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Long-term Health Effects of Malaria Exposure around Birth: Evidence from Colonial Taiwan

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> Living things are "plastic" in their early lives: their growth and development are molded by the environment. (David J. P. Barker 2000)

The Barker hypothesis postulates that health shocks before birth and during infancy could permanently change one's physiology and metabolism and further shape her ability and health trajectory in adult life (Barker 1992, 1993, 1994, 1995, 2000).[1] Empirically, epidemiologists have found that, for example, cardiovascular and lung diseases in adulthood are associated with intrauterine health conditions (Rasmussen 2001). Yet, to go beyond correlational studies, one needs either exogenous health shocks or other effective ways to address potential confounders (Almond and Currie 2011). A distinguishing feature of this article is to exploit exogenous variation in malaria deaths *around* birth—the birth year and the year before birth—resulting from an eradication campaign in colonial Taiwan in the early twentieth century.

Malaria is an infectious disease caused by parasites called *Plasmodium* and mainly transmitted by female *Anopheles* mosquitoes.[2] It also can be vertically transmitted from an infected mother to her fetus (Manendez and Mayor 2007;

[1] The Barker hypothesis is also known as the Fetal Origins hypothesis. However, it is noteworthy that the recent development in this line of research concerns both the intrauterine period and early childhood after birth. See http://www.thebarkertheory.org/index.php.

[2] There are four strains of *Plasmodium* that are known to infect humans: *P. falciparum, P. vivax, P. ovale*, and *P. malariae.* Further information is available at http://who.int/topics/malaria/en.

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Poespoprodjo et al. 2010). Pregnant women and infants typically have low immunity and are thus particularly vulnerable to malaria infection. Once transmitted into the human body, the parasites would destroy red blood cells, leading to disruption of the supply of oxygen and nutrients to organs and tissues. Since most human organs are being developed around birth, contracting malaria in this critical period is likely to have a long-term negative effect on health. In this article, we aim to estimate the causal effects of malaria exposure around birth on the health of elderly who were born during the Japanese colonial period in Taiwan.

Although we consider the malaria eradication campaign as a natural experiment, we are still challenged by potential measurement errors and omitted variables that are correlated with both malaria deaths around birth and later outcomes. We will describe these concerns later. To mitigate potential bias caused by these concerns, we employ climatic factors such as rainfall, rainy days, and relative humidity to instrument for malaria deaths. These factors are important in determining the breeding and survival of *Anopheles* mosquitoes but are presumably uncorrelated with the potential measurement errors and other omitted variables in our study.

One problem that we are not able to deal with is a mortality selection issue. The data that we use only consist of the colonial cohorts who survived about half a century after the colonial period ended. If the abler people among them were more likely to survive malaria and live long enough to be included in our data, we are likely to underestimate the malaria effects. Accordingly, we consider our estimates to be lower bound estimates of the true malaria effects.

We show that a higher malaria exposure risk around birth leads to worse health outcomes at old age. Specifically, elderly people who were born into a high malaria risk environment are more likely to have high blood pressure, heart disease, and worse cognitive functions. They are also more likely to self-report poor health.

Our findings add to the growing literature in economics that treats the relationship between early-life health shocks and adult outcomes (Almond and Mazumder 2005; Almond 2006; Chen and Zhou 2007; Case and Paxson 2009, 2010; Meng and Qian 2009; Almond et al. 2010; Barreca 2010; Bleakley 2010; Cutler et al. 2010; Lucas 2010; Kim et al. 2014). Yet, to the best of our knowledge, our article is the first to document the malaria effects on health outcomes in old age.

The rest of this article is organized as follows. In the next section, we delineate the malaria eradication campaign in colonial Taiwan. Section II describes the data. In Section III, we present our empirical model and identification

strategy, while Section IV reports the estimation results. We conclude in Section V.

I. Malaria Eradication in Colonial Taiwan

In the early stage of the Japanese ruling period (1895–1945), malaria was not only rampant but also deadly in Taiwan.[3] Between 1906 and 1909, for example, more than 10,000 residents died of malaria each year, while the population was only about 3 million (Centers for Disease Control 1991). To fight against malaria, the Japanese colonial government initiated an island-wide eradication campaign in 1911, which lasted until the eruption of World War II (Ting 2008; Ku 2009; Liu 2009).

An island-wide monitoring system was first established. All malaria cases had to be reported to local physicians by the police and the local self-policing system. So-called antimalaria districts were gradually set up throughout the whole island.[4] Residents within the districts were required to provide blood smears to test for malaria, and, if found to carry malaria parasites, they were forced to take quinine for 18 consecutive days under the supervision of a policeman. In 1919, the campaign expanded to areas beyond the districts and emphasized more on vector-control measures such as cleaning up the environment, while blood tests and quinine prophylaxis continued.

