample size for all regressions is 11,131.

Table 1-12 The Effect of Home Appliances on Male Outcomes

VARIABLES	Instrumental Variable Estimates	
Panel I: Weight Outcomes		
BMI	0.292	(0.454)
Underweight	0.0118	(0.0400)
Normal Weight	-0.0750	(0.0557)
Overweight	0.0633	(0.0680)
Obese	0.0446	(0.0351)
Panel II: Energy Intake		
Calorie	488.3***	(150.0)
Fat	25.34***	(6.745)
Carbohydrate	49.88**	(22.50)
Protein	8.840*	(5.015)
Panel III: Number of Meals		
Breakfast	0.213	(0.183)
Lunch	0.101	(0.104)
Dinner	0.146	(0.129)
Snack	-0.722**	(0.325)
Panel IV: Sleep Time and Sedentary Activity Time		
Weekday Sedentary Activity Time	0.164	(0.112)
Weekend Sedentary Activity Time	0.00568	(0.0930)
Sleep Time	0.628	(0.613)
Panel V: MET Hours of Physical Activity		
Physical Leisure Activity	-2.918	(3.127)
Occupational Activity	-1.605	(3.311)
Transportation	6.583	(15.37)
Home Production	-9.362	(28.64)

Standard errors, reported in parentheses, are adjusted for clustering by 18 province-rural cells.

All regressions include age, age square, household income, household size, the interaction term of household income and household size, work status, insurance status, education dummies, wave fixed effects, individual fixed effects, and interaction between wave and province dummies.

*** p<0.01, ** p<0.05, * p<0.1

The sample size for all regressions is 9,853.

Appendix Tables

Table 1-A1: Classification of Weight Status according to BMI

	Asian Standard[1]	International Standard[2]
Classification	BMI(kg/m2)	
Underweight	<18.5	<18.5
Normal range	18.5-22.9	18.5-24.9
Overweight	>23.0	>25.0
Obese	>25.0	>30.0

[1] Source: World Health Organization/International Association for Study of Obesity /Interagency Oncology Task Force (2000)

[2]Source: World Health Organization (1998)

Table 1-A2: Technology Index (Partial Results of First Stage of IV Approach)

	Main Results[1]	Robustness Checks[2]			Alternative Explanations[3]			
		Technology Index	Subsidy Eligibility	Famine	Non-subsidized	Rural Insurance	Food Prices	Food and Other Prices
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Rural x Post	0.313***	0.402***	0.577***	0.295***	0.0807	0.294***	0.312***	0.304***
	(0.0664)	(0.0951)	(0.0414)	(0.0554)	(0.0763)	(0.0643)	(0.0538)	(0.0580)
t-ratio	4.72	4.22	13.93	5.33	1.06	4.57	5.81	5.23
F-ratio	22.28	17.81	194.04	28.41	1.12	20.88	33.76	27.35
Sample Size	11,131	11,131	7,877	9,648	11,131	11,131	7,460	6,954

[1] First stage of Tables 1-2 through 1-7

[2] First stage of Table 1-8

[3] First stage of Tables 1-9 & 1-10

Standard errors, reported in parentheses, are adjusted for clustering by 18 province-rural cells.

All regressions include age, age square, household income, household size, the interaction term of household income and household size, work status, insurance status, education dummies, wave fixed effects, individual fixed effects, and interaction between wave and province dummies.

*** p<0.01, ** p<0.05, * p<0.1

Table 1-A3: First Stage of Table 1-11

	Color TV	Washing Machine	Refrigerator	Cell Phone
Low Penetration_Color TV × Post × Rural	0.164***	0.0258	0.104***	-0.00638
	(0.0244)	(0.0394)	(0.0264)	(0.0316)
Low Penetration_Washing Machine × Post × Rural	0.0312	0.116***	0.0369	-0.0422
	(0.0204)	(0.0195)	(0.0367)	(0.0365)
Low Penetration_Refrigerator × Post × Rural	-0.0734***	0.0574	0.539***	-0.302***
	(0.0125)	(0.0408)	(0.0238)	(0.0216)
Low Penetration_Cell Phone × Post × Rural	0.0125	0.0184	-0.204***	0.548***
	(0.00996)	(0.0270)	(0.0249)	(0.0398)
F-ratio	47.73	11.53	127.02	78.15

Standard errors, reported in parentheses, are adjusted for clustering by 18 province-rural cells.
All regressions include age, age square, household income, household size, the interaction term of household income and household size, work status, insurance status, education dummies, wave fixed effects, individual fixed effects, and interaction between wave and province dummies.
*** p<0.01, ** p<0.05, * p<0.1
The sample size for all regressions is 11,131.

2. The Long-term Effects of Early Life Malaria Exposure: Evidence from the Malaria Eradication Program in Taiwan

2.1 Introduction

Malaria is a parasitic disease that involves headache, fever, fatigue, pain, chills, dry cough, nausea, and enlarged spleen. It was one of the leading causes of death in the nineteenth century, and is still a major public disease in many developing countries. In 2010, there were about 219 million cases of malaria and an estimated 660,000 malaria deaths globally (World Health Organization, 2013).[19] The most vulnerable groups are pregnant women and children under the age of five.[20] There are also multiple channels by which malaria impedes a county's economic development, including its effects on fertility, population growth, saving and investment, worker productivity, absenteeism, premature mortality and medical costs (Sachs and Malaney, 2002). For example, in 1995, countries with intensive malaria incidence had income levels of only 33% as high as those of countries without malaria (Gallup and Sachs, 2011).

The short-term effects of malaria exposure have been studied thoroughly. However, the long-term effects of malaria exposure are not easily identified. First of all, the prevalence of malaria is often related to poor health care systems and insufficient research funds, so a correlation between malaria exposure and worse future outcomes does not necessarily indicate a causal relationship. Second, some unobserved factors, such as genes and life styles, are correlated with both malaria infection and future outcomes. One possible solution to this quandary is to find an external shock that can isolate the effects of malaria exposure.

World Health Organization (WHO) launched the Global Malaria Eradication Program in

[19]http://www.who.int/features/factfiles/malaria/en/
[20]http://www.cdc.gov/malaria/malaria_worldwide/impact.ht

the 1950s, when the potent tools DDT and chloroquine became available.[21] This led to 37 of the 143 countries that were endemic in 1950 becoming free from malaria by 1978. That included 27 countries in Europe and the Americas (Mendis et al., 2009). Based on malaria eradication programs, three earlier papers study the long-term effects of early life malaria exposure on future education and labor outcomes. Studying the nationwide eradication program in India in the 1950s, Cutler et al. (2010) find that the program led to modest increases in household consumption per capita for prime age men; the effects were larger for men than for women in most specifications. They find no evidence of increased educational attainment for men and mixed evidence for women. Bleakley (2010) uses the malaria eradication campaigns in the United State (circa 1920) and in Brazil, Colombia, and Mexico (circa 1955) to measure how much childhood exposure to malaria depresses labor productivity. His results suggest that, relative to non-malarious areas, cohorts born after eradication of the disease have higher income as adults than the preceding generation. Lucas (2010) uses the malaria eradication programs in Paraguay and Sri Lanka to estimate the effect of malaria exposure on lifetime female educational attainment. He obtains similar results from both countries, concluding that malaria eradication increases years of educational attainment and literacy. All these three papers focus on education and labor outcomes; none of them study long-term health effects.

Some researchers use the instrumental variable (IV) approach to solve the endogeneity problem. Because malaria prevalence is highly related to environmental factors such as temperature, rainfall, relative humidity, a potential IV for the malaria prevalence is a combination of the environmental variables. For example, Barreca (2010) uses the variation in

[21]DDT (dichlorodiphenyltrichloroethane) is an organochlorine insecticide which is a colorless, crystalline solid, tasteless and almost odorless chemical compound. Chloroquine is a 4-aminoquinoline drug used in the treatment or prevention of malaria.

malaria-ideal temperatures, or temperatures between $22\,^{o}C$ and $28\,^{o}C$, to instrument for malaria exposure at the time of birth. His finding indicates that both *in utero* and postnatal exposures to malaria lead to considerably lower levels of educational attainment and higher rates of poverty later in life. Chang et al. (2011) study the long-term effects of early childhood malaria exposure on education and health in Taiwan during the colonial period. The authors use the number of public health physicians per 10,000 people and the number of other medical personnel per 10,000 people to instrument the early life malaria exposure. Their results imply that malaria exposure around birth leads to lower life-time educational attainment and worse mental and physical health outcomes in old age. The impact of malaria exposure is reflected in particular in worse cognitive function, a higher likelihood of cardiovascular diseases, and a higher mortality hazard. Hong (2013) uses three approaches to study the effect of early life malaria exposure on later disease and work level. First, he uses longitudinal lifetime records of Union Army veterans and finds that a malarial environment in early life substantially increases the likelihood of having various chronic diseases and not working in old age; second, he uses the US anti-malaria campaign began in 1921 as a natural experiment, and finds that the program significantly decreases the level of disability in old ages; third, by comparing the DALYs (disability-adjusted life years) in each country, he also finds that the early life malaria exposure is associated with worse future health outcomes.

This paper extends the existing literature, providing more detailed evidence of the long-term effects of *in utero* and early childhood (hereafter termed early life) malaria exposure on later life health outcomes.[22] It makes four contributions to the literature. First, we use

[22] The Fetal Origin of Adult Disease (FOAD) hypothesis suggests an association between an unfavorable environment during pregnancy and the development of chronic diseases in adulthood (Barker and Osmond, 1986; Sachs and Malaney, 2002). Those women who were pregnant in an unfavorable environment tended to have less nutritional intake and to have feelings of stress. Both can lead to chronic diseases in their offspring's adulthood.

comprehensive data from Taiwan's nation health insurance (NHI) to study the long-term effects of malaria exposure on the likelihood of heart disease, stroke, and unique major illness outcomes (such as malignant neoplasm, chronic renal failure, cerebral palsy, and so on). Second, we have better measure of early life malaria exposure: we are able to link birth place and malaria prevalence, varying by county/city (hereafter termed county).[23] Most previous studies have used malaria prevalence measured at more aggregated levels, then linking that to current residence. But using current residence is problematic, unless people have never moved since the time they were born. Third, we have better defined birth cohorts, and we use only two years before and two years after the malaria eradication program. Previous studies have used a much wider range of birth cohorts: 60 years for Cutler et al. (2010) and 140 years for Bleakley (2010). Their results are very likely confounded by other shocks that occur during such a long period of time. Fourth, the data used in this paper, both census and NHI data, cover the entire population in Taiwan. The large number of observations helps to eliminate the selection bias, and also provides more precise and convincing results.

By using the sudden drop in malaria spleen rates due to the eradication program in Taiwan, we are able to isolate the effects of malaria exposure. Our findings on health outcomes show that malaria eradication is associated with better future health outcomes for both genders: the eradication program lowered the probabilities of women having heart disease, stroke, renal failure and lowered the probabilities of men having ischemic stroke and cerebral palsy. Our

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Falade et al. (2010) find that malaria during pregnancy results in symmetric fetal growth restriction. This leads to a natural question: is *in utero* exposure to malaria associated with some health problems in adulthood?

[23]Every citizen has a unique ID number. A valid national identification number consists of one letter and nine-digits. The initial letter indicates a person's birth county/city. (see Table 2-A1 for detail) We drop two counties outside the main island --- Lienchiang and Kinmen --- for which there are no census or NHI data available. There are 23 counties/cities left in our sample.

results on education outcomes are consistent with earlier findings of Lucas (2010) and Cutler et al. (2010): malaria eradication significantly improves educational attainment of females, but not males. For labor market outcomes, we find that females, but not males, are more likely to have a job due to the malaria eradication program, unlike previous studies that find some beneficial effects on male labor outcomes. For example, Cutler et al. (2010) find that household consumption per capita for males increases significantly, and Bleakley (2010) finds that labor productivity and adult income of males increase after malaria eradication.

2.2 Background

2.2.1 Early Life Environment and Future Outcomes

The concept of developmental origins of health and disease arose from epidemiological observations relating small birth size to an altered risk of cardiovascular disease (Baker and Osmond, 1986). In 1995, the British Medical Journal named this the Barker Hypothesis. Following Barker's finding, many researchers found evidence on the link between size or proportions at birth and cardiovascular diseases, including stroke (Martyn et al., 1996), congenital heart disease (Forsen et al., 1997; Stein et al., 1996), and circulatory disease (Koupilova et al., 1997a). Other diseases associated with smaller size at birth include insulin resistance syndrome (Phillips et al., 1994; Law et al., 1995; Barker et al, 1993), type-2 diabetes (Hales et al., 1991), and high blood pressure (Law and Shiell, 1996).[24] Some researchers also suggest a potential impact of early life environment on other diseases, including breast cancer (Michels et al., 1996) and renal disease (Hoy et al., 1999).

[24]There is evidence of the inverse relationship between adult high blood pressure and birth weight in different countries: for example, Sweden (Koupiova et al., 1997b), USA (Curhan et al., 1996), Finland (Forsen et al., 1997), southern India (Stein et al., 1996), and China (Mi et al., 2000).

Poor nutritional status at the onset of (and throughout) pregnancy and insufficient energy and nutrient intake during pregnancy are major factors of fetal growth impairment in developing countries and in poorer population groups. The former may even be more critical than the latter for fetal growth. Malaria prevalence can lead to lower income levels or fewer resources in endemic areas (Gallup and Sachs, 2001). Even if the pregnant women are not directly infected, their nutritional status at the onset of pregnancy, and their nutritional intake during pregnancy, are worse than among pregnant women in malaria-free areas. In this paper, we do not attempt to study the impact of malaria infection, but rather study the impact of unfavorable early life environment due to malaria prevalence on later life outcomes.

2.2.2 Malaria and the Eradication Campaign in Taiwan

The island of Taiwan is 245 miles long, north to south, and 90 miles wide at the broadest part. Its total area is 13,785 square miles, of which three-fifths are hills and mountains of more than 820 feet. Taiwan has a subtropical climate, with an annual average temperature of $74.3\,^{o}F$ in the north and $76\,^{o}F$ in the south. In 1905 when the first census was taken, there were about 3 million people in Taiwan. In 1952, when the WHO DDT spraying plan was initiated, the population was 8.2 million, and it increased to 9.2 million when the attack phase of the malaria eradication program ended in 1957. The earliest record of malaria in Taiwan was in 1874, when the Japanese army invaded Southern Taiwan. During the fifty years of Japan's colonization of Taiwan, malaria was considered to be the most severe public disease. From 1906--1911, malaria killed more than 10,000 people each year. Given that the total population was slightly over 3 million, malaria was therefore the leading cause of death. From 1910 on, following Koch's method, monthly blood tests were conducted in highly malarious areas, and people with positive results for malaria parasites were treated with anti-malaria drugs. Although this did not eradicate

malaria in Taiwan, it maintained the malaria spleen rate at 2--3% for the next 30 years.[25]

This malaria control system ended in 1942 with World War II (WWII). As a result, malaria once again became an important public problem in Taiwan. Infection rates were about 20--40% among primary school children, according to parasite surveys conducted in 1946 in northern, central, and southern Taiwan. Fortunately, the availability of DDT and other residual insecticides provided an opportunity to stop this disaster. As a member country of WHO, Taiwan began a DDT spraying plan in May 1952. The Chishan District of Kaohusiung Hsien was chosen as the area for initial spraying operations, because of its high malaria spleen rate and large population (37,000). This program was extended to areas with a spleen rate of 35% or more in 1953, and in 1954 areas with a spleen rate of 10% or more were included. In 1955, these same areas were sprayed again. In 1956 and 1957, another two rounds of island-wide DDT spraying were conducted, except for those areas which proved to be definitely non-malarious. For these spraying operations, the island was divided into four operational regions: central, northern, eastern, and southern Taiwan. They were sprayed in rotation at the same time each year. Figure 2-1 shows the four-year island-wide malaria control program from 1952--1955.

