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# ECONOMICS OF THE ENVIRONMENT AND INFANT MORTALITY IN SUB-SAHARA AFRICA

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## ECONOMICS OF THE ENVIRONMENT AND INFANT MORTALITY IN SUB-SAHARA AFRICA

#### Abstract

Studies have used ambient air pollutant concentrations to explain infant mortality. Thus, this study investigates the impact of carbon dioxide emission on infant mortality, while controlling for other covariates of human well-being in sub-Sahara Africa using a panel FGLS and GMM for the period 1990-2012. Our findings reveal that: there is a positive relationship between carbon dioxide emission and infant mortality; access to water has a significant negative impact on infant mortality rate; there is a significant positive relationship between access to sanitation and infant mortality rate; food production index has a significant negative impact on infant mortality rate; GDP per capita has a significant negative relationship with infant mortality rate; urban population as a percentage of total population is significantly positively related with infant mortality rate while relative change in fertility rate has a significant inverse relationship with relative change in fertility rate has a significant inverse relationship with relative change in infant mortality rate.

Keywords: carbon dioxide, infant mortality, FGLS, GMM

JEL Classification: C33, I12, Q53

#### **1.0 Introduction**

The explanation of infant mortality has been studied to be based on economic, biological and physiological reasons (see: Davanzo et al., 2004; Machado and Hill, 2005). Some studies have gone further to use ambient air pollutant concentrations (like: particulate matter, nitrogen oxides, sulfur dioxide, and bacteria in water) to explain infant mortality. Currie et al. (2009) found that a unit decrease in mean levels of carbon monoxide in the first two weeks of life would save roughly 18 lives per 100,000 births in New Jersey, USA. Arceo et al. (2012) also found significant effects of particulate matter and carbon monoxide on both neonatal and infant deaths for 48 municipalities across Mexico City.

Though air pollution affects everybody but children have greatest susceptibility. Their bodies perform metabolic activity and their immune systems are weak so they are less able to

metabolize to remove pollutants like adults. Children, also, have greater surface-to-volume ratios than adults as they play on the ground and engage in oral exploratory behavior. This increases their skin's potential exposure, potential ingestion of contaminants in the soil and dust, and exposure through respiration by inhaling air closer to the ground than adults do. In the case of infant death the link between cause and effect is immediate, whereas for adults diseases today may reflect pollution exposure that occurred many years ago (Fitzgerald et al., 1998; Currie et al, 2009).

Because their respiratory systems are under-developed, infants breather more air per body weight than adults and also engage in a lot of mouth breathing. The mouth breathing, which bypass the filter of the nose, may pull air pollutants deeper into infants respiratory system thus making the effect of air pollution to be suffocation and death or Sudden Infant Death Syndrome (SIDS i.e. an unexplained and unexpected death of an apparently healthy infant) (Cao et al., 2007; Pickett and Bell, 2011).

An indirect susceptibility of infants to air pollution has been argued to be through the health of the pregnant mothers. The weakening of her immune system when exposed to air pollution could be detrimental to her fetus. For illustration: Prenatal exposure to virile atmospheric pollutants, like polycyclic hydrocarbons which are released by combustion of crude oil and coal, has been linked to poorer birth outcomes, lower birth weight and smaller head circumference (Choi et al., 2006; Cesur et al., 2013).

Although, given income distribution, the level of infant mortality in a society should be optimal in the absence of market imperfections such as externalities (Grabowski and Shields, 1996). But with the effect of global climate change, as mentioned by McGranahan et al. (1999), carbon emission has a major impact on the spread of infectious diseases. This makes infant mortality rate an indicator useful in exploring the link between environmental and population health.

