When The Last Tree Dies, The Last Man Dies: Do Forests Hold The Key To Survival In Ghana? A Critical Analysis Using The Bootstrapped Rolling Window Granger Causality Test Approach

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Abstract

This paper investigates the role of forests in the life expectancy of people in Ghana. We test whether the extinction of forests will inevitably lead to extinction of people in Ghana. We first examined the causal relationship between life expectancy and deforestation using the full sample bootstrap Granger causality test approach and find causality to run from deforestation to life expectancy with no feedback from life expectancy to deforestation. Testing for parameter stability, the parameters of the VAR model were found to be unstable in the short and long run. Consequently, the Bootstrapped Rolling Window granger causality test, a time-varying approach was then employed to examine the true nature of the causal relationship that exists between life expectancy and deforestation. The results showed that deforestation has a negative effect on life expectancy, confirming the widely accepted saying that the health of forests is inextricably linked to the health of mankind. The empirical results further show that, on trend higher life expectancy increases the rate of deforestation in Ghana. Highlighting the importance of the role of forests in influencing life expectancy in Ghana, we recommend awareness creation on the role of forests in supporting human life and also extensive afforestation programs to reduce the rate of deforestation in Ghana. This, we believe, will reduce the spread of vector borne diseases such as malaria and reduce the surge in respiratory diseases which shorten the life span of Ghanaians.

keywords

Ghana; deforestation; life expectancy; rolling window bootstrap Granger causality.

JEL codes

Q23, Q50, Q53, Q58, Q58

1.0 Introduction

There can be very little denying the fact that the quality of environmental resources and human health, and consequently life expectancy are intricately interlinked. As scientists all over the world seek to find solutions to slowing down the raging forces of climate change, preserve wildlife and consequently support lives of billions of people, it emerges that trees most form a substantial component of the solution to the problem. Trees are a very important environmental resource that have been proclaimed to hold the key for the very existence and survival of the human race. The

major concern among policy makers is that the high rate deforestation, mostly caused by anthropogenic activities sacrifices the long term benefits derived from trees for the short term economic gains obtained when trees are cut down[1].The last decade has been environmentally challenging globally considering the many environmental ills the world has had to battle with, chief among them being the twin destructive environmental forces, climate change and deforestation. Though independently destructive, to a very large extent, global climate change and deforestation are interlinked, with deforestation playing a major role in global climate change through the absorption and storage of carbon in soil and biomass. As posited by many climate change scientists, tropical forests are important carbon sinks, hence the reduction of forest cover does not only exacerbate carbon emissions into the atmosphere but gradually degenerates the carbon sink which is known to absorb approximately 18% of the carbon emitted into the Earth's atmosphere[2]. Forests, traditionally are known to occupy a unique position in terms of safeguarding the health and even the very life of humankind. Forests are traditionally thought of as holding the very key to the very existence of man. As cleverly encrypted in the ever simple but powerful saying "When the last tree dies the last man dies", we can see in this adage that the protection of forests is even ever more paramount now.

The relationship between human health and environmental quality has been a subject of intense intellectual and research scrutiny, with many people holding the view that the health of a people in a society is somewhat linked to the quality of the environment in which they find themselves. Even though the relationship is obscured, not straight forward and may be subject to a little controversy, there can also be no denying that environmental quality plays some role in the quality of health of humans and consequently influences their life expectancy. According to Smith et al.

[1]https://www.nationalgeographic.com/environment/global-warming/deforestation/
2 http://www.cbc.ca/news

(1999), environmental quality, being a key direct and indirect determinant of human health in any society and nation, also has negative spillover effects and externalities into other nations. Employing data from Global Burden of Disease (GBD) database, the authors estimated that approximately 25%-33% of the global disease burden in modern times can be attributed to environmental factors. The findings from their study shows that while it is the activities of wealthy nations that mostly threatens the global atmosphere, it is the people in the poorest countries and middle income countries who are at most risk from household-related and community-related environmental quality problems respectively.

In a joint report by *Lancet* and University College London Institute for Global Health Commission, assessing the linkage between health and climate change, Costello et al. (2009) traced the directs effects of global warming, and inherently climate change to the increase in the rate of transmissions of vector-borne and rodent-borne diseases such as malaria and dengue fever. The report further showed that, indirectly, climate change affects economic development, which subsequently affects health. In a related study,Gulis (2000) used data on 156 countries to quantitatively estimate the effect of environmental degradation on life expectancy. Using access to safe drinking water as a proxy for quality of the environment, it emerged from the study that there existed a strong direct correlation between life expectancy and the quality of the environment. Moreover, applying OLS regression models, the authors found a statistically strong positive effect of environmental quality on life expectancy, enforcing the assertion that environmental quality is a strong determinant of the life expectancy of humans. In another empirical investigation of the effect of physical environment on life expectancy, Idrovo (2011) employed exploratory factor analysis using data on life expectancy and 50 environmental indicators from 32 Mexican states. Four factors were extracted and used as independent variables in an OLS regression model. The authors found

environmental quality, proxied by environmental sustainability, ecological resistance and plague-free environments to exert a statistically significant positive effect on total life expectancy in Mexico.

