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THE PRO-POOR ECONOMIC GROWTH AND CHILD MORTALITY IN THE SSA SUB-REGION: EVIDENCE OF A STRUCTURAL BREAK

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Abstract

This study investigates the question of whether the change in economic growth pattern introduced with the Millennium Development Goals has a structural break effect on child mortality across SSA countries, and over time. An unbalanced panel data set for 48 countries classified by the World Bank as constituting SSA was constructed from the World Bank open data catalogue and other sources. The data contain time-varying covariates, including under-five mortality rate, GDP per capita, corruption, political regime, ethnic fragmentation, conflict, public service delivery, and anomalies of temperature and rainfall. The panel structure of the data was exploited using the Arrelano and Bond system GMM to estimate the growth elasticity of mortality. The result from the basic model specification indicates that under-five mortality falls with income per capita with an elasticity of -0.42. In the expanded model, the result was consistent, though with a reduction in the magnitude of growth elasticity (-0.206). Also, the study findings reveal that the growth policy in the post-reform era may not be associated more with pro-poor growth in comparison with the pre-reform era. Since the growth elasticity is not stronger after the introduction of the reforms, we may conclude that a structural break that is not pro-poor has occurred. Indeed, the evidence suggests that mortality may still be largely concentrated among the poor despite the adoption of pro-poor growth policies by countries in the sub-region. An inclusive growth that will increase the number of children having access to basic capabilities, nourishment, and consequently long and satisfying lives is still needed.

Keywords: Pro-Poor Economic growth, Child mortality, Structural break, SSA

INTRODUCTION

It has been argued that about half a million under-five deaths would have been averted in Africa in 1990 alone if the continent’s economic growth were 1.5% higher in the 1980s (Pritchett and Summers, 1996). The Millennium Development Goals (MDGs) advocated a pro-poor growth, which is expected to be facilitated by pro-poor economic policies, hence regarded as pro-MDG economic growth (Sarkar, 2007; p.2). The pro-poor growth is supposed to occur through output expansion in pro-poor sectors such as manufacturing and agriculture. This is expected to increase national and individual-level resources differently from *traditional growth*. Aside from enhancing the incomes of the poor, which will increase their access to food, safe water, sanitation, health, and education, the pro-poor growth policy could be a pathway to improve development indicators for the SSA sub-region. Understanding how this form of growth reduces mortality could, therefore, be vital in attaining the ongoing child health targets of the Sustainable Development Goals (SDGs).

The pro-poor growth advocated resonates with the paradigm of sustainable human development in the *Human Development Report,* which suggests that development must enable all individuals to enlarge their human capabilities to full potential and to put those capabilities to optimum use (UNDP, 1994). It is supposed to occur in labour-intensive sectors with high concentrations of the poor, such as agriculture, education, and health. Growth occurring in pro-poor sectors through output expansion reinforced by strong linkage effects between sectors can increase resources both at the national level for public investment and at the household level for the poor to improve their well-being.

Aside from enhancing the incomes of the poor, the pro-poor growth policy could be a pathway to improve development indicators for the SSA sub-region. Despite the potential of pro-poor growth in influencing health outcomes in developing countries, it remains understudied. Though there is a vast literature on the effect of growth on mortality, there appears to be a paucity of evidence on the relationship between the economic growth advocated in the international development strategy and child mortality in SSA countries, and particularly, Nigeria. This chapter fills the existing empirical gap by addressing two research questions: i) What is the relationship between economic growth and child mortality? ii) Whether there is a structural break in the effect of growth on child mortality following the introduction of the MDGs. Investigating how changes in the economic growth patterns influence child mortality outcomes across countries in the SSA sub-region, and over time, could provide further insight into the debate of whether more attention should be given to socio-economic interventions.

In explaining the dynamics and heterogeneity of the impact of growth on mortality, the roles of non-economic factors could be vital. The endogeneity of economic growth may explain the failure of growth models and policies in Africa (Adika, 2020). For instance, economic outcomes may be influenced by the quality of institutions in a country. More precisely, economic institutions could determine the creation of incentives for investment in physical and human capital and technology, which is needed to spur economic growth (Acemoglu, Johnson, and Robinson, 2005). Invariably, a weak economic institution may provide few incentives for investment, which can constrain the economic prosperity of a society. Similarly, political institutions could determine the constraints as well as the incentives for political groups in societies to make policies that will influence economic outcomes. In effect, a political system with an independent judiciary and regulatory bodies is likely to restrict the ability of the government to engage in rent seeking but will promote accountability to taxpayers as pointed out by North (1990). Hence, I hypothesize that the growth elasticity of child mortality will depend on the efficiency of institutions across countries in the sub-region, and over time.[1]

Additionally, it is plausible to suggest that the economic growth rate could be volatile in a resource-rich and oil-producing country, and this may be attributed to volatility in oil prices in the international market. Such volatility in growth may adversely affect the economic prosperity of a country, as pointed out by Ogwumike and Ogunleye (2008). Perhaps this explains why Nigeria, a country with the biggest economy in Africa, is considered a resource curse. Despite the abundance of natural resources such as oil and solid minerals, the number of people living in poverty appears to be relatively high. According to the Nigeria Living Standard Measurement Survey (NLSS 2018-19), 40.1% of the total population is classified as poor. This translates to over 82.9 million Nigerians who are considered poor by the national standards (NBS, 2020). Other examples of countries in Africa that are considered to be resource curse include Sierra Leone, DR Congo, and Angola. Thus, there is the argument in the literature that resource-rich countries tend to underperform in terms of economic development compared to resource-poor countries (see, for example, Sachs and Warner, 1995). Specifically, it is argued that point-source non-renewable resources such as oil and minerals could adversely affect development. It is further argued that the natural resource curse only occurs in countries with low institutional quality (Mehlum, Moene, and Torvik, 2006). It is likely that the natural resource curse for countries with point natural resources may exist because such resources are more prone to rent seeking and conflicts (Boschini, Pettersson, and Roine, 2007). On the contrary, there are studies suggesting that point natural resources are potential sources of growth and not a curse (Cavalcanti, Mohaddes, and Raissi, 2011; Smith, 2015). Hence, I hypothesize that there is heterogeneity in the effect of growth on mortality between resource-rich and resource-poor

1 In Acemoglu, Johnson, and Robinson (2005, p.395), *good* could be interpreted as efficient.

countries. Finally, I investigate whether there is a structural break in the effect of growth on child mortality across countries in the SSA sub-region, and over time following the introduction of the pro-poor growth strategy.

