Barker's Hypothesis Among the Global Poor: Positive Long-term Cardiovascular Effects of In-utero Famine Exposure

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Abstract

An influential literature on the Barker's hypothesis (or the Developmental Origins of Health and Disease, DOHaD) has documented that poor conditions in utero lead to higher risk of hypertension, diabetes, stroke and heart disease in middle age in middle- and high-income contexts. One of the main explanations is that periods of high calorie intake after birth are inconsistent with the adaptations that the fetus makes to prepare for a poor resources environment (thrifty phenotype hypothesis). Using data from a persistently low-income country, Malawi, we find that individuals exposed in utero to a substantial famine in 1949, have lower levels of blood pressure and blood sugar and less symptoms associated with stroke over half a century later. These findings may be explained by a prolonged period of malnutrition following the famine in contrast to most of the contexts studied in the previous literature.

Introduction

This study is among the first to document the lasting effects of severe early life adversity on health at older ages in a low-income country (LIC) cohort that has experienced extreme poverty throughout life (Figure 1A). Contrary to the predominant findings from higher-income contexts on early life origins of adult health and aging [1, 2], our analyses show that persons exposed to in-utero adversity during the 1949 Nyasaland Famine in Malawi have *better* cardiovascular health at older ages across multiple domains: they have lower blood pressure (Figure 1B), reduced age-related increase in blood pressure (Figure 1C), lower levels of blood sugar and fewer stroke symptoms. This result is not driven by mortality selection, as evidenced by analyses of census age patterns, but is possibly due to a "reverse"

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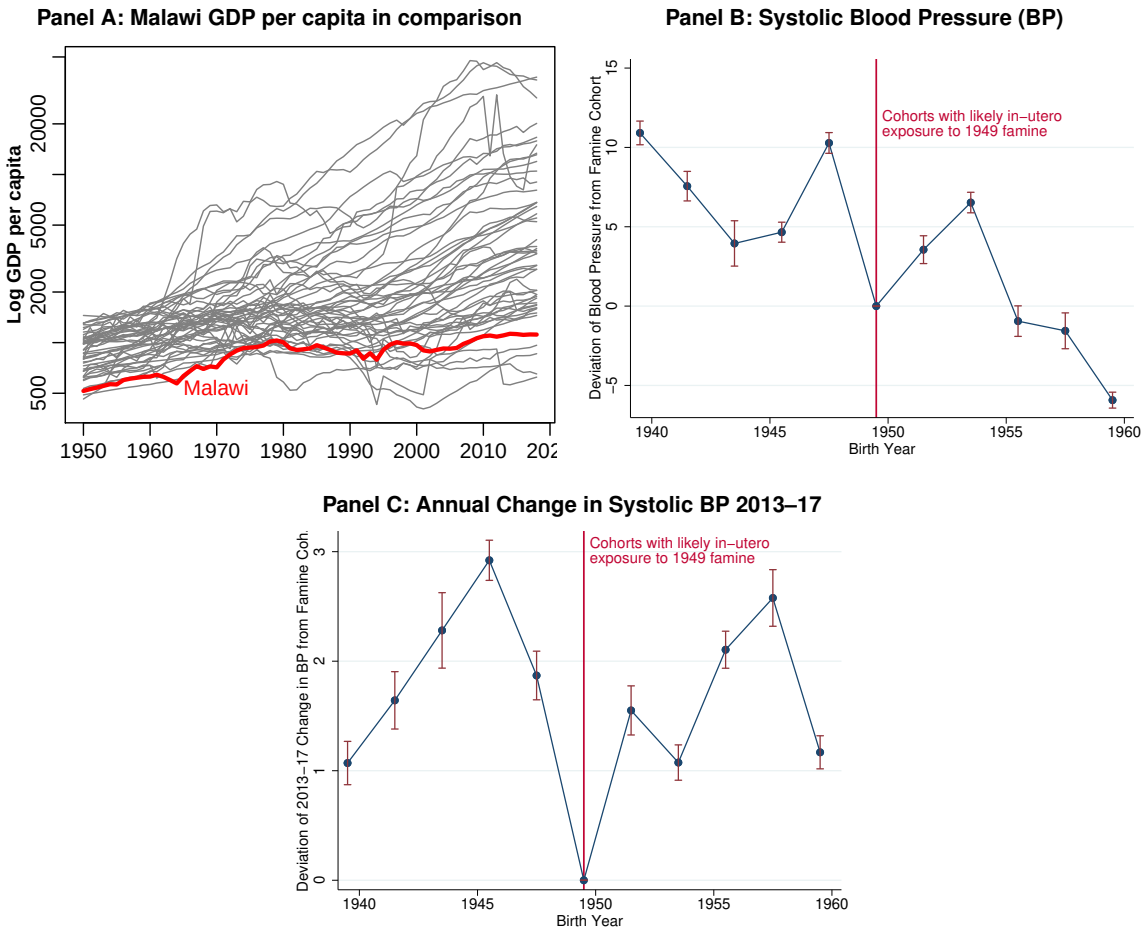

Figure 1: Panel A: GDP per capita during 1950–2018 of 50 poorest countries in 1950s based on cross-sectionally and longitudinally comparable income data. Panel B: Systolic blood pressure among MLSFH respondents exposed to the 1949 famine is lower than among those born before or a few years after the famine. Panel C: Famine-exposed individuals have smaller increases in systolic blood pressure per year of age during 2013–17

Notes: Panel A: Based on Maddison Project Database 2020 [4]. Panel B: Results are based on a OLS regression of systolic blood pressure on year pairs (e.g. 1939-1940, 1941-1942,...), gender, region of residence and birth and a dummy for whether the survey was administered in 2017 or 2019. The reference category corresponds to the cohorts with likely in-utero exposure to the famine, that is, 1949 and 1950. The graph shows 95% confidence intervals with standard errors clustered at the year pair level. Panel C: Results are based on a OLS regression of the change in systolic blood pressure from 2013 to 2017 on year pairs (e.g. 1939-1940, 1941-1942,...), gender, region of residence and birth. The reference category corresponds to the cohorts with likely in-utero exposure to the famine, that is, 1949 and 1950.The graph shows 95% confidence intervals with standard errors clustered at the year-pair level.

Barker hypothesis [3]: severe early life adversity in these cohorts might have resulted in increased resilience that partially protected individuals during lives with frequent exposures to poverty and adverse shocks. Against the odds, in this context with frequent and widespread lifecourse adversities and high levels of early- to mid-life mortality, individuals exposed to the Nyasaland famine maintained (relatively) better cardiovascular health across multiple domains as compared to their peers with less-severe in-utero adversities.

The first 1000 days of life, from conception to age 24 months, is a period of tremendous

physiological and neurological development [5, 6]. In this period, children's development is affected by their genetic endowments, psychosocial and biological factors and their environments. Because of the substantial changes and developments in the first 1000 days, this also is a period of great vulnerability where early life adversities may result in long-term health and socioeconomic consequences that persist throughout the life course. Importantly, in what became known as the *Barker hypothesis*, a series of studies on the importance of in-utero nutrition for health at older ages documented that early life deprivation is associated with the onset of a number of chronic conditions in late-middle age, including coronary heart disease, diabetes and hypertension [3, 7]. A parallel literature has documented analogous negative effects of early life adversities on a broad range of socioeconomic outcomes in adolescence and adulthood (e.g., schooling, income, occupations, marital status, timing of fertility) [8]. The hypothesized mechanism underlying these early life origins of later-life health is that different physical systems and organs—including importantly the cardiovascular system—may not be able to recover fully from lack of adequate nutrition at critical periods in fetal life and infancy, which causes long-run health problems from being subsequently exposed to a "normal" diet [3, 9].