2.2.3 GIS Digitized Maps

In order to study the effects of malaria eradication, we have to find a suitable indicator of the prevalence of the disease. The eradication program took place around 60 years ago, and no numerical records on pre-eradication malaria spleen rates are available. We thus use the malaria

[25]Spleen rate is commonly used to measure malaria prevalence. This is the proportion of children (2--10 years) in a community who have an enlarged spleen. In the 1950s, WHO suggested using spleen rates as a proxy for malaria prevalence. Another measure of malaria prevalence is the parasite rate, which is the proportion of persons in a given community who show parasites in their peripheral blood. In this paper, we use malaria spleen rate as the measure of malaria prevalence.

spleen rate information that is based on maps from the WHO named Malaria endemicity in Taiwan, 1953 (Figure 2-2) and Malaria endemicity in Taiwan, 1955 (Figure 2-3). It shows that the island was classified into five categories according to the malaria spleen rates: areas with spleen rates of 0-10%, 10-25%, 20-25%, 50-75%, and more than 75%.[26]

Because the malaria endemicity is highly related to local climate and landscape, there is a wide variation of prevalence rates within a county (Figure 2-4). Therefore, we assign malaria spleen rates to the township level first, and then aggregate spleen rates to the county level in the second step.[27] In order to assign malaria spleen rates to township levels, we use the geographic information system (GIS) to locate 369 townships of Taiwan according to their coordinates, and then overlie these locations onto the digitized malaria spleen rate map. Based on Figure 2-5 (or Figure 2-6 for malaria spleen rate in 1955), each township is then assigned to one of the above mentioned five spleen rate categories.

Next, to aggregate malaria spleen rates from township to county level, we convert the township-level rates from categorical to numerical by taking the midpoint of each interval.[28] We then calculate county-level malaria spleen rates using the weighted mean[29]:

$$Spleen\ Rate_j = \frac{\sum w_{jk} r_{jk}}{\sum w_{jk}}, \tag{6}$$

where $Spleen\ \ Rate_j$ is 1953 (or 1955) malaria spleen rate in county j, w_{jk} is 1953 (or 1955) population in county j town k, and r_{jk} is 1953 (or 1955) malaria spleen rate in county j town k.

[26]The surveys were conducted simultaneously throughout the island on December 17.
[27] We only have the birth county information from the first letter of ID.
[28]The five malaria intervals 0-10%, 0-25%, 25-50%, 50-75%, and above 75% correspond to the midpoints 5%, 17.5%, 37.5%, 62.5%, and 87.5% respectively.
[29]We use the township population in 1953 (or 1955) as the weight. Population data are from "The Annual Population Statistics, published by the Department of Interior Affairs. Most previous studies use simple mean or mode, which leads to larger measurement errors than weighted mean.

2.3 Data and Sample

2.3.1 Taiwan National Health Insurance

Taiwan implemented National Health Insurance (NHI), which is compulsory for all citizens from birth, in March 1995. Prior to that, health insurance was provided primarily through various employment based programs covering 12.3 million people, or 57% of the total population in 1994. Nearly half of the total population was still uninsured, of which the majority were children under age 14 or the elderly over age 65. Universal health insurance was first discussed in 1992 and finally implemented in 1995. By the end of 1995, the insured rate had jumped to 92%. The rate has stayed above 97% since 1997, and the coverage had reached 99% by the end of 2004.[30]

NHI is modeled after Medicare (Dr. Michael Chen, Vice President and CFO of Taiwan's National Health Insurance Bureau), but differs from it in at least three aspects. First, it covers an even wider range of services, including outpatient, inpatient, dental, mental health and even Chinese medicine; in other words, NHI covers almost all medical services. Second, NHI reimburses health providers, at least in the period we analyze, on a fee-for-service basis, while Medicare switched to a prospective payment system for hospitals in 1984. Finally, hospitals in Taiwan are categorized into three groups based on their accreditations: major teaching hospitals, minor teaching hospitals, and community or unaccredited hospitals; the hospitals with higher accreditation are paid more generously.[31] For instance, the daily rate for an ordinary bed (four persons in a room) is NT512 for major teaching hospitals, NT456 for minor teaching hospitals,

[30]The insured rate is calculated on the basis of being Taiwanese citizens. Under some exceptions, Taiwanese citizens are allowed to drop out the insurance coverage, of which the majority of cases are citizens residing in places other than Taiwan for over 6 months. The rate will be even higher (approximately 99%) when calculating at the resident basis.

[31] In Taiwan, there are four types of accreditation (medical center, regional hospital, district hospital, and unaccredited hospital) and three types of teaching hospitals (major teaching hospital, minor teaching hospital, and community hospital). Due to institutional reasons, the first three types of accreditation correspond almost identically to three types of teaching status.

and NT395 for community and unaccredited hospitals.

This paper uses NHI data covering from 1997--2011. In order to study the impact of early life malaria exposure, our sample must contain birth cohorts both before and after the eradication program. Therefore, we keep people who were born between 1951 and 1960. We use both inpatient and major illness information from the NHI data in our research. The inpatient records include information about the incidence of heart disease and stroke. The certificate of major illness/injury, designed to relieve financial burdens of patients with serious illness who need long-term proactive medical treatment, is issued by the Department of Health; the application and approval process for certificate of major illness is long and complicated. If the person is found to meet the criteria after the review, a major illness/injury certificate will be issued. Some medical fees may be waived for insured persons holding a valid major illness/injury certificate when they consult a doctor. We only study the impact of malaria on the top ten major illnesses in Taiwan (listed in Panel B of Table 2-1).

2.3.2 Taiwan Census

The second data source for this study is the Taiwan census, which is conducted every ten years by the Directorate of General Budgeting, Accounting, and Statistics (DGBAS). The Taiwan census information comes from a fairly detailed questionnaire, similar to those used to create the PUMS files for the U.S. censuses (long-form), except that Taiwan excludes income related variables. For each household, the interviewer records every individual member's basic demographics (race, sex, age, and marital status), highest completed education level, relationship to the head of household, working and employment status in the past two weeks, and the industry in which he or she works. Another advantage of using the Taiwan census is that the files contain the full sample of Taiwan's residents --- around 22 million in total.

We use the 2000 census survey for two reasons. First, people in our sample should have stable education and labor outcomes, so they cannot be too young. On the other hand, they cannot be too old or there will be sample selection due to mortality. In year 2000, ages of our analysis sample (born between 1950 and 1960) range from 40--50. More importantly, information about the first letter of ID, which is used to identify the birth county, is only available in the 2000 census survey.

2.3.3 Outcome Measures

Health outcomes come from NHI claims data; diseases are identified by ICD-9 codes. In this paper, we are interested in the long-term effects of early life malaria exposure on both common diseases, including heart disease and stroke, and major illnesses. There are three major types of heart diseases: Ischemic Heart Disease (IHD), Acute Myocardial Infarction (AMI), and Congestive Heart Failure (CHF). Both IHD and AMI occur when blood flow to the heart muscle is decreased: IHD is caused by narrowed heart arteries, while AMI is due to a partial or complete blockage of heart's arteries. CHF occurs when the heart is not able to pump enough blood to meet the needs of the body. A stroke is a condition in which the brain cells suddenly die because of a lack of oxygen: the two main types of stroke are ischemic stroke and hemorrhagic stroke.

In this paper, we study the effect of malaria eradication on the top 10 major illnesses in Taiwan (listed in Table 2-1).[32] Previous studies show that malaria infection is related to the defects and abnormalities of a wide range of organs, such as 1) bone marrow and sickle (warrell et al., 2010; Wickramasinghe and Abdalla, 2000), 2) Cardiovascular (Herr et al., 2011; WHO, 2000), 3) Respiratory (Murphy and Breman, 2001; Perlmann and Wigzell, 1988), 4) Diarrhea, eye, and ear, and 5) Brian and neurologic damage (Idro et al., 2010). Chronic renal failure, which

[32]Currently, there are 30 eligible major illnesses covered by NHI. We have results for all major illnesses. Due to the low incidence rates of major illnesses, we only report the results for the top 10 prevalent major illnesses.

describes the gradual loss of kidney function, is the most prevalent major illness in Taiwan. Disorders of the immune system can result in autoimmune diseases, inflammatory diseases, and cancer. The immune system, when functioning properly, identifies a variety of threats including viruses, bacteria and parasites, and distinguishes them from the body's own healthy tissue. A chromosome abnormality may reflect a problem of chromosome number or structure. Maternal age and environment are highly related to the risk of chromosome abnormalities.[33] Organ transplantation is the moving of an organ from one body to another, or from a donor site to another location on the patient's own body, for the purpose of replacing the recipient's damaged or absent organ. Of all these diseases, the results for cerebral palsy are perhaps most important, because this type of disease is less likely to be affected by SES.[34]

The education and labor outcomes come from Taiwan census data. We use elementary school completion to measure education level, and current work status to measure the labor market outcomes. We also construct a measure of activities of daily living (ADL), which includes eating, bathing, dressing, toileting, transferring (walking), and bedding --- a larger value of ADL implies that more help is needed to conduct those activities. A dummy variable, *Homekeeping*, is used to indicate whether help is needed to conduct homekeeping activities.

2.4 Empirical Specifications

The variation in malaria reduction that is generated by the malaria eradication program

[33]Women are born with all the eggs they will ever have. Therefore, when a woman is 30 years old, so are her eggs. Some researchers believe that errors can crop up in the eggs' genetic material as they age over time. Therefore, older women are at higher risk of giving birth to babies with chromosome abnormalities than younger women. Since men produce new sperm throughout their life, paternal age does not increase the risk of chromosome abnormalities. Although there is no conclusive evidence that specific environmental factors cause chromosome abnormalities, it is still possible that the environment may play a role in the occurrence of genetic errors. (source: http://www.genome.gov/11508982#)

[34]Other congenital diseases covered by NHI include congenital abnormality of coagulation factors, congenital metabolic disease, impairment in premature infants, congenital immunodeficiency, congenital muscular dystrophy, and congenital vesicular epidermolysis.

helps us to isolate the impact of malaria exposure on later life outcomes. The malaria eradication program is based on the external innovation of a new treatment of malaria (DDT), which is truly exogenous. In addition, the malaria spleen rate in Taiwan dropped sharply to a very low level in the 1950s, because of the effectiveness of DDT. Therefore, our results are less likely to be confounded by other contemporaneous shocks. Finally, the DDT spraying plan was developed by the WHO, not the local government, which means that it was unexpected and not related to the financial and development situation in local areas.[35]

We capitalize on the differential impact of malaria eradication on areas with varying pre-eradication malaria spleen rates. We use a difference-in-differences (DD) framework, comparing outcomes for cohorts born before and after the eradication era in counties with higher malaria spleen rates (hereafter endemic counties) to the outcomes for those cohorts in counties with lower malaria spleen rates (hereafter control counties). The endemic counties should benefit more from the malaria eradication program. Thus, the outcome difference for cohorts born before and after the eradication era in endemic counties will capture the effect of the eradication program as well as other contemporaneous changes, while the outcome difference in the control counties will only capture the effect of other contemporaneous changes. The differenced outcome differences will reflect the effects of the eradication program. Because the island-wide DDT spraying plan started in 1953 and malaria spleen rates dropped to nearly zero in 1958, people born in 1951 and 1952 were exposed to malaria during their early life, and people born in 1959 and 1960 had almost no malaria exposure. Another advantage of this specification is that

[35]Plans for an island-wide malaria control program were laid in 1951 at a meeting between representatives of the Public Health Administration, Taiwan Provincial Malaria Research Institute (TAMRI), the International Co-operation Administration (ICA), the Council for United States Aid (CUSA), the Joint Commission on Rural Reconstruction (JCRR), and WHO (TAMRI and WHO malaria team in Taiwan, 1958).

the results are less likely to be confounded by DDT exposure during *in utero* period.[36] According to the pre-eradication malaria spleen rates (malaria spleen rate in 1953), the island is divided into three areas: control areas (areas with low malaria prevalence), high malaria-endemic areas, and very high malaria-endemic areas. The equation used to estimate the impact of malaria eradication is given below:

$$\begin{aligned} Outcome_{icj} = {} & \alpha + \beta_1 High\_Endemic_j \times Post\_Eradication_c \\ & + \beta_2 Very\_High\_Endemic_j \times Post\_Eradication_c \\ & + \gamma X_{icj} + \lambda_c + \delta_j + \varepsilon_{icj}. \end{aligned} \tag{7}$$

In this model, i indexes individual, c indexes birth cohort, and j indexes birth county. In our main analyses, $Post\_Eradication = 0$ for birth year = 1951 or 1952, and $=1$ for birth year = 1959 or 1960. In the basic setup, we define $High\_Endemic = 1$ for counties that have pre-eradication malaria spleen rates between 18.5% (median) and 33% (90^{th} percentile) and $Very\_High\_Endemic = 1$ for counties that have spleen rates above 33% (90^{th} percentile). The coefficients of interest are β_1 and β_2: β_1 captures the effects of malaria eradication in high malaria-endemic counties, while β_2 captures the effects of malaria eradication in very high malaria-endemic counties. If diseases are the outcome variables, then negative βs indicate that malaria eradication reduces the likelihood of having diseases for people who were born in 1951 and 1952 in endemic counties, after the eradication program. Based on the assumption that areas with higher pre-eradication malaria spleen rates benefit more from malaria eradication, the estimates of β_2 should be more significant and larger than the estimates of β_1. We also include

[36]Some previous studies have established a positive relationship between DDT exposure and cancer risks (Cohn et al., 2007). Because of this positive association, our estimate of the effect of the malaria eradication program will be understated. Because people in areas with higher levels of malaria prevalence were also exposed to more DDT spraying, failing to consider the impact of DDT will underestimate the beneficial effects of malaria eradication. We will address this issue in the appendix.

birth-cohort fixed effect λ_c and birth-county fixed effect δ_j.

X_{icj} are background characteristics such as current residences and local mortality rates. Big cities have more job opportunities, schools, and medical resources, so failing to control for current residence can lead to biased results. In our robustness checks, we control more individual background information, such as marital status, number of family members, and races.[37]

2.5 Results

2.5.1 Data Description

Table 2-1 presents some initial evidence of the effects of the eradication program on future health and other outcomes. As mentioned early, the island is classified into control, high-malaria endemic and very high-malaria endemic areas according to the pre-eradication malaria spleen rate. In general, areas with the highest level of malaria prevalence before the eradication program have the largest changes in adult outcomes. The summaries of chronic diseases and major illnesses are shown in Panels A and B respectively: these values tell us the number of patients with certain diseases in every million people.[38] For example, the number 559 in the first column of Panel I means that in control areas, the number of female AMI patients per one million women was 599 before the eradication program. For heart disease and stroke, we observe a clear trend across areas with different levels of pre-eradication malaria prevalence (expect for AMI): the higher level of prevalence, the more benefits there were from the malaria eradication program (or, the larger is the decrease in the incidence of diseases.) Such trends are weaker for major illnesses: there is only evidence that the probability for females to have renal failure decreases

[37] The marital status, number of family members and race information are only available in Censes data, but not in the NHI data.

[38] In the original data, health outcomes are dummy variables indicating whether the individual has this type of disease. Because the magnitude is too small, we multiply the values by 1,000,000.

tremendously, especially in very high-malaria endemic areas, after the eradication program. For the education labor market outcomes, the gaps between areas with different pre-eradication malaria prevalence become much smaller after the eradication program.

2.5.2 Empirical Results

The effects of malaria eradication on long-term outcomes of females and males are shown in Panels A and B of Table 2-2, respectively. The estimates of β_1 and β_2 in Equation 7 are presented, and the baseline results are shown in Panels A1 and B1. Consistent with our expectations, the estimates of β_2 are more significant and larger (or more negative) than those of β_1. This implies that the eradication program has greater effects on very high endemic counties than on high endemic counties. For health outcomes, the estimate of β_1 is significant at the 10% level only when the cerebral palsy is the outcome. Thus, the significant effects of the eradication program on endemic counties are likely driven mainly by the effects on very high endemic counties. In order to control for current residence, we include the current county of each individual in our regression, and the results are shown in Panels A2 and B2. The selection due to mortality is another concern: to make it into our sample, people need to survive at least to the age of 40 for the treatment cohorts and to 47 for the control cohorts. If only the healthiest children in the control cohorts (exposed to malaria) are more likely to make into the sample, then the effects of the malaria eradication program will be underestimated. As a result, in Panels A3 and B3 we future control for the estimated early life death rates at the county level[39].