Carbon dioxide (CO<sub>2</sub>) is the most dangerous greenhouse gas (GHG)-other types of GHGs are water vapor, methane, nitrous oxide, and ozone- and it can persist for over a hundred years once emitted (Cunha-e-Sá, 2008). For every gallon of gasoline manufactured, distributed, and then burned in a vehicle, 25 pounds of carbon dioxide are produced (Donohoe, 2003). Simple activities like change in patterns of land use in developing countries like those in sub-Sahara Africa (SSA) can increase carbon dioxide. This would occur because clearing of forest will reduce the environment's capacity to absorb  $CO_2$ , since plants absorb carbon dioxide and emit oxygen. UNCTAD (2012) states that deforestation, alone, is estimated to account for about 25% of  $CO_2$  emitted world-wide.

Panel studies on the role of carbon dioxide in explaining infant mortality are terse in SSA. Adeyemi et al. (2008) examined the environment of health on under-five mortality rate in Nigeria using a multiple regression. They found that most deaths associated to children under the age of five are accounted for by these variables: expenditure on health as a percentage of GDP, access to portable water and health care, female illiteracy rate, daily calorie per capital, total fertility rate and carbon dioxide emission. Sanglimsuwan (2013) considered carbon dioxide as one of the environmental indicators (others are rainfall, temperature, safe water, sanitation, government effectiveness index, population density, urban population density, and per capita GDP) he used to explain infant mortality rate. He found that an increase in carbon dioxide leads to an increase in infant mortality rate.

Our objective is to investigate the impact of carbon dioxide emission on infant mortality, while considering other factors that determine wellbeing, in sub-Sahara Africa using a panel data for the period 1990-2012. The paper proceeds as follows: section two describes our methodology, section three provides discussion of our findings, and section four summarizes and concludes our study.

#### 2.0 Methodology

#### 2.1 Data Regressand

Infant mortality is defined as mortality in the first year of life. Most infant deaths occur in the first month of life and this is often from some form of respiratory failure (Currie and Neidell, 2003). The infant mortality rate used is measured per 1,000 live births while carbon dioxide is measured as metric tons per capita.

#### Regressors

In addition to carbon dioxide this study adopts some variables of wellbeing to explain infant mortality. According to the 1991 UNICEF report, inadequate and unsafe water supply and sanitation are environmental problems responsible for three quarter of all child deaths in the developing world. Many countries facing water scarcity are low-income countries that have a

rapidly growing population and are generally unable to make investments in water-saving technologies. These factors contribute to the deaths of more than 5 million people, of whom more than half are children (United Nations, 2001). Improved water sources and improved sanitation facility are measured based on percentage of population with access to them.

An adequate and dependable supply of food is essential for health. As at July, 2013, 27 out of the 34 countries the Food and Agriculture Organization (FAO) identified to require external assistance for food are sub-Sahara African countries. Food production index is measured using 2004-2006 as the base year. Economic welfare is measured most commonly by GDP per capita. Studies like Prichett and Summers (1996) and Deaton (2001) found that child deaths in developing countries can be attributed to poor economic status (Sanglimsuwan, 2013). GDP per capita measured as constant 2005 US\$ is used.

Epidemiological studies, conducted for SSA, have provided clues that overcrowding in urban environment is associated with infant mortality. That is rapid urbanization has severe effect on factors (e.g. poor housing conditions and emergence of slums) that affect infant diseases hence death (Asun, 1992). Urban population as percentage of total population is used as proxy for overcrowding in urban environment.

The flaw in explaining infant mortality without considering fertility has long been emphasized by Williams (1976); Olsen (1980); Eckstein et al. (1981); Lee (1981) and so on. The UNICEF report (1991) mentioned that no developing country has achieved a reduction in birth rates without achieving a reduction in child deaths. The Malthusian theory also states that a reduction in fertility rate will decrease infant mortality (Yamada, 1983). Total fertility rate, measured as birth per woman, is considered as a control variable for this study.

All these variables are obtained from the World Bank database for the period 1990-2012 using an unbalanced panel data of a sample of forty four countries out of the forty eight sub-Sahara African countries, as categorized by the World Bank (2014). Lesotho, Seychelles, Somalia, and South Sudan are not considered for this study due to high attrition rate of the respective countries data availability.