The impacts of these early life-influences are not dwarfed by adult socioeconomic status or health behaviors; instead, they interact. The *thrifty phenotype concept* [10, 11], for example, implies that exposures and behaviors later in life (e.g., diet, physical activity, and health and socioeconomic contexts) modify the long-term implications of early life adversities: the greater the discrepancy between early childhood conditions such as nutritional deprivation, and relative nutritional affluence in later life, the greater are potentially the consequences of in-utero deprivation on later-life health. High rates of cardiovascular disease and diabetes among older persons in middle- and high-income countries (MHICs) are partially attributed to this mechanism [12].

Yet, most of this literature on the lifecourse implications of early life shocks is from MHICs where population aging and chronic diseases are long-standing health and policy concerns, and where longitudinal cohort studies allow lifecourse linkages between early life events and health at adult and older ages. The underrepresentation of low-income countries (LICs) in this literature, however, is unfortunate for at least two reasons. First, globally, major sources of negative shocks in the first 1000 days are weather fluctuations. Despite the high levels of and increases in extreme weather events in LICs as a result of climate change [13], there has been relatively little investigation of the impact of weather fluctuations in LICs, even though such countries would seem to be where children in early life are most vulnerable to such fluctuations due to high dependence on rainfed agriculture, poor transportation and health infrastructure, and limited institutionalized safeguards that buffer individuals and their families from the consequences of extreme weather. We currently don't have adequate evidence on the long-lasting influences of weather-related early life shocks among a large and growing population that was highly vulnerable to such

shocks: individuals born in LICs and now reaching old age.

Second, the focus on MHICs in research on the developmental origins of adult health also implies that analyses might miss important context-specificity in the effects of early life adversities on later-life health. Importantly, existing MHIC studies mostly investigate individuals whose early life adversities were followed by better—and often considerably better—health and socioeconomic contexts during later childhood, adult and old ages (e.g, the Dutch hunger-winter cohort [1], individuals born during the Great Depression in the 1930s [14], or individuals born during the early 1900s in countries that experienced substantial economic development during the second half of the 20th century [15], etc.). The experiences of these cohorts differ dramatically from those experienced by the global poor reaching older ages: their lives were characterized by poverty and adversities *throughout* (Figure 1A). For example, the individuals studied in our analyses lived most of their lives in subsistence-agriculture contexts with per capita incomes of less than \$1/day [16]. Members of the cohort were born when under-5 mortality was almost one out of three (≈30%) [17], and they have survived sustained poverty, repeated famines and the HIV/AIDS epidemic. As a matter of fact, among the globally poorest countries in the 1950s, Malawi stands out as one of the few countries that remained very poor in subsequent decades (Figure 1A).

The exposure to repeated and sustained adversities throughout the lifecourse has given rise at older ages to *accelerated aging*. Accelerated aging is a distinctive hallmark of health in LICs that starts in mid-adulthood and extends to older ages, and that entails an earlier onset and faster pace-of-decline in physical, mental and cognitive health [18, 19]. Major disease burdens occurring at younger ages in LICs than in MHICs include poor cognitive health, depression, anxiety, cardiovascular diseases, and chronic pain [20, 21].

Importantly, in LICs with accelerated aging, early life adversities may have very different consequences on adult health from those that have been documented in MHICs. On one hand, early life adversities might matter less for health at older ages when adversities are common throughout the lifecourse, as is the case in LICs; on the other hand, individuals exposed to severe in-utero adversities might even be protected from worse health at older ages if their lifecourse includes repeated and sustained exposure to adversities. This latter possibility has previously been raised in the literature on early life effects on later-life health, in part based on animal studies that have documented predictive adaptive responses based on in-utero environments (e.g., in-utero nutrient deprivation linked to adaptations for a limited post-natal diet) [9, 22, 23]. Contrary to the predictions of the Developmental Origin of Health and Disease literature, while not finding positive later-life health consequences as we do in our analyses of a LIC cohort (Figure 1B+C), some HIC studies have shown *null-effects*, including absence of elevated late-life mortality after famine exposure [24] or lack of worse health among children born during the American Dust Bowl [25].

Protective effects of early life adversities on health at older ages have rarely been documented in human populations. In part, such patterns are difficult to detect given available data: much of the research on the Barker hypothesis and the developmental origins of adult diseases is based on MHIC data where early life adversities were often followed by substantially improved circumstances in later childhood, adulthood and old age. Our study points to the importance of investigating the early life origins or later-life health in LICs where poverty and adversities prevail throughout the lifecourse, and where accelerated aging is common. Our findings indicate that some well-established relations between early life adversities and later-life health might be distinctly different in these contexts.

The 1949 Nyasaland Famine

Malawi is one of the poorest countries in the world with current GDP per capita around 4% of the global average. Now, as was the case in the 1940s, the main economic activity is centered on rain-fed agriculture. Maize is the main crop, and sweet potatoes and cassava are distant seconds. Tobacco has been the main cash crop since the early decades of the 20th century, creating a significant competition for land available for subsistence agriculture. All crops are planted at the start of the rainy season which runs from November to April (Supplemental Figure A.1). Harvest time is between April and June. This harvest needs to last for a year until the end of the next growing season, resulting in an annual "*hungry season*" from December, when food supplies tend to start running low, to the time of the new harvest. Our study population, the Malawi Longitudinal Study of Families and Health (MLSFH) cohort [26, 27], was selected to represent these rural contexts where the majority of Malawians (85%) live in conditions similar to those in other rural sub-Saharan African LICs: subsistence agriculture, poor health conditions, over-burdened health facilities, and frequently unmet nutritional needs.

The 1949 famine occurred in Nyasaland, the British colony that became Malawi after independence in 1964. The famine is relatively well-documented in a 1987 book "The story of an African famine" [28]. While famines occur with some regularity in Malawi, and have also been the subject of a well-known book and film *The Boy Who Harnessed the Wind* [29], the 1949 famine was an outlier. It was substantially more severe than preceding or subsequent famines, and its impacts are extensively recorded in colonial reports and the country's oral history.

The 1949 Nyasaland famine was preluded by lower-than-average rainfall in 1948 that led to poor harvests and depleted maize reserves. When the rains almost stopped in the first part of 1949, the extremely poor 1949 harvest occurred at a time when food storage was already almost exhausted and little other supplies were available for the population. The culmination of three years of exceedingly low rainfall that resulted in the almost complete loss of the 1949 harvest, therefore, extended the annual *hungry season* from late 1948

until the new harvest in April 1950. While poor rains triggered the famine, as in other cases [30], the weather impacts on food supply were exacerbated by other factors: limited access to farmland, poor management by colonial authorities who failed to keep sufficient maize in storage, and a failure to adequately import food from other countries [28]. Even though most common accounts of the famine focus on the Southern Region of Malawi, the resulting crisis was a national one: weather data show much lower than usual levels of precipitation in central and northern regions as well (Figure A.2), colonial harvest records point to clear impacts in the Central Region in addition to the Southern Region, and maize prices increased nationally as a result of redistribution efforts within overall severely limited national food supplies [28].

Data

To identify the long-term health effects of the Nyasaland famine, our analyses investigate individuals who were affected by the famine while in utero. Because the famine lasted from the start of the hungry season of 1948/49 (with severe hunger beginning in late 1948) to the harvest of 1950s (mid 1950), we consider individuals born in 1949 and 1950 as having had in-utero exposure to the famine. We then compare individuals with in-utero exposure to the famine with individuals born ± 10 years of the 1949 famine.