The effects of malaria eradication on future chronic diseases are shown in Columns 1--5 of

[39]In "The Annual Population Statistics", there are population information for each age group in every year. By comparing the change in population from age group 0--5 to age group 5--10 in five years, we can estimate the death rate in the five years, assuming that young children are not likely to move to another county.

Table 2-2. From Panel A1, we find none of the estimates of β_1 are significant, and the estimates of β_2 capture the effect of malaria eradication program on very high endemic areas. This finding implies that the malaria eradication program significantly decreases the likelihood of getting IHD, CHF, ischemic stroke, and hemorrhagic stroke for females in very high endemic areas. Because there were 12,951 female IHD patients per one million women in very high-malaria endemic areas before the eradication program, the point estimates of -6,245 in Column 2 of Panel A1 indicates a 50% (=6,245/12,591) decrease in the incidence of IHD in very high endemic counties. Using a similar calculation, we find that the eradication program leads to a 60%, 68%, and 72% decrease in the incidence of CHF, hemorrhagic stroke, and ischemic stroke for females in very high endemic areas. The eradication program has weaker impact on the chronic disease for males (Panel B1): the eradication program decreases the likelihood of getting ischemic stroke by 34%, and has no significant impact on other chronic diseases for males. After we further control for the current residence (Panels A2 and B2) and local mortality rates (Panels A3 and B3), the results remain robust.

The incidence of major illnesses also decreases in very high endemic counties due to the eradication program (Columns 6--15): the incidence of chronic renal failure and cerebral palsy decrease by 9% (=943/10,476) and 23% (=338/1,703) for females, respectively; and the incidence of cerebral palsy decreases by 28% (=304/1092) for males. Overall, results are robust (except on cerebral palsy for females) across various specifications.

The malaria eradication also significantly improves female educational attainment: the probability of completing elementary school increases by 8 percentage points or 20% (Column 17). Job participation rate for females is significantly affected by malaria eradication only in high endemic areas (Column 16): there are some evidences that help is less needed to conduct the

activities of daily living or homekeeping after the eradication program for females, but the results are not robust (Column 18). There is no evidence about the impact of malaria eradication on male educational attainment, labor market outcomes, and care levels.

2.5.3 Different Cohorts

In the basic setup, we define birth cohorts 1951 and 1952 as pre-eradication cohorts, and birth cohorts 1959 and 1960 as post-eradication cohorts. The difference in the ages between pre and post cohorts is 8 years on average. If there were any events during these 8 years that had different effects on future outcomes in control and malaria-endemic areas, or if any diseases are age sensitive, our results will be biased.

To solve this problem, we use closer birth cohorts to study the impact of malaria eradication. Utilizing the variation in malaria spleen rate in 1955 (Figure 2-6), after two rounds of DDT spray, we have the following two alternative specifications. In the first alternative specification, we still use the birth cohorts 1951 and 1952 as the pre cohorts, but use 1954 and 1955 as the post cohorts. In this specification, the counties are classified into three categories (control, high endemic, and very high endemic areas) based on the change in malaria spleen rate between 1953 and 1955. In the second alternative specification, we use 1954 and 1955 as pre cohorts, and 1959 and 1960 as the post cohorts. The counties are classified into three categories based on the malaria spleen rate in 1955. Under these two specifications, we are not only able to identify the impact of malaria eradication using closer cohorts, but also able to separate the effects the first two rounds of DDT spraying (in years 1953 and 1954) and the effects of the last three rounds of DDT spraying (in years 1955, 1956, and 1957).

Compared to results in Panels A1 and B1 of Table 2-2 (the baseline results), the results in Table 2-3 are less significant in general. It is reasonable because the change in malaria spleen

rates are smaller (the pre and post cohorts are closer). Taking both the significance levels and magnitudes of the estimates into consideration, we find that the decrease in the incidence of chronic diseases can be explained by both the first two rounds and the last three rounds of DDT spraying, but mainly due to the first two rounds (the magnitudes of estimates in Panels A1 and B1 are larger than the ones in Panel A2 and B2, and close to the baseline results); the decreases in the incidences of female renal failure and male cerebral palsy are mainly due to the last three rounds of DDT spraying (Panels A2 and B2), and the first two rounds of DDT spraying have no impact on the incidence of major illnesses (Panels A1 and B1); the improvement in educational attainment for both females and males and the change in ADL for females can be explained by both effects.

2.6 Robustness Checks

2.6.1 Specification Checks

Table 2-4 presents the results of our three specification checks, using variants of Equation 7.

Dose Response Specification

Equation 7 above does not consider the effects caused by the intensity of childhood malaria exposure within the birth cohorts before the eradication program.[40] People who were born after the eradication program had no malaria exposure, either *in utero* or during childhood. People who were born before the eradication program were exposed to malaria *in utero* and had different years of malaria exposure during their childhood. Because the malaria spleen rate dropped to nearly zero in 1958, for people who were born in 1951 and 1952, the years of

[40]Equation 7 assumes that following the fetal origin hypothesis, the malaria effect is limited to the year of birth.

exposure to malaria during childhood are 8 and 7, respectively. In this specification, instead of using the $Post\_Eradication$ dummy to capture the impact of malaria eradication, we define $Post\_Eradication\_\%$ of cohort c as the percentage of years prior to age 8 that are free from malaria exposure. We then estimate the following equation:

$$\begin{aligned} Outcome_{icj} = \alpha &+ \beta_1 High\_Endemic_j \times Post\_Eradication\_\%_c \\ &+ \beta_2 Very\_High\_Endemic_j \times Post\_Eradication\_\%_c \\ &+ \gamma X_{icj} + \lambda_c + \delta_j + \varepsilon_{icj}. \end{aligned} \quad (8)$$

where $Post\_Eradication\_\% = 1$ (or 100%) for birth year = 1959 or 1960, and $= 0$ and $\frac{1}{8}$ for birth year = 1951 and 1952, respectively. The estimates of β_1 and β_2 in Equation 8 are presented in Panel A1 and B1 of Table 2-4: consistent with the results under the basic specification (Table 2-2), both genders benefit from the malaria eradication program. The estimate of -19,314 in Column 2 of Panel A1 indicates that the one-year reduction in childhood malaria exposure is associated with a 2,414 (=19,314/8) or 25%(=2,414/9,715) decrease in the incidence of IHD for females. People born in 1952 had 2 years of *Post Eradication* during their first 8 years of life, and the incidence of IHD decrease by 1.8% due to the malaria eradication program. Using a similar calculation, we find that the one-year reduction in childhood malaria exposure leads to 1.1% and 1.2% decreases in the incidence of CHF and stroke, 0.4% and 1.1% increases in the job participation rate and elementary school completion, and a 1.7% and 2.2% decrease in the probability of needing help with ADL and homekeeping.

Exclude the Very High Endemic Counties

From Figures 2-2 and 2-3, we can find that counties with very high malaria endemic all locate on the east coast of the island. The Jade Mountain divides the Taiwan island into two parts, and the climate and demography are different between the west and east coasts. One assumption of our basic specification is that there is no different trends in areas with different

pre-eradication spleen rate that are also related to the future outcomes. As a result, we exclude counties with very high malaria spleen rate (red areas in Figure 2-7), and compare the control counties with high endemic counties using the following regression:

$$Outcome_{icj} = \alpha + \beta High\_Endemic_j \times Post\_Eradication_c + \gamma X_{icj} + \lambda_c + \delta_j + \varepsilon_{icj}. \quad (9)$$

The results in Panels A2 and B2 of Table 2-4 indicate that in high endemic areas, malaria eradication leads to higher job participation rate and elementary completion rate for females, and lower incidence of cerebral palsy for males.

Aggregate Data

In the third specification check, we aggregate the data from individual levels to county levels. Aggregating the data makes sense for several reasons. First, no individual-level variables are used as regressors --- malaria spleen rates vary by county, and control variables, such as current residence and mortality rates, also vary by counties. Second, some diseases are very rare, and it is very hard to identify the effect on a single disease at the individual level. Therefore, aggregating the data can provide more variations. Third, the random measurement error of outcome can be greatly reduced or eliminated in aggregate data.

Because NHI inpatient records are annual, we aggregate the data into patients' county of birth, year of birth, and year of survey. There are 23 cells for county of birth, 6 cells for year of birth, and 15 cells for year of survey, resulting in 2,576 potential cells. For major illnesses, the records were kept on file until the certificate is expired once a patient was diagnosed. We aggregate NHI major illness data into patients' county of birth and year of birth, resulting in 184 potential cells (23 birth county cells × 8 birth year cells). Similarly, we aggregate the year 2000 census data into 184 cells according to birth county and birth year. We then obtain weighted coefficients, where the set of weights is the number of observations in each cell. We estimate the

following equation:

$$\begin{aligned} Outcome_{cj} = {} & \alpha + \beta_1 High\_Endemic_j \times Post\_Eradication_c \\ & + \beta_2 Very\_High\_Endemic_j \times Post\_Eradication_c \\ & + \lambda_c + \delta_j + \varepsilon_{cj}. \end{aligned} \tag{10}$$

Results are shown in Panels A3 and B3 of Table 2-4. Under this specification, the malaria eradication program decreases the incidence of cerebral palsy for both genders, and also makes people need less help for homekeeping.

2.6.2 Other Robustness Checks

Robustness Check for Heart Disease and Stroke

Our sample covers people who were born in 1951, 1952, 1959, and 1960, so the age gap between these two groups is 8 years on average. The 1997--2011 NHI data contain health information for people in the control cohorts ($Post\_Eradication = 0$) during their fifties, and the health outcomes for people in the post eradication cohorts ($Post\_Eradication = 1$) during their forties. Because the incidence of heart disease or stroke is highly related to age, we check our conclusions for robustness after putting these restrictions on age. For the robustness check, we use only inpatient records for those ages 46--51.[41] The results in Table 2-5 further confirms that the malaria eradication program significantly decreases the likelihood of getting IHD and ischemic stroke for females, but there is mixed evidence about the impact of the malaria eradication program on male chronic diseases.

Robustness Check for Education and Labor Outcomes

Taiwan census data have rich individual background information including marital status,

[41]People in the oldest cohort (born in 1951) are 46--60 years old during the NHI period, while people in the youngest cohort (born in 1960) are 37--51 years old. We use the overlap group, ages 46--51.

race, and number of family members, all of which are related to adult outcomes. In this robustness check, we run regressions including all of these abovementioned control variables. In Table 2-6, we find that the effects of the eradication program on female work status and education levels are very robust --- the estimates remain significant and the magnitudes do not change very much. However, we again do not find any significant impact on male educational attainment and labor outcomes.

2.7 Conclusion

Based on the malaria eradication program in Taiwan, we examine the long-term effects of early life malaria exposure on future health, education, and labor outcomes. Our results show that both *in utero* and childhood malaria exposure are associated with worse later life health and education outcomes, especially for females. For example, the malaria eradication program decreases the incidence of IHD and ischemic stroke for women by 50% and 68% respectively in very high malaria-endemic areas. The effects of the eradication program on female major illnesses are significant only when the chronic renal failure is the outcome variable. Malaria eradication also increases the job participation and educational attainment for women. For men, the impact of malaria eradication is less significant and robust in general --- we only find a significant decrease in the incidence of cerebral palsy.

Malaria eradication also has implications for early life environment and post-natal investment in children. As mentioned earlier, we are trying to identify the effects of being born in areas with malaria prevalence, not the effects of being infected. Our findings suggest that investments in improving early life environment are worthwhile, because they will improve health and education outcomes in adulthood. Furthermore, the insignificant impact on males may be explained by gender-biased post-natal investments --- if there is a preference for males in the

culture, then it is easier to buffer the negative impact of an unfavorable early life environment through higher post-natal investments.

2.8 Appendix: Impact of Malaria Exposure and DDT Exposure on Cancer

Here, we extend our study to the long-term effects of malaria exposure on cancer, which has been the leading cause of death in Taiwan for the past 31 years (according to the Department of Health). From our empirical results, we witness that the malaria eradication program increases the incidence of cancers in some cases. Given the potential link between DDT exposure and cancer, we discuss these effects on cancer risk in a separate section. Many previous studies have established a positive relationship between DDT exposure and cancer risks (Cohn et al., 2007). Because of this positive association, our estimate of the effect of the malaria eradication program will be understated. Because people in areas with higher levels of malaria prevalence were also exposed to more DDT spraying, failing to consider the impact of DDT will underestimate the beneficial effects of malaria eradication.

We first identify 12 types of cancer, based on the ICD-9 codes[42], then run regressions based on Equation 7. The results are shown in Appendix Table 2-A2. Although malaria reduction and DDT spraying are highly correlated, they are not perfectly correlated. In order to have a more precise measure of DDT exposure, we use information on the DDT spraying plan based on a map from the WHO called "Four-year island-wide malaria control program" (Figure 2-1). It shows Taiwan island is divided into four regions: 1) the pioneer plan area with DDT spraying from 1952 to 1955, 2) a highly endemic area with DDT spraying from 1953 to 1955, 3) a moderately endemic area with DDT spraying from 1954 to 1955, and 4) low endemic and non-malarious areas with no DDT spraying.

[42] The ICD-9 codes to identify cancer type can be found in the following link: https://cris.bhp.doh.gov.tw/pagepub/Home.aspx?itemNo=cr.h.10&helperNo=ICD

$$
\begin{aligned}
Outcome_{icj} = \alpha &+ \beta_1 High\_DDT_j \times Post\_Eradicatoin_c \\
&+ \beta_2 Very\_High\_DDT_j \times Post\_Eradicatoin_c \\
&+ \lambda_c + \delta_j + \varepsilon_{icj}.
\end{aligned} \tag{11}
$$

Here, *High_DDT* is a dummy variable that equals one for counties with DDT spraying frequency between the median and 90th percentile, and *Very_High_DDT* is another dummy variable which equals one for counties with DDT spraying higher than the 90th percentile. We use GIS software to digitize the DDT spraying plan map, and then overlie the locations of townships onto this map. Based on Figure 2-8, each township is then assigned to one of the above mentioned four DDT spraying plan regions.

The DD Results based on Equation 11 are shown in Table 2-A3, and we find mixed evidence of the impact of early life exposure to malaria and DDT on the incidence of cancers. For females, the incidence of oral cancer decreases and the incidences of respiratory, breast and endocrine cancers increase due to the malaria eradication program. For males, the incidence of gland and brain cancers decrease and the incidence of oral and urinary cancer increases due to the eradication program. These estimates indicate the joint effect of malaria exposure and DDT exposure. Because people born in areas with high malaria endemicity should benefit from less malaria exposure but are worse off due to more DDT exposure, the positive sign of the coefficients could be explained by more DDT exposure while the negative sign could be explained by less malaria exposure.