To investigate the effect of growth on child mortality, I construct a unique panel dataset for 48 SSA countries from 1990 to 2023. The data contain time-varying covariates, including under-five mortality rate, GDP per capita, corruption, political regime, ethnic fragmentation, conflict, public service delivery, and anomalies of temperature and rainfall. There are three potential statistical problems in estimating the effect of income on mortality: endogeneity, simultaneity, and unobserved country-specific effects. Though income per capita is likely to reduce mortality, it is also possible that the reduction in mortality could translate into higher income. Also, there could be the problem of omitted variable bias if there is the presence of a country effect. Further, mortality rates tend to produce high persistence within a country, and this can bias the estimates. To minimize the extent of these biases, I exploit the panel structure of the data using the Arrelano and Bond system GMM to estimate the growth elasticity of mortality. I employ an estimation strategy that relies on instrumental variables generated by the estimator to correct for endogeneity. To attenuate the potential bias from unobserved country effects, I control for country-specific effects. Also, I include year dummies to remove time-related shocks from the errors. Finally, to deal with the problem of high persistence of mortality rates, I include lagged values in the empirical specification. The regressions use instruments based on the second lag of the endogenous variables and adjust for robust standard errors.

Section 2 reviews related literature on the effect of economic growth on child mortality in developing countries and analyses the evidence on pro-and anti-poor growth patterns. In section 3, I discuss the sources of data, variables, and estimation strategy. In Section 4, I present results from a system GMM estimation. Section 5 discusses the findings of the study, and conclusions are drawn in the last section.

LITERATURE REVIEW

In a World Bank report, Wang et, al. (1999) provides quantitative measures of 155 countries performances over the period 1960-90 on six health indicators (under-five mortality rates, total fertility rates, adult mortality rates for males and females, and life expectancy at birth) by gender relative to income and education, and over time. They used random-effects generalized least square regression since it allows for country effects to vary over different periods, to provide a weighted average of between and within country results. The model allows the estimates to be conditioned by the sample in that the country effect has a distribution, and it is not fixed. The result indicates an income elasticity for the under-five mortality rate as -0.38, holding education constant. Compared with the effect of education, income was reported to predict health outcomes better over time. In validating the results, they used hierarchical linear modelling (HLM), a method they argued reduces bias in the standard errors and found that the results are identical.

Pritchett and Summers (1996), using instrumental variables estimation and data at five-year intervals over the period of 1960 up to 1985, across 58 developing countries, find a causal and structural positive relationship between income and health. Their motivation to use five-year instead of annual differences hinges on data quality and the need to reduce measurement error and smoothen short-term fluctuations as pointed out by Bhalotra (2008). The results of IV estimation using instruments including terms of trade shocks, investment ratios, price level distortion, and black-market premium indicate a growth elasticity of -0.29. The result implies that if GDP per capita were 1% higher in developing countries, about 50,000 child deaths are likely to be averted annually. They estimated the global health consequences of various growth paths of income, which were 2.5 lower, on average, for Africa and Latin America regions compared to other regions of the world. The result suggests that over 400,000 child deaths would have been averted across Africa and Latin America in 1990 alone, had the two regions experienced the same growth in the 1980s as they did in the period from 1960 through 1980.

A closely related paper with a significant contribution to the present study is by Bhalotra (2008). The Bhalotra paper investigates to what extent economic growth is likely to reduce mortality rates across India's 15 states over the period of 1970-1998. She estimates growth elasticities before and after a 1980 economic reform and uses the growth elasticity of the post-reform period to determine the required growth rate to achieve the MDG target. The results indicate that economic growth reduces child mortality with an unconditional growth elasticity of -0.7, and that growth in the post-reform era is less effective in reducing child mortality. This study extends the work of Bhalotra in a number of ways. It uses a panel data set of 48 SSA countries, over a more recent period (1990-2018), and with specific reference to the growth reforms introduced with the MDGs across the SSA sub-region. Also, in contrast to the Bhalotra paper, where the growth elasticity of mortality was effectively estimated using the least squares dummy variable method, as I will show, this study exploits the panel structure of the data using the Arrelano and Bond system GMM to estimate the relationship. The estimator's ability to generate GMM-style instruments to correct for endogeneity concerns in the model makes it appealing. Also, this study uses a unique set of time-varying confounding factors, including institution, ethnic fractionalization, climate, and conflict. It considers these factors to be relevant in estimating the growth elasticity for the study area. Like the Bhalotra paper, this study includes a time effect in the model to allow for the identification of the distinct effect of economic growth from other time-related shocks. Due to data unavailability, the study does not investigate income distribution effects on child mortality inequalities. This limits the understanding of the effect of changes in income inequality on equity in child mortality outcomes.

Other studies that have estimated the relationship between economic growth and child health outcomes for developing countries include Stuckler, Basu, and McKee (2010) and Nishiyama (2011). Using data from 164 countries, Stuckler, Basu, and McKee (2010) investigate whether differential progress in attaining the MDGs child health goals is related to GDP per capita. The effect of growth alongside other covariates was estimated on the measured distances of countries from achieving the child mortality target, unmet MDG progress. The results indicate that a 10% increase in GDP per capita is associated with 1.8% and 1.64% increases in progress towards attaining the infant and under-five mortality targets, respectively. Nishiyama (2011), using panel data from 83 developing countries over a period of 40 years, shows that economic growth broadly reduces infant mortality, and the impact could be asymmetrical during periods of booms (weak and mixed effect on mortality reduction) and slumps (strong adverse impact).

The above review suggests that an increase in GDP per capita will improve child health outcomes. However, it is possible that there could be an increase in GDP per capita without corresponding child health gains if the increase in income is disproportionately accruing to the top 1% of the income distribution, as pointed out by Pritchett and Summers (1996). This introduces the debate in the economic growth literature about the extent to which the poor benefit from growth. There are two extremes to the debate. At one end of the spectrum is the view that liberal economic policies resulting in growth can raise the income of the general population proportionately: the equiproportionate proposition. At the other extreme is the view that sharp increases in inequalities are inherent in rapid economic growth, and this tends to undermine the potential benefits of growth for the poor or even offset it entirely: the anti-poor bias proposition. An exhaustive review of related literature along these two broad extremes has been done elsewhere (Atsiya and Atsiya, 2024)

METHODOLOGY

Model and estimation strategy

In line with Bhalotra (2008), I specify the child mortality outcome as a function of GDP per capita and a vector of country-level controls. The Bhalotra specification includes demographic variables such as gender, religion, ethnicity, age of mother, maternal, and paternal education. These were measured at the child or family level but aggregated up to the state level. Unlike the Bhalotra paper, where the relationship between growth and mortality was effectively estimated using the least squares dummy variables method, I investigate persistence in child mortality using an alternative technique. I argue that child mortality, the outcome variable, GDP per capita and other control variables can be persistent with current levels depending on their past realisations, hence a dynamic model may better reflect the data-generating process. I specify a dynamic panel data model as:

$$lnU5m_{i,t} = \Phi\ lnU5m_{i,t-1} + \propto lnGDPpc_{i,t} + \gamma lnGDPpc_{i,t-1} + X_{i,t}\beta + \delta_t + \mu_t + u_{i,t}, ---- (2.1)$$