The Malawi Longitudinal Study of Family and Health (MLSFH) [26, 27] is a rare dataset enabling these analyses because (*a*) it includes older respondents, which are often omitted in LIC surveys, (*b*) provides extensive—and often longitudinal—health measures that cover all aspects of the metabolic syndrome (hypertension, diabetes, heart disease and obesity) as well as information on mortality after enrollment in the study, and (*c*) includes study areas—Balaka in the Southern and Mchinji in the Central Region—that experienced the largest shortfalls in rain in 1949, while respondents in Rumphi in the Northern Region were affected by the spillovers on national food supplies and economy that resulted from the famine. Our analyses sample consists of about 850 MLSFH respondents born in 1939–1960 (1949 ± 10 years). Robustness tests also use MLSFH respondents born in 1929–70 (1949 ± 20 years).

The MLSFH is one of very few long-standing publicly-available cohort studies in a Sub-Saharan African LIC, and it provides a rare record of more than two decades of demographic, socioeconomic and health conditions in one of the world's poorest countries. The MLSFH currently consists of 12 data collection rounds from 1998–2020 with extensive lifecourse, socioeconomic and health information [26, 27, 31]. While the MLSFH is not nationally representative, comparisons with the rural samples of the Malawi DHS [32] and IHS [33] confirm that the MLSFH study population continues to match closely the characteristics of nationally representative surveys [26, 27]. The initial MLSFH sample was established using a cluster random sampling strategy (Mchinji and Rumphi) and by drawing

a subset of an earlier representative population survey (Balaka). The initial sample characteristics closely matched the characteristics of the rural population of the 1996 Malawi Demographic and Health Survey (MDHS) [26]. Importantly for this study, during the 2008 MLSFH round, a sample of parents of the original MLSFH respondents was added to the MLSFH to increase the suitability of the MLSFH for studying intergenerational aspects and the health of older individuals. The MLSFH study population has been followed up until 2020 (including migration follow-ups), with 2012–18 data collections focusing on a subset of MLSFH respondents aged 45+ (MLSFH Mature Adults Cohort), and the 2019 data collection following-up on the remaining MLSFH respondents (including older MLSFH respondents who previously were not included in the MLSFH Mature Adults cohort). MLSFH Cohort Profiles [26, 27] provide detailed information about sampling, study instruments, attrition/follow-up rates, and data quality. Non-mortality-related attrition among MLSFH respondents is *not* different for respondents with in-utero exposure to the famine as compared to their peers born in the years before or after the 1949 famine, while the mortality among famine-affected respondents is lower (Supplemental Table A.6; discussed also in more detail below).

The primary outcomes for our analyses are systolic/diastolic blood pressures and blood glucose. Secondary outcomes include anthropometric measures (weight, height, waist and hip circumference) and symptoms of heart disease and stroke. Systolic and diastolic blood pressures were collected in 2013, 2017 and 2019, each round making three measurements using upper-arm blood-pressure monitors. We take the average of the three measures and determine hypertension stage 1 (systolic > 130mmHg or diastolic > 80mmHg) and stage 2 (systolic > 140mmHg or diastolic > 90mmHg). Fasting blood glucose was collected in 2017. We classify diabetes as blood glucose > 7mmo/L and pre-diabetes as glucose > 5.6mmo/L. For 51 respondents who did not fast, the thresholds were set as 11.1mmo/L for diabetes and 7.8mmo/L for pre-diabetes. Body mass index (BMI) was calculated based on *measured* height and weight. Measured waist and hip circumferences are combined to construct the waist-hip ratio (2017 only). Symptoms of stroke are taken from the Questionnaire for Verifying Stroke-Free Status (QVSFS) [34] and include sudden weakness in one part of the body, sudden numbness in one part of the body, sudden loss of vision, sudden loss of ability to speak and ability to understand. For symptoms of heart disease, we rely on the WHO Rose Angina Questionnaire, which elicits experience of chest pain, exertion and symptom resolution [35]. We use the number of stroke symptoms, the presence of any symptoms, and whether respondents experienced any stroke or angina as secondary outcomes in our analyses. In all cases when multiple health measures are available for an individual, we select the most recent available measure to assess the effect of the famine. In addition, for blood pressures, we also compute the *annual changes* in blood pressures during 2013–17 (for respondents surveyed in 2013 and 2017 only; for those surveyed in 2019, no prior blood-pressure measurements are available).

Summary statistics for our analysis sample are reported in Table A.1. Women represent slightly more than 50% of the sample. About 25% of respondents do not have any formal schooling. This low schooling attainment is expected as respondents in our analyses sample were born too early to benefit from the dramatic increases in school enrollments since the 1990s. Our study population is characterized by relatively high prevalence of hypertension, despite the relative absence of risk factors such as Western diets or obesity (only 5% are classified as obese based on BMI). 48% of individuals in our analyses sample are stage-2 hypertensive, and more than two thirds (71%) are at least pre-hypertensive (stage-1 or -2 hypertension). Diabetes is much less prevalent. Only 2% have diabetes based on fasting blood glucose. 7% are either pre-diabetic or diabetic. For this reason, we will only focus on pre-diabetes in the analysis. MLSFH anthropometric measures document an average height (159cm), weight (55kg) and BMI (21.9) that are much lower than those commonly observed in HIC cohorts. Only 2% of respondents have been diagnosed by medical doctors with a stroke and only 2% with angina (as of 2017).

Methods

Age measurement and exposure to the famine: A complication arises in our context as the measurement of birth year is affected by measurement error. For the cohorts exposed to the famine, neither Malawi nor other sub-Saharan LICs had reliable birth registration [36]. Birth year needs to be inferred from respondents' own recollections, which are often subject to measurement errors [37–39]. An advantage of our analyses is that we can rely on two different and independent data collection efforts to obtain birth years. Our first analyses rely on the birth years as reflected on Malawi's National Identity Card. The National Registration Bureau (NRB) started to issue National IDs in 2007 [36], and the MLSFH has been recording birth years from the National IDs since 2017 (MLSFH interviewers obtained birth years directly from the ID card). In some cases, when a respondent did not have a National ID but a Voter Registration Card, birth years were recorded based on the Voter Registration Cards. For individuals in our analyses data, birth years from National IDs are available for 91% of respondents, and from Voter Registration Cards for 9%. Appendix Figure A.3 shows a histogram of the birth-year distribution of our analyses sample; there is no clear evidence of heaping at 5 or 10 years intervals. Robustness tests for different measures of famine exposure will be reported below.

While National ID cards are issued by the NRB, it is important to recognize that the birth dates on the National ID cards for our analysis sample are based on respondents' reports to NRB staff without external validation via birth registration linkages. Thus, respondents' imperfect knowledge about their exact birth years and dates also affects the information on National IDs. Hence, there are likely measurement errors in the birth-year information on the National IDs.

The MLSFH provides an alternative way to assess exposure to the 1949 famine based on reported ages in the survey. While each age report elicited in a survey is likely subject to some measurement error, as is common for surveys in SSA [38, 39], individuals in our analysis sample have been asked about their ages multiple times during 1998–2019 (on average 11 times, with an interquartile range from 9 to 15 times). Rather than relying on a single age report to infer birth year, therefore, we first infer birth year as $(= \textit{survey year} - \textit{age report})$ and then estimate a *robust birth year* for each respondent as the median of all available MLSFH birth year data. If measurement/recall error of age is random across different measurements, combining multiple age reports reduces measurement error. If NRB staff faced similar misreporting as the MLSFH, robust age will have smaller measurement error than the National ID birth year. The median used to infer robust birth year is also not affected by single outliers, thereby reducing concerns about age heaping. A *robust* measure of each respondent's age is obtained as $(= \textit{survey year} - \textit{robust birth year})$, providing our "best" (and intertemporarily consistent) measure of a respondent's actual age.