2.9 Reference

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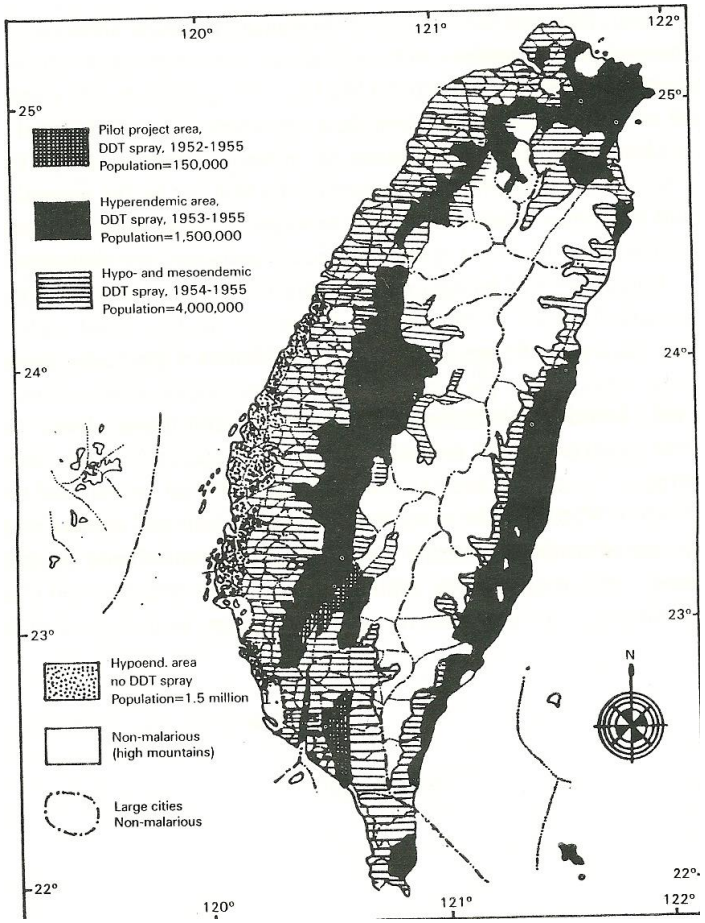

Figure 2-1 Four-year Island-wide Malaria Control Program (Source: WHO)

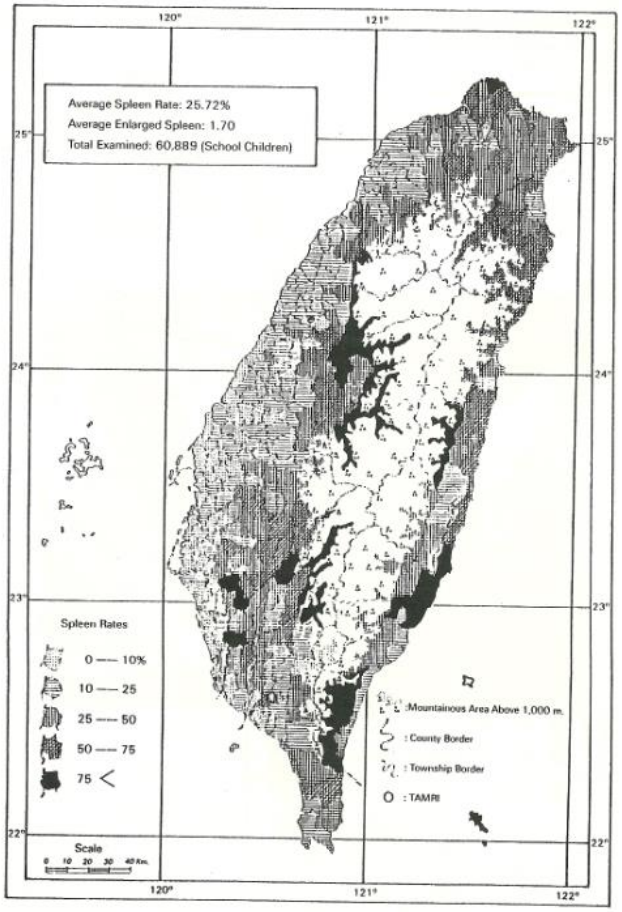

Figure 2-2 Malaria Spleen Rates in Taiwan, 1953 (Source: WHO)

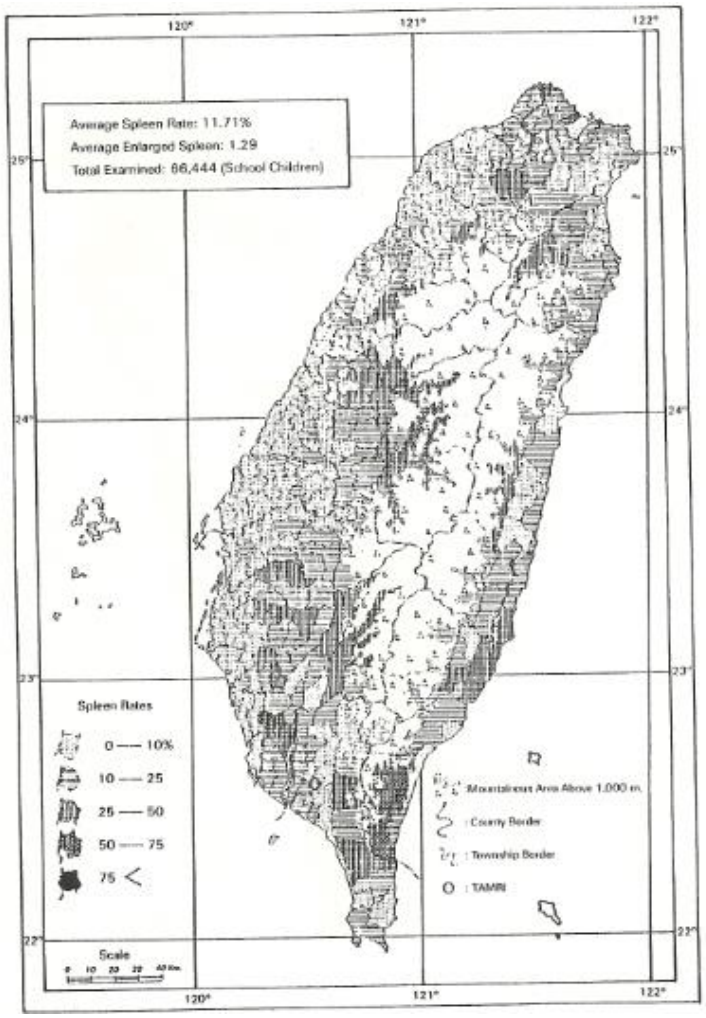

Figure 2-3 Malaria Spleen Rates in Taiwan, 1955 (Source: WHO)

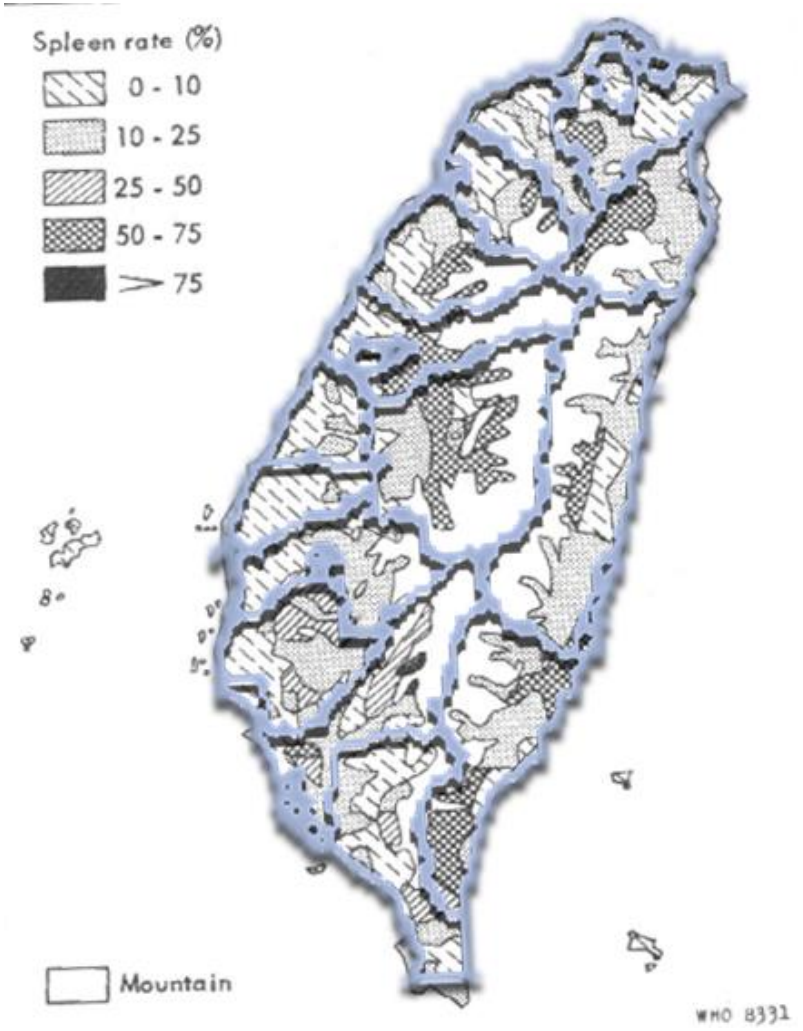

Figure 2-4 "Malaria Spleen Rates in Taiwan, 1953" with county boundaries

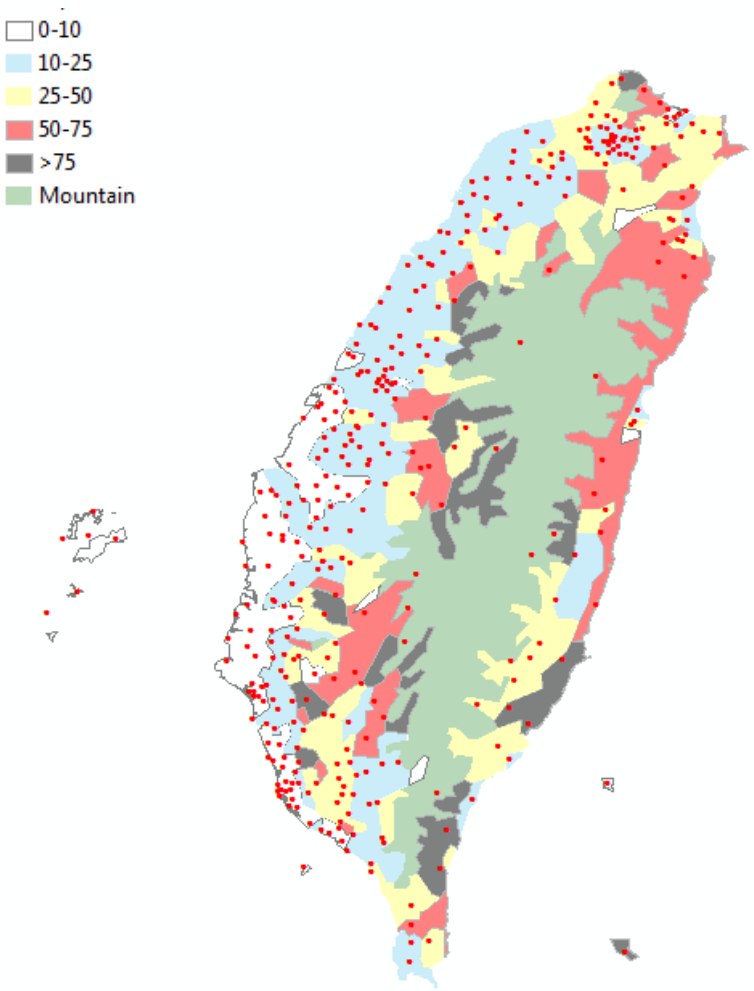

Figure 2-5 Digitized "Malaria Spleen Rates in Taiwan, 1953"

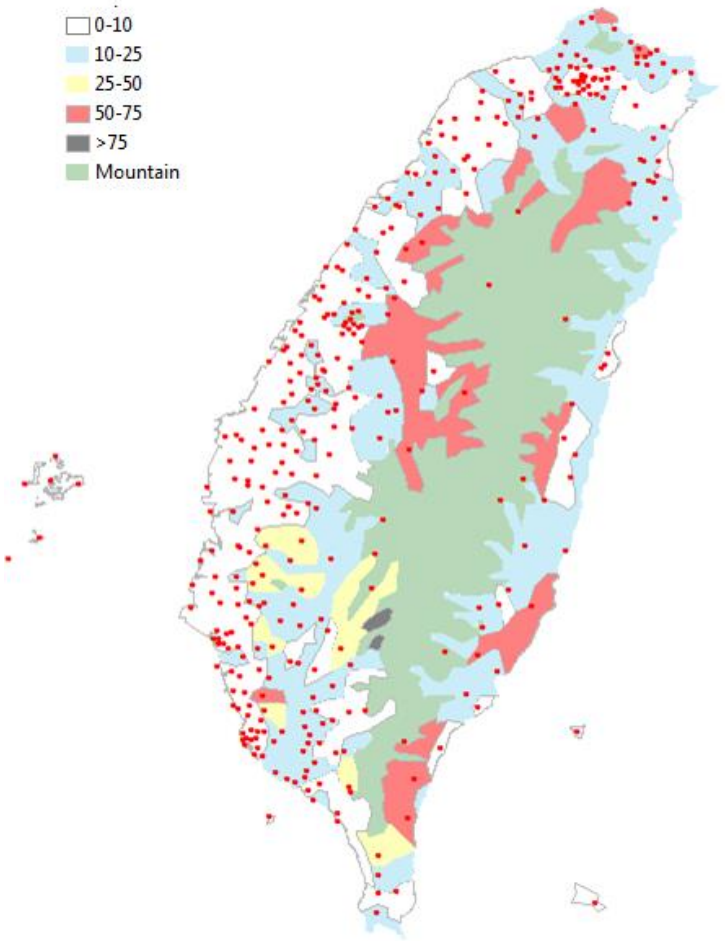

Figure 2-6 Digitized "Malaria Spleen Rates in Taiwan, 1955"