The direction of causality between environmental quality and health has been a subject of controversy. While some argue for a unidirectional causality running from environmental quality to health, others have asserted that there exists a bi-causal relationship between the two variables. In a recent study, Mariani et al. (2010), proposed a bi-causal relationship between life expectancy and environmental quality. Employing an Over Lapping Generations model, the authors theorized that, overtime whereas environmental quality depends on life expectancy, given that people who expect to live for longer periods of time have a stronger concern for the future and therefore invest more in environmental care, the longevity of people is also somewhat affected by their prevailing environmental conditions.

The forgoing analysis reveals that empirical studies in the past have concentrated on other measures of environmental quality while implicitly overlooking one of the key indicators of environmental quality, i.e. deforestation. From literature, we observe that empirical studies on the causal relationship between deforestation and life expectancy is very scarce and virtually non-existent. However, deforestation is a fast occurring environmental ill which has been repeatedly cited for being responsible for many health conditions which subsequently threatens life expectancies.

The channels through which deforestation threatens life expectancies can be explained by the repercussions that arise from the loss of forest cover. Over the years, research shows increasing

numbers of respiration diseases globally[3], which may be traceable to the reduction in the quality of air that we breathe, as the role of trees as carbon sinks and filters is gradually terminated. It can thus be implicitly inferred that deforestation is partly responsible for aggravating the health conditions of humans through poor air quality and shortening their life expectancy. Moreover, in addition to the therapeutic value of forest, Bodeker et al. (1997) reports that forests are key sources of traditional medicine for approximately 80% of the population in tropical developing countries. It is worth noting that clearing of forests, which mostly clears plants of medicinal value, to a long extent denies humans the benefits of traditional medicine that will otherwise have contributed to improving life expectancy. Deforestation, therefore, is not only destructive but also prevents humans from having access to key medicinal resources that can improve our health and to a large extent improve our life expectancy. The other channel through which deforestation is hypothesized to affect health and therefore reduce life expectancy is creating conditions that promote the thriving of vector-borne and rodent-borne diseases. In Sub Saharan Africa, deforestation has been responsible for creating conditions that are climatically conducive for breeding the female anopheles mosquito which is responsible for transmitting the malaria parasite. The malaria epidemic in Sub-Saharan Africa has severe consequences on the quality of life of humans. In an overview of the link between deforestation and Malaria in Sub-Saharan Africa , Uneke (2008) estimated that the joint mortality and morbidity impacts of malaria are estimated to account for 11% of all infectious diseases, which translates into approximately 45 million *DALYs* (Disability Adjusted Life Years). This shows that deforestation has a very key role to play in influencing life expectancies of human.

[3] http://ghdx.healthdata.org/gbd-results-tool

Ghana was chosen for this study because it exhibits characteristics that reflect the channels through deforestation influences human health in Sub Saharan Africa. Ghana is basically an epitome of the Sun Saharan experience with reference to the effect of deforestation on life expectancy. Ghana has experienced high rates of deforestation, high incidences of Malaria and respiratory diseases in recent years. According to Acheampong et al. (2019), Ghana has suffered average deforestation rates of 0.7%, 0.5%, 0.4% and 0.6% for the periods 1990-2000, 2000-2005, 2005-2010 and 2010-2015 respectively. In the year 2019, malaria accounted for approximately 19 % of all recorded death in Ghana, with malaria admissions also increasing from 280,000 to 340,000 between 2000 and 2017. Also, respiratory diseases have accounted for approximately 13.54% of all deaths in Ghana between 1990 and 2017[4].