We subsequently determined in-utero exposure to the 1949 famine based on this *robust* age. As the MLSFH surveys are primarily conducted in June and July, a respondent aged 69 years at the time of the survey in 2019 has a roughly 50% chance of having being born in the 2nd half of 1949 or the first half of 1950 (and analogously for any other age). In our analyses, we therefore consider respondents with a 2019 *robust* age of 69 as being exposed in-utero to the 1949 famine: they were approximately born during the 2nd half of 1949 and the first half of 1950, and thus were in-utero during the most severe periods of the famine. The sample selected using robust age shows very similar characteristics (Table A.2) to the sample selected using the registration card. Robustness tests using alternative assumptions are explored below.

All of our key findings are consistent across both measures of famine exposure derived using either the National IDs or robust age reports. Moreover, random measurement error in each of the derived indicators for in-utero famine exposure tends to bias our estimated coefficients towards zero [40], and the identified effects of the 1949 famine on subsequent health therefore are likely conservative: effects would likely be larger if a more-accurate measure of birth year were available for this study population.

In-utero famine exposure and long-term health: The effects of famine exposure on subsequent health are estimated using

$$y_i = \alpha + \beta T_i + \gamma_1 \, year_i + \gamma_2 \, year_i^2 + \gamma_3 X_i + \epsilon_i, \tag{1}$$

where y_i is health for individual i measured during 2012–19, T_i is a dummy for being affected by the famine in utero (born in 1949 or 1950), *year* is the year of birth minus 1938 –the oldest cohort in our sample–, X_i are individual controls including gender, region of birth, region of current location and survey wave dummies corresponding to the survey

wave used for y_i. Standard errors are clustered at the year of birth. In the main specification, we do not include schooling in X_i because schooling is potentially an endogenous variable that could be affected by the famine.

Survival of individuals exposed to the famine: The MLSFH is not suitable to identify potential differential survival to older ages among individuals with and without in-utero exposure to the 1949 famine. Instead, we use complete (100%) Malawi census data from 1987 and 1998 that has been made available through the African Census Analysis Project (ACAP) [41]. Data were aggregated by single-age cohort. Analyses are restricted to cohorts who were between 40 and 60 years old by 1998 (29–49 in 1987). Robustness tests also use cohorts aged 30–70 years in 1998. Census data are affected by age heaping at multiples of 5 and 10 (Supplemental Figure A.4), for which we control in our analyses. Estimates for potentially differential survival to adulthood are obtained from the census data using

$$y_i = \alpha + \beta T_i + \gamma_1 age_i + \gamma_2 age_i^2 + \gamma_3 d_5 + \gamma_4 d_{10} + \epsilon_i, \quad (2)$$

where y_i is the number of individuals alive in cohort i, T_i is a dummy for being affected by the famine in utero (born in second semester of 1949 or first semester of 1950), d_5 is a dummy equal to 1 if age is a multiple of 5 and d_{10} is a dummy equal to 1 if age is a multiple of 10. The dummies d_5 and d_{10} control for the tendency to round age to 5 or 10 years intervals. Because the two census are 11 years apart, this age heaping affects the 1987 and 1998 censuses differentially, adding to the robustness of our findings.

Results

In-utero famine exposure and long-term health: Panel A of Table 1 presents the effects of in-utero famine exposure on hypertension and diabetes risk, using birth year (1949 or 1950) from the National ID cards to identify individuals affected by the famine. 70 years after the in-utero exposure to the famine, affected individuals have systolic and diastolic blood pressures that are on average 6 mmHg and 3 mmHg respectively lower than those of individuals without in-utero exposure to the famine. These results are both statistically significant and large in magnitude (e.g., for systolic blood pressure corresponding to a reduction by 4.5%, or .25 std. deviations), and confirm our previous descriptive findings in Figure 1 (Panel B). Given average blood pressures of 139 systolic and 85 diastolic in the study population, these reductions indicate a positive effect on cardiovascular health. The famine effects, however, are not sufficiently large to significantly reduce the proportion of people who are defined to be hypertensive (columns 5 and 6). Famine exposure also leads to a statistically significant decrease in blood glucose by 0.13mmol/L, but given the overall low prevalence of diabetes and pre-diabetes, this magnitude of this effect is not sufficiently large to reduce the proportion of respondents who are prediabetic (blood glucose

Table 1: In-utero famine exposure and long-term health: Exposure identified based on birth year (1949 or 1950) recorded in National Registration Card (National ID)

Panel A: Hypertension and Diabetes Risk

	Blood pressure				Hypertension		Diabetes risk	
	Systolic (1)	Diastolic (2)	Δ sys (3)	Δ dias (4)	Stage 1 (5)	Stage 2 (6)	Glucose (7)	Prediabetes (8)
Famine exposure	-6.24***	-2.99**	-1.87***	-0.96***	-0.07	-0.01	-0.13**	0.01
	(1.84)	(1.20)	(0.42)	(0.20)	(0.07)	(0.03)	(0.05)	(0.01)
N	818	818	561	561	818	818	639	621

Panel B: Stroke and Heart Disease Symptoms

	Stroke		Heart disease
	Number of symptoms (1)	Any symptoms (2)	Rose angina (3)
Famine exposure	-0.19**	-0.06	0.04
	(0.08)	(0.07)	(0.04)
N	642	642	642

Panel C: Anthropometrics

	Height (1)	Weight (2)	BMI (3)	Waist/hip ratio (4)	Obese (5)
Famine exposure	-0.69	-0.22	0.19	0.00	0.02
	(0.44)	(1.26)	(0.52)	(0.00)	(0.02)
N	822	822	810	823	810

Notes: The table shows regression estimates for in-utero exposure to the famine following econometric specification (1) using a sample of MLSFH respondents born between 1939 and 1960. In-utero exposure is defined as being born in 1949 or 1950 according to the National Registration Card. Estimates for control variables are not shown. Standard errors clustered at the age level are reported in parentheses * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

$\geq$ 7mmol/L).

In our study population, blood pressures increased with age by about .2 mmHG per year on average during 2013–17 (longitudinal measures are available for 561 respondents in our analysis sample). These age gradients are reduced by about 2 mmHg for systolic and 1 mmHg for diastolic per year for individuals with in-utero exposure to the famine (columns 3 and 4 in Table 1), confirming our previous descriptive findings in Figure 1 (Panel C).

Panel B of Table 1 documents the effects of in-utero famine exposure on stroke and heart-disease symptoms. We find a negative effect on the number of stroke symptoms, but no effect on reporting any stroke symptoms or on the Rose angina indicator. We also do not find any famine effects on anthropometrics (Panel C in Table 1), although the negative coefficient on height is almost statistically significant (p-value = 0.12) and in line with the literature on negative economic shocks in utero and stunting [42, 43].