The specification suggests that under-five mortality ($lnU5m_{i,t}$) in a country i, can be explained by its past level ($lnU5m_{i,t-1}$), by GDP per capita ($lnGDPpc_{i,t}$) as well as its lagged values ($lnGDPpc_{i,t-1}$), and a vector of time-varying factors ($X_{i,t}$) including education (as well as its lagged values), corruption, regime type, conflict, ethnic fractionization, public health programmes, and anomalies of weather variables (temperature and rainfall). As earlier mentioned, while there is some form of persistence in mortality with current under-five mortality correlating with past levels, in the case of income, endogeneity due to reverse causality is possible since health may influence productivity and consequently, socio-economic condition of households, which in turn could impact health outcomes. This introduces the problem of dynamic endogeneity. Therefore, I include lags of under-five mortality and GDP per capita as instruments. Lags as instrumental variables can have the desirable properties of being correlated with the regressors, yet uncorrelated with the error terms. Also, in the model, δ_t denotes a full set of country dummies and μ_t denotes a full set of time effects capturing common shocks to mortality across all countries and $u_{i,t}$ is an error term capturing all other omitted factors, with $E(\mu_t) = 0$ for all i and t.

In identifying the effect of income on under-five mortality, I employ a dynamic panel estimator, the system GMM developed by Arrelano (1995) and Blundell & Bond (1998). The problem of potential dynamic endogeneity in the model motivates the use of an estimation strategy that controls for lagged values of the variables of interest directly. That is, past realisations are used as valid instrumental variables to correct for potential endogeneity issues. The estimator's ability to generate GMM-style instruments to correct for endogeneity makes it a plausible candidate amongst dynamic panel models, especially when it is difficult to find appropriate time-varying geographical or historical variables that could be used to instrument for income. Also, with multiple instruments in the GMM procedure, it is possible to investigate whether the assumption of serial correlation in $u_{i,t}$ can be rejected, and to test for overidentifying restrictions. I only report results from models that pass the specification test (the Hasen J-test) and the serial correlation test. In all specifications, I use robust standard errors and a double lag to instrument for all variables with potential endogeneity concerns.

In the modelling, I include year dummies to remove time-related shocks from the errors. In implementing the estimator, there is the likelihood of proliferation of instruments as the number of instruments tends to increase exponentially with the number of time periods. This may result in the overfitting of endogenous variables and increases the likelihood of false positive results (Heid, Langer, and Larch, 2011). I control for such potential bias by estimating the model with a collapsed instrument matrix. More so, with the GMM procedure, it is possible to use instruments outside the baseline specification as excluded instruments for the endogenous independent variable. It was difficult to identify additional valid instruments. The results are sensitive to the addition of excluded instruments.

In addition, I control for a unique set of time-varying confounding factors, including corruption, political regime, ethnic fractionalization, conflict, and climate-related variables, in order to reduce the problem of omitted/unobserved variable biases. These factors are likely to influence the success of growth polices in the SSA sub-region. I rely on the fixed effects component of the GMM to treat all time-invariant country-specific characteristics that are likely to bias the income parameter estimate. However, likely, there might still be some unobserved effects that are both country-specific and time-variant. For instance, there might be some country-specific policies on inequality that could determine the effect of income per head on health outcomes. Due to data unavailability, I did not control for inequality or policy on redistribution, but it is unclear whether this will significantly affect our results, given the homogeneity of institutional structures across countries in our sample. In any case, the GMM framework is known to overcome estimation problems introduced by unobserved panel heterogeneity, dynamic endogeneity, and simultaneity and produce unbiased and consistent estimates under mild assumptions (Schultz, Tan, and Walsh, 2010).

In identifying the structural break effect of economic growth on child mortality post-pro-poor growth reforms, I first estimate the relationship between economic growth and child mortality. I address the question of whether changes in GDP per capita influence under-five mortality outcomes across countries in the SSA sub-region over time. I find a statistically significant relationship, and this allows for the assessment of the impact of growth after the introduction of the growth reforms. I argued that, as a pattern of growth that is inclusive, the pro-poor growth is likely to allow a greater proportion of children access to basic nourishment and health care. It is supposed to occur in sectors with a high concentration of the poor. Hence, policies and programmes that expand output in these sectors will increase resources both at the national level for public investment and at the household level for the poor to improve their well-being. A priori, I expect a stronger relationship between economic growth and child mortality post-pro-poor growth reforms. I use changes in GDP per capita to proxy the impact of changes in growth policy before and after its introduction with the MDGs. Due to data unavailability, I use the GDP per capita series aggregated at the country level, though this limits our understanding of how changes in income inequality due to the reform have impacted equity in child health outcomes. I interpret the study findings in the context of this limitation.

Further, I split the data at the year 2003, a potential structural break-point, and estimate the growth elasticities in the different sample periods. I allow a 3-year lag period after the introduction of the campaign in 2000 for the implementation of policies and agreements by development agencies. As in Bhalotra (2008), I compare the growth elasticities for the two sample periods and test whether they differ statistically. If growth elasticity is stronger after the introduction of the growth reforms, I may conclude that a structural break that is pro-poor has occurred with the introduction of the intervention. A problem with using the GMM approach is the inability to statistically test whether the growth elasticities differ between the sample periods. A classical test for structural break is the Chow test (Chow, 1960).[2] In implementing the Chow test, I naively re-estimated the model using the Least Squares Dummy Variable (LSDV) estimator. Using the above procedure, I conduct a series of heterogeneous analyses. For instance, I test the hypothesis whether the structural break effect identified is different for resource-rich countries. This hypothesis is based on the argument that it will matter whether a country is resource-rich in assessing the impact of growth on other economic outcomes.

Data and variables description

The World Bank open data catalogue is the main source for this study. The dataset comes from demographic health surveys and censuses and is available at annual frequencies. Other data sources used include the Worldwide Governance Indicators (WGI) project database, Centre for Systemic Peace/Integrated Network for Societal Conflict Research data resources, the University of East Anglia Climatic Research Unit database, the historical index of ethnic fractionalization dataset, and the Uppsala Conflict Data Programme/Peace Research Institute Oslo dataset version 20.1. In brief, I construct an unbalanced panel data set for 48 countries classified by the World Bank as constituting SSA. The panel data which covers a time span of 33 years (1990-2023), is meant to account for the period prior to, and during, the introduction of the pro-poor growth policy in the SSA sub-region. The period of analysis allows for the assessment of the impact of the pro-poor economic growth reforms on child mortality.

2 The classical test for structural break as in Chow (1960) was used. In applying the Chow test, the sample is split around the break-point into two sub-periods and the parameters for each sub-period are estimated. The F-test statistics is used to test the equality of the set of parameters.