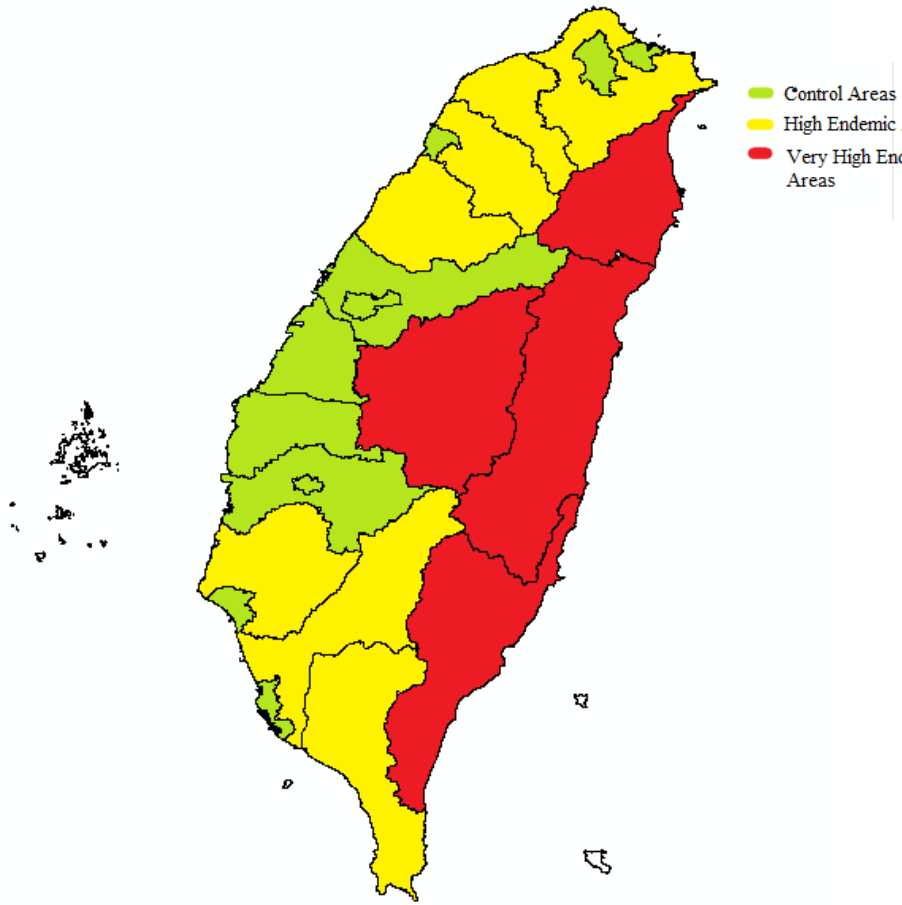

Figure 2-7 Control, High Endemic, and Very High Endemic Areas

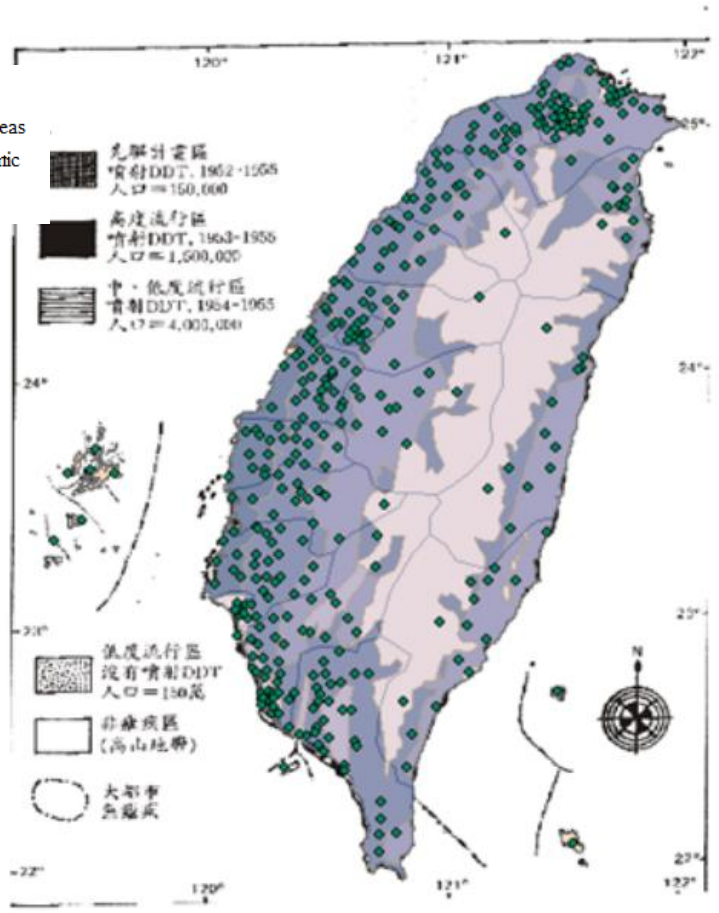

Figure 2-8 Four-year Island-wide Malaria Control Program

Table 2-1 Data Description

	Female						Male					
	Pre-Eradication			Post-Eradication			Pre-Eradication			Post-Eradication		
Variable	Control	High	Very High	Control	High	Very High	Control	High	Very High	Control	High	Very High
Panel A: Chronic Diseases												
AMI	559	676	777	390	610	789	3201	3682	3104	3848	3766	3539
IHD	8435	9715	12951	3789	4000	5454	14381	14884	15091	11204	10810	11277
CHF	2224	2295	3281	1003	1121	1842	3194	3092	3392	1893	2130	2100
Hemorrhagic stroke	2012	2287	3166	1601	1831	2512	4816	5011	6008	3822	4123	4836
Ischemic stroke	2893	3215	4202	2289	2110	2488	5373	6160	5893	5110	5149	5355
Sample Size	155545	127223	34747	187397	150740	150740	161535	128725	34789	194929	154020	42387
Panel B: Major Illnesses												
Malignant Neoplasm	74917	72322	68783	48619	47074	44898	75179	69948	70913	39019	38787	38715
Chronic Renal Failure	9618	10250	10476	4771	5035	4784	11669	11482	10578	5684	5473	5072
Autoimmune Disease	12787	11452	13670	9141	8359	9712	2736	2905	2874	1821	1727	2359
Psychoses	17268	16184	20088	15715	14316	19567	13793	12266	16183	15236	13420	16491
Organ Malformation	1472	1344	1669	1206	1194	1268	1127	1041	920	1036	1026	991
Organ Transplant	1087	794	1094	736	610	646	1591	1453	1466	1293	987	1014
Cerebral Palsy	167	157	115	114	90	84	198	225	201	1108	844	613
Trauma	2237	1949	2792	1254	1393	1842	5411	5112	7962	4463	4733	7479
Long-term Mechanical Ventilation	3118	2790	3856	1313	1287	1674	5417	5438	5117	2760	2552	3326
Cirrhosis	2006	1863	2475	720	710	1866	8778	7885	9773	6407	6175	8352
Sample Size	155545	127223	34747	187397	150740	41806	161535	128725	34789	194929	154020	42387
Panel C: Education and Labor Market Outcomes												
Work	553624	549062	565654	647222	658435	659773	879769	878270	892402	916556	913654	923913
Elementary School Completion	474651	441887	408732	787133	791909	782451	637013	635426	590656	902955	907885	897706
Need Care for ADL	6238	6435	6961	4399	4243	4023	7267	7322	8432	5509	5611	6558
Need Care for Homekeeping	6620	6541	7938	4754	4591	4895	6698	6501	8337	5536	5142	6637
Sample Size	146670	122305	32755	174583	143759	39021	149446	121819	31785	179175	145079	38272

In the original data, health outcomes are dummy variables indicating whether the individual has this type of disease. Because the magnitude is too small, we multiply the values by 1,000,000. Thus, values in Panel I indicate the number of patients with certain diseases in every million people.

Table 2-2 Effects of Malaria Eradication on Long-Term Outcomes

Dependent variable:	Chronic Disease					Major Illness										Education and Labor			
	AMI	IHD	CHF	Ischemic Stroke	Hemorrhagic Stroke	Malignant Neoplasm	Chronic Renal Failure	Autoimmune Disease	Psychoses	Organ Malform	Organ Transplant	Cerebral Palsy	Trauma	Long-term Mechanical Ventilation	Cirrhosis	Work	Elementary School Completion	Need Care for ADL	Need Care for Homekeeping
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)
Panel A: Female																			
A1. Basic Specification (Omitted Category: Low Endemic x Post Eradication)																			
High Endemic ×	-151	-685	436	-199	-1,010	1,415	-378	460	-34	119	172	-228*	396	277	100	0.017**	0.052	-346.648	-35.607
Post Eradication	[207]	[1,591]	[533]	[555]	[957]	[2,126]	[280]	[571]	[705]	[166]	[160]	[129]	[296]	[195]	[315]	[0.007]	[0.031]	[286.067]	[371.095]
Very High	-68	-6,245**	-1,969*	-2,155***	-3,051***	2,300	-943***	-448	1,234	-145	-113	-254**	-52	-432	619	0.001	0.079**	1,112.366*	-1,135.165
Endemic × Post	[240]	[2,248]	[1,039]	[677]	[950]	[1,706]	[306]	[547]	[1,309]	[192]	[219]	[115]	[307]	[827]	[415]	[0.004]	[0.036]	[531.939]	[771.842]
Sample Size	688,430	688,430	688,430	688,430	688,430	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	659,093	659,094	659,095	659,096
A2. Control For Current Residence																			
High Endemic ×	-159	-659	459	-185	-986	1,555	-357	473	50	117	173	-231*	405	288	113	15,370**	52,899*	-372	-56
Post Eradication	[207]	[1,579]	[531]	[564]	[953]	[2,123]	[263]	[575]	[662]	[167]	[160]	[129]	[303]	[197]	[308]	[7,242]	[29,656]	[277]	[357]
Very High	-47	-5,988**	-1,885*	-2,131***	-2,931***	2,362	-901**	-441	1,623	-141	-105	-247**	-8	-385	691	2,389	75,394**	-990*	-964
Endemic × Post	[245]	[2,172]	[1,005]	[678]	[915]	[1,654]	[332]	[568]	[1,387]	[196]	[223]	[115]	[306]	[827]	[441]	[3,933]	[36,022]	[503]	[778]
Sample Size	688,375	688,375	688,375	688,375	688,375	681,746	681,746	681,746	681,746	681,746	681,746	681,746	681,746	681,746	681,746	659,093	659,093	659,093	659,093
A3. Control For Death During Early Life																			
High Endemic ×	-105	-1,057	359	-237	-1,347*	1,854	-444*	462	-177	123	152	-209	334	226	-74	13,623***	62,231**	-412	-171
Post Eradication	[213]	[1,380]	[525]	[581]	[731]	[1,874]	[240]	[583]	[691]	[178]	[153]	[126]	[312]	[218]	[203]	[4,202]	[27,146]	[297]	[334]
Very High	24	-7,002***	-2,125*	-2,234***	-3,735***	3,190*	-1,077***	-443	946	-137	-154	-216*	-178	-536	267	-5,049	99,293***	-1,244**	-1,409**
Endemic × Post	[290]	[2,374]	[1,081]	[718]	[1,130]	[1,737]	[328]	[536]	[1,203]	[210]	[232]	[115]	[276]	[827]	[287]	[4,797]	[30,792]	[509]	[655]
Sample Size	688,430	688,430	688,430	688,430	688,430	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	659,093	659,093	659,093	659,093
Panel B: Male																			
B1. Basic Specification (Omitted Category: Low Endemic x Post Eradication)																			
High Endemic ×	-1,269*	-1,237	908	477	-1,089	4,905*	-4	-312	-57	100	-212	-304**	473	-283	511	-0.002	0.015	56.929	-177.202
Post Eradication	[677]	[1,040]	[619]	[749]	[1,396]	[2,601]	[713]	[276]	[741]	[195]	[227]	[120]	[302]	[504]	[403]	[0.003]	[0.033]	[368.828]	[340.182]
Very High	-1,439	-22	309	-1,718**	-774	3,019	431	360	-1,069	195	-194	-518***	198	789	671	-0.005	0.052	-157.187	-590.022
Endemic × Post	[953]	[1,273]	[538]	[823]	[1,174]	[2,815]	[1,151]	[274]	[960]	[247]	[381]	[125]	[453]	[489]	[604]	[0.003]	[0.033]	[590.589]	[593.068]
Sample Size	699,571	699,572	699,573	699,574	699,575	678,186	678,187	678,188	678,189	678,190	678,191	678,192	678,193	678,194	678,195	665,576	665,577	665,578	665,579
B2. Control For Current Residence																			
High Endemic ×	-1,289*	-1,270	925	454	-1,104	5,110*	-22	-308	21	100	-202	-301**	520	-260	630	-1,651	16,998	17	-149
Post Eradication	[692]	[1,091]	[620]	[741]	[1,389]	[2,521]	[706]	[279]	[739]	[197]	[222]	[122]	[318]	[509]	[433]	[3,440]	[33,208]	[341]	[344]
Very High	-1,463	-29	339	-1,650*	-755	3,197	429	378	-661	199	-176	-518***	237	818	846	-5,655*	50,300	-149	-570
Endemic × Post	[955]	[1,318]	[555]	[830]	[1,216]	[2,826]	[1,156]	[279]	[700]	[246]	[378]	[124]	[502]	[525]	[681]	[3,073]	[33,818]	[655]	[681]
Sample Size	699,432	699,432	699,432	699,432	699,432	678,093	678,093	678,093	678,093	678,093	678,093	678,093	678,093	678,093	678,093	665,576	665,576	665,576	665,576
B3. Control For Death During Early Life																			
High Endemic ×	-1,261*	-1,256	815	414	-1,327	4,520*	-173	-308	-121	135	-191	-306**	440	-387	465	-2,086	21,937	8	-206
Post Eradication	[685]	[1,067]	[541]	[697]	[1,072]	[2,329]	[555]	[284]	[735]	[155]	[212]	[122]	[310]	[424]	[377]	[3,309]	[28,243]	[362]	[336]
Very High	-1,407	-101	-76	-1,976**	-1,754	1,462	-253	376	-1,328	336	-108	-526***	63	369	488	-5,989*	76,513**	-345	-699
Endemic × Post	[1,018]	[1,337]	[559]	[939]	[1,205]	[2,621]	[1,139]	[282]	[1,086]	[218]	[344]	[135]	[394]	[377]	[600]	[3,443]	[29,984]	[590]	[607]
Sample Size	699,571	699,571	699,571	699,571	699,571	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	665,576	665,576	665,576	665,576

Standard errors, reported in parentheses, are adjusted for clustering by 46 county-post cells.
*** p<0.01, ** p<0.05, *p<0.1

Table 2-3 Effects of Malaria Eradication on Long-Term Outcomes (Difference Cohorts)

Dependent variable:	Chronic Disease					Major Illness										Education and Labor			
	AMI	IHD	CHF	Ischemic Stroke	Hemorrhagic Stroke	Malignant Neoplasm	Chronic Renal Failure	Autoimmune Disease	Psychoses	Organ Malform	Organ Transplant	Cerebral Palsy	Trauma	Long-term Mechanical Ventilation	Cirrhosis	Work	Elementary School Completion	Need Care for ADL	Need Care for Homekeeping
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)
Panel A: Female																			
A1. The Effect of The First Two Rounds of DDT Spraying -- Birth Cohorts 51 52 (Pre) VS 54 55 (Post)																			
High Endemic ×	41	-1,173	-283	-95	-1,930*	2,231	114	-372	-1,054	30	396**	23	157	461	-31	7,631	30,391***	-70	-97
Post Eradication	[265]	[1,323]	[596]	[465]	[1,031]	[1,650]	[560]	[424]	[650]	[243]	[185]	[111]	[255]	[366]	[214]	[6,538]	[8,986]	[474]	[459]
Very High	485	-6,375***	-1,148	-1,679	-5,752***	620	705	-516	444	-457	-22	-264*	-468	733	299	7,743	21,821**	-1,165***	-542
Endemic × Post	[592]	[1,410]	[777]	[1,185]	[1,430]	[2,782]	[831]	[692]	[2,130]	[355]	[298]	[128]	[685]	[605]	[519]	[4,628]	[8,883]	[321]	[792]
Sample Size	631,171	631,171	631,171	631,171	631,171	624,268	624,268	624,268	624,268	624,268	624,268	624,268	624,268	624,268	624,268	603,872	603,872	603,872	603,872
A2. The Effect of The Last Three Rounds of DDT Spraying -- Birth Cohorts 54 55 (Pre) VS 59 60 (Post)																			
High Endemic ×	97	47	77	-204	-310	-239	-504	707	1,211*	38	-21	-115	306	-191	141	2,751	49,745**	-613**	-546*
Post Eradication	[153]	[728]	[204]	[380]	[367]	[1,499]	[340]	[570]	[646]	[202]	[159]	[113]	[192]	[169]	[153]	[2,712]	[21,166]	[242]	[284]
Very High	-313	-2,910**	-913	-1,053**	374	975	-1,300***	-51	1,749*	26	118	-36	199	-562*	217	-8,212**	83,594***	-441*	-605
Endemic × Post	[225]	[1,107]	[615]	[413]	[648]	[1,637]	[446]	[731]	[970]	[191]	[152]	[138]	[343]	[325]	[149]	[3,190]	[21,495]	[251]	[399]
Sample Size	712,580	712,580	712,580	712,580	712,580	706,650	706,650	706,650	706,650	706,650	706,650	706,650	706,650	706,650	706,650	682,335	682,335	682,335	682,335
Panel B: Male																			
B1. The Effect of The First Two Rounds of DDT Spraying -- Birth Cohorts 51 52 (Pre) VS 54 55 (Post)																			
High Endemic ×	105	516	464	251	-1,779	2,419	528	-132	-1,069*	243	-103	-113	789*	940**	34	-3,391	15,161	-507	-751**
Post Eradication	[589]	[1,117]	[577]	[1,100]	[1,288]	[2,277]	[748]	[268]	[560]	[150]	[183]	[91]	[412]	[350]	[494]	[2,527]	[10,324]	[326]	[336]
Very High	901	-2,424*	205	-1,983**	-1,120	995	667	-123	-1,007	137	149	-16	210	615	451	1,612	20,473*	-890	-567
Endemic × Post	[919]	[1,189]	[643]	[725]	[1,161]	[1,197]	[527]	[477]	[692]	[174]	[157]	[259]	[553]	[548]	[1,040]	[4,106]	[10,974]	[988]	[860]
Sample Size	639,456	639,456	639,456	639,456	639,456	617,314	617,314	617,314	617,314	617,314	617,314	617,314	617,314	617,314	617,314	608,079	608,079	608,079	608,079
B2. The Effect of the Last Three Rounds of DDT Spraying -- Birth Cohorts 54 55 (Pre) VS 59 60 (Post)																			
High Endemic ×	7	1,704	-650*	-1,203**	-1,283**	3,227*	-585	197	1,471**	-104	-375*	-209**	-104	-278	-139	713	27,396	245	-1
Post Eradication	[586]	[1,352]	[348]	[556]	[539]	[1,608]	[423]	[192]	[573]	[186]	[201]	[81]	[300]	[280]	[343]	[1,770]	[20,705]	[433]	[421]
Very High	-421	1,250	-1,466*	-1,047	1,315*	3,901*	-908	538	777	124	-225	-240	161	137	-358	246	57,825**	23	-645
Endemic × Post	[596]	[1,510]	[720]	[683]	[717]	[2,188]	[698]	[409]	[826]	[238]	[288]	[224]	[460]	[329]	[527]	[2,092]	[22,244]	[377]	[499]
Sample Size	725,351	725,351	725,351	725,351	725,351	704,728	704,728	704,728	704,728	704,728	704,728	704,728	704,728	704,728	704,728	690,660	690,660	690,660	690,660