At the beginning, malaria distribution varied considerably from the north to the south. Figure 1 portrays the five regions in colonial Taiwan in 1920, with darker areas characterized by higher malaria death rates.[5] As illustrated, malaria was generally more serious in the south than in the north. The trends of malaria death rates in figure 2 clearly exhibit the effect of the eradication campaign. After the eradication campaign was initiated in the early 1910s, the malaria death rate dropped dramatically from four deaths per 1,000 people in the early 1910s to less than one in the late 1920s, remaining under control

[3] It is worth noting that malaria was not the only infectious disease in the colonial period. Others include plague, dysentery, and cholera. However, malaria was the most prevalent and the deadliest. For example, in 1902, malaria caused 13,444 deaths, while plague caused 1,853, dysentery 754, and cholera 613 (*Yearly Statistics Book of the Office of the Governor-General in Taiwan*, 1903, available at http://tcsd.lib.ntu.edu.tw). Besides, plague had been under control by 1910 and was eradicated in 1917, while dysentery and cholera never became endemic island-wide (Tetsuzou 2007). Later, we provide additional evidence to show that other diseases during the colonial period are unlikely to bias our estimates.

[4] The number of antimalaria districts increased from 12 in 1911 to 185 in 1942 (Liu 2009).

[5] The size of each region is roughly equivalent to 2–3 counties today. Nankao in the south and Huadong in the east each actually consisted of two prefectures—the administrative division in the colonial period—in 1920. Between 1901 and 1941, there were two changes in administrative divisions, one in 1909 and the other in 1920. The changes make the prefecture-level data not comparable for these prefectures. Hence, we merge these prefectures together to make historical data comparable across years.

Figure 1. Malaria severity in 1920. Darker areas are characterized with higher malaria mortality

through the 1930s.[6] The Nankao region in the south experienced the largest drop, and the north-south disparity disappeared in the 1930s.

II. Data

To implement our empirical study, we employ both regional- and individual-level data to match individual health outcomes at old age to the malaria severity around birth in his or her birth region.

[6] It is worth noting that malaria was not completely eradicated by the end of the colonial period (Ku 2009). The eruption of World War II curtailed the eradication campaign and malaria burst out again. The Chinese Nationalist government took over Taiwan after the war and initiated another eradication campaign in the 1950s with the help of dichloro-diphenyl-trichloro-ethane (DDT), which was not available in the colonial period. In 1965, the World Health Organization (WHO) finally declared Taiwan as malaria-free.

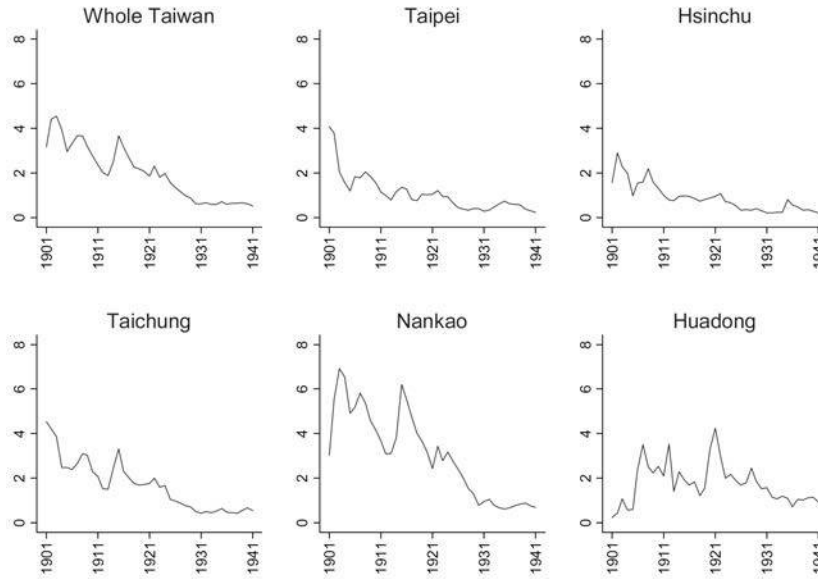

Figure 2. Trend of malaria death rate by region. Malaria death rate is defined as malaria deaths per 1,000 people. Source: malaria death rates are authors' own calculation using statistics from various *Yearly Statistics Books of the Office of the Governor-General in Taiwan* published by the colonial government.