Given that malaria, respiratory diseases and deforestation are occurring at high rates in Ghana, one would expect some kind of correlation between them, with deforestation influencing human health and consequently life expectancies through the occurrences of malaria and respiratory diseases. Deforestation thus, to a large extent, exacerbating the incidence of malaria and respiratory diseases affects the human health and thus the life expectancy of mankind. Form the foregoing analysis, though we can observe that deforestation clearly has both direct and indirect effects on human health and thus life expectancy. However, empirical studies to test this assertion are virtually nonexistent. To this end this study empirically verifies whether indeed deforestation leads to a reduction in human life expectancy in Ghana. Also we test the assertion that the people's desire to invest in environmental quality is motivated by how long they expect to live. The significance of this paper is driven by the compelling fact that, laying aside the empirical works of Smith et al. (1999) ,Gulis (2000), Idrovo (2011) , Mariani et al. (2010) and a few other studies, empirical

[4] http://ghdx.healthdata.org/gbd-results-tool

studies on the causal relationship between environmental quality and life expectancy is scarce. Considering the very important role that deforestation plays in influencing life expectancies as expounded above, it is worthy of note that there exists virtually no empirical study on the causal relationship between deforestation and life expectancy. It appears that there exists an obvious scarcity of empirical studies providing empirical validation of the theoretical and logical linkages between deforestation and life expectancy in Ghana. Our paper thus seeks to close this gap in literature. Further, our paper adopts the Rolling Window Bootstrap Granger causality test approach to examining the causal relationship between deforestation and life expectancy in Ghana. This approach has that advantage that it tests causality between two variables within sub samples of the study's data set as opposed to other procedures that test causality over the full sample data. By testing causality within sub samples, we are able to determine the exact nature of the causality between the two variables, i.e. whether it is linear or non-linear. This contrasts approaches by former empirical works that have assumed that the causal relationship between environmental quality and life expectancy is strictly linear. Our paper is significant because the adoption of the Rolling Window Bootstrap Granger causality approach allows testing for the existence of feedback effects from life expectancy to deforestation. This is a build-up on past empirical studies that explicitly assumed that the direction of causality runs from environmental quality for life expectancy with no feedback from life expectancy to the environment. The remainder of our paper is structured in the following manner: Section 2 discusses the theoretical framework for our study. Section 3 discusses the methodology of the paper. Section 4 discusses the empirical results of the estimations in section 3. Section 5 concludes the paper.

2.0 Theoretical framework

The notion that the extinction of trees may lead to the extinction of human life is inextricably linked to the fact that deforestation may create conditions for the spread of life-threatening diseases such as malaria and dengue fever. Also, trees serve as a carbon sink, filtering the air that we breathe. In this regard, the fewer trees we have, the poorer the quality of air that we breathe, the higher the risk of suffering respiratory diseases; thereby leading to a reduction in the quality of our health, which will invariably lead to a reduction in our life expectancy. A study by Pattanayak and Yasuoka (2012) established that regions with higher rates of deforestation experienced higher incidences of malaria. Deductively, the foregoing analysis shows that, deforestation leading to an increase in the rate of diseases such as malaria and respiratory diseases, may to a large extent have an effect on the quality of life and hence life expectancy of people. Denoting Life expectancy by *LIFE*, health quality by *HEALTH,* and rate of deforestation by *DEF*, our study theorizes the relationship among life expectancy, health and deforestation as follows. Firstly, we assume propose that life expectancy of individuals depends on the quality of their health. We express this in Equation (1) as:

$$LIFE = f(HEALTH) \qquad (1)$$

Where *HEALTH* is determined by factors such as incidences of malaria, dengue fever and respiratory illnesses. All other things being equal, we expect $\frac{dLIFE}{dHEALTH} > 0$, implying that high health quality leads to higher life expectancies.

Secondly, we propose that the health status of individuals in a country is influenced by the rate of deforestation in the country. We express this in Equation (2) as:

$$HEALTH = f(DEF) \qquad (2)$$

All other things being equal, we expect that $\frac{dHEALTH}{dDEF} < 0$, implying that higher rates of deforestation ameliorates the quality of health.

From equation (1) and equation (2), we can conceptualize that:

$$LIFE = f(DEF) \quad (3)$$

Following Mariani et al. (2010), we then assume feedback effect from life expectancy to deforestation. All other things equal, we propose that:

$$DEF = f(LIFE) \quad (4)$$

The actual nature of the causal relationship between *LIFE* and *DEF* as defined in equations (3) and (4) may be linear or non-linear.

This paper, seeking to explain the role of trees in sustaining human life investigates the true nature of the causal relationship that exists between *LIFE* and *DEF* by using the bivariate Bootstrapped Rolling Window Granger causality test by Balcilar et al. (2010). The methodology of the paper is divided into three stages. The first stage requires the performance of the full sample bootstrapped Granger causality test. In the second stage, the parameters of the VAR model employed in the first stage is tested for short run as well as long run parameter constancy. In the final stage, in the presence of non-constancy of the parameters of the VAR model, the Bootstrapped Rolling Window Granger causality test is conducted to examine the true nature of the relationship between *LIFE* and *DEF*. The three steps are carefully explained below.