Standard errors, reported in parentheses, are adjusted for clustering by 46 county-post cells.
*** p<0.01, ** p<0.05, *p<0.1

Table 2-4 Specification Test

Dependent variable:	Choronic Disease					Major Illness										Education and Labor			
	AMI	IHD	CHF	Ischemic Stroke	Hemorrhagic Stroke	Malignant Neoplasm	Chronic Renal Failure	Autoimmune Disease	Psychoses	Organ Malform	Organ Transplant	Cerebral Palsy	Trauma	Long-term Mechanical Ventilation	Cirrhosis	Work	Elementary School Completion	Need Care for ADL	Need Care for Homekeeping
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)
Panel A: Female																			
A1. Dose Response Model																			
High Endemic ×	-337	-2,814	1,001	-613	-3,915*	4,655	-1,482**	1,376	-392	337	426	-572	858	710	-142	36,271***	169,011**	-1,192	-593
Post Eradication	[574]	[3,859]	[1,412]	[1,596]	[2,073]	[5,127]	[679]	[1,602]	[1,944]	[512]	[415]	[349]	[862]	[607]	[576]	[12,050]	[74,460]	[864]	[938]
Very High	225	-19,314***	-6,008**	-6,478***	-10,719***	9,266*	-3,427***	-1,330	2,234	-354	-425	-602*	-633	-1,517	859	-14,379	269,768***	-3,815***	-4,396**
Endemic × Post	[772]	[6,544]	[2,897]	[2,030]	[3,187]	[4,605]	[855]	[1,576]	[3,059]	[630]	[693]	[318]	[731]	[2,309]	[775]	[13,040]	[84,097]	[1,318]	[1,828]
Sample Size	688,430	688,430	688,430	688,430	688,430	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	659,093	659,093	659,093	659,093
A2. Exclude Areas With Very High Malaria Endemicity																			
High Endemic ×	-93	-1,203	289	-231	-1,403*	1,846	-452*	475	-113	119	141	-212	338	227	-71	13,403***	61,985**	-391	-130
Post Eradication	[214]	[1,376]	[527]	[587]	[727]	[1,881]	[239]	[586]	[702]	[179]	[155]	[127]	[315]	[218]	[204]	[4,167]	[27,359]	[295]	[337]
Sample Size	612,652	612,652	612,652	612,652	612,652	607,005	607,005	607,005	607,005	607,005	607,005	607,005	607,005	607,005	607,005	587,317	587,317	587,317	587,317
A3. Aggregate Data																			
High Endemic ×	-309	-386	654	118	-1,064	2,542	-277	224	14	90	175	-251	252	237	43	16,758*	52,302	-5	-318
Post Eradication	[489]	[937]	[569]	[638]	[1,259]	[1,799]	[372]	[616]	[694]	[151]	[144]	[165]	[285]	[250]	[288]	[8,647]	[32,994]	[6]	[262]
Very High	-419	-460	-470	-1,537*	-1,647	1,682	-774	-372	2,194	-428*	58	-358**	301	-391	73	1,597	77,160*	-4	-1,865**
Endemic × Post	[566]	[1,753]	[515]	[855]	[1,573]	[1,724]	[667]	[667]	[1,457]	[244]	[211]	[170]	[261]	[778]	[399]	[4,839]	[39,445]	[5]	[699]
Sample Size	2,072	2,072	2,072	2,072	2,072	138	138	138	138	138	138	138	138	138	138	132	132	132	132
Panel B: Male																			
B1. Dose Response Model																			
High Endemic ×	-3,530*	-2,907	2,057	949	-4,034	13,074*	-321	-704	-532	382	-586	-858**	1,264	-918	1,083	-5,496	59,185	32	-542
Post Eradication	[1,807]	[2,834]	[1,552]	[1,980]	[3,109]	[6,431]	[1,508]	[741]	[1,962]	[421]	[569]	[330]	[878]	[1,208]	[1,088]	[9,023]	[77,439]	[1,022]	[976]
Very High	-3,984	-608	-563	-5,461**	-5,442	3,921	-801	967	-3,787	962	-474	-1,459***	-48	1,196	1,475	-16,053	208,242**	-836	-1,970
Endemic × Post	[2,574]	[3,386]	[1,624]	[2,632]	[3,415]	[7,159]	[3,231]	[771]	[3,063]	[606]	[965]	[338]	[1,075]	[1,023]	[1,670]	[9,418]	[82,401]	[1,450]	[1,534]
Sample Size	699,571	699,571	699,571	699,571	699,571	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	665,576	665,576	665,576	665,576
B2. Exclude Areas With Very High Malaria Endemicity																			
High Endemic ×	-1,241*	-1,267	813	408	-1,329	4,539*	-182	-305	-142	135	-197	-306**	449	-384	455	-2,034	22,193	2	-207
Post Eradication	[697]	[1,076]	[544]	[698]	[1,077]	[2,345]	[559]	[286]	[737]	[155]	[215]	[123]	[306]	[425]	[377]	[3,347]	[28,403]	[366]	[338]
Sample Size	624,037	624,037	624,037	624,037	624,037	605,645	605,645	605,645	605,645	605,645	605,645	605,645	605,645	605,645	605,645	595,519	595,519	595,519	595,519
B3. Aggregate Data																			
High Endemic ×	-1,269*	-1,237	908	477	-1,089	4,766*	-295	-85	483	84	-137	-243	493*	380	521	-1,476	16,605	-193	-191
Post Eradication	[684]	[1,051]	[625]	[756]	[1,410]	[2,745]	[708]	[278]	[616]	[161]	[240]	[168]	[241]	[523]	[486]	[3,661]	[35,621]	[236]	[469]
Very High	-1,439	-22	309	-1,718*	-774	3,250	351	162	-356	255	-66	-440***	-69	1,011**	1,075	-9,138***	50,429	-193	-1,126***
Endemic × Post	[963]	[1,286]	[544]	[831]	[1,186]	[2,394]	[960]	[444]	[658]	[232]	[360]	[121]	[638]	[476]	[730]	[2,383]	[35,580]	[236]	[338]
Sample Size	1,383	1,383	1,383	1,383	1,383	138	138	138	138	138	138	138	138	138	138	132	132	132	132

Standard errors, reported in parentheses, are adjusted for clustering by 46 county-post cells.
*** p<0.01, ** p<0.05, *p<0.1

Table 2-5 Long-term Impact on Chronic Diseases with Age Restriction

Dependent variable:	AMI	IHD	CHF	Ischemic Stroke	Hemorrhagic Stroke	AMI	IHD	CHF	Ischemic Stroke	Hemorrhagic Stroke
Panel A: Female						*Panel B: Male*				
A1. The Effect of Malaria Eradication: 51 52 (Pre) VS 59 60 (Post)						B1. Birth Cohorts 51 52 (Pre) VS 59 60 (Post)				
High Endemic × Post Eradication	170	-1,434*	67	-49	-571	-569	-1,005	342	140	-702**
	[148]	[758]	[321]	[259]	[378]	[347]	[821]	[358]	[453]	[273]
Very High Endemic × Post Eradication	289	-3,709***	-215	-302	-1,280**	-273	-1,311*	-249	-232	-147
	[279]	[1,062]	[548]	[297]	[500]	[276]	[745]	[405]	[543]	[344]
Sample Size	677,752	677,752	677,752	677,752	677,752	674,696	674,696	674,696	674,696	674,696
A2. The Effect of the Last Three Rounds of DDT Spraying: 54 55 (Pre) VS 59 60 (Post)						B2. Birth Cohorts 54 55 (Pre) VS 59 60 (Post)				
High Endemic × Post Eradication	55	-422	-141	-410*	-231	20	906	-347	-327	-204
	[127]	[472]	[143]	[222]	[191]	[283]	[688]	[244]	[311]	[266]
Very High Endemic × Post Eradication	-268*	-2,677***	-542	-542	269	-492	-71	-672**	-179	1,071***
	[133]	[338]	[346]	[333]	[326]	[287]	[592]	[294]	[460]	[354]
Sample Size	702,840	702,840	702,840	702,840	702,840	701,411	701,411	701,411	701,411	701,411
A3. The Effect of the First Two Rounds of DDT Spraying: 51 52 (Pre) VS 54 55 (Post)						B3. Birth Cohorts 51 52 (Pre) VS 54 55 (Post)				
High Endemic × Post Eradication	85	-1,097**	230	185	-475	-453	-1,806***	512**	242	-322
	[169]	[488]	[252]	[269]	[384]	[356]	[463]	[197]	[290]	[248]
Very High Endemic × Post Eradication	507**	-3,357***	178	-21	-1,701***	80	-2,283***	440	149	-290
	[226]	[498]	[512]	[311]	[451]	[321]	[378]	[365]	[418]	[469]
Sample Size	642,400	642,400	642,400	642,400	642,400	637,225	637,225	637,225	637,225	637,225

Standard errors, reported in parentheses, are adjusted for clustering by 46 county-post cells.
*** p<0.01, ** p<0.05, *p<0.1

Table 2-6 Effects of Malaria Eradication on Long-term Education and Labor Market Outcomes

-- Control for More Individual Level Background

Dependent variable:	Work			Elementary school Completion			Need Care for ADL			Need Care for Homekeeping		
Panel A: Female												
High Endemic × Post	17,347**	17,282**	17,103**	52,746	53,587*	53,259*	-276	-289	-287	43	33	33
Eradication	[6,484]	[6,523]	[6,604]	[30,970]	[30,974]	[30,862]	[304]	[307]	[308]	[386]	[386]	[387]
Very High Endemic ×	2,208	2,020	2,008	79,769**	82,480**	82,457**	-1,004*	-1,066*	-1,066*	-1,014	-1,067	-1,067
Post Eradication	[3,759]	[3,762]	[3,808]	[36,040]	[35,747]	[35,536]	[545]	[557]	[557]	[775]	[780]	[780]
Sample Size	659,093	659,093	659,093	659,093	659,093	659,093	659,093	659,093	659,093	659,093	659,093	659,093
Panel B: Male												
High Endemic × Post	-2,005	-1,982	-1,988	15,379	16,236	16,305	67	48	45	-167	-189	-191
Eradication	[3,556]	[3,566]	[3,569]	[32,372]	[32,401]	[32,403]	[413]	[415]	[414]	[377]	[383]	[382]
Very High Endemic ×	-6,693*	-6,400*	-6,403*	50,847	54,337	54,360	35	-109	-110	-383	-524	-525
Post Eradication	[3,246]	[3,273]	[3,273]	[32,324]	[32,109]	[32,122]	[534]	[513]	[513]	[494]	[454]	[454]
Sample Size	665,576	665,576	665,576	665,576	665,576	665,576	665,576	665,576	665,576	665,576	665,576	665,576
Marriage Status	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Race	N	Y	Y	N	Y	Y	N	Y	Y	N	Y	Y
Number of Family Member	N	N	Y	N	N	Y	N	N	Y	N	N	Y

Standard errors, reported in parentheses, are adjusted for clustering by 46 county-post cells.

*** p<0.01, ** p<0.05, *p<0.1

Table 2-A1 The First Letter of ID and The Corresponding Counties

First Letter	Counties Name
A	Taipei Municipality
B	Taichung Municipality
C	Keelung Municipality
D	Tainan Municipality
E	Kaohsiung Municipality
F	Taipei County
G	Ilan County
H	Taiyuan County
I	Chia-I Municipality
J	Hsinchu County
K	Miaoli County
L	Taichung County
M	Nantou County
N	Changhua County
O	Hsinchu Municipality
P	Yunlin County
Q	Chia-I County
R	Tainan County
S	Kaohsiung County
T	Pingtung County
U	Hualien County
V	Taitung County
W	Kinmen County
X	Penghua County
Y	-
Z	Lienchiang County

Table 2-A2 Effects of Malaria Eradication on Cancer

Dependent variable:	Oral (1)	Gland (2)	Digestive (3)	Respiratory (4)	Bone (5)	Breast (6)	Urinary (7)	Eye (8)	Brain (9)	Endocrine (10)	Lymphoma (11)	Leukemia (12)
Panel A: Female												
High Endemic × Post Eradication	-148	82	-386	214	-34	1,927	-137	-32	-61	164	-300*	41
	[318]	[75]	[657]	[351]	[150]	[1,783]	[250]	[29]	[114]	[264]	[162]	[166]
Very High Endemic × Post Eradication	-353	-1	-124	15	-234	2,048	75	-51	263**	585**	-199	-369*
	[461]	[163]	[546]	[365]	[161]	[1,684]	[183]	[32]	[111]	[250]	[338]	[188]
Sample Size	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794
Panel B: Male												
High Endemic × Post Eradication	2,407**	-5	1,282	680	116	-148	137	-15	-33	286*	115	-51
	[991]	[66]	[1,284]	[753]	[203]	[147]	[436]	[23]	[134]	[145]	[159]	[134]
Very High Endemic × Post Eradication	-879	-286***	1,866	405	92	183	708	3	-247	-298	8	282
	[1,563]	[64]	[1,688]	[1,022]	[163]	[308]	[466]	[13]	[297]	[248]	[267]	[189]
Sample Size	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186

Standard errors, reported in parentheses, are adjusted for clustering by 46 county-post cells.
*** p<0.01, ** p<0.05, *p<0.1

Table 2-A3 Effects of DDT Exposure on Cancer

Dependent variable:	Oral (1)	Gland (2)	Digestive (3)	Respiratory (4)	Bone (5)	Breast (6)	Urinary (7)	Eye (8)	Brain (9)	Endocrine (10)	Lymphoma (11)	Leukemia (12)
Panel A: Female												
High Endemic × Post Eradication	324	117	-608	803**	-17	1,601	150	12	23	41	-289	7
	[199]	[74]	[669]	[285]	[165]	[1,866]	[234]	[26]	[120]	[236]	[197]	[152]
Very High Endemic × Post Eradication	-1,111**	-160	-19	-187	-59	2,970**	239	-32	152	753***	-39	155
	[402]	[101]	[480]	[365]	[98]	[1,096]	[217]	[36]	[184]	[231]	[110]	[270]
Sample Size	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794	681,794
Panel B: Male												
High Endemic × Post Eradication	2,486**	-159**	1,244	1,434*	227	-290*	920**	-28	12	1	-110	-88
	[941]	[60]	[1,335]	[701]	[205]	[158]	[391]	[21]	[146]	[143]	[152]	[139]
Very High Endemic × Post Eradication	-485	-216*	2,282*	244	-8	-11	550	-2	-368**	-12	-66	136
	[1,733]	[107]	[1,296]	[765]	[116]	[243]	[330]	[14]	[138]	[433]	[302]	[148]
Sample Size	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186	678,186

Standard errors, reported in parentheses, are adjusted for clustering by 46 county-post cells.
*** p<0.01, ** p<0.05, *p<0.1

3. The Intergenerational Transition of Fetal Programming: Evidences from the Great Chinese Famine from 1959 to 1961

3.1 Introduction

According to Baker's fetal origins hypothesis, unfavorable shocks *in utero* or during early life can lead to negative health outcomes in later stages of life. Putting the fetal origins hypothesis to test, recent researches have shown that individuals who were exposed to shocks such as famines, droughts, wars, and epidemics in the early stages of their gestation tend to have worse health outcomes in their adulthood (Meng and Qian 2006; Yang et al. 2007; Mu and Zhang 2008). If the impact of *in utero* environment is permanent, an important question that emerges is whether the environmental experience of one generation will affect the offspring of subsequent generations. Using the Great Chinese Famine as a natural experiment, in this paper we examine the effect of *in utero* exposure to famine on the health outcomes of offspring based on the China Health and Nutrition Survey (CHNS) data and Nutrition and Health Survey in Taiwan (NAHSIT). In particular, we focus on the effects of having parents exposed to the famine during the early stages of their gestation on the anthropometric outcomes of their children. The socio-economic status at birth, parental education, and the anthropometric outcomes of parents may echo family behavioral traits such as eating habits and nutritional intake and family socio-economic background. The CHNS has a panel data design and contains information that allow us to control for an individuals' socio-economic status (SES) as well as a rich set of information on dietary habits and nutritional intake. Exploiting the availability of this rich set of background information, we also assess whether parents with higher SES are able to buffer the negative impact of their own exposure to the famine on their children's anthropometric outcomes. Lastly, we examine whether the strength of the intergenerational link between parental exposure to the

famine and the child's outcomes varies by the child's gender. In order to have a control group that did not expose to famine at all, we also use data from NAHSIT, which has a similar design and variable as CHNS.