Historical Regional-Level Data

We collect historical data on malaria deaths, population, climate, education, and economic variables from various *Yearly Statistics Books of the Office of the Governor-General in Taiwan* published by the colonial government.[7] Because malaria death information is only available for the period 1901–41, we restrict our study period accordingly. In addition, malaria death information is annual. We define the key malaria variable, MALARIA, as the average malaria deaths per 1,000 people in the year of birth and the year before birth in the region of birth. Since human pregnancy lasts approximately 40 weeks, our definition of MALARIA *around birth* covers the entire intrauterine period and a postnatal period, the length of which depends on one's birth date in a year.[8]

For climatic factors, we collect data on historical annual rainfall, annual rainy days, and relative humidity. Similar to the way we construct MALARIA, we use the average of climatic measure in the year of birth and the year before birth as instrumental variables. Unfortunately, climate data for the Hsinchu region was

[7] An online archive of the *Yearly Statistics Books of the Office of the Governor-General in Taiwan* is available at http://tcsd.lib.ntu.edu.tw.

[8] Due to lack of monthly data on births and malaria deaths, we are not able to separate the intrauterine and the postnatal period.

not available until the late 1930s. Therefore, we have to exclude this region in our IV estimation. For more details about historical data, see the appendix.

Individual-Level Data

The individual-level data come from a longitudinal survey: Survey of Health and Living Status of the Middle-Aged and the Elderly in Taiwan. This survey was initiated in 1989 using a representative sample of the elderly age 60 and older. In 1996, it drew another sample representing the middle-aged population between 55 and 66. We combine these two samples and discard observations on individuals who were not born in Taiwan to form our main sample. Our final sample consists of 3,577 elderly people who were born between 1901 and 1941. In addition to basic demographic and socioeconomic variables, this survey of the elderly provides us information on birth year and place, as well as health outcomes such as high blood pressure, heart disease, cognitive ability measures, and self-reported general health. Unfortunately, we only have information on exact birthplace for about two-thirds of our sample. For the remaining one-third, we use their current residence as proxy for their birthplace.

Sample Characteristics

Table 1 summarizes the sample characteristics for the whole sample and the regional subsamples. All outcomes are evaluated in 1996. The average age is

TABLE 1
SAMPLE CHARACTERISTICS BY REGION

	All Regions (1)	Taipei (2)	Hsinchu (3)	Taichung (4)	Nankao (5)	Huadong (6)
Age	67.96	66.95	68.75	68.30	67.92	66.31
Female (%)	51.97	54.29	50.16	52.26	51.09	58.24
Minnan people (%)	77.89	94.49	32.90	89.55	85.61	37.36
Father's years of schooling	1.24	1.50	1.21	1.27	1.15	.74
Father was farmer (%)	63.27	47.33	71.99	67.03	63.60	69.23
Observations	3,577	617	614	928	1327	91
Outcome variables:						
Cardiovascular conditions:						
High blood pressure (%)	28.29	28.04	30.46	26.29	29.09	24.18
Heart disease (%)	17.11	17.50	16.78	18.53	15.90	19.78
Self-reported poor health (%)	29.27	23.82	28.99	28.99	31.27	41.76
Observations	3,577	617	614	928	1,327	91
Cognitive ability measures:						
Immediate recall	3.90	4.33	3.82	3.81	3.81	3.55
Backward counting (%)	19.00	27.37	13.08	19.68	18.17	10.00
Observations	3,294	548	581	869	1,216	80

Note. All characteristics are evaluated in 1996; self-reported poor health is constructed from a 5-point scale, i.e., very good, good, fair, poor, and very poor; we merge poor and very poor together; immediate recall is a sum of correctly recalled objects out of a total of 10 objects; backward counting is whether one can backwardly count five single-digit numbers.

about 68. About 52% of the elderly are female, and 78% of them belong to the largest ethnic group, the Minnan people. The majority of their fathers were farmers who had only slightly more than 1 year of schooling. In 1996, 28% of them reported high blood pressure, 17% of them heart disease, and 29% said they have poor or very poor health on a 5-point scale. The cognitive ability measures show that on average they can recall about 4 out of 10 objects that are just read to them, and less than 20% of them can correctly count a sequence of five single-digit numbers backward. Across regions, elderly people in Taipei generally seem to have better health outcomes than their counterparts in other regions.

III. Empirical Model and Identification Strategy

Our benchmark model is

$$y_{ipt} = \beta Exposure_{pt} + \alpha_p + \gamma_t + \theta X_{ipt} + \varepsilon_{ipt}, \quad (1)$$

where y_{ipt} denotes a health outcome at old age for individual i born in region p in year t; *Exposure* is regional malaria exposure risk around birth; α is a region fixed effect; γ is a cohort fixed effect; X is a vector of controls, including sex, ethnicity, father's education, father's major occupation, and regional economic and education resource indicators that are time-varying;[9] and ε is an error term that may be correlated across individuals within the same region.

