

# HIV/AIDS AND MACROECONOMIC PERFORMANCE: EMPIRICAL EVIDENCE FROM KENYA

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**Abstract:** *This paper contributes to the investigation and exploration of the impacts of HIV/AIDS on the economic performance of Kenya. It evaluates the HIV/AIDS profile and socio-economic performance of the Kenyan economy before and during the epidemic era. The paper attempted a robust examination of the relationship between HIV/AIDS and major economic, education, demographic and health indicators in Kenya. The paper applied the step by step Wiener-Granger causality tests; and found that HIV/AIDS Granger-causes tuberculosis, life expectancy at birth, population growth, gross primary school enrolment and economic growth while urbanization, proportion of population under 15 years, rural population and gross primary school enrolment were identified as the determinants of the prevalence of HIV/AIDS in Kenya. The paper recommends a combined HIV mitigation surveillance approach and a post epidemic reconstruction of both human and social capital.*

**Keywords:** *HIV/AIDS Epidemic, Economic growth, Kenya, Wiener-Granger Causality.*

**JEL Classification:** *O15, J11, C18.*

## **Introduction**

Sub-Saharan Africa has been described as the epicenter of the prevalence of HIV/AIDS over the past decades [29]. It is however argued that, the degree of prevalence of the HIV epidemic was not correlatively distributed among the countries in the region [20], since some countries in Africa are believed to belong to the high HIV prevalence group while others are described as the low HIV prevalence countries. Countries with high HIV/AIDS prevalence were further categorized by [20] as very high HIV prevalence countries, moderately high HIV prevalence countries and low high HIV prevalence countries. Kenya once belonged to the middle high HIV prevalence countries category but has experienced a dramatic reversal of the incidence and severity of the epidemic.

## **1 Problem Statement**

Although Kenya has experienced a somewhat dramatic reversal of the epidemic: its impact on the economic performance of Kenya has been an issue of serious controversy. Some studies have argued that although Kenya is one of the first countries in Sub-Saharan Africa to experience the disease, its impacts on economic performance is only in the short-run. Other studies argued that the impact of the epidemic in Kenya was not widespread in terms of its sectoral coverage and dimension. Yet there are other studies who believe that the effects of the epidemic are not phenomenal in the affected economies.[32] observed that this explains why there is paucity of empirical studies on the impact of HIV/AIDS on economic performance in Kenya. However, no doubt HIV/AIDS has affected Kenya's economic development and is bound to inhibit future significant economic progress because compared to other diseases HIV/AIDS is a more recent epidemic in Kenya.

Therefore, the main objective of this paper is to examine the impact of the HIV epidemic on economic performance in Kenya since the early 1990s. The paper also evaluates its impact on population growth; population under 15, rural population and urbanization, and other selected macroeconomic indicators (such as per capita GDP, economy-wide savings, school enrolment, under-five mortality, life expectancy at birth, and the incidence of tuberculosis). Finally, the paper presents the methodological elucidation of the simplified (step by step) method of implementing the Wiener-Granger causality test using both HIV prevalence and selected indicators' data from Kenya. This is a major contribution to knowledge.

The rest of the paper is structured as follows: section two focuses on a review of Kenya's socio-economic performance in the era of the epidemic. It also examines some epidemiological characteristics of the HIV disease in Kenya. Section three describes the methodology of the research: Section four discusses the results and section five concludes the paper with recommendations and empirically-induced policy implications.

### **1.1 Kenya: HIV/AIDS Profile**

The HIV/AIDS epidemic in Kenya started in the early 1980s (precisely in 1984) among the sex hawkers' population [29]. Since then it has spread to the proportion of the population in their most productive/reproductive ages and to most areas of the country [32]. In 1999, the Kenyan government declared AIDS a national disaster. However, in the past few years, declines in HIV prevalence were observed in both high risk and low risk population groups as well as in urban and rural areas. Population-based HIV testing further confirms the decline in levels among the general population.

From the early 1990s to 2000, HIV sero-prevalence levels stabilized at around 16 percent among pregnant women tested in all four city council clinics in Nairobi (the capital city). By 2004, the rate declined to 10 percent. This decreased to 5 percent in 2008. HIV levels rose rapidly among sex "hawkers" in Nairobi between the mid-1980s and 1992 from 7 percent to nearly 86 percent. But it has dropped to less than 27 percent as at 2008. A similar trend was noticed in *Mombassa*, where over half of sex hawkers tested were HIV positive between 1993 and 1997. In 2000, the rate dropped to about 30 percent. For more information on Kenya's current HIV profile see [29].