I specify child mortality as a function of GDP per capita and a vector of covariates including education, corruption, weather variables, regime type, conflict, and public health programmes (sanitation, safe water provision, and immunization). While the *under-five mortality rate* is defined as the probability per 1,000 that a new-born baby will die before reaching the age of five, *infant mortality rate* is the number of infants dying before reaching one year of age, per 1,000 live births in a given year. For income, I use *GDP per capita* based on Purchasing Power Parity (PPP) in constant 2011 international dollars. The use of GDP per capita PPP makes it possible to compare the effect of growth on mortality on an international scale whilst controlling for inflation. It is expected that the value of a dollar in Ghana should be the same as in South Africa. The basic intuition is that accelerated pro-poor economic growth through output expansion in pro-poor sectors will increase national and individual resources, reduce inequality, and improve on household outcomes. To assess the impact of pro-poor growth on mortality, it is vital that the analysis captures changes in child mortality inequalities by income quintiles.

The effect of maternal *education* is measured using the primary school completion rates for females. In the World Bank dataset, it is measured as the number of new entrants in the last grade of primary education, regardless of age, divided by the population at the entrance age for the last grade of primary education. In line with the literature, I argue that the probability of a mother without schooling losing a child is higher compared to a mother with schooling (Cadwell, 1979; UNESCO, 2010; Summers, 1992; Semba, 2008). To control for the role of institutions, in line with the literature, I use the *control of corruption* as a proxy (see Mauro, 1995). As opined by Vian (2008), a strong control over corruption, especially in developing countries, will influence the provision of health care through the construction and rehabilitation of health facilities, control of quality health products, medical research, purchase and supply of health equipment, and drugs. The corruption index constructed by the World Bank Worldwide Governance Indicators (WGIs) was used. It has a value of -2.5 to 2.5: the higher value signifying a stronger institution. Further, I control for regime type by using a constructed index that measures institutionalised democracy and autocracy. It is a regime score that ranges from +10 (full democracy) to -10 (full autocracy). I expect a higher regime score to reduce child mortality as documented in the literature (Ruger, 2005; Safaei, 2006; Bollyky et al, 2019).

In controlling for *weather variables*, I use annual series on rainfall and temperature obtained from the University of East Anglia Climatic Research Unit (UEA-CRU, Harris, Jones, and Osborn, 2020). Specifically, I use annual temperature and rainfall anomalies in line with the literature (see, for example, Marchiori, Maystadt, and Schumacher, 2012; Henry and Dos Santos, 2013; Maystadt and Ecker, 2014). In line with the literature, I expect deficits in rainfall and high temperatures, especially in agrarian subsistence societies, to increase the risk of childhood mortality through malnutrition and changes in disease environments (Kudamatsu, Persson, and Stromberg, 2012; Baker and Anttila-Hughes, 2020).

I controlled for *Ethnic Fractionalization,* the probability that two persons drawn at random from a country's population will belong to the same ethnolinguistic group (0, where all individuals are members of the same ethnic group, to 1, where everyone belongs to his/her own ethnic group). In line with Wimmer (2015), I argue that countries with a more fractionalized ethnic structure may be less able or willing to provide their citizens with public goods such as education, physical infrastructure, and health. This is because different ethnic groups are likely to have divergent preferences, values, and beliefs, and this might increase collective action and coordination problems with lower levels of public goods provision, consequently (Alesina, Baqir, and Easterly, 1999).

For the conflict variable, I use an indicator of a conflict year and the intensity of conflict from the Uppsala Conflict Data Programme/Georeferenced Events Dataset (UCDP/GED). The dataset provides a yearly series of armed conflicts. I argued that conflict has a persistent adverse effect on children over their life course and on subsequent generations given birth to after the conflict has ended, as suggested by Wagner et al. (2018). Hence, in line with O'Hare and Southall (2007), I expect countries with recent conflicts to have a higher under-five mortality rate compared to countries without recent conflicts (O'Hare and Southall, 2007).

Finally, I control for the influence of public health programmes such as the provision of safe drinking water, sanitation and immunisation. For *safe water*, I use access to safe water, measured as the percentage of a country's population using an improved drinking water source. In the case of *sanitation*, I use the percentage of the population of a country using improved sanitation facilities. I control for *immunisation* because it is identified as the most cost-effective health intervention and a crucial determinant in child survival efforts in developing countries (Nelson 2004). It is argued that major causes of under-five mortality are preventable by vaccination. Immunisation in this study is measured using the diphtheria, pertussis (or whooping cough), and tetanus (DPT) vaccination coverage rate.

RESULTS

Descriptive Statistics

Table 1 presents descriptive statistics of the variables in the model over the study period. To motivate descriptive findings, the study period is divided into two eras: pre-pro-growth policy and post-pro-poor growth policy. From Table 1, there appears to have been progress in reducing child mortality over time. To be specific, the average under-five mortality rate falls from 133 to 71 deaths per 1,000 live births before and after the introduction of the pro-poor growth policy. I investigate the drivers of this evidence of decline in mortality subsequently. Additionally, as shown in Table 1, there has been an increase in the average GDP per capita (GDPpc) from $3,789 in 2000 to $5,392 in 2015.

Table 1: Summary statistics for SSA sub-region: pre-and post-pro-poor growth policy periods.

	Pre-Pro-Poor Growth			Post-Pro-Poor Growth		
Variable	Mean	Min	Max	Mean	Min	Max
U5MR	133.19	13.70	234.00	71.31	14.30	130.50
	(50.48)			(29.58)		
GDPpc	3789.83	630.68	19011.92	5392.10	852.75	28313.60
	(4331)			(6513.98)		
Female Education	29.23	4.70	85.50	49.50	13.86	108.75
	(25.26)			(25.79)		
Ethnic Fragmentation	0.67	0.057	0.889	0.679	0.054	0.889
	(0.2144)			(0.205)		
Rainfall	1082.09	66.6	2834.9	1108.21	78.1	2808.6
	(612.63)			(609.05)		
Temperature	24.52	11.6	29.1	24.85	11.6	29.4
	(3.28)			(3.30)		
Immunization	61.84	19.00	98.00	79.93	16.00	98.00
	(21.49)			(18.54)		
Sanitation	26.04	3.40	94.14	35.14	6.86	100.00
	(21.25)			(22.41)		
Safe water	55.96	18.70	99.28	65.88	38.98	99.87
	(19.14)			(15.36)		
Conflict	0.386	0	2	0.204	0	2
	(0.689)			(0.461)		
Political regime	1.07	-6	10	2.833	-7	10
	(5.0286)			(4.873)		
Corruption	-0.574	-1.83	1.2	-0.541	-1.72	1.14
	(0.638)			(0.656)		

Source: World Bank Poverty Data (2024)

In addition to income, changes in certain covariates are associated with child mortality. For instance, education, the means of female primary school completion, has improved by 20% between 2000 and 2015. Similarly, there have been increases in the percentages of people having access to safe water (10%), improved sanitation (9%), and immunisation against DPT

(18%). Also, on average, the sub-region appears to be experiencing improvement in the political regime scores, which rose from 1.07 before the introduction of the pro-poor growth policy to 2.83 in post post-pro-poor growth policy era. However, ethnic diversity across the sub-region seems to be high and stable over time. The mean value of the ethnic fractionalization index, measured between 0 and 1, has remained stable across the periods. Finally, it is unclear if any SSA countries achieved a sustained 7% annual real GDP growth rate target over the study periods. It appears that only four countries, Equatorial Guinea (13%), Cape Verde (12%), Comoros (8%), and Mauritius (7%) attained a growth rate above 7% in 2015. While 6 countries had a zero growth rate, 17 countries were experiencing negative growth in 2023.