The key parameter β measures the average change in individual outcomes at old age as a response to a one-unit increase in local malaria exposure risk in the year of birth. We acknowledge that it is the malaria infection per se that can affect subsequent outcomes. However, we assume that there exists a stable stochastic relationship between exposure risk and infection.

Our major challenge is to infer the exposure risk from observed malaria death rates, which are the best information available to us.[10] To make our empirical model operational, we assume that malaria death rates are proportional to the exposure risk. Therefore, we can use malaria death rates at the regional level as a proxy for the malaria exposure risk. Formally, we assume that the stochastic relationship between malaria death rates and the malaria exposure risk with a potential measurement error is as follows.

[9] Taiwan was an agricultural economy in the colonial period. To measure regional economy, we use the output of rice and sugar cane, which were the two most important agricultural products then. Moreover, we include the number of primary schools in the region to control for local educational resources.

[10] One might model the true risk as a function of many factors such as nearby malaria hosts, mosquitoes, parasites, medical facilities, government intervention, mother's immunity, and other environmental and individual characteristics. Many of these factors are not observed, and the exact modeling of their interaction is unknown.

$$Malaria_{pt} = \delta Exposure_{pt} - \eta_{pt}, \tag{2}$$

where *Malaria* is the malaria death rate, δ is a proportionality coefficient, η is an unobserved measurement error, and other notations are the same as in equation (1). Following the classical errors-in-variables assumption, we assume *Exposure* and η are uncorrelated. Plug equation (2) into (1) and, after some arrangements, we get an operational model:

$$y_{ipt} = \lambda Malaria_{pt} + \alpha_p + \gamma_t + \theta X_{ipt} + \lambda\eta_{pt} + \varepsilon_{ipt}, \tag{3}$$

where

$$\lambda = \frac{\beta}{\delta}.$$

The parameter λ now measures the average change in an individual outcome at old age as a response to a one-unit increase in local malaria death rate in the year of birth. However, under the classical errors-in-variables assumption, *Malaria* and η must be correlated, leading to an attenuated estimate of λ. We discuss possible sources of measurement errors in more details later.

In the following regressions, we use MALARIA, which is previously defined as the average malaria deaths per 1,000 people in the year of birth and the year before birth, in place of *Malaria*.

For statistical inference, we use robust standard errors clustered at regions. However, the usual Huber-White cluster robust standard errors require the number of clusters to be large, while we have only five clusters. To address this concern, we additionally calculate the wild-bootstrap cluster robust standard errors, which can reduce overrejection in the case of few clusters, based on the method proposed by Cameron et al. (2008).

Measurement Errors

In addition to simple observation errors, measurement errors in equation (3) could arise for two reasons. First, malaria death rates may deviate away from proportionality to exposure risk due to unobserved factors. For example, three parasite strains were found among malaria patients in colonial Taiwan: *P. falciparum, P. vivax*, and *P. malariae* (Sawa 1931; Morishita 1976). Of these, only *P. falciparum* would typically cause mortality, but all three strains could cause morbidity. The parasites' composition could vary geographically and over time. Unfortunately, information on geographic distribution of the parasites does not exist. Second, measurement errors could arise from misidentification of birthplace for a subset of our sample, as indicated in the data section.

Omitted Confounders

There also exist some unobserved family characteristics that may lead to a biased estimate of λ. For example, it is possible that a better-off family has more means to protect their children by moving to a low-malaria region and also invests more in their human and health capital. Parents' education may play a similar role. In our empirical model, we control for father's education and occupation, but we do not observe family wealth or income.

Instrumental Variables

To mitigate potential biases caused by the concerns listed above, we employ climatic factors such as rainfall, rainy days, and relative humidity to instrument for malaria death rates. These climatic factors are important in determining the breeding and survival of Anopheles mosquitoes, although their interactions are quite complicated. For example, *Anopheles* mosquitoes breed on the surface of a body of water, but too much rainfall could flush away their larvae. The survival and activity are related with humidity in a nonlinear fashion. Some other researchers argue that the number of rainy days is more important than the amount of rainfall in determining the wetness in environment (Pampana 1969).[11]

Our purpose is not to model the true nonlinear interactions between malaria death rate and the climatic factors. Instead, we just need a linear correlation between malaria death rate and climate through *Anopheles* mosquitoes. More importantly, we argue these factors are presumably uncorrelated with the measurement errors and other omitted confounders and satisfy the exclusion restriction.