### **1.2 Kenya Socio-Economic and Demographic Performance**

Since the epidemic started in Kenya, serious demographic changes specifically in terms of rising mortality rates and declining population growth have been experienced. Between 1960 and 1990, life expectancy at birth in Kenya rose steadily.

With respect to health human capital, life expectancy in Kenya started declining specifically few years after the HIV/AIDS epidemic started and is currently more than 10 years lower than the pre-HIV/AIDS era. The male life expectancy experienced -12% changes between 1990 and 2000; and a -11% change between 2000 and 2005 [36].

**Tab. 1: HIV/AIDS-Induced Life Expectancy Changes in Kenya (1980 – 2013)**

	1980	1990	2000	2005	2008	2010	2013	% Change 1990- 2000	% Change 1990- 2005	% Change 2010- 2013
Male	56	57	50	51	54	58	60	-12%	-11%	3.45%
Female	60	61	53	53	55	61	64	-13%	-13%	4.92%
Total	59	59	50	52	54	60	62	-15%	-12%	3.33%

Source: Author's Computation using WDI(2015)

From Table 1 above, it can be inferred that in Kenya, female life expectancy experienced an HIV/AIDS-induced change of -13% between 1990 and 2000; and between 2000 and 2005 respectively. Total life expectancy in Kenya experienced a -15% change between 1990 and 2000; and a -12% percentage change between 2000 and 2005. However, total life expectancy at birth experienced a 3.33% change during the 2010/2013 period. The statistical observations shown in Table 1 is in consonance with [2].

**Tab. 2: The Growth Rate of Population in Kenya (1960-2005)**

Population	YEARS					Percentage (%) Change			
	1980	1990	2000	2010	2013	80-90	90-00	00-10	00-13
(Annual Growth %)	3.78	3.36	2.58	2.70	2.70	-11%	-23%	4.65%	4.65%

Source: Author's Computation using WDI(2015)

The evaluation of the annual growth rate of population in Kenya shows that the pre-HIV growth rates of 1980 was above 3 percent per annum. However; from 1990 to 2000 it started declining. The HIV-induced percentage changes in population growth between 1980 and 1990, and 1990 to 2000 in Kenya experienced negative growth rates of -11% and -23% respectively. This means that HIV/AIDS has had a negative impact on Kenyan population growth during these periods ([33],[23],[2]. For instance, [23] concluded that Kenya had experienced serious demographic changes during the epidemic era through the decline in population growth rates as a result of rising death rates (for both adults and children).

### 1.3 Review of Related Literature

Among the earliest studies on HIV/AIDS in Sub-Saharan is [4],[5] and [6]. They used a one sector and two factor neoclassical growth model to predict the economic impact of HIV on growth in Malawi and Tanzania. They found that over the period 1985-2010, GDP growth would be reduced by about 1.5 percentage points in Malawi and 11 percentage points in Tanzania: [22] applying an eleven sector computable general equilibrium (CGE) to evaluate the impact of HIV in Cameroon. The study found that over a period of five years, the loss of an urban worker had seven times the negative impact on production as would the loss of a rural worker. The negative impact would be 100 times more when the lost workers were skilled and urban. [26] distinguished between three categories of workers and between urban and rural production in his model.

[1] used cross-national regressions to estimate relationships among economic growth and other parameters of interest such as policy, institutional variables and HIV prevalence [16],[17] and [18].

The impact of the HIV pandemic on the economy of Kenya has been an issue of serious debate and research interest since the epidemic began in the 1980s. Hence, apart from the region-wide or African-wide studies of the economic/macroeconomic impacts of HIV/AIDS on economic growth and other macroeconomic variables, there are country-specific studies. Regrettably, very few of these studies focus on the Kenyan epidemic scenario. For instance, [28] used a stochastic model of growth to assess the impact of the risks of an HIV epidemic on economic growth in Kenya [21].

[24] explored the geographical characteristics of HIV/AIDS in Kenya; and identified gender, age, marital status, education level, income, occupation, number of sexual partners, urbanization, demographic structure and migration as the major determinants of the prevalence of the disease in Kenya. The study adopted a qualitative method of estimation and found that: HIV/AIDS epidemic affected all the provinces as at 1990 (the period when the systematic surveillance started in Kenya) with the epidemic progressing at almost similar rates; HIV/AIDS epidemic has a positive relationship with income, urbanization, migration and poverty; religion has a reducing impact on HIV/AIDS prevalence, and densely populated regions had the highest prevalence of the disease.