Regression results

Economic growth and under-five mortality

First, I answer the question of whether changes in GDP per capita influence child mortality outcomes across countries in the SSA sub-region, and over time. From the results, I find that economic growth is negatively associated with under-five mortality rate in the SSA region, but the statistical significance depends on the number of controls. In the second phase, I test the hypothesis whether the economic growth reforms introduced are pro-poor. I failed to reject the null hypothesis that the growth elasticities in the two sample periods are equal. That is, there is evidence of growth in the post-policy era not being more pro-poor compared to the pre-policy era.

In the GMM estimation results, tests for the validity of the overidentifying restrictions are carried out, and the p-values for the null hypotheses are reported on the rows for the Hansen J-test (instrument validity test). In all specifications, I accepted the null hypothesis, indicating that the models are well-specified. Additionally, the first-order and second-order tests for autocorrelated disturbances are carried out with reported p-values on the rows for AR(1) and AR(2), respectively. Our results suggest that there is no evidence of first and second-order autocorrelation. That is, the specified test statistics suggest that the models are properly specified. Finally, the GMM estimations use standard errors that are robust to autocorrelation and heteroskedasticity of unknown form and treat the lagged under-five mortality variable as pre-determined. All GMM estimations are carried out using the xtbond2 package in Stata/MP 17.0 and in line with the procedure in Roodman (2009).

In model 1 (Table 2), I estimate the effect of economic growth on under-five mortality, controlling for income and education in the baseline specification. This is in line with Wang et al. (1999). In their study, under-five mortality is expressed as a function of real GDP per capita, education, and time. Results from the baseline specification suggest that under-five mortality falls with income with an elasticity of -0.42. That is, a random SSA country at the sample mean would avert 42 under-five deaths per 1000 live births if income per head were raised by 1%. Also, the result in model 1 indicates that improving female education will reduce the under-five mortality rate (-0.0462). Maternal education will likely reduce mortality by influencing fertility schedule, response to illness and decision-making during illness, reception and utilisation of health information, nutrition, and empowerment, a factor that changes the traditional balance of familial relationships (Caldwell 1979; Gakidou, Cowling, Lozano, Murray, 2010). Next, I control for time-varying confounding factors sequentially in models 3-5. When I control for corruption and political regime in model 2, the income elasticity (-0.206) and education (-0.036) estimates are robust, though with reductions in their effect sizes. Also, the result suggests that the political regime score and the corruption control index are negatively associated with under-five mortality. Precisely, while a unit increase in the political regime score will reduce under-five mortality by -0.075, increasing the intensity of control over corruption by a unit will reduce mortality by -0.266, respectively.

In models 3 and 4, I test the hypotheses whether the quality of institutions matters in the effect of growth on under-five mortality. I argue that the quality of institutions may explain the dynamics and heterogeneity of the impact of growth on child health. The results show that increases in the political regime score and control over corruption index may reduce mortality through economic growth, but in a statistically non-significant relationship.

Table 2: Economic growth and child mortality: A two-step system GMM estimation

Variables	(1)	(2)	(3)	(4)	(5)
L1_lnU5MR	1.001***	0.833***	0.992	0.980	0.342
	(0.0437)	(0.0633)			(0.683)
lnGDPpc	-0.420**	-0.206***	-0.208*	-0.060	-0.037
	(0.209)	(0.093)	(0.124)	(0.550)	(0.871)
L1_lnGDPpc	0.257	-1.280**	-0.229	-0.818	-0.523
	(0.363)	(0.616)	(0.399)	(0.529)	(1.028)
Education	-0.0462**	-0.036**	-0.0128	-0.00514	-0.0620
	(0.00562)	(0.0147)	(0.0141)	(0.00845)	(0.0704)
L1_Education	-0.00237	-0.0137**	-0.00919	-0.00510	0.0718
	(0.00591)	(0.00557)	(0.00892)	(0.00798)	(0.0782)
Pol_regime		-0.0747**		-0.0177	-0.0547
		(0.0318)			(0.0729)
Corruption		-0.266**	-0.0279		
		(0.125)	(0.0808)		

Table 2…

GDP*Pol_regime			-0.00294		
			(0.00365)		
GDP*Corruption				-0.00945	
				(0.0137)	
Sanitation					-0.0125
					(0.00760)
Safe Water					-0.0238
					(0.0221)
Immunization					-0.00563
					(0.00742)
Rainfall					0.166
					(0.174)
Ethnic Fraction					3.931
					(4.334)
Conflict					0.137*
					(0.0731)
Temperature					0.0328
					(0.0443)
Constant	0.735	6.042	3.0255	1.923	0.3239
	(1.911)	(3.997)	(2.118)	(1.899)	(0.5187)
Observations	377	332	332	332	101
Number of country2	33	30	30	30	25
AR(1)	0.525	0.0867	0.105	0.337	0.455
AR(2)	0.521	0.477	0.417	0.514	0.502
Spec Test (p-value)					
Hansen J-test	0.9599	0.999	0.9877	0.464	0.9997
Number of Instruments	95	93	93	93	61

Standard errors in parentheses; *** p<0.01, ** p<0.05, * p<0.1

Notes: Results in all columns use the GMM of Arellano and Bond (1991), with robust standard errors. The dependent variable is under-five mortality. Year dummies are included in all regressions. The AR(1 &2) tests and the Hansen J test indicate that there is no serial correlation, and the overidentifying restrictions are not rejected.

In theory, I expect *good* economic institutions through their ability to create incentives for investment in physical and human capital and technology, to spur economic growth (Acemoglu, Johnson, and Robinson, 2005). Similarly, I expect a political system with incentives to reduce rent-seeking activities by the government while promoting accountability to taxpayers, to reduce child mortality through the income pathway. However, from the results in models 3 and 4, I fail to reject the hypothesis that, on average, the quality of institutions may not matter in influencing economic growth and consequently child mortality outcomes.