Table 2: In-utero famine exposure and long-term health: Exposure identified based on robust age (age 51 in mid-2019, which implies being born in second half of 1949 or first part of 1950)

Panel A: Hypertension and Diabetes risk

	Blood pressure				Hypertension		Diabetes risk	
	Systolic (1)	Diastolic (2)	Δ sys (3)	Δ dias (4)	Stage 1 (5)	Stage 2 (6)	Glucose (7)	Prediabetes (8)
Famine exposure	-2.97**	-1.70**	-1.20***	-0.89***	-0.09***	0.02	-0.20***	-0.03**
	(1.20)	(0.72)	(0.39)	(0.22)	(0.02)	(0.03)	(0.05)	(0.01)
N	888	888	609	609	888	888	700	678

Panel B: Stroke and heart disease symptoms

	Stroke		Heart disease
	Number of symptoms (1)	Any symptoms (2)	Rose angina (3)
Famine exposure	-0.44***	-0.17***	-0.10***
	(0.05)	(0.03)	(0.02)
N	699	699	699

Panel C: Anthropometrics

	Height (1)	Weight (2)	BMI (3)	Waist/hip ratio (4)	Obese (5)
Famine exposure	0.09	-1.78***	-1.29***	-0.01***	-0.03**
	(0.29)	(0.57)	(0.17)	(0.00)	(0.01)
N	893	891	870	892	870

Notes: The table shows regression estimates for in-utero exposure to the famine following econometric specification (1). In-utero exposure is defined as being born in the second semester of 1949 or first semester of 1950 according to our robust measure of age. Estimates for control variables are not shown. Standard errors clustered at the year of birth level are reported in parentheses * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 2 presents the corresponding estimates of in-utero famine exposure on long-term health using *robust age* to identify individuals affected by the famine. Across most outcomes, the famine effects are estimated more precisely (smaller std. error) using robust age rather than birth year from the National ID. The reductions in systolic and diastolic blood pressure are somewhat smaller in magnitude (Panel A), but remain statistically significant. The analyses in Table 2 also document a statistically significant reduction as a result of famine exposure in the proportion being pre-hypertensive or having pre-diabetes (Panel 1). We also observe a negative significant effect on the likelihood of reporting any stroke symptom and having heart disease based on the Rosa angina metric (Panel B). There are also significant negative coefficient estimates for weight, BMI, waist/hip ratio and obesity (Panel C).

Table 3: Mortality Selection: Census Data

	Outcome: Number of persons by single year of age			
	Census 1998		Census 1987	
	(1)	(2)	(3)	(4)
Famine exposure	29807	29391	157	2228
	(19718)	(17664)	(11806)	(14176)
Age multiple of 5	29389***	23014*	26279***	31923***
	(10168)	(12597)	(6092)	(10147)
Age multiple of 10	28498**	31927*	9389	16144
	(12966)	(16054)	(7768)	(12925)
Years of birth	1928–68	1938–58	1927–67	1937–57
N	41	21	41	21

Notes: The table shows regression estimates for in-utero exposure to the famine following econometric specification (2). Analyses additionally control for age and age^2. In-utero exposure is defined as being born in the second semester of 1949 or first semester of 1950 according to the 1998 or 1987 Census. The sample is limited to 20 years before and after the cohort born in 1949 in columns (1) and (3) and to 10 years before and after in columns (2) and (4). Standard errors in parentheses * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Robustness: In light of the measurement errors that are inherent in any age or birth-year reports for sub-Saharan African LIC cohorts born early- to mid-20th century, the consistency of our findings across different and independent approaches to determine famine exposure in Tables 1–2 is an important robustness test for our key findings (robustness also extends to alternative specifications using survey-reported age to identify famine exposure that are not reported here in detail). Our key findings are also robust if analyses additionally control for schooling (Table A.3), and the remain unchanged when extending the analyses sample to include respondents born twenty years before and after the famine (Table A.4). A placebo analysis for cohorts born in 1951–52, that is, cohorts without any famine exposure in early life, does not document any long-term health effect (Table A.5) (cohorts born before 1949 do not provide a valid placebo test as they had early-life exposure to the famine).

Survival of individuals exposed to the famine: Mortality selection is one potential concern in interpreting the previous results as causal estimates. All cohorts in our analyses sample faced very high early- and mid-life mortality, and all survivors to older ages in these cohorts are fairly selected (period life expectancy at birth was around 35 years in the 1950s, and remained below 46 years until the early 2000s [17]). Hence, if individuals exposed to the famine faced additional mortality and were more likely to die prior to reaching adulthood or older ages, then the survivors may be more healthy due to *additional* mortality selection.

No accurate data on infant and under-5yr mortality exist for the period around the 1949 Nyasaland famine; yet, since under-5 mortality is estimated to have been around 1

in 3 (≈35%) [17], among the highest in the world at this time (Supplemental Figure A.5), a further substantial spike in under-5yr mortality seems unlikely (albeit the age-pattern of under-5yr mortality might have changed with more children dying early [44]). While we cannot directly estimate variation in under-5yr mortality as a result of the famine, analyses of census data allow us to investigate differences in cohort sizes. Importantly, our analyses of the 100% Malawi censuses from 1987 and 1997 in Table 3 show that famine-exposed cohorts are not smaller than cohorts born before or after the famine, after controlling for the trend in overall cohort sizes and patterns of age heaping. This result is consistent for both the 1987 and 1998 census, which is noteworthy as these censuses are differentially affected by age-heaping given the 11-year gap between census years. Our analyses of census data in Table 3 thus do *not* provide evidence for a reduced survival to adulthood among famine-exposed cohorts; to the contrary, survival to adult ages might even have been somewhat enhanced for famine cohorts as famines and related crises generally depress fertility (albeit effects can be small in pre-demographic transition populations) [44]. This conclusion is further supported by analyses of mortality and attrition in the MLSFH cohort itself (Supplemental Table A.6). These analyses focus on respondents who were successfully interviewed in 2008/10, i.e., the survey rounds when MLSFH first expanded to older ages, and focus on three outcomes observed in 2018/19: being interviewed, being recorded as deceased, and being interviewed conditional on survival. Individuals exposed to the famine were slightly more likely to be surveyed during 2018–19, and this effect is driven by *lower* mortality during 2008/10– 2018/19 among famine-exposed respondents (Supplemental Table A.6). Related findings of reduced older-age mortality have also been documented for Finish cohorts exposed to famine in early life [24].

Acknowledging limitations due to the lack of detailed fertility and mortality data for the relevant time periods, the analyses in Tables 3 and A.6 nonetheless suggest that the positive effects of in-utero famine exposure on long-term cardiovascular health (Tables 1–2) are *not* the result of *additional* mortality selection among famine-exposed cohorts. There is no evidence that survival to or during adulthood was lower in these cohorts.

Discussion and Conclusion

The Barker (or Developmental Origins of Health and Disease, DOHaD) hypothesis that adversities in utero have long-run negative effects on adult health has had substantial influence on understanding the origins of diseases. Numerous studies have presented results that are consistent with this hypothesis. But almost all of these studies are in middle or high-income contexts (HMICs). In these HMICs, importantly, the studied adversities are generally restricted to early life as a result of specific events (famine, depression, etc.), and adversities do not persist as the individuals pass through the life course in countries that are relatively highly developed.

This study is one of the first, if not the first, to examine the impact of adversities in utero in a persistently low-income context. The individuals we study experienced not only an in-utero adversity in the form of a severe famine, but they also spent their childhood and adulthood in a very poor context with high overall mortality levels and frequent and diverse lifecourse adversities. We conjecture that in such a setting the adversities in utero might cause adaptations that turned out to be advantageous in terms of cardiovascular health given that individuals were subsequently exposed to persistently low incomes and poor living standards.

We investigate such possibilities by examining individuals who were and were not exposed in utero to the 1949 Nyasaland Famine, a well-documented and severe famine in Malawi. Relying on the Malawi Longitudinal Survey on Families and Health, we document the long-run impacts of this in-utero famine exposure on cardiovascular and related health almost 70 years later. We find significant effects of the famine in terms of *reducing* systolic and diastolic blood pressures, changes in systolic and diastolic blood pressures during 2013–17, diabetes risk as measured by glucose levels, and the number of reported stroke symptoms. These results are robust across analytic specifications, and consistent across alternative inferences of year of birth. Results disappear—as is expected—when using a placebo exposure one year after the famine. Analyses of census and MLSFH attrition data confirm our interpretation that additional mortality selection among famine-affected cohorts is not likely to drive our results.