3.0 Methodology

3.1. Bootstrap full-sample Granger causality test

The traditional full sample Granger causality test has some defects which have been identified by Sims et al. (1990) and other researchers. After carefully examining the characteristics of standard

test statistics such as the Likelihood Ratio test and the Lagrange Multiplier test, the researchers unanimously concluded that standard test statistics employed in the traditional full sample Granger causality tests are likely to be denied the standard asymptotic distributions owing to certain impertinent changes structural changes which are always inevitably found in time series and VAR (Sims et al. (1990),Toda and Phillips (1993),Toda and Phillips (1994)). Consequently, Toda and Yamamoto (1995), in remedying the situation, proposed a modified Wald test. This modified Wald test possess the standard asymptotic distribution properties and is obtained by by estimating an augmented VAR model with variables integrated of order one. However, the effectiveness of this modified Wald test is still in doubt as prediction from Monte Carlo simulations show that it is bound to fail in small and medium samples. Shukur and Mantalos (2000) therefore derived the critical values of residual-based bootstrap method (the RB method), which are very effective even when the variables in the VAR model are not co-integrated (Mantalos and Shukur (1998);Shukur and Mantalos (2000); Mantalos (2000); Balcilar et al. (2010)). Shukur and Mantalos (2000) asserts that the excellency of the RB method is especially suitable for standard asymptotic tests and for power and size properties in sample corrected LR tests. Proceeding on the excellent properties of residual-based bootstrap method, the residual based bootstrapped method based on modified-LR statistics is adopted by this paper. The starting point of the full sample bootstrapped Granger causality test is the modelling of a Vector Auto Regressive (VAR) process between the two endogenous variables under consideration as follows:

$$X_t = \theta_0 + \theta_1 X_{t-1} + ... + \theta_p X_{t-p} + \varepsilon_t \qquad t=1, 2, ..., T \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (5)$$

p, the optimal lag order is determined using the minimum value of the Schwartz Information Criterion (SIC). In the VAR (*p*) process in equation (2) above , X represents a vector denoting

Life expectancy (*LIFE*) and deforestation (*DEF*), i.e. $X_t = (LIFE_t, DEF_t)'$. Then, we can rewrite the Equation (5) as follow:

$$\begin{bmatrix} LIFE_t \\ DEF_t \end{bmatrix} = \begin{bmatrix} \theta_{10} \\ \theta_{20} \end{bmatrix} + \begin{bmatrix} \theta_{11}(L) & \theta_{12}(L) \\ \theta_{21}(L) & \theta_{22}(L) \end{bmatrix} \begin{bmatrix} LIFE_t \\ DEF_t \end{bmatrix} + \begin{bmatrix} \varepsilon_{1t} \\ \varepsilon_{2t} \end{bmatrix} \quad \ldots\ldots(6)$$

where $\varepsilon_t = (\varepsilon_{1t}, \varepsilon_{2t})'$ is a white-noise process with zero mean and covariance matrix Σ. $\theta_{12,k} = \theta(L) = \sum_{k=1}^{p} \theta_{ij,k} L^k$, i, j=1, 2 and L denotes the lag operator, defined as $L^k X_t = X_{t-k}$. Using equation (6) we can test the null hypothesis that *LIFE* has no impact on *DEF* ($\theta_{12,k} = 0$ for k=1, 2, ... , p). Equation (6) also allows us to test the null hypothesis that null hypothesis that *DEF* exerts no influence on *LIFE* ($L_c \theta_{21,k} = 0$ for k=1, 2, ... , p).

3.2. Parameter stability tests

The fundamental assumption underlying the full sample bootstrapped Granger causality test approach is the short run and long run parameters of the VAR model. When this fundamental assumption breaks down, it leads to the invalidity of the results of the full sample Bootstrapped Granger causality test. To verify the short run stability of the parameters of the VAR, the *Sup-F*, *Ave-F* and *Exp-F* tests, developed by Andrews (1993) and Andrews and Ploberger (1994) is employed. To verify the long run stability of the parameters of the VAR model, the L_c test from Nyblom (1989) and Hanson (2002) is carried out.

3.3. Bootstrap sub-sample rolling-window causality test

Consequent to the short run and long run instability of the parameters in the VAR model, the invalidity of the results of the full sample bootstrapped granger causality test becomes more inevitable. As a result, a new time-varying approach, the Bootstrap sub-sample rolling-window causality test developed by Balcilar et al. (2010) is implemented. This new time-varying approach