In the final model of Table 2, the result suggests that the statistical significance of the relationship between income and mortality is dependent on the number of controls introduced. That is, income elasticity loses its statistical significance with additional controls. I note the relationship between conflict and the under-five mortality rate. The result in model 5 suggests that in any conflict year, under-five mortality will increase by 0.137. This effect could persist over the life-course of children as suggested by Wagner et al. (2018), and may be operational, in a gendered pattern, even amongst the unborn as pointed out by Dagnelie, De Luca, and Maystadt (2014). Also, I note that public health programmes such as safe water provision, improved sanitation and immunisation could reduce child mortality. The finding that child mortality is strongly associated with access to safe water is supported in the literature (Shi, 2000). Access to safe water reduces the exposure of children to diarrhoea and other communicable diseases risk factors. Increasing access to safe water is expected to stimulate the simple but effective act of hand washing, and this has been shown to reduce the transmission of diarrhoea by one-third (WHO/UNICEF, 2000). Also, immunisation, proxy by the DPT immunisation coverage rate, may reduce child mortality in the region. This is because the DPT vaccine, which is a class of combination vaccines, is known to be effective against three infectious diseases (diphtheria, pertussis, and tetanus), which are major causes in childhood. This finding points to a critical role that the Expanded Program on Immunisation (EPI) in countries within the sub-region can provide in reducing child mortality.

Finally, the results show that the relationship between climate variables—temperature and rainfall anomalies—and mortality is positive, though statistically insignificant. In the literature, this relationship can be ambiguous. While high temperature is known to increase mortality risk in children (see, for example, Kudamatsu, Persson, and Stromberg, 2012; Baker and Anttila-Hughes, 2020), infant mortality is reported to reduce with high temperature but increases with low temperature in rural Bangladesh (Lindeboom, Alam, Begum, Streatfield, 2012; Hashizume, Wagatsuma, Hayashi, Saha, Streatfield, Yunusy, 2009). However, the relationship between the climate variables and mortality may be more direct in a micro-level analysis, with spatial heterogeneity in effects. In contrast to La Porta et al. (1999), I did not find a strong association between ethnic fractionalization and child mortality. But the result suggests that the more fractionalized a country is, the probability of under-five survival declines. This is because, with more different ethnic groups having divergent preferences, values, and beliefs, chances for collective action reduce and coordination problems increase and consequently, lower levels of public goods provision (Alesina, Baqir, and Easterly, 1999).

Structural break in the effect of economic growth on child mortality

I have established elsewhere that the pattern of economic growth advocated in the development strategy of the MDGs is pro-poor growth, and it is supposed to be facilitated by pro-poor economic reform policies. Such a pattern of growth is expected to be sustainable, inclusive, and equitable. It is therefore argued that this will result in income poverty reduction and a consequent increase in the consumption of goods and services, promoting well-being. Hence, I expect a stronger relationship between economic growth and child mortality post-growth reforms. To this end, I test the hypothesis that the pro-poor growth elasticity is greater compared to the traditional growth elasticity. To achieve this, the sample period is truncated into pre-pro-poor growth reform period (1990-2003) and post-pro-poor growth reform era (2004-2023) and the growth elasticities are estimated. I have allowed a 3-year lag period after the introduction of the campaign in 2000 for the implementation of policies and agreements by development agencies. As in Bhalotra (2006), I compare the growth elasticities for the two sample periods and test whether they differ statistically. If growth elasticity is stronger after the introduction of the MDGs, then we may conclude that a structural break that is pro-poor has occurred.

I achieve the above by first estimating a GMM model. The results reported in columns 1 and 2 (Table 3) show conditional income elasticities of the pre-and post-reform era to be -0.292 and 0.124, respectively. Tentatively, I reject the null hypothesis that the income elasticities do not differ in both eras. The income elasticity is greater in magnitude (and significant) in the pre-reform era compared to the income elasticity post-reform. To statistically test for the equality of the growth elasticity parameters in both periods, I employ the Chow test. In implementing the Chow test, I naively re-estimated the model using the Least Squares Dummy Variable (LSDV) estimator and the results are presented in columns 3 and 4 (Table 3). The Chow test requires the residual sums of squares (RSS) from both the sub-samples and the full sample, respectively.

Table 3: Structural break in the effect of economic growth on child mortality

	(1)	(2)	(3)	(4)
	Two-System AB estimation		LSDV	
VARIABLES	PreMDGs	PostMDGs	PreMDGs	PreMDGs
L1_lnU5MR	1.091***	0.945***	0.987***	0.985***
	(0.135)	(0.0724)	(0.0475)	(0.0176)
lnGDPpc	-0.292**	-0.124	-0.114**	0.0182
	(0.118)	(0.554)	(0.0492)	(0.0440)
L1_lnGDPpc	-0.0526	0.0161	-0.00776	0.00756
	(0.942)	(0.513)	(0.0577)	(0.0428)
Literacy	-0.0290	-0.00240	-0.00313**	-0.000365
	(0.0377)	(0.00329)	(0.00144)	(0.000674)
L1_Literacy	0.00703	-0.000265	-0.000150	0.00127*
	(0.00803)	(0.00306)	(0.00122)	(0.000746)
Sanitation	-0.0278	-0.00129	-0.00479**	-0.000236
	(0.0409)	(0.00238)	(0.00213)	(0.000768)
Safe_water	0.00439	0.00256	-0.00536**	-0.00179**
	(0.00746)	(0.00523)	(0.00229)	(0.000748)
Immunization	-0.00106	-0.000527	-0.000296	-0.000109
	(0.00333)	(0.00110)	(0.000263)	(0.000264)
Rainfall	-0.0652	-0.00280	-0.00322	0.00120
	(0.0842)	(0.0108)	(0.00350)	(0.00305)
Ethnic	0.792	0.327	0.703	0.0224
	(1.433)	(0.354)	(1.082)	(0.336)
Conflict	0.00410	0.0245	0.00715	0.119*
	(0.223)	(0.0264)	(0.00663)	(0.0666)
Temperature	-0.0162	0.00722	0.00598	0.00682*
	(0.0459)	(0.0155)	(0.00453)	(0.00359)
Pol_regime	-0.0419	-0.00722	-0.000158	-0.00129
	(0.0565)	(0.0112)	(0.00157)	(0.00131)
Constant	0.305	0.637	4.513	6.060
	(0.518)	(0.906)	(6.155)	(4.888)
Observations	81	200	81	200
R-squared			0.705	0.892
Number of country2	25	30		
AR(1)	0.346	0.667		
AR(2)	0.587	0.363		
Spec test (p-value)				
Hansen J-test	0.201	0.316		
Number of Instruments	58	84		

Standard errors in parentheses; *** p<0.01, ** p<0.05, * p<0.1

Notes: Results in all columns use the GMM of Arellano and Bond (1991), with robust standard errors. The dependent variable is under-five mortality. Year dummies are included in all regressions. The AR(1 &2) tests and the Hansen J test indicate that there is no serial correlation, and the overidentifying restrictions are not rejected.