These findings need to be interpreted within the context of the cohorts that are studied. All individuals born prior to 1960 in Malawi faced significant lifecourse adversities. Under 5-year mortality for these cohorts was about 1 in 3, and period life expectancies at birth were around 35 years in the 1950s, and remained below 46 years until the early 2000s [17]. Survivors to older ages in all of these cohorts are thus very selected. But despite this selection, health at older ages is often poor, and individuals experience accelerated aging with an early onset of morbidity across multiple domains [18–21]. Cardiovascular health in particular is often poor, as we document in our analyses above and elsewhere [27, 45], with widespread hypertension in the absence of risk factors such as obesity or Western diets. In the particular context of these cohorts, our findings indicate that exposure to the 1949 Nyasaland famine had *positive* effects on cardiovascular health. This finding is important because it extends evidence on the Barker (or DOHaD) hypothesis to persistently low-income contexts that have to-date received very little attention in this literature. This finding is also important because it indicates that early-life adversities may have distinctive implications for some aspects of health—cardiovascular health in our study—when cohorts experience not only early but also severe lifecourse adversities.

But we want to emphasize that our findings should *not* be construed as evidence against the large body of findings that have documented that early-life adversities in general have negative health and socioeconomic consequences later in life [6, 8, 9], and that policies that

target early-life adversities to improve developmental outcomes of children have high returns and should be prioritized [46, 47]. In Malawi and globally, recent cohorts did face, and future cohorts are likely to face much lower levels of early life *and* subsequent adversities than the cohorts studied in this paper [48]. Further improving early-life *and* ongoing environments of recent and future cohorts in Malawi and globally is an important priority that should not be interpreted to be contradicted by the evidence provided in this paper. Indeed, as subsequent adversities are reduced, the impacts of early life adversities in Malawi are likely to have the negative longer-run Barker hypothesis effects on cardiovascular health that have been reported in other contexts with less ongoing adversities than experienced by the cohorts that we study [1, 3, 9].

Several limitations of our analyses need to be acknowledged. As in other LIC studies, age and birth year cannot be verified by registration data. Individuals' recall of age or birth year is subject to measurement errors that affect both survey responses and information on the National ID card [37–39]. An advantage of our study is that we can rely on multiple sources of information including the National ID card (for birth year) and repeated measures of age obtained during the MLSFH 1998–2019. Moreover, measurement errors in years of birth are likely to bias our estimates towards zero, and our estimated famine effects of later-life health probably are thus conservative. Another limitation is that we are not able to identify the biological and detailed social mechanisms through which the famine worked to affect the health of aging Malawians. Future research that can utilize epigenetic or biomarker data for this cohort might be able to identify possible pathways, but such data are not yet available in the MLSFH or any other LIC aging datasets. We can also represent the famine only in terms of the time period when it prevailed in its most severe form, and we lack direct measures of food shortages or localized market prices. These limitations also imply that our analyses probably underestimate of the true effects of the famine.

Overall, despite the above limitations, our analyses provide robust evidence that, in a persistently low-income context such as rural Malawi, in-utero adversities may help adapt to longer-run persistent lifecourse adversities. Importantly, individuals with in-utero exposure to the 1949 Nyasaland famine are documented in this study to have *better* cardiovascular health 70 years after the famine. These effects do not seem to be the result of additional mortality selection. The findings in this paper thus complement the substantial evidence on the Barker (or DOHaD) hypothesis that to date has mostly been informed by higher-income contexts, and has generally shown negative consequences of early-life adversities, especially with regard to cardiovascular health. Our analyses show that these effects may be context-specific, with possibly opposite consequences of in-utero adversities prevailing in persistently low-income and adverse contexts. Our results highlight the need to further study the implications of early-life adversities on later-life health, including the pathways through which they operate, in populations that face sustained poverty

and high levels of morbidity and mortality throughout the lifecourse.

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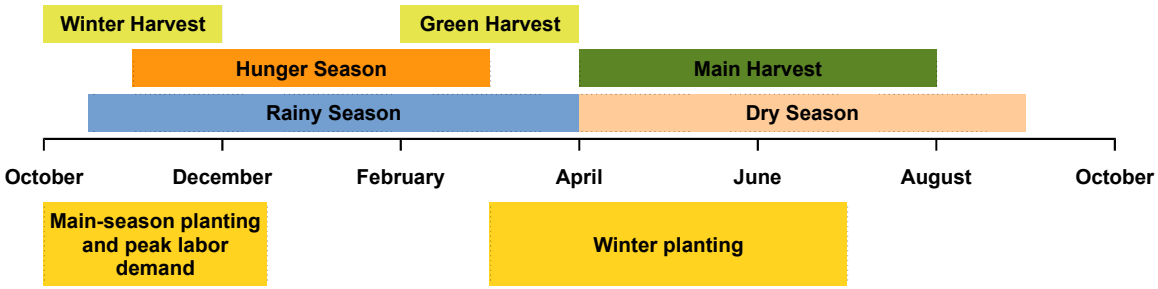
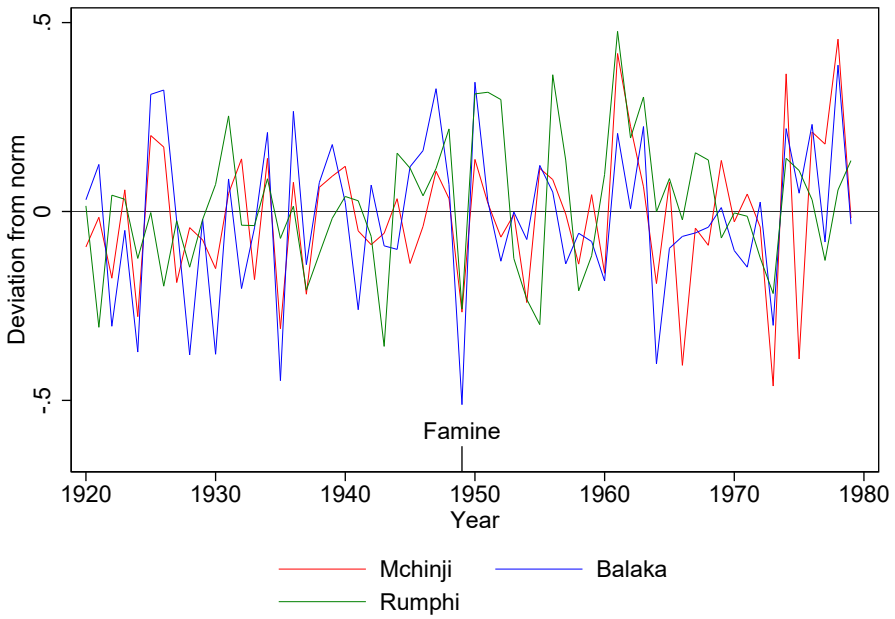
A Supplemental Materials

Figure A.1: Seasonality of harvest and labor demand in Malawi



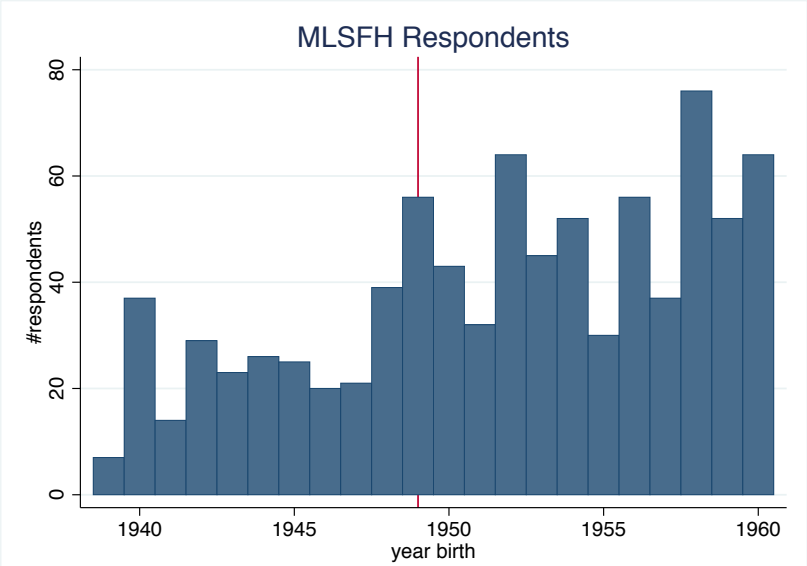
Source: Adapted from USAID & FEWS NET [49]