Our sample includes: Angola, Benin, Botswana, Burkina Faso, Burundi, Cameroon, Cape Verde, Central African Republic, Chad, Comoros, Congo Democratic Republic, Congo Republic, Cote d'Ivoire, Equatorial Guinea, Eritrea, Ethiopia, Gabon, Gambia, Ghana, Guinea, Guinea-Bissau, Kenya, Liberia, Madagascar, Malawi, Mali, Mauritania, Mauritius, Mozambique, Namibia, Niger, Nigeria, Rwanda, Sao tome, Senegal, Sierra Leone, South Africa, Sudan, Swaziland, Tanzania, Togo, Uganda, Zambia, Zimbabwe.

#### 2.2 Model for the Study

The relationship between infant mortality and carbon dioxide and other variables discussed in the section above can be expressed in equation 1:

$$IM_{it} = f(CO_{2it}, AW_{it}, AS_{it}, FP_{it}, GDP_{it}, UP_{it}). \qquad . \qquad .1$$

IM represents infant mortality rate in country i at time t,  $CO_2$  is carbon dioxide per capita in country i at time t, AW is access to water in country i at time t, AS is access to sanitation in country i at time t, FP is food production index in country i at time t, GDP is gross domestic product per capita in country i at time t, and UP is urban population as a percentage of total population in country i at time t.

$$\begin{split} lnIM_{it} &= \beta_0 + \beta_1 lnCO_{2it} + \beta_2 lnAW_{it} + \beta_3 lnAS_{it} + \beta_4 lnFP_{it} + \beta_5 lnGDP_{it} + \beta_6 lnUP_{it} + \delta_i + \gamma_t \\ &+ \varepsilon_{it}. \end{split}$$

Equation 2 specifies a panel model of equation 1. The  $\delta_i$  and  $\gamma_t$  represent cross section and period specific effects. Each of the variables is logged so as to bring them to the same base and reduce any level of heteroskedasticity in the model (Gujarati, 2007).

Carbon dioxide emitted can remain in the atmosphere for over a hundred years after it has been emitted (Cunha-e-Sá 2008). This implies that  $CO_2$  emission can affect health outcomes not only in the current generation but also in subsequent generation. In other words, holding additional emission constant, health outcomes today could be as a result of emission from previous period(s). To identify any likely effect  $CO_2$  emission of preceding periods has on infant mortality rate we specify a dynamic panel model in equation 3.

$$lnIM_{it} = \beta_0 + \beta_1 lnCO_{2it} + \beta_2 lnCO_{2it-1} + \beta_3 lnCO_{2it-2} + \beta_4 lnAW_{it} + \beta_5 lnAS_{it} + \beta_6 lnFP_{it} + \beta_7 lnGDP_{it} + \beta_8 lnUP_{it} + \delta_i + \gamma_t + \varepsilon_{it}.$$

In equation 4 and 5 we introduce the control variable fertility rate in country i at time t (FR).  $lnIM_{it} = \beta_0 + \beta_1 lnCO_{2it} + \beta_2 lnAW_{it} + \beta_3 lnAS_{it} + \beta_4 lnFP_{it} + \beta_5 lnGDP_{it} + \beta_6 lnUP_{it} + \beta_7 lnFR_{it} + \delta_i + \gamma_t + \varepsilon_{it}.$ 

$$lnIM_{it} = \beta_0 + \beta_1 lnCO_{2it} + \beta_2 lnCO_{2it-1} + \beta_3 lnCO_{2it-2} + \beta_4 lnAW_{it} + \beta_5 lnAS_{it} + \beta_6 lnFP_{it} + \beta_7 lnGDP_{it} + \beta_8 lnUP_{it} + \beta_9 lnFR_{it} + \delta_i + \gamma_t + \varepsilon_{it}.$$

#### **2.3 Estimation Procedure**

We estimate equation 2 and 3 with Panel Least Squares considering fixed effects. We further conduct redundant fixed effects test to test for the significance of the cross-section and period effects. We estimate with Panel Feasible Generalized Least Squares (FGLS) where serial correlation is present under the Panel Least Squares. After estimating equation 2 and 3, we test the relevance of our control variable fertility rate using the omitted variables test. We then estimate equation 4 and 5 using Panel Least Squares and Panel FGLS.