The GMM estimator, by default, does not return the RSS.[3] By applying the Chow test, I can reject the null hypothesis of equality of sub-sample growth elasticities at 5% level of significance. This finding resonates with the Bhalotra (2006) study. It implies that the growth policy in the post-reform era may not be associated more with pro-poor growth in comparison with the pre-reform era. Indeed, when I compared the sizes of the growth elasticities in the different sub-sample periods, it is likely that the growth in the post-reform era is anti-poor.

Finally, I investigate the above finding further by testing the hypothesis whether there is heterogeneity in the effect of growth on mortality between resource-rich and resource-poor countries. I argued that economic growth in resource-rich countries can be volatile, which may adversely affect a country's economic prosperity (Ogwumike and Ogunleye, 2008). Thus, resource-rich countries are likely to exhibit different economic development outcomes compared to resource-poor countries. To test this hypothesis, first, I split the sample using the presence of natural endowments into resource-rich and resource-poor countries and estimate the income elasticities. The results in columns 1 and 4 (Table 4) show that the impact of growth on mortality was only significant amongst resource-poor countries (0.0488). This is in line with the argument in the literature that resource-rich countries tend to underperform in terms of economic development compared to resource-poor countries (see, for example, Sachs and Warner, 1995). This evidence further supports the natural resource curse hypothesis that points to natural resource-led growth being prone to rent seeking and conflicts (Boschini, Pettersson, and Roine, 2007).

Table 4: Structural break in the effect of economic growth: Resource-rich vs. resource-poor countries

	(1)	(2)	(3)	(4)	(5)	(6)
		Resource-Rich			Resource-Poor	
VARIABLES	Full	PreMDGs	PostMDGs	Full	PreMDGs	PostMDGs
L1_lnU5MR	0.977***	0.916***	1.052***	0.891***	0.756***	0.928***
	(0.0336)	(0.0730)	(0.0477)	(0.0298)	(0.0656)	(0.0395)
lnGDPpc	-0.00209	-0.0173	-0.119	-0.0488**	-0.122*	-0.0994
	(0.0577)	(0.0353)	(0.0887)	(0.0020)	(0.0621)	(0.0811)
L1_lnGDPpc	0.0572	0.0543	0.0116	0.0928	-0.0417	-0.0521
	(0.0526)	(0.0779)	(0.0673)	(0.0670)	(0.0750)	(0.0800)

3 In Roodman (2009 p.103), the LSDV estimator was suggested as a plausible candidate for a dynamic panel data estimation. It transforms the data as a way to remove the fixed effects, drawing them out of the error term by entering dummies for each country, in order to resolve the dynamic panel bias associated with our specification. Aside the LSDV estimator, there are other more efficient ways such as the difference and system GMM that deals with dynamic panel bias through data transformation but are limited in testing for structural breaks.

Table 4…

Literacy	-0.00105	-0.00452	-0.00378	-0.00200*	-0.00157	-0.000511
	(0.00110)	(0.00983)	(0.0131)	(0.00111)	(0.00173)	(0.000887)
L1_Literacy	-0.000777	-0.00116	-0.00163	0.00304***	0.00788***	0.00177*
	(0.00134)	(0.000891)	(0.00184)	(0.00105)	(0.00187)	(0.00105)
Sanitation	-0.00813***	-0.000636	-0.00995***	-0.00187*	-0.00847**	-0.000133
	(0.00145)	(0.00452)	(0.00280)	(0.00104)	(0.00331)	(0.00213)
Safe_water	-0.00905***	-1.48e-05	0.0122***	-0.00186*	-0.00829***	-0.000327
	(0.00154)	(0.00279)	(0.00308)	(0.000985)	(0.00182)	(0.00106)
Immunization	-6.93e-05	0.000106	-7.19e-05	-0.00618**	0.000456	-0.000724
	(0.000305)	(0.000300)	(0.000389)	(0.00300)	(0.000301)	(0.000577)
Rainfall	-0.00254	-0.00116	-0.00124	-0.00928**	0.00127	0.00457
	(0.00447)	(0.00293)	(0.00556)	(0.00405)	(0.00465)	(0.00359)
Ethnic	0.917	3.357**	4.844**	4.640***	8.303***	1.220
	(0.845)	(1.470)	(1.821)	(1.007)	(2.157)	(1.461)
Conflict	0.00614	0.00221	0.00110	0.0120*	0.00396	0.00466
	(0.00551)	(0.00697)	(0.00608)	(0.00665)	(0.00986)	(0.00581)
Temperature	0.00524	-0.00581	0.00666	0.00519	0.000542	0.00424
	(0.00587)	(0.00408)	(0.00721)	(0.00486)	(0.00588)	(0.00399)
Pol_regime	-0.00193	-0.00115	1.51e-05	0.0950***	-0.0145**	-0.00207
	(0.00172)	(0.00156)	(0.00201)	(0.00213)	(0.00211)	(0.00287)
Year	-0.00503**	-0.00566	0.00215	-0.00428*	-0.0146***	-0.00506*
	(0.00229)	(0.00442)	(0.00358)	(0.00252)	(0.00427)	(0.00282)
Constant	10.31**	8.563	-0.0595	10.61**	34.90***	10.38*
	(4.322)	(7.862)	(6.861)	(5.224)	(9.050)	(5.961)
Observations	107	46	70	132	50	92
R-squared	0.999	1.000	0.999	1.000	1.000	1.000

Standard errors in parentheses; *** p<0.01, ** p<0.05, * p<0.1

DISCUSSION

This study adds to the literature that examines the impact of economic growth on child mortality reduction in the SSA sub-region by investigating the effect of the pro-poor growth advocated in the MDGs on child mortality reduction. It is unclear if studies exist that have assessed the relationship for a sub-region that lagged in attaining the child mortality target. Using a constructed annual panel dataset, the GMM was implemented, and the study's main finding suggests that the growth policy adopted in the post-MDG era may not be more pro-poor comparatively.

I investigate this using panel data for 46 SSA countries over the period 1990-2023, I find evidence of a growth elasticity of -0.42, which is supported by similar studies such as Pritchett and Summers (1996) and Bhalotra (2008). In particular, it is smaller than the unconditional growth elasticity of -0.7 in the Bhalotra paper for the possible reasons that I have: conditioned growth elasticity on education; used a different data set involving countries that are not Indian states; controlled for a different set of covariates; and implemented a different estimation strategy. Further, controlling for corruption and regime type results in the reduction of the magnitude of growth elasticity by about a half (-0.206). In the fuller model with all the controls, the effect of GDP per capita on child mortality disappears. This is in contrast with the findings from the Bhalotra paper. Though there was a reduction in the magnitude of the unconditional growth elasticity compared to the estimated elasticities with controls, the growth elasticity is robust to different model specifications in the Bhalotra paper. While the reason for the non-robustness of the growth elasticity estimate in this study is unclear, it is plausible to attribute it to the role of country-specific underlying constraints influencing child mortality outcomes in the region. This study shed new light on the effect of confounders such as culture, institutions, geography, and conflict on mortality for countries in the SSA sub-region.