adopts the bootstrapped version of the Toda and Yamamoto (1995) Granger causality testing approach. Given a full time series data of size *T*, the bootstrapped sub-sample rolling window Granger causality test segments the full data into small sample spaces based on the rolling window width of size *l.* Starting from the beginning of the time series data, the segmented sub samples are rolled to the end of the time series. The detailed steps Bootstrap sub-sample rolling-window Granger causality test are as follows: From the full sample, *T,* a sequence of *T-l* subsamples are generated, given by $t = \theta - l + 1, \theta - l, ..., \theta$;where $\theta = l, l+1, ..., T$. We can then obtain a causality test result of each sub-sample by employing the *RB*-based modified-*LR* test. The results of the bootstrap sub-sample rolling-window causality test can be obtained by summarizing all the observed *p*-values and *LR* statistics in chronological order. The average of quite a large number of estimation $N_b^{-1}\sum_{k=1}^{p}\hat{\theta}^*_{12,k}$ represents the effect from *LIFE* to *DEF* and $N_b^{-1}\sum_{k=1}^{p}\hat{\theta}^*_{21,k}$ has the opposite meaning, where N_b denotes the times of bootstrap repetitions. $\hat{\theta}^*_{12,k}$ and $\hat{\theta}^*_{21,k}$ are estimations from the bivariate VAR system. This study considers a 90% confidence interval, as well as the corresponding lower and upper bounds being the 5th and 95th quantiles of $\hat{\theta}^*_{12,k}$ and $\hat{\theta}^*_{21,k}$, respectively (Balcilar et al., 2010).

3.4. Data source for the study

To empirically verify the widely accepted saying that the health of forests is linked to the lifespan of humans, the paper employs annual data on forest area from FAOSTAT database of the Food Agricultural Organization (FAO) for the time period 1962 to 2011[5][6]. This data period is

[5] Data from 1961 to 1989 are from previous versions of the FAOSTAT database

[6] The ending period of the latest African Development indicators (ADI) is 2012.

strategically chosen because it is the longest time period available on the study variables, annual rate of deforestation (*DEF*)[7] and life expectancy (*LIFE*). The annual rate of deforestation is calculated as the annual percentage decrease in forest area, whereas Life expectancy (in years) measures how long individuals are expected to live given socio-economic and prevailing health conditions. To correct for skewness of the variables, the natural logarithms of the two endogenous variables are derived and used.

4.0. Empirical results

Table 1 presents the results of the full sample Bootstrapped granger causality test. The optimal lag chosen by the Schwartz Information Criterion is 2. A cursory observation of the results suggests that *DEF* granger causes *LIFE* at 10% level of significance, whereas *LIFE* is found not to granger cause *DEF* at 10% level of significance. The full sample bootstrap Granger causality test results show that causality runs from deforestation to life expectancy with no feedback from life expectancy to deforestation.

< Table 1 is inserted here>

There may be little doubt on the reliability of the results presented in Table 1 given that the results generated from the full sample bootstrapped Granger causality tests are based on the implicit assumption that the parameters of the VAR model in equation (6) are constant in the short and long run. The results of the short run and long run parameter stability test are reported in Table 2.

[7] $DEF = \frac{(F_{t-1} - F_t)}{F_{t-1}}$, where F_t and F_{t-1} are defined as the total forest area in Ghana at time and time t-1 respectively

< Table 2 is inserted here>

The results of the *Sup-F* test and *Ave-F* test, at 1% statistical significance level, shows the existence of structural changes and gradual evolution of the parameters of *DEF, LIFE* and the VAR system as a whole in the short run. The results in Table 2 further reveal that the long run parameters are significantly non-constant in the time series (*DEF* and *LIFE*) and the VAR system. That simply implies that the results of the full-sample causality tests are very unreliable and hence the sub-sample test, based on the RB-based modified LR method should be used to investigate the time-varying causality between *DEF* and *LIFE*. Following Pesaran and Timmermann (2005) and the work of Aye (2015), this paper chooses a rolling –window size of 15 years. The null hypothesis of the test is similar to the full sample bootstrap Granger causality test: *DEF* does not granger cause *LIFE* and vice versa. Figure 1 and 2 present the bootstrap probability value and the direction of the effect from *DEF* to *LIFE*, respectively. The results show that the null hypothesis that *DEF* does not Granger cause *LIFE* is rejected in the year 2004 at the 10% level of significance. In the 2004 sub sample period, *DEF* had a negative effect on *LIFE* in the period 2004 signifying that during that period deforestation threatened life expectancy. Even though deforestation threatens life expectancy in only one sample period, it can be said to be a weak confirmation of the widely accepted saying that reduction in forests is inextricably linked to the life spans of humans. The ameliorating effect of deforestation on life expectancy can be traced to the incidences of malaria that has been on the rise since the early 2000s in Ghana; infamously linked with the death of a number of Ghanaians. According to World Malaria Report on Ghana in 2008, malaria accounted for approximately 22% of death in Ghana in the year 2004. It is worth noting that the early 2000s in Ghana was in a period of massive industrialization and urbanization, which had a heavy toll on the forests of Ghana, aggravating the spread of vector borne disease. In this regard, we can attribute

the negative effect of *DEF* on *LIFE* to the negative effect that deforestation has on the quality of human health. It is also worth noting that trees serve as a common sink, thus purifying the air that we breathe. Deforestation therefore deprives us of this very important function of trees, leading to poor air quality, exacerbating respiratory illnesses and subsequently reducing life expectancies. Research shows that upper and lower respiratory diseases in Ghana accounted for approximately 8.14% of all deaths in Ghana in the year 2004.[8]