The Malthusian and Modern Economic theories on population suggest that the relationship between fertility and infant mortality reflects lagged causality in both directions. Williams (1976) says that those who seek to explain mortality view fertility as an exogenously given independent variable, hence such estimates are marred by simultaneous equation bias. Thus, fertility and infant mortality are not mutually independent but mutually interdependent i.e. jointly determined (Yamada, 1983). As theory suggests, causality may run in one or both directions of infant mortality and fertility rate. To affirm this we first estimate lagged causality between fertility and infant mortality using the Pairwise Granger Causality. To solve the problem of causality we adopt the difference Generalized Method of Moments (GMM) as developed by Arellano and Bond (1991) for the estimation of equation 4 and 5. This is because the GMM would account for the likely endogeneity that exist in these models (see: Angeles 2008). Thus, first-differencing the specification in equation 4 and 5 eliminates the effects and produces equation 6 and 7. Also, the time-invariant country characteristics (such as demography, etc.) in our sample would be removed after the first differencing.

$$\Delta lnIM_{it} = \beta_1 \Delta lnCO_{2it} + \beta_2 \Delta lnAW_{it} + \beta_3 \Delta lnAS_{it} + \beta_4 \Delta lnFP_{it} + \beta_5 \Delta lnGDP_{it} + \beta_6 \Delta lnUP_{it} + \beta_7 \Delta lnFR_{it} + \Delta \varepsilon_{it}.$$

 $\Delta lnIM_{it} = \beta_1 \Delta lnCO_{2it} + \beta_2 \Delta lnCO_{2it-1} + \beta_3 \Delta lnCO_{2it-2} + \beta_4 \Delta lnAW_{it} + \beta_5 \Delta lnAS_{it} + \beta_6 \Delta lnFP_{it} + \beta_7 \Delta lnGDP_{it} + \beta_9 \Delta lnPR_{it} + \beta_8 \Delta lnPR_{it} + \Delta \varepsilon_{it}.$ 

Arceo et al (2012) and Currie et al (2009) used Instrumental Variables (IV) while Sanglimsuwan (2013) used OLS. GMM, unlike these methods of estimation, would allow us use lagged variables as instruments which would make endogenous variables to be predetermined. This would help to avoid having weak instruments and biased estimators which are likely under IV and OLS. Although for a panel data GMM is designated for small T (time) and large N

(countries) panels, but when T is large as in this study shocks to country's fixed effects will decline with time and correlation of lagged dependent variables with error term will be insignificant (Roodman, 2006). Thus, GMM becomes not necessary to use but it is not sufficient to disregard GMM for such estimation. This is affirmed by Grunewald and Martinez-Zarzoso (2011) who used T that is 50 years and N that is 213 countries. After estimating equation 6 and 7, we estimate the Arellano-Bond serial correlation test. This procedure is done using E-views 8.0.

#### **3.0 Discussion of Findings**

#### 3.1 Equation 2

Table 1 shows the estimated results for equation 2. The constant coefficient and coefficients of all the regressors are statistically significant at all levels under Panel least squares. The result shows that access to water, access to sanitation, food production index, GDP per capita, and urban population to total population all have a negative relationship with infant mortality while  $CO_2$  per capita has a positive impact on infant mortality in our panel sample.

The adjusted coefficient of determination (Adjusted  $R^2$ ) shows that a high percentage of total variation in the natural log of infant mortality is explained by the natural log of the regressors. The p-value of the F-statistic allows us to reject the null hypothesis that the regressors have no impact on infant mortality, at all levels of significance. The Durbin Watson statistic shows evidence of positive autocorrelation. The F-statistic and Chi-square for the diagnostic test (redundant fixed effects) strongly rejects the null hypothesis that both cross-section and period effects are redundant. This suggests that both effects are statistically significant at all levels.