When I test the hypothesis whether the growth policy in the post-MDG era was more pro-poor, I find evidence of a possible anti-poor growth. Similar findings suggesting that growth could be anti-poor exist in the literature (see, for example, Kakwani, 2000; Szekely, 2000; Bhalotra, 2008). Precisely, the study findings resonate with the Bhalotra paper that growth in a post-reform era is less effective in reducing child mortality. This implies that child mortality is still domiciled amongst the poor despite the pro-poor growth policy adopted. That is, on average, there is no evidence that the post-MDGs growth policy has improved the quality of life amongst households in SSA countries. Aside from child mortality being concentrated among the poor, the rate of child mortality in a society can be informative about the nature of social inequalities, gender bias, population health, and changing economic and social situations (Sen, 1998). Thus, it is likely that the growth pattern in post-MDG may be marked by rising inequality such that the potential benefit of growth is more than offset by the adverse impact of rising inequality. Perhaps, these findings provide additional support to the assertion by Kakwani (2000) that growth is not necessarily always good for the poor and that income distribution could matter in determining the effect of growth on child mortality outcomes.

Further, I test the hypothesis whether there is heterogeneity in the effect of growth on mortality between resource-rich and resource-poor countries. The results show that the impact of growth on mortality was only significant amongst resource-poor countries. These findings support an existing argument in the literature that resource-rich countries tend to underperform in terms of economic development compared to resource-poor countries (Sachs and Warner, 1995). Also, they can be considered as an additional support for the resource curse hypothesis. The volatility of economic growth in resource-rich countries may likely hurt their economic prosperity (Ogwumike and Ogunleye, 2008). Nigeria is considered a resource-rich and oil-producing country. Perhaps this in part, may explain why Nigeria, an emerging economy with the largest economy in Africa, has a significant number of people living in poverty and with the highest number of under-five deaths in Africa. It is likely that the natural resource curse exists for Nigeria. Windfall gains from the discovery of oil in commercial quantities in the early 1970s crowded out investment from other sectors of the economy, particularly the agricultural sector, a pro-poor sector. It is also likely that the point natural resources may have introduced rent seeking, corruption, and conflicts in Nigeria in line with the argument in the literature (for example, Boschini, Pettersson, and Roine, 2007). The inverse relationship between resource abundance and economic outcomes may exist for other countries in the SSA sub-region (for example, Angola, Sierra Leone, Congo DR, and Liberia).[4]

On the other hand, pro-poor growth may influence mortality differently in resource-poor countries. For such countries, growth may generate more resources both at the national level for public investment and at the household level for the poor to purchase child health. At the national level, it may result in greater efficiency (given the absence of resource curse) in public spending on services such as health care, education, safe water provision and sanitation, immunisation coverage, and employment provision. At the individual level, increases in per capita income from pro-poor growth may result in a greater reduction in household poverty in resource-poor economies. This will facilitate greater access to basic MDG goods and services by the poor.

LIMITATIONS AND FURTHER STUDY

The study findings on the structural break effect of economic growth on child mortality should be considered within the context of some limitations. First, the income and mortality data are aggregated at the country level, and this limits our understanding of how changes in income inequality due to the growth reform have impacted equity in child mortality outcomes. The underlying argument of the pro-poor growth strategy suggests the generation of growth from the pro-poor sectors in order to reduce inequality and improve household outcomes. In assessing the impact of pro-poor growth, it will be important to measure changes in child mortality inequalities by income quintiles. It is argued that a stronger association between income and

4 There are also counterexamples of countries in the SSA with abundance of natural resources but with better growth performance such as Botswana, South Africa

health may exist at the lowest quintile of the income distribution. Hence, using disaggregated mortality data by socioeconomic status may shed a different light on the structural break effect of growth on mortality. The study uses aggregate income and health outcome data limits our understanding of the precise effect of an increase in income growth of the bottom 10% on mortality amongst the population in the lowest socioeconomic status, and this could be a future research priority for evaluating the impact of socio-economic interventions. Effectively, a less aggregated dataset with heterogeneity variables could allow for the assessment of how the impact of income growth on child mortality outcomes varies across socioeconomic groups (poor and non-poor) and regions (rural and urban). Uncovering such heterogeneity across various groups and regions should be an agenda for future study.

Second, it is unlikely that the pro-poor economic growth reforms are implemented within a similar time frame across the SSA countries. So, the conclusion from an assessment of changes in income elasticity between two sub-sample periods using a common structural break point may need to be interpreted in the context of a strong assumption of a common break point. Due to data unavailability, I did not exploit the variability in economic reforms between and within countries. Finally, child mortality data in developing countries are usually underestimated. Reported deaths are likely to be higher because births and deaths of neonates occurring at home are less likely to be counted due to the near absence of a vital birth and death registration system. Importantly, the distribution of unreported deaths might be systematic, concentrating amongst the lowest income quintiles. This could lead to an inaccurate estimation of the effect of economic growth on mortality, especially amongst the different income quintiles.

CONCLUSION

From the study findings, I can conclude that growth in the post-reform period was less associated with childhood mortality across SSA countries compared to growth in the pre-reform era. In fact, the evidence suggests that mortality may still be largely concentrated among the poor despite the adoption of pro-poor growth policies by countries in the sub-region. As an inclusive growth, it is expected that there will be an increase in the number of children having access to basic capabilities, nourishment, and consequently long and satisfying lives but why this is not the case may be a scope for future study. However, the heterogeneous effect analysis suggests that growth may matter more in reducing mortality amongst resource-poor countries, and it may do so through greater efficiency in public spending. These results have important policy implications, especially for Nigeria, a resource-rich country but a significant contributor to global under-five mortality. Perhaps, generating growth through expansion in pro-poor sectors such as manufacturing and agriculture rather than crude oil exportation can be a valid policy suggestion. This will involve deliberate actions in diversifying the economy from its primary dependence on oil to allow for growth driven by output expansion in the pro-poor sectors. Hence, directing interventions into, particularly the agricultural sector, by international institutions and national governments can have the dual impact of reducing poverty and child mortality. In addition to promoting programmes that increase literacy among women, scaling up interventions in the provision of safe water and improved sanitation should be considered in the ongoing SDGs. From the study findings on the role of conflict in influencing mortality rate, it will also be important to promote actions that encourage the entrenchment of peace and conflict resolution at all levels. The effect of the on-going armed conflict in Nigeria may persist over the life course of children and to subsequent generations given birth to after the conflict has ended. Hence, the study findings on conflict provide justification for the prioritization of the sustainable development goal of promoting peace, justice, and strong institution and in particular the target of reducing conflict-related deaths per 100,000 population, by sex, age, and cause in the on-going SDGs.

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