< Figure 1 and 2 are inserted here>

Figures 3 and 4 present the bootstrap probability value and the direction of the effect from *LIFE* to *DEF*, respectively. The results show that the null hypothesis that *LIFE h*as no impact on *DEF* is rejected in the 1988-1997, 2001-2003, and 2005-2011 sub sample periods at the 10% level of significance level. During these sub sample periods *LIFE* has a negative effect on *DEF* in the 1988 -1997 period, and positive effects in the 2001-2003 and 2005 -2011 periods. The 1988-1997 sub sample period corresponded to the latter days of the military era and the very early days of constitutional rule in Ghana. The military era was characterized by strict discipline by the military and severe punishments for flouting the law. The severe punishment for exploiting forest resources served as a deterrent to illegal loggers and as such forest resources were protected. The military thus instilled in the citizenry the culture of maintaining the quality of the environment. The same culture was carried on unto the early years of democratic rule and hence we observe a negative effect of life expectancy on the rate of deforestation in this sub sample period. People thus lived in those periods with the consciousness of protecting forest resources.

The 2001-2003 and 2005-2011 subsample periods corresponded to later years in the democratic dispensation in Ghana. With the military rule faded out, the strict protection of forest resources

[8] http://ghdx.healthdata.org/gbd-results-tool

relaxed and the punishment for exploiting forest resources reduced, citizens lost their awareness of the need to protect the forests. These sub-samples were characterized by urbanization and industrialization which inherently led to the clearing of forest resources for residential and industrial structures. The positive trend effect of *LIFE* on *DEF* in these two sub sample periods can be due to the fact that as people live for longer periods of time they tend to exert more pressure on the carrying capacity of natural resources like forestry for their daily survival hence leading to an increase in the rate of deforestation. The results of our study thus reveals that, while deforestation influences life expectancy, there exists feedback effect from life expectancy to deforestation.

< Figure 3 and 4 are inserted about here>

5.0 Conclusion and recommendation

This study tested the widely accepted saying that "when the last tree dies, the last man dies", implying that the state of forests is linked with the survival of the human race. The study employed the bootstrapped full sample and sub sample rolling window Granger causality tests using annual data on the two endogenous variables, *LIFE* and *DEF*. The results of the full sample bootstrap Granger causality tests showed that causality from *DEF* to *LIFE* and not from *LIFE* to *DEF*. The VAR model failing the parameter stability tests, we use the sub sample bootstrapped rolling window Granger causality test, revealing Granger causality running from *DEF* to *LIFE,* with feedback effect from *LIFE* to *DEF*. The study finds that the DEF has a negative effect on LIFE, implying that deforestation indeed leads to a reduction in life expectancy. On trend, LIFE is found to have a positive effect on DEF, implying that increases in life expectancy lead to an increase in deforestation over time. The study recommends extensive afforestation programs to reduce the rate of deforestation in Ghana. This, the study believes will go a long way to reduce the spread of

vector borne diseases such as Malaria which affects the life span of humans. Further, extensive afforestation programs will also reduce the impacts of urbanization and industrialization on the nation's forest resources. We also recommend increased awareness of the citizenry on the important role that trees play in the very existence of human kind.

APPENDIX

Table 1. Full-sample Granger causality tests

Tests	H_0: DEF does not Granger cause LIFE		H_0: LIFE does not Granger cause DEF	
	Statistics	*p*-value	Statistics	*p*-value
Bootstrap *LR* test	9.9452	0.0583	0.6293	0.732

The *p*-values for this test are generated using 10,000 bootstrap repetitions.

Table 2. The results of parameter stability test

	DEF		LIFE		VAR system	
	Statistics	*p*-value	Statistics	*p*-value	Statistics	*p*-value
Sup-F	39.6077***	0.0000	32.288***	0.0000	207.8371***	0.000
Ave-F	15.7373***	0.0000	14.511***	0.0000	53.3687***	0.000
Exp-F	15.935***	0.0001	12.511***	0.0002	100.6227***	0.000
L_c					3.6573***	0.005

The *p*-values for this test are generated using 10,000 bootstrap repetitions.