Because we are using an unbalanced data, equation 2 is re-estimated using Panel FGLS with Period Seemingly Unrelated Regression (SUR) which corrects for both period heteroskedasticity and general correlation. Although the Adjusted  $R^2$  is low due to the diverse cross sectional units but the p-value of the F-statistic is significant at all levels which implies that the estimated regression is significant and that the  $R^2$  is not zero. The Durbin Watson statistic is much higher than that of the Panel Least Squares. This suggests that the Panel FGLS is a better specification.

It is observed under Panel FGLS that the constant coefficient and coefficients of the natural log of  $CO_2$  per capita, access to water, food production index and GDP per capita are statistically

significant at all levels while the coefficients of the natural log of access to sanitation and urban population as a percentage of total population are not statistically significant. It is observed that  $CO_2$  per capita, access to water, food production index and GDP per capita have a negative impact on infant mortality.

#### 3.2 Equation 3

Table 1 shows the estimated results of equation 3. The constant coefficient and coefficients of the regressors under Panel Least Squares, except for two periods lagged  $CO_2$  per capita ( $CO_{2t-2}$ ), are statistically significant. Our result shows that access to water, access to sanitation, food production index, GDP per capita, and urban population to total population have a negative relationship with infant mortality while  $CO_2$  per capita and one period lagged  $CO_2$  per capita have a positive impact on infant mortality in our panel sample.

The Adjusted  $R^2$ , p-value of the F-statistic, Durbin Watson statistic, and the redundant fixed effects test have the same interpretation as the result for equation 2 under Panel Least Squares.

Equation 3 is re-estimated using Panel FGLS. Although the Adjusted  $R^2$  is lower but the p-value of the F-statistic is significant at all levels which implies that the estimated regression is significant. The Durbin Watson statistic is much higher than that of the Panel Least Squares. This suggests that the Panel FGLS is a better specification.

It is observed that the constant coefficient and coefficients of the natural log of access to water, food production index, GDP per capita, and urban population as a percentage of total population are statistically significant while the coefficients of the natural log of  $CO_2$  per capita, its lagged period regressors and access to sanitation are not statistically significant. It is also observed that access to water, food production index, and GDP per capita have a negative relationship with infant mortality while urban population as a percentage of total population has a positive relationship with infant mortality.

#### 3.3 Equation 4

An Omitted variable test conducted under equation 2, as shown on Table 1, gives evidence that fertility rate is relevant for our modeling at all levels of statistical significance. Thus, Table 2 shows the estimated results for equation 4.

The constant coefficient and coefficients of all the regressors are statistically significant under the Panel Least Squares. The result shows that access to water, access to sanitation, food production index, GDP per capita, and urban population to total population all have a negative relationship with infant mortality while  $CO_2$  per capita has a positive impact on infant mortality. On the other hand, Fertility rate has a negative relationship with infant mortality.

The Adjusted  $R^2$  shows that a high percentage of total variation in the natural log of infant mortality is explained by the natural log of the regressors. The p-value of the F-statistic allows us to reject the null hypothesis that the regressors have no impact on infant mortality, at all levels of significance. The Durbin Watson statistic shows evidence of positive autocorrelation. The redundant fixed effects test suggests that both cross-section and period effects are statistically significant at all levels for the model.

Equation 4 is re-estimated using Panel FGLS. The Adjusted  $R^2$  is fair under the Panel FGLS and the p-value of the F-statistic is significant at all levels. This implies that the estimated regression is significant. The Durbin Watson statistic is much higher than that of the Panel Least Squares. This suggests that the Panel FGLS is a better specification compared to not only the estimation of equation 4 using Panel Least Squares but also the estimation of equation 2 using Panel FGLS because both the Adjusted  $R^2$  and the Durbin Watson statistic are higher.

It is observed that the constant coefficient and coefficients of the natural log of access to water, access to sanitation, food production index, GDP per capita, urban population as a percentage of total population and fertility rate are statistically significant while the coefficient of the natural log of  $CO_2$  per capita is not statistically significant. It is observed that access to water, food production index, and GDP per capita have a negative relationship with infant mortality while access to sanitation, urban population as a percentage of total population, and fertility rate have a positive relationship with infant mortality.