Our climatic variables may be invalid instruments if they are also correlated with other infectious diseases in the colonial period, which in turn could also have long-term health effects. If this is the case, the exclusion restriction would be violated. To verify the exclusion restriction, we first plot the mortality of major infectious diseases in the colonial period in figure 3. As shown, compared to malaria, the mortality of the other eight infectious diseases was almost negligible, except for plague in the 1900s and cholera in the second half of the 1910s. However, none of these diseases are known to be correlated with our climatic factors.

To further verify this, we first separately regress each infectious disease mortality and climatic factor on the exogenous variables, other than *Malaria*, in-

[11] Barreca (2010) uses temperature as an instrumental variable. However, the annual temperature across the whole island of Taiwan normally ranges from 21°C to about 26°C, which is suitable for the breeding of mosquitoes and the growth of malaria parasites year-round.

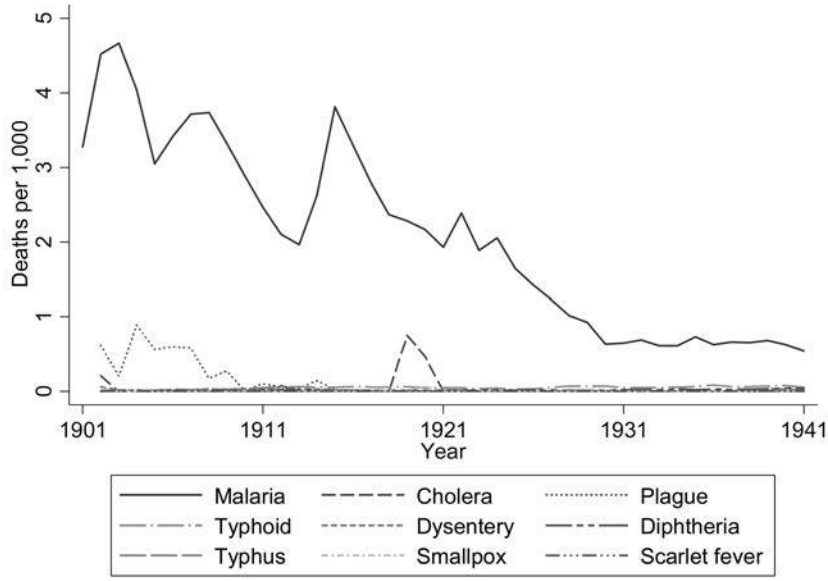

Figure 3. Mortalities of major infectious diseases in the colonial period

cluded on the right-hand side of equation (3), namely sex, ethnicity, father's education, father's major occupation, regional economic and education resource indicators, and a full set of region and cohort dummies. We then predict the residuals, which represent the variation in these infectious disease mortalities and climatic factors unexplained by the regressors. We relate the residuals for each pair of infectious disease mortality and climatic factor in figure 4. As shown, there is almost no correlation with the climatic factors for plague and cholera, while malaria is observed to have pronounced correlations with the three climatic factors. Later, we provide more statistical evidence on the correlation between malaria and the climatic factors in the discussion of our formal estimation results.

Mortality Selection

Our sample consists of individuals in later life, and a mortality selection problem must be considered. If the survivors and those who died of either malaria in early childhood or other causes in later life were different in some unobserved aspects that are also correlated with the later-life outcomes, our estimation results will be biased. For example, it is possible that people with higher innate ability are more likely to survive malaria and live longer and healthier. In this case, we are likely to underestimate the malaria effect on health. Since we do not observe the dead, we are not able to correct for such