We find that famine survivors have higher BMIs for both genders compared to people who were not affected by the famine. In addition, daughters of famine survivors have lower z-score body mass index (BMI) than children of parents that were not exposed to the famine, but the effects on sons are not significant.

3.2 Literature Review and Background

3.2.1 Literature Review

The Long-term Effects of the Great Chinese Famine

Baker's fetal origins hypothesis suggests that an adverse environment during early life or even *in utero* may lead to negative health outcomes in later stages of life. Recently, many researchers use natural experiments, such as disasters, to investigate the long-term impact of early life environment. Exploiting the Great Chinese Famine, several researchers find supporting evidence for the fetal origins hypothesis. For example, merging the 1990 Population Census to both the 1989 China Health and Nutritional Survey (CHNS) and the 1997 Agricultural Census, Meng and Qian (2006) estimate the impact of the exposure to the famine on the upper quintiles of the distribution of anthropometric to solve the selection problem. If stronger (or taller) people are more likely to survive the famine, the attenuation bias should be smaller on the upper quintiles of the distribution of the outcome because backgrounds of the control and treatment groups are similar on the upper quintile. Their results indicate that *in utero* and early childhood exposure to famine has large negative effects on adult height, weight, weight-for-height, educational attainment and labor supply. Yang et al. (2007) also find that women in the famine

groups are more likely to become overweight in their adulthood, but such differences were not found in men. Mu and Zhang (2008) also find that the negative impact of exposure to the famine varies by gender. They use data from the 2000 China Population Census and the 1998 China Agricultural Census, and find that *in utero* exposure to famine significantly increases the likelihood of disability and illiteracy for rural females, but has a lesser impact on males. They further indicate that more than two-third of the gender difference in the long-term impact can be explained by selection bias caused by gender differences in mortality. In general, the existing literature suggests that *in utero* or early life exposure to adverse shocks such as famines will lead to negative outcomes in later stages of life, being particularly deleterious for women.

The Intergenerational Effects of the Great Chinese Famine

Some researchers take a further step, and study the impact of early life famine exposure on the offspring's outcomes. Using the 1% sample of the 2000 Chinese Census, Almond et al. (2007) find that maternal malnutrition reduces the male to female ratio. They use Hong Kong natality micro data from 1984--2004 to further confirm this pattern of female offspring among mainland-born residents exposed to malnutrition *in utero*. Using CHNS data, Fung and Ha (2010) find that although individuals born during the famine have a higher BMI, their children have a lower height-for-age and weight-for-age. The effect of mothers' famine exposure is larger than the effect of fathers' exposure, and the negative effect for boys is much stronger than for girls.

3.2.2 Background

The Great Chinese famine lasted three years, from 1958 to 1961. According to data from the China Statistical Yearbook (1984), during this period there was a dramatic decrease in crop production in China from 200 million tons (1958) to 143.5 million tons (1960). The Famine was

called “three years of natural disaster” (san nian zi ran zai hai), which implies that the cause of the famine was a fall in grain output due to the bad weather. However, although this three-year period was characterized by unusual weather conditions, the current perception is that the main cause of the famine was a sequence of policy mistakes. In particular, the Great Leap Forward (da yue jin) from 1958--1961, which aimed at rapidly raising industrial production, resulted in a big drop in grain production. During the Great Leap Forward, almost all Chinese villages had been reformed into working communes of several thousand people in size within a year. People stopped agricultural production and produced shoddy and unsellable goods in small “backyard furnaces”. The result of this policy was bewildering. The urban population, which had protected legal rights to receive a stated amount of grain consumption, was the least affected. On the other hand, local officials in the countryside competed to over-report the levels that their communes produced, in response to the new economic organization. As a result, the very grain producers from rural areas suffer the most from the grain shortages, ultimately leading the country to the great famine.

The Great Chinese Famine is a disaster in human history. According to the official record, the total death (birth) was 21.24 million (60.48 million) from 1956--1958, 36.02 million (42.25 million) from 1959--1961, and 21.52 million (81.43 million) from 1962--1964. The estimates suggest that the excess death from the Great Famine was about 15 million. However, under the record system at that time, if a child died before getting a *hukou*[43], his birth and death would not be officially recorded. Moreover, the validity of the existing official records are dubious as, during the Great Leap Forward, the national population surveys and in particular the death records are believed to have been altered by the prevailing political forces of the time. Thus, the

[43] A *hukou* is a household registration or resident permit given by the government of China. *hukou* contain the basic information of a person, such as name, date of birth, marital status and so on. It is also related to the eligibility for medical insurance, policies and certain jobs.

official records are believed to underestimate the true mortality due to the famine. Johnson (1998) and Coale et al. (1984) suggest the excess deaths range somewhere between 20 million and 30 million.

3.3 Data

3.3.1 China Health and Nutrition Survey (CHNS)

Our primary data set is the China Health and Nutrition Survey (CHNS). The CHNS survey rounds collected data in the years 1989, 1991, 1993, 1997, 2000, 2004, 2006 and 2009, and were gathered over a three-day period using a multistage, random cluster process. Samples were drawn from nine provinces (Guangxi, Guizhou, Heilongjiang, Henan, Hubei, Hunan, Jiangsu, Liaoning, and Shandong). Counties in the nine provinces were stratified by income, and a weighted sampling scheme was used to randomly select four counties in each province. If possible, the same households were interviewed over time.

The weight and height outcomes are important indicators of children's health status. Thus, in our paper, we attempt to compare weight outcomes of famine survivals' offspring with those whose parents were not affected by the great famine. To achieve this purpose, we first restrict our sample to the families with both parents that are present and have at least one child. The CHNS data only ask interviewees' current residence, instead of the birth place. Because the immigration between provinces are very rare before 1995 due to the *hukou* system, we use waves 1989, 1991, and 1993 from CHNS data. The famine was from 1959--1961, so we restrict analysis to those whose mothers were born from 1955--1965 (four years before and after the Chinese Great Famine). The relatively narrow birth interval is intended to increase the similarity

of the unobserved characteristics, and can also help to avoid the effect from other shocks.[44]

The weight and height outcomes of each individual are examined by medical specialists. The wealth of information on physical activity, time allocation, dietary habits and energy intake allow us to further investigate the possible underlying mechanism. We drop children without complete parents' education and income information, and restrict the age range of children to 2--20 years old. In the end, our sample contains 312 girls and 374 boys.

3.3.2 Nutrition and Health Survey in Taiwan (NAHSIT)

Our second data set is NAHSIT data, which was designed to monitor the nutritional status, health status, life-styles, and health and nutrition relation knowledge/attitude/practice in the whole population and in various age, sex, geographical, and ethnic groups. A multi-staged, stratified, clustered probability sampling scheme was used. Useful information include weight outcomes based on health examinations, nutrition balance based on 24-hour dietary recall, and household background such as parents' income levels.

This survey has similar designs, contents, and operations as the CHNS. Thus, we want to use sample from the NAHSIT as a control group, to examine the impact of famine exposure. Because waves 1989, 1991, and 1993 from CHNS data are used, we use the closest wave of NAHSIT, which was conducted from 1993--1996. After we restrict it to mothers who were born between 1956 and 1964 and children from 2--20 year old, the final sample with complete information is 304 girls and 342 boys.

3.4 Empirical Specification

We first need to clarify the notations of the generations. The famine happened between

[44] The People's Republic of China was founded in 1949. In the early 1950s, there were a massive economic and social reconstruction, and the 10-year Cultural Revolution (Wen Hua Da Ge Min) which started in 1966.

1959 and 1961, we denote people who were adults during that time as the first generation (G1). The children of G1, or those who were born around the famine period are called the second generation (G2). And the children of G2 are considered to be the third generation (G3).

Our research questions are: 1) whether the adverse environmental experience during early life stages will have a long term effect on future outcomes, and 2) whether the adverse environmental experience of parents will affect their children. According to Barker's Fetal Origins Hypothesis (1988), there is an inverse relationship between birth size and long-term disease risk. It implies that the *in utero* exposure to famine of G2 may have a long-term impact on their future outcomes. There are several possible explanations for intergenerational effects (the impact of early life famine exposure of G2 on the outcomes of G3). First, fetal programming is durable through several generations. This is the effect we are trying to identify. However, it is also possible that genetic attributes of weight outcomes may manifest themselves similarly in mother and offspring. Or adverse environmental conditions may persist across generations. Or finally, postnatal investment may affect the mother's health, and therefore the offspring's health. Because the famine survivors (G2) are the healthy and strong ones, this will lead to a positive selection effect that will bias our estimates downwards.

This paper focuses on the second question: the intergenerational effects of early life famine exposure. We compare the weight and height outcomes of the children (G3) of famine survivors with children whose parents were not exposed to famine during their *in utero* period. The results for the long-term health effect of famine will be reported in the discussion section.

3.4.1 Baseline Model

In the baseline model, the following equation is used to estimate the impact of mothers' *in utero* environment on their children's outcomes:

$$Outcome_{ipt} = \beta_0 + \beta_1 \times Fa\min e_i + \beta_2 \times f(age_{ipt}) + \beta_3 Parents\_Education_i + \beta_4 \times Parents\_Income_i + \beta_4 \times Rural_i + \tau_t + \eta_p + \varepsilon_{ipt} \quad (12)$$

In this model, *i* indexes individuals, *p* indexes provinces, and *t* indexes waves. We control for age, age square, their residence (rural or urban), and their background information such as their parents' education and income levels. Province fixed effects (η_P) and wave fixed effects (τ_t) are included. We also add other control variables, such as their nutrition intakes and ownership of color TV (as a measure of nutrition expenditure), to check whether the difference in weight status can be explained by caloric intake and expenditure. Finally, ε_{ipt} represents individual idiosyncratic errors.

Our main outcomes are weight and height outcomes. Because the weight and height of children in different ages cannot be compared directly, we transform children's BMI, weight, and height to z-score. The transformation of child anthropometric data to z-scores is based on WHO 2007 standard, which is more suitable for Asian children.

The variable of interest is *Famine*, which equals 1 if their mothers were born during the famine, and equals 0 otherwise. The parameter β_1 captures the effects of being the children of the famine survivors. For example, if the dependent variable is BMI-for-age, a negative sign of β_1 indicates the BMI-for-age is lower if their mothers were exposed to famine during their *in utero* period. Because the errors within each province may be correlated, all regressions are clustered by provinces.

3.4.2 Difference-in Difference (DD)

In order to find a truly unexposed control group, we use observations from health and nutrition survey data in Taiwan. Because Taiwan was not affected by the famine from 1959 to 1961, under this specification, we use people born in Taiwan as the control group and use people

born in mainland as the treatment group. To address the potential bias due to the mortality, we control for yearly Excess Death Rate (EDR) in difference provinces, and assume the EDR in Taiwan is 0, because Taiwan was not affected by the great famine.

$$Outcome_{ipt} = \beta_0 + \beta_1(\text{Famine})_i \times Mainland_p + \beta_2(\text{Famine})_i + \beta_3 Mainland_p + \beta_4 f(age_{ipt}) + \beta_5 Parents\_Education_i + \beta_6 EDR_{pt} + \tau_t + \eta_p + \varepsilon_{ipt} \quad (13)$$

In this model, i indexes individuals, p indexes provinces, and t indexes waves. Mainland is a dummy variable which equals 1 for people born in Mainland, and equals 0 for people born in Taiwan. The coefficient of interest is β_1, which captures the DD effect.

3.5 Empirical Results

3.5.1 Baseline Results

The impact of mothers' famine exposure on G3's outcomes is shown in Table 3-1. Regressions are based on Equation 12. Considering the potential gender difference, we run the regressions for boys and girls separately. The results for girls and boys are presented in Panels I and II, respectively. The outcome variables include weight-for-age, BMI-for-age, and height-for-age. By comparing the impact of mothers' *in utero* famine exposure on girls and boys, we find that the effects on boys' weight and height outcomes are not significant at all. On the other hand, the weight-for-age and BMI-for-age of girls whose mothers were exposed to famine during the *in utero* period are significantly smaller. The estimate of -0.38 in Column 2 of Panel I indicates that the BMI-for-age of girls are 0.38 lower on average, and it is mainly due to lower weight (0.30 lower in weight-for-age). As a result, our study will only focus on the weight and height outcomes of daughters of famine survivors.

3.5.2 Caloric Intake and Caloric Expenditure

BMIs are highly related to caloric balance. Thus, we further control for the caloric intake

and expenditure, to check whether the effects of mothers' *in utero* famine exposure on their daughters' weight and height outcomes remain significant. The caloric intake information is based on the three consecutive 24-hour recalls. During the survey, the interviewers ask individuals about the ingredient names and amounts for each meal. Based on the information, they calculate the total calorie intake, fat consumption, carbohydrates consumption, and protein consumption. Table 3-2 shows the results when controlling for caloric intake, as well as the consumption of carbohydrate, fat, and protein. The results are very robust. And we find no evidence about the impact of caloric intake on girls' weight and height outcomes.

Because there is little variation in the physical activity participation among girls, we use the ownership of color TV to estimate their physical activity levels (assume that Color TV significantly increases girls' sedentary activities and decrease their physical activities). The results are shown in Table 3-3: the ownership of color TV is associated with increases in the weight-for-age and height-for-age, but the results about the effects of famine exposure remain robust after controlling for color TV ownership.

3.5.3 Parents' SES

We already showed that the impact of unfavorable *in utero* events can pass to their children. Then an important question will be whether higher SES of parents can help eliminate the negative effect on their children? To answer this question, we interact the parents' SES with the *Famine* dummy. The "high SES" is defined as: (1) finished upper middle school (2) income is higher than the average. The effects of mothers' and fathers' SES are evaluated separately, which leads to four regressions. The outcome variable is children's BMI-for-age.

If higher SES can dampen the negative impact, the coefficient of "High SES × Famine" should not be significant, while the coefficient of "Low SES × Famine" should remain

significant. In Table 3-4, Columns 2, 3, and 4 meet such conditions, which implies that it is possible to modify the intergenerational transmission of programming effect when 1) father has at least upper middle school education, or 2) either father or mother has income higher than the average. On the other hand, mothers' education level cannot buffer the negative impact of the bad *in utero* environment on their offspring (Column 1).

The comparison between girls from Mainland and Taiwan are shown in Table 3-5. We can find that both the height-for-age and weight-for-age of girls from Taiwan are larger than that of girls from mainland; the educational attainments of Taiwan parents are better than mainland parents, especially for mothers. The results for DD model are shown in Table 3-6. Consistent with the conclusion from our baseline model (Panel I of Table 3-1), girls have lower weight-for-age and BMI-for-age if their mothers were exposed to famine during the *in utero* period, but the results become less significant. In this specification, we also observe that sons of famine survivors tend to have a lower BMI-for-age.

3.6 Discussion

The results for G2 based on the basic setup are shown in Table 3-7 and Table 3-8. In different columns, we show results based on different sets of control variables. There are three EDR values for each province: 1) *in utero,* 2) in the first year of birth, and 3) in the second year of birth.

The results for fathers are shown in Table 3-7: both *in utero* and early childhood famine exposure will make fathers heavier and shorter in their adulthood, which leads to a larger BMI. Controlling for education will not affect the results. However, the impact on height becomes insignificant after controlling for the EDR. The results for mothers are shown in Table 3-8: mothers who were born during or right before the famine tend to be shorter and have larger BMIs

in their adulthood. The results are robust when we further control for their education levels and the EDR.