#### 3.4 Equation 5

The Omitted variable test conducted under equation 3 on the Panel FGLS model as shown on Table 1 gives evidence that fertility rate is relevant for our modeling at all levels of statistical significance. Thus, Table 2 shows the estimated results of equation 5.

The Panel Least Squares shows that the constant coefficient and coefficients of all the regressors, except for two periods lagged  $CO_2$  per capita ( $CO_{2t-2}$ ) and fertility rate, are statistically

significant. The fixed effect model shows that access to water, access to sanitation, food production index, GDP per capita, and urban population to total population have a negative relationship with infant mortality while  $CO_2$  per capita and one period lagged  $CO_2$  per capita have a positive impact on infant mortality.

The Adjusted  $R^2$ , p-value of the F-statistic, Durbin Watson statistic, and the redundant fixed effects test have the same interpretation as the result for equation 4 under Panel Least Squares.

Equation 5 is re-estimated using Panel FGLS. The Adjusted  $R^2$  is fair and the p-value of the Fstatistic is significant at all levels. This implies that the estimated regression is significant. The Durbin Watson statistic is much higher. This suggests that the Panel FGLS model is a better specification compared to not only the estimation of equation 5 using Panel Least Squares but also the estimation of equation 3 using Panel FGLS because both the Adjusted  $R^2$  and the Durbin Watson statistic are higher.

It is observed that the constant coefficient and coefficients of the natural log of access to water, access to sanitation, food production index, GDP per capita, urban population as a percentage of total population and fertility rate are statistically significant while the coefficients of the natural log of  $CO_2$  per capita and its lagged period regressors are not statistically significant. It is also observed that access to water, food production index, and GDP per capita have a negative relationship with infant mortality while access to sanitation, urban population as a percentage of total population, and fertility rate have a positive relationship with infant mortality.

#### 3.5 Equation 6

The Granger causality test on Table 3 was conducted using lag 10. From this result, we reject the null hypothesis that fertility rate does not Granger cause infant mortality and also reject the null hypothesis that infant mortality does not Granger cause fertility rate at all levels of statistical significance. In other words, Granger causality runs both ways between fertility rate and infant mortality. This supports the Malthusian and Modern Economic theories on population and allows us to take into consideration endogeneity in estimating equation 4 and 5 which becomes equation 6 and 7.

Table 4 shows the difference GMM results of equation 6: the coefficients of the regressors, except the relative change in  $CO_2$  per capita and urban population as a percentage of total population, are statistically significant. The relative change in access to sanitation, food

production index, GDP per capita and fertility rate have a negative relationship with relative change in infant mortality while relative change in access to water has a positive relationship with relative change in infant mortality.

The p-value of the Sargan test allows us to accept the null hypothesis that the over-identifying restrictions are valid at 1% level of significance. The Arellano-Bond Serial Correlation test shows that the first order statistic (AR(1)) is statistically significant at 1% whereas the second order statistic (AR(2)) is not statistically significant at 1%. This is expected as it means that the model's error terms are serially uncorrelated, i.e. no serial correlation at 1% significance.

#### 3.6 Equation 7

Table 4 also shows the difference GMM results for equation 7: the coefficients of the regressors, except the relative change in  $CO_2$  per capita, access to water, access to sanitation, and food production index, are statistically significant. The relative change in GDP per capita and fertility rate have a negative relationship with relative change in infant mortality while relative change in lagged regressors of  $CO_2$  per capita and urban population as a percentage of total population have a positive relationship with relative change in infant mortality. The p-value of the Sargan test makes us not to reject the null hypothesis that the over-identifying restrictions are valid at all levels of significance. The Arellano-Bond Serial Correlation test shows that the first order statistic (AR(1)) is statistically significant at 1% whereas the second order statistic (AR(2)) is not statistically significant at 1%.