Figure A.2: Precipitation in three districts of Malawi between 1920 and 1980



Notes: Deviations in precipitation from the norm in the three districts considered in the analysis (Mchinji, Balaka and Rumphi). The norms are district-specific, and the deviations are computed using the difference between the amount of precipitation in a given calendar year (in *ln*) and the norm (also in *ln*). The norm is derived using the mean annual rainfall over the period, excluding the observation for which the deviation in a particular year is calculated. Deviation from norm should be interpreted (approximately) as the % deviation from the mean rainfall, that is, a value of 0.1 roughly corresponds to a 10% positive deviation from the norm. The dataset used to generate this graph is derived from data made available by the Terrestrial Hydrology Research Group at Princeton University. We use gridded monthly data points (0.5 by 0.5 degrees, which corresponds roughly to 55km by 55km) that we match to each district GPS coordinate.The data can be downloaded from the Group's webpage: http://hydrology.princeton.edu/home.php. See Sheffield *et al.* [50] for more details on how the data are constructed, including the bias correction and downscaling methodology they use.

Figure A.3: MLSFH Sample



Notes: The figure shows the number of MLSFH respondents by year of birth. Year of birth is taken from the national registration card. The red line corresponds to 1949, the first year of the famine.

Figure A.4: Census 1998 Age Distribution

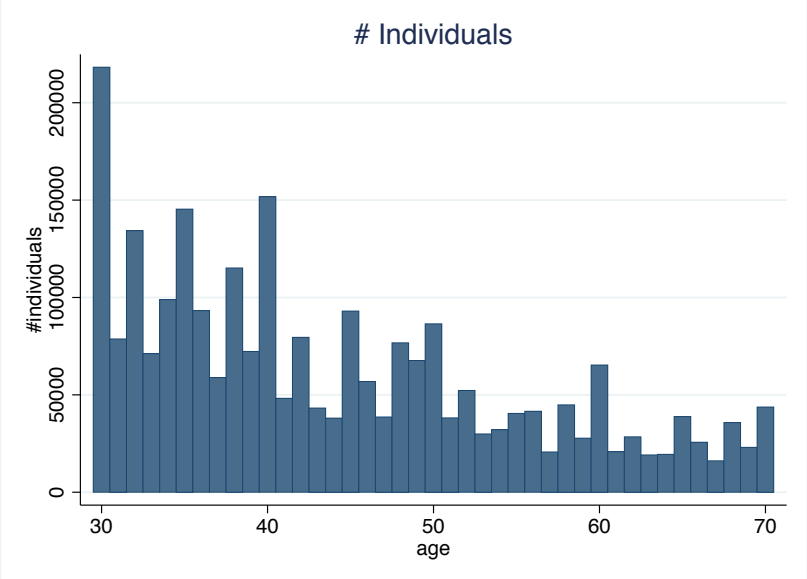
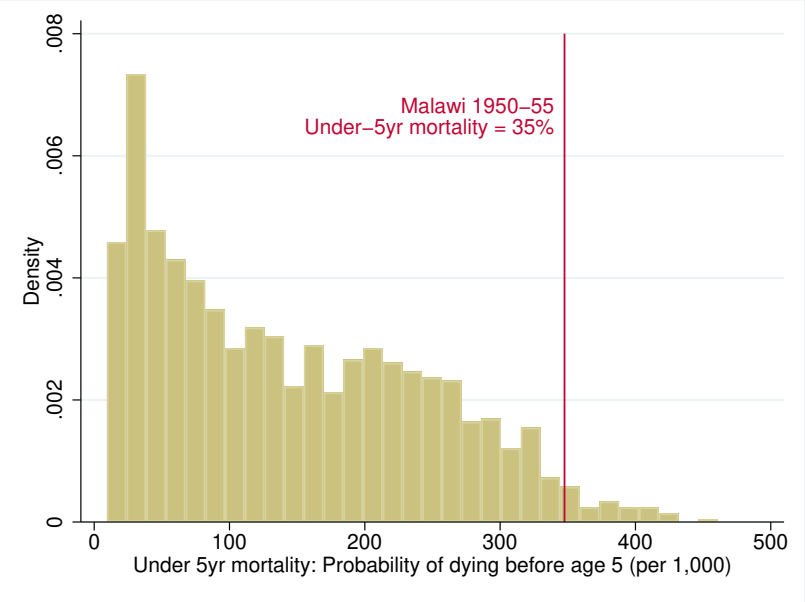


Figure A.5: Probability of dying in the first 5 years of life during 1950–1980 (under-5yr mortality): Malawi in global comparison (histogram)



Notes: Graph depicts a histogram of all under-5 year mortality estimates in the 2019 UN Population Prospects [17] for all 5-year periods 1950–1980 (6 time periods, 273 countries). In 1950–55, under-5 mortality in Malawi is estimated to be around 35%, or at the 98th percentile of the histogram distribution for under-5 mortality during 1950–80. Malawi remained in the top 10% of this distribution until 1970–75.

Table A.1: Descriptive statistics: year of birth sample

	mean	min	max	obs
Demographics				
Year of birth	1951.70	1939.00	1960.00	848
Female	0.53	0.00	1.00	848
Primary	0.69	0.00	1.00	835
Secondary	0.06	0.00	1.00	835
School years	3.50	0.00	12.00	835
Hypertension and diabetes risk				
Systolic	138.87	75.33	218.33	818
Diastolic	84.90	55.67	143.33	818
Hypertension stage 1	0.71	0.00	1.00	818
Hypertension stage 2	0.48	0.00	1.00	818
Blood glucose	4.51	1.90	17.30	641
Diabetes	0.02	0.00	1.00	641
Pre-diabetes	0.07	0.00	1.00	623
2013-2017 change in systolic	0.18	-28.00	25.92	561
2013-2017 change in diastolic	-0.74	-12.50	9.00	561
Anthropometric measures				
BMI	21.94	12.78	42.55	810
Height	159.18	129.00	185.50	822
Weight	55.15	34.00	101.90	822
Waist / hip	0.89	0.70	1.37	823
Obese	0.05	0.00	1.00	810
Cardiovascular disease diagnosis and symptoms				
Stroke diagnosis	0.02	0.00	1.00	642
Angina diagnosis	0.02	0.00	1.00	642
Stroke: N symptoms	0.80	0.00	5.00	642
Stroke: any symptoms	0.42	0.00	1.00	642
Rose angina	0.21	0.00	1.00	642
Observations	848			

Notes: The table shows summary statistics for the variables used in the analysis. The sample is defined as respondents born between 1939 and 1960 according to the national registration card. Hypertension stage 1 is defined as having systolic blood pressure 130 or higher or diastolic blood pressure 80 or higher. Hypertension stage 2 is defined as having systolic blood pressure 140 or higher or diastolic blood pressure 90 or higher. Diabetes is defined as having fasting blood glucose greater than 7 mmol/L or non-fasting greater than 11.1 mmol/L. Obesity is defined as having a BMI greater than 30.