The results for G2 using the DD specification are shown in Table 3-9 and Table 3-10. In general, the results are consistent with the baseline model results, expect for the impact of early famine exposure on mothers' future weight: the DD model implies that mothers will become heavier if they were exposed to famine during their early life.

3.7 Conclusion

Our results indicate that *in utero* famine exposure has significant effects on future weight and height outcomes, and the effects can pass on to their offspring. However, the effects of famine exposure on G2 and G3 are different: parents (G2) tend to have a larger BMI in their adulthood due to their *in utero* famine exposure, but the girls whose mothers were born during the famine tend to have a smaller BMI-for-age. These results are robust under various specifications. Moreover, higher income and education levels for parents can help to buffer the bad impacts of their unfavorable early life environments on their daughters' weight outcomes.

3.8 Reference

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Table 3-1 The Impact of Mothers' Famine Exposure

VARIABLES	Weight-for-age	BMI-for-age	Height-for-age
Panel I: Girls			
Mothers Born During the Famine	-0.3015***	-0.3780**	-0.0307
	[0.079]	[0.143]	[0.180]
Observations	312	312	312
Panel II: Boys			
Mothers Born During the Famine	0.1195	-0.1067	0.3104
	[0.153]	[0.153]	[0.182]
Observations	374	374	374

Standard errors in brackets
*** p<0.01, ** p<0.05, * p<0.1

Table 3-2 The Impact of Mothers' Famine Exposure Controlling for Caloric Intakes

VARIABLES	Weight-for-age	BMI-for-age	Height-for-age
Mothers Born During the Famine	-0.3022***	-0.3720**	-0.0364
	[0.082]	[0.134]	[0.182]
Calorie	-0.0230	-0.0320	0.0028
	[0.016]	[0.020]	[0.010]
Carbohydrate	0.0915	0.1294	-0.0132
	[0.063]	[0.079]	[0.041]
Fat	0.2083	0.2885	-0.0241
	[0.140]	[0.176]	[0.093]
Protein	0.0953	0.1259	-0.0043
	[0.064]	[0.079]	[0.040]
Observations	312	312	312

Standard errors in brackets

*** p<0.01, ** p<0.05, * p<0.1

Table 3-3 The Impact of Mothers' Famine Exposure Controlling for Caloric Expenditure

VARIABLES	Weight-for-age	BMI-for-age	Height-for-age
Mothers Born During the Famine	-0.3119***	-0.3609**	-0.0894
	[0.087]	[0.147]	[0.165]
Color TV	0.3463**	0.0850	0.3710**
	[0.115]	[0.111]	[0.138]
Observations	312	312	312

Standard errors in brackets
*** p<0.01, ** p<0.05, * p<0.1

Table 3-4 The Impact of Mothers' Famine Exposure Considering Parents' SES

VARIABLES	Mother High Education	Father High Education	Mother High Income	Father High Income
High SES ×	-0.8195***	-0.1282	-0.1354	-0.2630
Mothers Born During the Famine	[0.242]	[0.755]	[0.178]	[0.161]
Low SES ×	-0.2770***	-0.3242***	-0.3846***	-0.3289**
Mothers Born During the Famine	[0.072]	[0.083]	[0.104]	[0.104]
Observations	312	312	312	312

Standard errors in brackets

*** p<0.01, ** p<0.05, * p<0.1

Table 3-5 Comparison between Mainland and Taiwan Samples

Variable	Taiwan	Mainland
Age	9.79	11.56
BMI-for-age	-0.11	-0.52
Height-for-age	0.23	-1.02
Weight-for-age	0.05	-0.97
Mother No Education	0.05	0.24
Father No Education	0.06	0.07
Observations	304	312

Table 3-6 The Impact of Mothers' Famine Exposure (DD Results)

	Weight-for-age	BMI-for-age	Height-for-age
Panel I: Girls			
Mothers Born During the Famine	-0.1410***	-0.1584***	0.0071
	[0.018]	[0.013]	[0.019]
Mainland	-1.5469**	-0.6533	-1.3726***
	[0.583]	[0.604]	[0.397]
Mothers Born During the Famine * Mainland	-0.1680*	-0.2211*	-0.0730
	[0.087]	[0.119]	[0.184]
Observations	618	618	618
Panel II: Boys			
Mothers Born During the Famine	0.1341***	0.1458***	0.1160***
	[0.013]	[0.012]	[0.022]
Mainland	-1.2303**	-0.3551	-1.2930*
	[0.469]	[0.487]	[0.667]
Mothers Born During the Famine * Mainland	-0.0866	-0.3038*	0.1291
	[0.135]	[0.152]	[0.172]
Observations	716	716	716

Standard errors in brackets

*** p<0.01, ** p<0.05, * p<0.1

Table 3-7 Weight and Height Outcomes for G2 Fathers

Panel I: BMI				
Father born during the famine * Mainland	1.3380***	1.3198***	0.9754**	1.1028**
(born from 1959-1961)	[0.175]	[0.178]	[0.351]	[0.372]
Father born before the famine * Mainland	1.1403***	1.1568***	1.1008***	1.1475***
(born from 1954-1958)	[0.210]	[0.177]	[0.161]	[0.179]
EDR in utero	-	-	0.0242	0.0221
	-	-	[0.024]	[0.026]
EDR in first year of birth	-	-	0.0311	0.0301
	-	-	[0.031]	[0.030]
EDR in second year of birth	-	-	-0.0155	-0.0158
	-	-	[0.018]	[0.018]
Panel II: Weight				
Father born during the famine * Mainland	2.1895**	2.3965**	2.7337	2.8732*
(born from 1959-1961)	[0.779]	[0.857]	[1.493]	[1.544]
Father born before the famine * Mainland	2.1374**	2.9114***	2.9487***	3.0931***
(born from 1954-1958)	[0.847]	[0.685]	[0.777]	[0.727]
EDR in utero	-	-	0.0244	0.0194
	-	-	[0.100]	[0.113]
EDR in first year of birth	-	-	0.0662	0.0667
	-	-	[0.090]	[0.089]
EDR in second year of birth	-	-	-0.1696	-0.1625
	-	-	[0.107]	[0.098]
Panel III: Height				
Father born during the famine * Mainland	-2.1551**	-1.7129*	-0.0452	-0.3106
(born from 1959-1961)	[0.701]	[0.840]	[1.168]	[1.174]
Father born before the famine * Mainland	-1.5165*	-0.4261	-0.2000	-0.1464
(born from 1954-1958)	[0.739]	[0.636]	[0.674]	[0.585]
EDR in utero	-	-	-0.0404	-0.0380
	-	-	[0.079]	[0.090]
EDR in first year of birth	-	-	-0.0267	-0.0231
	-	-	[0.046]	[0.048]
EDR in second year of birth	-	-	-0.1769	-0.1652
	-	-	[0.113]	[0.107]
Father's Education	N	Y	N	Y

The omitted group is Father born after the famine (from 1962-1966).
All regressions include year of birth fixed effects, survey wave fixed effect, and province fixed effects.
The sample size is 1,453.

Table 3-8 Weight and Height Outcomes for G2 Mothers

Panel I: BMI			
Mother born during the famine * Mainland	0.2000	-0.2883	-0.1640
(born from 1959-1961)	[0.274]	[0.453]	[0.448]
Mother born before the famine * Mainland	0.6497***	0.3533**	0.5460***
(born from 1954-1958)	[0.184]	[0.140]	[0.151]
EDR in utero	-	-0.0383*	-0.0379**
	-	[0.017]	[0.016]
EDR in first year of birth	-	0.0463	0.0524
	-	[0.039]	[0.038]
EDR in second year of birth	-	0.0240	0.0243
	-	[0.028]	[0.028]
Panel II: Weight			
Mother born during the famine * Mainland	-1.3016*	-2.0848	-1.9817
(born from 1959-1961)	[0.673]	[1.268]	[1.178]
Mother born before the famine * Mainland	0.4478	-0.1160	0.3163
(born from 1954-1958)	[0.584]	[0.404]	[0.454]
`EDR in utero	-	-0.0560	-0.0487
	-	[0.066]	[0.064]
EDR in first year of birth	-	0.1061	0.1144
	-	[0.091]	[0.094]
EDR in second year of birth	-	-0.0080	0.0095
	-	[0.058]	[0.057]
Panel III: Height			
Mother born during the famine * Mainland	-2.5639***	-2.0168***	-2.2980***
(born from 1959-1961)	[0.388]	[0.541]	[0.542]
Mother born before the famine * Mainland	-1.5204***	-1.2853**	-1.3528**
(born from 1954-1958)	[0.434]	[0.474]	[0.491]
EDR in utero	-	0.0531	0.0615
	-	[0.056]	[0.051]
EDR in first year of birth	-	-0.0060	-0.0145
	-	[0.048]	[0.048]
EDR in second year of birth	-	-0.0937*	-0.0710
	-	[0.048]	[0.044]
Mother's Education	N	N	Y

The omitted group is Mother born after the famine (from 1962-1966).
All regressions include year of birth fixed effects, survey wave fixed effect, and province fixed effects.
The sample size is 1,949.

Table 3-9 Weight and Hight Outcomes for G2 Fathers (DD Results)

	BMI			Weight			Height		
Father born during the famine X Mainland	1.3380***	0.9754**	1.1028**	2.1895**	2.7337	2.8732*	-2.1551**	-0.0452	-0.3106
(born from 1959-1961)	[0.175]	[0.351]	[0.372]	[0.779]	[1.493]	[1.544]	[0.701]	[1.168]	[1.174]
Father born before the famine X Mainland	1.1403***	1.1008***	1.1475***	2.1374**	2.9487***	3.0931***	-1.5165*	-0.2000	-0.1464
(born from 1954-1958)	[0.210]	[0.161]	[0.179]	[0.847]	[0.777]	[0.727]	[0.739]	[0.674]	[0.585]
EDR in utero	-	0.0242	0.0221	-	0.0244	0.0194	-	-0.0404	-0.0380
	-	[0.024]	[0.026]	-	[0.100]	[0.113]	-	[0.079]	[0.090]
EDR in first year of birth	-	0.0311	0.0301	-	0.0662	0.0667	-	-0.0267	-0.0231
	-	[0.031]	[0.030]	-	[0.090]	[0.089]	-	[0.046]	[0.048]
EDR in second year of birth	-	-0.0155	-0.0158	-	-0.1696	-0.1625	-	-0.1769	-0.1652
	-	[0.018]	[0.018]	-	[0.107]	[0.098]	-	[0.113]	[0.107]
Father's Education	N	N	Y	N	N	Y	N	N	Y

Table 3-10 Weight and Height Outcomes for G2 Mothers (DD Results)

	BMI			Weight			Height		
Father born during the famine									
X Mainland	1.3380***	0.9754**	1.1028**	2.1895**	2.7337	2.8732*	-2.1551**	-0.0452	-0.3106
(born from 1959-1961)	[0.175]	[0.351]	[0.372]	[0.779]	[1.493]	[1.544]	[0.701]	[1.168]	[1.174]
Father born before the famine									
X Mainland	1.1403***	1.1008***	1.1475***	2.1374**	2.9487***	3.0931***	-1.5165*	-0.2000	-0.1464
(born from 1954-1958)	[0.210]	[0.161]	[0.179]	[0.847]	[0.777]	[0.727]	[0.739]	[0.674]	[0.585]
EDR in utero	-	0.0242	0.0221	-	0.0244	0.0194	-	-0.0404	-0.0380
	-	[0.024]	[0.026]	-	[0.100]	[0.113]	-	[0.079]	[0.090]
EDR in first year of birth	-	0.0311	0.0301	-	0.0662	0.0667	-	-0.0267	-0.0231
	-	[0.031]	[0.030]	-	[0.090]	[0.089]	-	[0.046]	[0.048]
EDR in second year of birth	-	-0.0155	-0.0158	-	-0.1696	-0.1625	-	-0.1769	-0.1652
	-	[0.018]	[0.018]	-	[0.107]	[0.098]	-	[0.113]	[0.107]
Father's Education	N	N	Y	N	N	Y	N	N	Y

Table 3-A1: Provincial Crude and Excess Death Rates in China, 1954-66 (Unit: 0.1%)

Province	1954	1955	1956	1957	1958	1959	1960	1961	1962	1963	1964	1965	1966	1954-58 Average
	Death rates													
Guangxi	15.20	14.60	12.50	12.40	11.70	17.50	29.50	19.50	10.30	10.10	10.60	9.00	7.50	13.28
Guizhou	12.20	16.20	13.00	12.40	15.30	20.30	52.30	23.30	11.60	17.20	20.70	15.20	13.50	13.82
Henan	13.30	11.80	14.00	11.80	12.70	14.10	39.60	10.20	8.00	9.40	10.60	8.50	8.20	12.72
Hubei	15.90	11.60	10.80	9.60	9.60	14.50	21.20	9.10	8.80	9.80	10.90	10.00	9.70	11.50
Hunan	17.50	16.40	11.50	10.40	11.70	13.00	29.40	17.50	10.20	10.30	12.90	11.20	10.20	13.50
Jiangsu	12.20	11.80	13.00	10.30	9.40	14.60	18.40	13.40	10.40	9.00	10.10	9.50	8.10	11.34
Liaoning	8.60	9.40	6.60	9.40	8.80	11.80	11.50	17.50	8.50	7.90	9.30	7.10	6.20	8.56
Shandong	11.70	13.70	12.10	12.10	12.80	18.20	23.60	18.40	12.40	11.80	12.00	10.20	9.90	12.48
Nation	12.80	12.00	10.80	10.70	12.00	15.10	23.20	13.10	9.50	9.80	11.80	9.40	8.80	11.66
	Excess death rates (Death rates minus 1954-58 average)													
Guangxi	1.92	1.32	-0.78	-0.88	-1.58	4.22	16.22	6.22	-2.98	-3.18	-2.68	-4.28	-5.78	
Guizhou	-1.62	2.38	-0.82	-1.42	1.48	6.48	38.48	9.48	-2.22	3.38	6.88	1.38	-0.32	
Henan	0.58	-0.92	1.28	-0.92	-0.02	1.38	26.88	-2.52	-4.72	-3.32	-2.12	-4.22	-4.52	
Hubei	4.40	0.10	-0.70	-1.90	-1.90	3.00	9.70	-2.40	-2.70	-1.70	-0.60	-1.50	-1.80	
Hunan	4.00	2.90	-2.00	-3.10	-1.80	-0.50	15.90	4.00	-3.30	-3.20	-0.60	-2.30	-3.30	
Jiangsu	0.86	0.46	1.66	-1.04	-1.94	3.26	7.06	2.06	-0.94	-2.34	-1.24	-1.84	-3.24	
Liaoning	0.04	0.84	-1.96	0.84	0.24	3.24	2.94	8.94	-0.06	-0.66	0.74	-1.46	-2.36	
Shandong	-0.78	1.22	-0.38	-0.38	0.32	5.72	11.12	5.92	-0.08	-0.68	-0.48	-2.28	-2.58	
Nation	1.14	0.34	-0.86	-0.96	0.34	3.44	11.54	1.44	-2.16	-1.86	0.14	-2.26	-2.86	

Source: Shi (2007, Table 1) and Funge and Ha (2010)'s calculation
Note: The shaded columns denote famine years.

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Research Papers

1. Cheng Chen, Shin-Yi Chou, Hsienming Lien, and Jin-Tan Liu, "The long-term health effects of early life malaria exposure: Evidence from the malaria eradication program in Taiwan," job market paper.
2. Cheng Chen, Shin-Yi Chou, and Robert J. Thornton, "The effect of household technology on obesity and weight gain among Chinese female adults: Evidence from China's home appliances going to the countryside policy," revise and resubmit, Journal of Human Capital.
3. Cheng Chen, Shin-Yi Chou, and Lea Gimenez Duarte, "The inter-generational effects of the 1959–1961 Great Chinese Famine on children's anthropometric outcomes."

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- Eastern Economic Association Annual Meeting, New York, NY, May 2013.

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"The inter-generational effects of the 1959–1961 Great Chinese Famine on children's anthropometric outcomes"

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