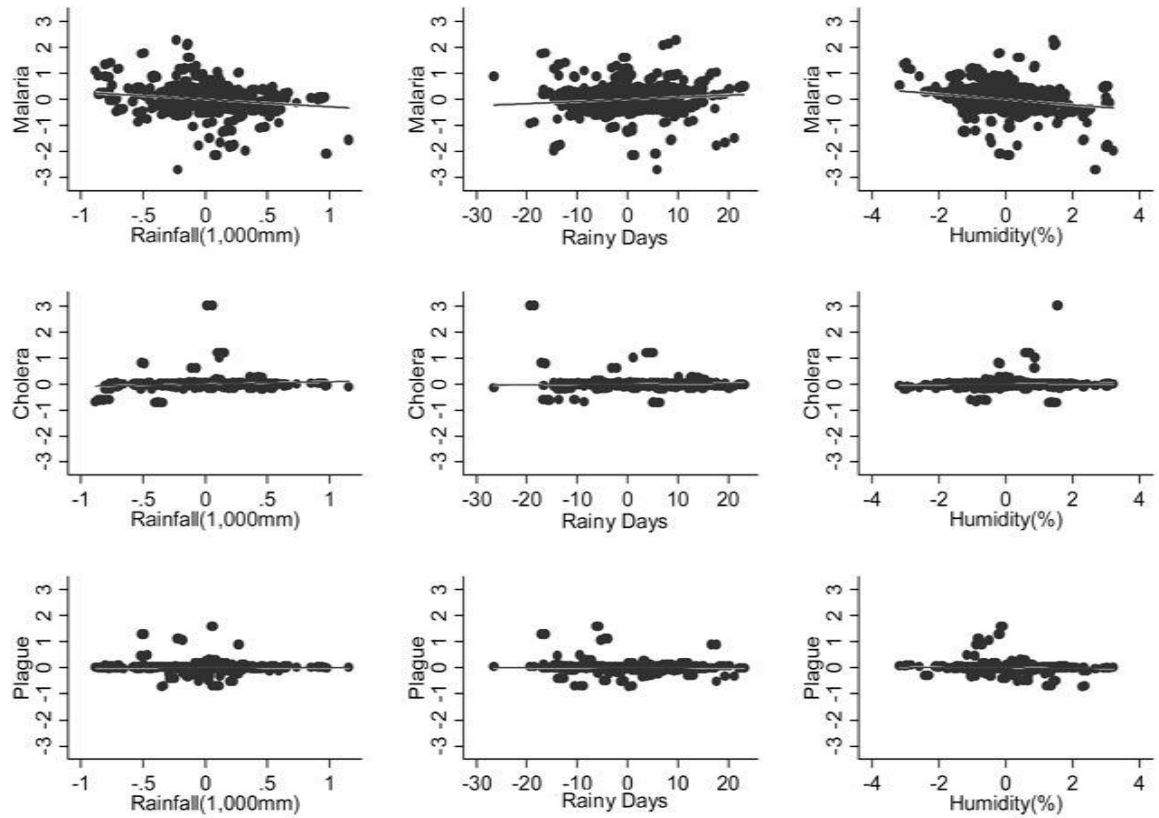

Figure 4. Partial correlation between weather and infectious diseases. Each dot represents a pair of residuals from two separate regressions. In each regression, the dependent variable is infectious disease mortality or a weather factor; regressors include right-hand side variables except for MALARIA.

selection bias without further information. However, we suspect that the selection bias attenuates the true malaria effect and thus consider our estimates as lower bound estimates of the true malaria effect.

IV. Estimation Results

OLS Estimates of Malaria Effects

Table 2 reports our ordinary least squares (OLS) estimates and two types of *p*-values, with the usual cluster robust *p*-value in parentheses and the wild-bootstrap cluster robust *p*-value in brackets. Note that the malaria death rate decreases from four deaths to one death per 1,000 people during the colonial period. Therefore, a decrease of one malaria death is equivalent to a decrease in the malaria death rate by about 25%. As shown in columns 1 and 2, a 25% increase in malaria death rate around birth is associated with a 9.5% ($2.7/28.29 \times 100\%$) increase in the probability of having high blood pressure and a 22% ($3.8/17.11 \times 100\%$) increase in the probability of having heart disease. Both types of *p*-value suggest the estimate is significant at 5% for heart disease, while it is not significant for high blood pressure.

Cognitive function results in columns 3 and 4 show that a 25% increase in malaria death rate around birth is associated with a 3.6% ($0.14/3.9 \times 100\%$) decrease in the number of objects that one can recall immediately and a 15.8% ($3/19 \times 100\%$) decrease in the probability of correctly counting backward a sequence of five single-digit numbers. The estimate for immediate recall is significant at 5% based on the wild-bootstrap cluster robust *p*-value, while the estimate for backward counting is significant at 5% based on the usual cluster robust standard *p*-value.

Finally, column 5 reports the estimate for self-reported poor health. A 25% increase in malaria death rate around birth is associated with a 16% (4.7/

TABLE 2
OLS ESTIMATES OF MALARIA EFFECT ON HEALTH OUTCOMES

	High Blood Pressure (1)	Heart Disease (2)	Immediate Recall (3)	Backward Counting (4)	Poor Health (5)
MALARIA	.027	.038	−.140	−.030	.047
	(.264)	(.012)	(.233)	(.035)	(.004)
	[.431]	[.033]	[.034]	[.143]	[.093]
Observations	3,572	3,572	3,293	3,293	3,572
R^2	.041	.033	.114	.103	.045

Note. Usual cluster-robust *p*-value in parentheses; wild-cluster bootstrap *p*-value in brackets; MALARIA is the average malaria deaths per 1,000 people in the birth region in birth year and the year before; other controls include sex, ethnicity, father's education, father's major occupation, numbers of primary schools per 100,000, and production of sugar cane and rice per capita (measured in 1929 prices) in birth year-region; all regressions include three dummies that indicate counties belonging to different regions across periods due to changes in administration divisions; all five regions are included.