*** and * denote significance at the 1% level of significance.

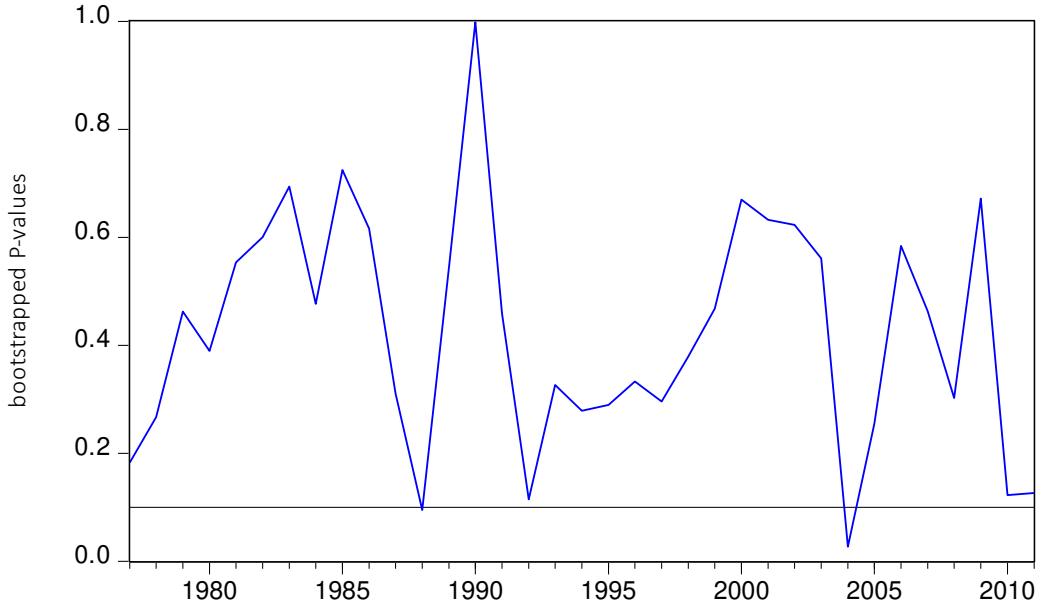

Figure 1. Bootstrap *p*-values of rolling test statistic testing the null hypothesis that DEF does not Granger cause LIFE

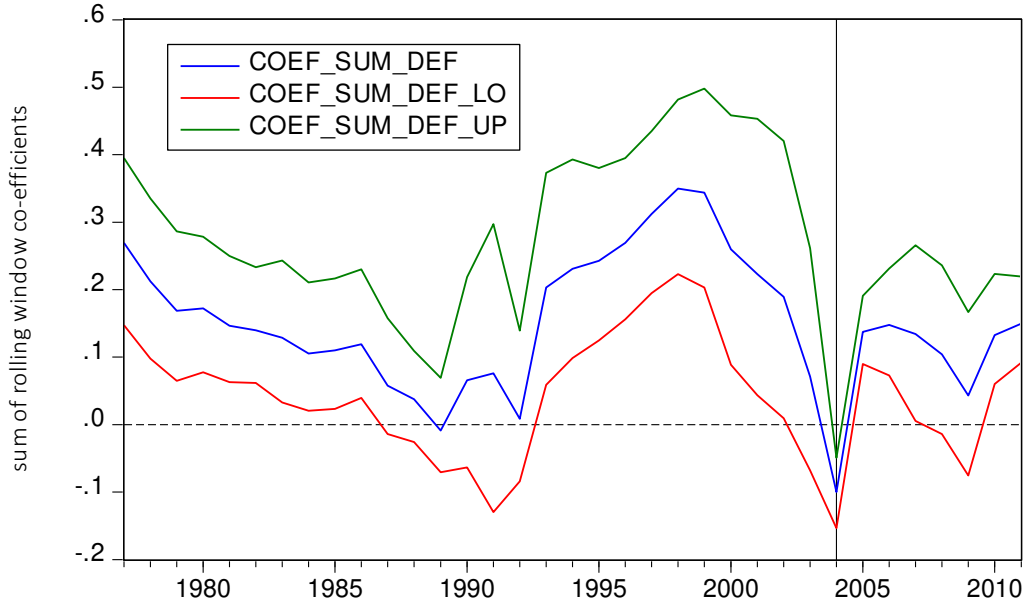

Figure 2. Bootstrap estimates of the sum of the rolling-window coefficients for the impact of DEF on LIFE