#### 4.0 Summary and Conclusion

This paper investigates the impact of carbon dioxide emission on infant mortality, while considering other factors that determine wellbeing, in sub-Sahara Africa using a panel study for the period 1990-2012. There is significant evidence that lagged periods of growth in carbon dioxide have a positive impact on growth in infant mortality rate. Thus, our findings suggest that a rise in the growth of carbon dioxide causes an increase in the growth of infant mortality rate in sub-Sahara Africa (SSA). This implies that the strong desires to attain robust economic growth in SSA countries are leading to drawbacks on concerns for global climate change which in turn leads to increase in infant mortality.

We found that access to water has a significant negative impact on infant mortality rate which means that those governments' efforts to provide access to clean water have positive influence on health. Growth in access to water has a significant positive impact on growth in infant mortality rate. This proffers that sub-Sahara African countries with less than fifty percent population with access to water need to increase creation of hygienic water. This suggestion is backed by Mahmood (2002) who found that households with piped water have a significantly lower post neonatal mortality than those who depend on wells.

There is a significant positive relationship between access to sanitation and infant mortality rate. Access to sanitation in sub-Sahara Africa is not making any positive impact on health. In other words, access to sanitation is extremely poor in most sub-Sahara African countries with Ethiopia having the least. Just three percent of Ethiopia's population has access to sanitation.

Food production index has a significant negative impact on infant mortality rate across in our findings. This complies that food has a positive influence on health. This notwithstanding, the region needs to give due regard to the FAO's (2013) signal of food crises in 27 sub-Sahara African countries.

We found that GDP per capita has a significant negative relationship with infant mortality rate. This supports Princhett and Summers (1996), Deaton (2001) and Sanglimsuwan (2013) that increase in economic status should lead to improvement in health.

The study found that urban population as a percentage of total population is significantly positively related with Infant mortality rate. This confirms Asun (1992) that in developing countries urban environment has severe effects on health like increase in slum areas, overcrowding, pollution, inadequate disposal of solid wastes, easy spread of communicable diseases, etc.

Lastly, we found that fertility rate has a significantly positive relationship with infant mortality rate. This complies with the Malthusian theory and the UNICEF report (1991) that no developing country has achieved a reduction in birth rates without achieving a reduction in child deaths. Considering endogeneity, growth in fertility rate has a significant inverse relationship with growth in infant mortality rate. The reason behind this could be ascribed to increase in primary health care facilities, access to orientation about breeding and conception, consistent foreign aid,

etc. Just as the links between environment and population health are receiving well deserved attention internationally, sub-Sahara African countries should also intensify efforts.

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# Appendix

# Table 1

	Equation 2		Equation 3	
Dependent variable: In	Denal Least Squares	Denal ECL S	Denal Least Squares	Denal ECL S
1111	Panel Least Squares	Pallel FOLS	Panel Least Squares	Pallel FOLS
constant	7.695*** [0.283]	7.596*** [0.205]	8.064*** [0.309]	7.506*** [0.241]
ln CO <sub>2</sub>	0.046*** [0.009]	-0.023*** [0.007]	0.032*** [0.010]	-0.011 [0.007]
ln CO <sub>2t-1</sub>	-	-	0.020* [0.011]	-0.003 [0.007]
ln CO <sub>2t-2</sub>	-	-	0.014 [0.011]	-0.003 [0.007]
ln AW	-0.119*** [0.024]	-0.564*** [0.062]	-0.123*** [0.027]	-0.588*** [0.066]
ln AS	-0.058*** [0.016]	0.025 [0.027]	-0.053*** [0.017]	0.028 [0.029]
ln FP	-0.171*** [0.026]	-0.142*** [0.019]	-0.137*** [0.027]	-0.138*** [0.019]
ln GDP	-0.184*** [0.023]	-0.096*** [0.017]	-0.214*** [0.028]	-0.086*** [0.019]
ln UP	-0.203*** [0.059]	0.056 [0.045]	-0.290*** [0.057]	0.086* [0.047]
Adjusted R <sup>2</sup>	0.949	0.471	0.955	0.431
Durbin Watson	0.112	1.074	0.108	1.037
F-statistic	241 (0.000)	130.842 (0.000)	250.629 (0.000)	76.826 (0.000)
Redundant Fixed Effects Tests:				
F-statistic	112.565 (0.000)	-	125.323 (0.000)	
Chi-square	1995.309 (0.000)	-	1953.377 (0.000)	
Omitted variable Test (fertility rate):				
t-statistic	-	14.130 (0.000)	-	14.405 (0.000)
F-statistic	-	199.664 (0.000)		207.494 (0.000)