29.27 × 100%) increase in the probability of reporting poor health. Both *p*-values suggest that this estimate is significant at least at the 10% level. Overall, the OLS estimates suggest that an increase in the malaria death rate around birth is associated with worse cardiovascular diseases, cognitive functions, and self-reported general health.

Two-Stage Least Squares Estimates

The first-stage estimation results are summarized in table 3. Due to a lack of climatic data for the Hsinchu region, we must exclude this region in all subsequent regressions. The sample size thus decreases by about 13%. Corresponding to our definition of MALARIA, we use the average climatic measures (i.e., rainfall, rainy days, and relative humidity) in the year of birth and the preceding year as our instrumental variables. Regressions in the first three columns include only one climatic measure, while the last column uses all three measures. In all four specifications, rainfall and relative humidity are negatively correlated with the malaria death rate, while the number of rainy days is positively correlated with the malaria death rate. We note in particular that all three estimates in column (4) are individually significant at the 5% level, and the *F*-statistic at the bottom shows that they are jointly significant as well, suggesting that these climatic factors are highly correlated with malaria deaths.

The malariology literature suggests that the interaction between malaria and climate could be complicated and highly nonlinear. Our goal is not to

TABLE 3
FIRST-STAGE ESTIMATES

	Dependent Variable: MALARIA			
	(1)	(2)	(3)	(4)
Total rainfall (100 mm.)	−.050			−.057
	(.045)			(.032)
	[.002]			[.002]
Total rainy days		.007		.028
		(.339)		(.019)
		[.476]		[.000]
Relative humidity			−.137	−.175
			(.096)	(.019)
			[.242]	[.002]
Observations	2,960	2,960	2,960	2,960
R^2	.931	.922	.929	.946
F				18.93
p				.019

Note. Usual cluster-robust *p*-value in parentheses; wild-cluster bootstrap *p*-value in brackets; all weather measures are average of the local weather measures in the birth year and the year before; other controls include all variables in the previous regression except for MALARIA; Hsinchu region excluded due to lack of weather data.

model the true relationship between malaria and climatic factors. Instead, we mainly want to use the variation in climatic factors to isolate the variation in malaria exposure risk orthogonal to measurement error and other omitted confounders.

Since we have only four regions in the two-stage least squares (2SLS) estimation, we cannot directly compare the results to the OLS estimates using all five regions in table 2. For comparison, we first reproduce OLS results using only four regions and report them in panel A of table 4. The 2SLS results are then reported in panel B. Compared to table 2, the OLS results using only four regions have the same signs as before, but they are generally larger in magnitude. Interestingly, the estimate for high blood pressure is not only larger but also highly significant, whereas it is insignificant in the table 2 estimation based on five regions.

In table 4, the 2SLS estimates have identical signs to the OLS estimates and are generally larger in magnitudes than the OLS estimates except for immediate recall and poor health. Compared to the means of these variables using only four regions (in parentheses below variable names), the 2SLS results suggest that a 25% increase in malaria death rate around birth leads to a 26% ($7.2/28 \times 100\%$) increase in the probability of having high blood pressure,

TABLE 4
OLS AND 2SLS ESTIMATES (HSINCHU REGION EXCLUDED)

	High Blood Pressure (Mean = .28) (1)	Heart Disease (Mean = .17) (2)	Immediate Recall (Mean = 3.9) (3)	Backward Counting (Mean = .20) (4)	Poor Health (Mean = .29) (5)
A. OLS estimates:					
MALARIA	.070	.047	−.283	−.044	.060
	(.033)	(.102)	(.204)	(.091)	(.009)
	[.000]	[.240]	[.002]	[.258]	[.058]
	2,960	2,960	2,712	2,712	2,960
	.047	.037	.127	.099	.049
B. 2SLS estimates:					
MALARIA	.072	.085	−.232	−.089	.043
	(.000)	(.000)	(.108)	(.000)	(.000)
	[.072]	[.061]	[.317]	[.072]	[.168]
Observations	2,960	2,960	2,712	2,712	2,960
Sargan χ^2	2.989	1.617	2.45	3.363	.023
p	.224	.446	.294	.186	.989