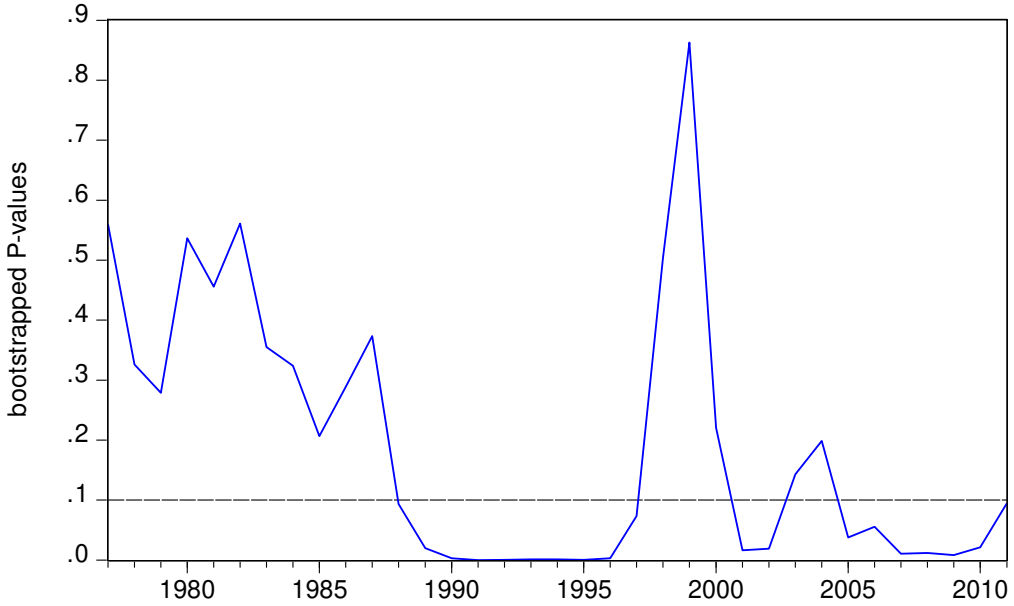

Figure 3. Bootstrap *p*-values of rolling test statistic testing the null hypothesis that LIFE does not Granger cause LIFE

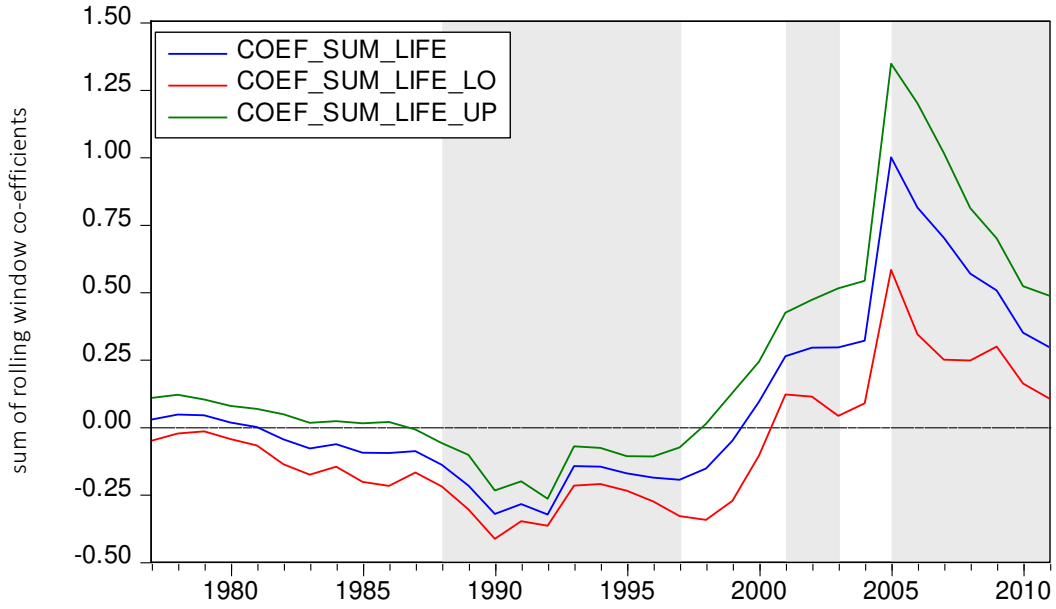

Figure 4. Bootstrap estimates of the sum of the rolling-window coefficients for the impact of LIFE on DEF

Declarations

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Consent for publication - Not Applicable

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Conflict of interest/competing interest

The authors declare no conflict of interest with regards to this study

Availability of data and material

The data that support the findings of this study are available in Harvard Dataverse repository with the identifier https://doi.org/10.7910/DVN/TSRVKN

Authors contributions

X.Z supervised the whole work. M.K.M conceptualized the whole work and performed all the econometric estimations. P.N. G and A.B did the literature review and did proof reading of the work.

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