Table A.2: Descriptive statistics: robust-age sample

	mean	min	max	obs
Demographics				
Female	0.53	0.00	1.00	1164
Primary	0.67	0.00	1.00	891
Secondary	0.06	0.00	1.00	891
School years	3.33	0.00	12.00	891
Hypertension and diabetes risk				
Systolic	139.01	75.33	223.67	870
Diastolic	84.99	56.33	135.67	870
Hypertension stage 1	0.71	0.00	1.00	870
Hypertension stage 2	0.48	0.00	1.00	870
Blood glucose	4.50	1.90	17.30	683
Diabetes	0.02	0.00	1.00	683
Pre-diabetes	0.06	0.00	1.00	683
2013-2017 change in systolic	0.06	-28.00	25.92	592
2013-2017 change in diastolic	-0.73	-12.75	9.00	592
Anthropometric measures				
BMI	21.98	12.78	42.55	854
Height	159.02	130.00	185.50	874
Weight	55.10	34.00	101.90	872
Waist / hip	0.88	0.70	1.48	874
Obese	0.05	0.00	1.00	854
Cardiovascular disease diagnosis and symptoms				
Stroke diagnosis	0.02	0.00	1.00	682
Angina diagnosis	0.02	0.00	1.00	682
Stroke: N symptoms	0.80	0.00	5.00	682
Stroke: any symptoms	0.42	0.00	1.00	682
Rose angina	0.21	0.00	1.00	682
Observations	1164			

Notes: The table shows summary statistics for the variables used in the analysis. The sample is defined as those born between 1939 and 1960 according to the robust-age estimate using the median of multiple age measures. Hypertension stage 1 is defined as having systolic blood pressure 130 or higher or diastolic blood pressure 80 or higher. Hypertension stage 2 is defined as having systolic blood pressure 140 or higher or diastolic blood pressure 90 or higher. Diabetes is defined as having fasting blood glucose greater than 7 mmol/L or non-fasting greater than 11.1 mmol/L. Obesity is defined as having a BMI greater than 30.

Table A.3: In-utero famine exposure and long-term health: Controlling for years of schooling

Panel A: Hypertension and Diabetes Risk

	Blood pressure				Hypertension		Diabetes risk	
	Systolic	Diastolic	Δ sys	Δ dias	Stage 1	Stage 2	Glucose	Prediabetes
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Famine exposure	-6.31***	-3.05**	-1.90***	-0.96***	-0.07	-0.01	-0.13**	0.00
	(1.86)	(1.20)	(0.42)	(0.20)	(0.07)	(0.03)	(0.05)	(0.02)
N	817	817	561	561	817	817	638	638

Panel B: Stroke and Heart Disease Symptoms

	Stroke		Heart disease
	Number of symptoms	Any symptoms	Rose angina
	(1)	(2)	(3)
Famine exposure	-0.19**	-0.06	0.04
	(0.08)	(0.08)	(0.04)
N	641	641	641

Panel C: Anthropometrics

	Height	Weight	BMI	Waist/hip ratio	Obese
	(1)	(2)	(3)	(4)	(5)
Famine exposure	-0.76*	-0.48	0.09	0.00	0.02
	(0.44)	(1.25)	(0.51)	(0.00)	(0.01)
N	822	822	810	823	810

Notes: The table shows regression estimates for in-utero exposure to the famine following econometric specification (1) using a sample of MLSFH respondents born between 1939 and 1960. In-utero exposure is defined as being born in 1949 or 1950 according to the National Registration Card. Estimates for control variables are not shown. Standard errors clustered at the age level are reported in parentheses * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A.4: In-utero famine exposure and long-term health: 1929-1970 extended sample

Panel A: Hypertension and Diabetes risk

	Blood pressure				Hypertension		Diabetes risk	
	Systolic (1)	Diastolic (2)	Δ sys (3)	Δ dias (4)	Stage 1 (5)	Stage 2 (6)	Glucose (7)	Prediabetes (8)
Famine exposure	-4.98***	-2.43***	-1.69***	-0.84***	-0.04	-0.01	-0.08*	0.02
	(1.10)	(0.73)	(0.31)	(0.15)	(0.06)	(0.02)	(0.04)	(0.01)
N	1592	1592	951	951	1592	1592	1232	1192

Panel B: Stroke and Heart-Disease symptoms

	Stroke		Heart disease
	Number of symptoms (1)	Any symptoms (2)	Rose angina (3)
Famine exposure	-0.16***	-0.03	0.04
	(0.05)	(0.07)	(0.03)
N	1234	1234	1234

Panel C: Anthropometrics

	Height (1)	Weight (2)	BMI (3)	Waist/hip ratio (4)	Obese (5)
Famine exposure	-0.98***	-0.97	0.09	0.00	0.01
	(0.33)	(1.10)	(0.46)	(0.00)	(0.01)
N	1601	1602	1574	1603	1574

Notes: The table shows regression estimates for in utero exposure to the famine following econometric specification (1) using a sample of MLSFH respondents born between 1929 and 1970. In utero exposure is defined as being born in 1949 or 1950 according to the national registration card. Estimates for control variables are not shown. Standard errors clustered at the year of birth level are reported in parentheses * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A.5: Placebo: In-utero famine exposure in 1951-52

Panel A: Hypertension and Diabetes risk

	Blood pressure				Hypertension		Diabetes risk	
	Systolic (1)	Diastolic (2)	Δ sys (3)	Δ dias (4)	Stage 1 (5)	Stage 2 (6)	Glucose (7)	Prediabetes (8)
Famine Exposure	0.46 (2.78)	-1.01 (1.31)	0.24 (0.92)	0.15 (0.32)	-0.04 (0.03)	-0.02 (0.04)	0.09 (0.09)	0.02* (0.01)
N	890	890	601	601	890	890	693	673

Panel B: Stroke and Heart-Disease symptoms

	Stroke		Heart disease
	Number of symptoms (1)	Any symptoms (2)	Rose angina (3)
Famine exposure	0.05 (0.10)	0.06 (0.06)	-0.06* (0.03)
N	694	694	694

Panel C: Anthropometrics

	Height (1)	Weight (2)	BMI (3)	Waist/hip ratio (4)	Obese (5)
Famine exposure	-0.98** (0.43)	-0.50 (0.96)	-0.23 (0.35)	0.00 (0.01)	-0.01 (0.02)
N	895	895	882	896	882

Notes: The table shows regression estimates for a placebo exercise where in utero exposure is defined as being born in 1951 or 1952 according to the national registration card. The econometric specification follows equation (1). Estimates for control variables are not shown. Standard errors clustered at the year of birth level are reported in parentheses * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A.6: MLSFH Attrition

	(1) Interviewed in 2018–19	(2) Dead in 2018–19	(3) Interviewed in 2018–19
Famine exposure	0.03** (0.01)	-0.04*** (0.01)	-0.01 (0.01)
Sample	All	All	Alive in 2018-19
N	1070	1070	940

Notes: The table shows the results of a test for differential attrition for MLSFH respondents exposed in utero to the famine. We use econometric specification (2). In utero exposure is defined as being born in 1949 or 1950 according to the 1998 or 1987 Census. The sample is limited to respondents born between 1939 and 1960 and who were interviewed in 2008 and/or 2010. The dependent variable in columns (1) and (3) is a dummy for being interviewed in 2018 or 2019. The dependent variable in column (2) is a dummy for being dead in 2018 or 2019. Column (1) looks at attrition from 2008-2010 to 2018-19. Column (2) looks at mortality from 2008-2010 to 2018-19. Column (3) looks at non-mortality attrition from 2008-2010 to 2018-19. Standard errors in parentheses * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$