Note. Hsinchu region is excluded; usual cluster-robust *p*-value in parentheses; wild-cluster bootstrap *p*-value in brackets; MALARIA is the average malaria deaths per 1,000 people in the birth region in birth year and the year before; other controls include sex, ethnicity, father's education, father's major occupation, numbers of primary schools per 100,000, and production of sugar cane and rice per capita (measured in 1929 prices) in birth year-region; all regressions include three dummies that indicate counties belonging to different regions across periods due to changes in administration divisions; in panel B, instrumental variables include rainfall, rainy days, and relative humidity.

a 50% (8.5/17 × 100%) increase in the probability of having heart disease, a 6% (0.232/3.9 × 100%) decrease in the number of objects that one can recall immediately, a 45% (8.9/20 × 100%) decrease in the probability of correctly counting backward a sequence of five single-digit numbers, and a 15% (4.3/29 × 100%) increase in the probability of reporting poor health.

The usual cluster robust *p*-values in panel B suggest that the 2SLS estimates are generally very significant. Although the wild-bootstrap cluster robust *p*-values are much larger, the 2SLS results are still significant at the 10% level except for immediate recall and general health. We take the 2SLS results overall to confirm our OLS estimation results, suggesting an increase in malaria death rate around birth leads to worse health outcomes at old age. Finally, at the bottom of panel B, we provide overidentification test statistics. They suggest that our climatic instrumental variables are exogenous.

V. Discussion and Conclusion

We exploit variation in malaria deaths around birth caused by a malaria eradication campaign in colonial Taiwan to estimate the malaria effects on health outcomes in later life. We find that people who were exposed to a higher risk of malaria, in terms of malaria death rate, during the critical period around birth are more likely to have worse health outcomes at old age. Specifically, they are more likely to develop cardiovascular diseases, have worse cognitive functions, and report poor health. Our findings provide causal evidence in support of the Barker hypothesis.

In principle, a child under age 5 is still subject to the high risk of contracting malaria. It is thus natural to ask whether our estimation picks up the effect in utero or in the early postnatal period. In our study, we are not able to separate the two periods. However, the spirit of the Barker hypothesis is that health shocks occurring in the critical period when our organs are developing could leave a long-term impact on health outcomes in adult life. Although the intrauterine period is important, not all human organs are fully developed at birth. During infancy and early childhood, for example, lungs continue to develop and the brain grows rapidly as neural connections are formed. Therefore, we focus on the malaria effect *around birth*.

Appendix

Administrative Division Changes

During the period 1901–41, administrative divisions were redrawn in 1909 and 1920. Before 1920, divisions were called *ting*; after 1920, some were called

zhou (prefecture) and others were still called *ting*. In general, a *zhou* is about the size of 3–4 *tings*. During 1901–8, there were 19 *tings* on the main island of Taiwan.[12] During 1909–19, there were 11 *tings*. After 1920, there were five *zhous* in the west and two *tings* in the east. In this article, we use the division after 1920 as basis. We group two *zhous* in the south and two *tings* in the east to minimize the inconsistency caused by the division changes. Therefore, we have five regions in the end. There are still three townships belonging to different prefectures according to our definition in different periods. For individuals born to these three townships, we include three dummy variables into all regressions to pick up the effect caused by the administrative division inconsistency.

Malaria Death Rate

We count the yearly malaria deaths for the five prefectures and then divide them by corresponding population (in thousands). Note that the malaria deaths and population include both Taiwanese and Japanese. The Japanese accounts for less than 5% of the population in the colonial period.

Climate Data

The climate data are collected from the following four weather stations: Taipei station (25°02′N, 123°31′E) for the Taipei region, Taichung station (24°09′N, 120°42′E) for the Taichung region, Tainan station (22°59′N, 120°12′E) for the Nankao region, and Taidong station (22°45′N, 121°08′E) for the Huadong region.

Agricultural Production

Taiwan was an agrarian economy during the colonial period. The two most important agricultural products then were rice and sugar cane. While sugar cane was most popular in the south, rice was widespread throughout the whole island. We thus calculate the per capita value of rice and sugar cane production in each prefecture to measure the local economic status. We use the prices of rice and sugar cane in 1929. This variable is included in all regressions as a control for time-varying local economic status.

Primary Schools

To control for local education resources, we use the number of primary schools per 100,000 people, and this variable is also included in every re-

[12] The jurisdiction of colonial Taiwan also includes an archipelago called Pescadores, which is off the southwestern coast.

gression. Primary schools were the most available education resource to native Taiwanese in the colonial period.

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