## Table 2

	Equation 4		Equation 5	
Dependent variable: ln	Donal Laget Squares	Danal FCI S	Danal Laget Squaras	Papel ECLS
1101	Faller Least Squares	Fallel FOLS	Faller Least Squales	Fallel FOLS
constant	8.168*** [0.258]	2.967*** [0.259]	8.206*** [0.285]	2.980*** [0.295]
ln CO <sub>2</sub>	0.043*** [0.009]	0.005 [0.003]	0.033*** [0.010]	-0.001 [0.004]
ln CO <sub>2t-1</sub>	-	-	0.019* [0.011]	0.004 [0.004]
ln CO <sub>2t-2</sub>	-	-	0.011 [0.011]	0.006 [0.005]
ln AW	-0.121*** [0.024]	-0.244*** [0.044]	-0.122*** [0.027]	-0.289*** [0.049]

ln AS	-0.065*** [0.016]	0.038** [0.018]	-0.057*** [0.018]	0.063*** [0.020]
ln FP	-0.144*** [0.023]	-0.094*** [0.012]	-0.130*** [0.027]	-0.088*** [0.013]
ln GDP	-0.197*** [0.023]	-0.029** [0.012]	-0.218*** [0.028]	-0.039*** [0.014]
ln UP	-0.221*** [0.056]	0.161*** [0.034]	-0.295*** [0.055]	0.192*** [0.037]
ln FR	-0.251*** [0.077]	1.348*** [0.073]	-0.075 [0.051]	1.365*** [0.079]
Adjusted R <sup>2</sup>	0.951	0.639	0.956	0.631
Durbin Watson	0.103	1.208	0.106	1.122
F-statistic	240.894 (0.000)	222.413 (0.000)	247.034 (0.000)	152.736 (0.000)
Redundant Fixed Effects Tests:				
F-statistic	58.977 (0.000)	-	64.296 (0.000)	-
Chi-square	1509.090 (0.000)	_	1483.471 (0.000)	-

# Table 3

Pairwise Granger Causality Tests		
Null Hypothesis:	F-statistic	
LOG(FR) does not Granger Cause LOG(IM)	3.634 (0.0001)	
LOG(IM) does not Granger Cause LOG(FR)	3.784 (6.E-05)	

# Table 4

Panel Generalized N	Panel Generalized Method of Moments		
	Equation 6	Equation 7	
Dependent variable: ∆ln IM			
$\Delta \ln \mathrm{CO}_2$	0.022 [0.037]	0.394 [0.528]	
Δln CO <sub>2t-1</sub>	-	0.784*** [0.248]	
Δln CO <sub>2t-2</sub>	-	0.297*** [0.084]	
Δln AW	2.178* [1.139]	-0.61 [1.722]	
Δln AS	-2.334* [1.22]	-0.607 [1.328]	
∆ln FP	-0.498** [0.206]	-0.188 [0.377]	
∆ln GDP	-1.008** [0.441]	-3.165*** [1.021]	
∆ln UP	0.354 [0.444]	2.253*** [0.721]	
∆ln FR	-1.122** [0.566]	-1.397* [0.844]	
J statistic	5.824	0.836	
Sargan test p-value	0.016	0.360	
Arellano-Bond Serial Correlation test:			
AR(1) p-value	0.004	0.003	
AR(2) p-value	0.051	0.039	