[2] conducted a theoretical appraisal of the potential impact of AIDS on population and economic growth rates in Kenya and other countries and concluded that AIDS is likely to reduce the growth of the population of Kenya and other countries globally. The study also concluded that the impact of HIV/AIDS on economic growth is expected to manifest in many ways and dimensions (either directly or indirectly). Also, the study found that food security was threatened by the high prevalence of the pandemic in Kenya.

[1] explored the economic implications of HIV/AIDS in Kenya. The study focused on the impacts of the epidemic on economic agents (households and selected firms), and some selected sectors (agriculture) of the economy including the macro economy. It reviewed studies on HIV/AIDS as it relates to the Kenyan economy. The study found that the impact of the epidemic on economic agents and specific sectors of the economy was unexaggeratingly profound. It recommends treatment of the disease as a national priority; and it emphasizes the indispensability of government commitment in mitigating the HIV epidemic.

[7] used a retrospective cohort study design to investigate the productivity and work attendance of tea estate workers in Kenya. Out of the 271 tea pluckers studied between 1997 and 2002, 54 died of HIV/AIDS. The study used longitudinal regression and found that HIV infection reduces the productivity rate and income earned by such tea pluckers.

[25] obtained results that are similar to [7]. The study assessed the impact of HIV/AIDS on the development of micro-enterprises in Kenya. The Obunga slum in *Kisumu* used as the case study is one of the most populated slums in Kisumu. A total of 50 micro-enterprises were randomly selected out of 250. The study found that apart from HIV/AIDS, other opportunistic diseases such as malaria, tuberculosis affect micro-enterprises in Kisumu, Kenya. However; the sample sizes of these studies are not large enough to inform policy conclusions for country like Kenya.

[4] assessed the long-run economic impacts of HIV/AIDS in Kenya with focus on fertility, education and child labor. Human capital accumulation was treated as input into the production process. The study used a calibrated model in analysing data for the period 1920 to 2000. The long-run impact of the epidemic was estimated for the period spanning

from 2000 to 2040. The study concludes that the impact of the HIV epidemic in Kenya (economy and people) is unarguably substantial and monumental.

## 2 Material and Methods

### 2.1 Source and Description of Data

The data used for this study were mainly drawn from the World Development Indicators (WDI) and the African Development Indicators (ADI) of the World Bank for various years. But the data on HIV prevalence were drawn from both UNAIDS and the US Census Bureau. The study covered the period 1990-2013. The series are grouped into economic, demographic, education and health indicators.

### 2.2 Estimation Technique

#### 2.2.1 Wiener-Granger Causality Tests.

[8],[9],[10] and [11] have assumed that, in causal test like the Wiener-Granger, there are two series  $X_t$  and  $Y_t$  ( $X_t$  represents the vector for HIV/AIDS and  $Y_t$  is the vector of the other indicators selected for this study).  $X_t$  fails to (successfully) Granger-cause  $Y_t$  if the conditional expectation of equality holds (does not hold).

$$E(Y_t | Y_{t-1}, X_{t-1}, X_{t-2}, \dots, X_{t-n}) = E(Y_t | Y_{t-1}) \quad (1)$$

According to [14], apart from the step by step implementation and application of the Wiener-Granger causality test, the following conditions must be satisfied:

#### Condition one:

The variables included in the bivariate model must be stationary or integrated of order zero or one (i.e. must be integrated of the same order).

#### Condition two:

The null hypothesis of the Wiener-Granger causality test must be stated and tested.

#### Condition three:

The number of lagged terms must be accurately determined as the direction of causality may depend on the appropriate selection of lag lengths.

#### Condition four:

The error terms entering the Wiener-Granger causality model must be uncorrelated ([13],[8],[9],[10],[11]). These conditions are implemented step-by-step below:

**Step One: Determine the stationarity or order of integration of the variables included in the Wiener-Granger causality model.** The stationarity test is a necessary condition for the implementation of the Wiener-Granger causality test; as such it is expected to precede the Wiener-Granger causality test. One of the most prominently applied stationarity tests in the econometric literature is the unit root test. The unit root test adopted in this study is the Augmented Dickey Fuller (ADF) test. [20] in line with [14] opined that a Standard implementation of the ADF unit root test is done in stages with relevant and suitably adequate hypotheses stated. The ADF unit root test models are stated as:

$$\Delta Y_t = \phi Y_{t-1} + u_t \quad (2)$$

$$\Delta Y_t = \alpha_1 + \phi Y_{t-1} + u_t \quad (3)$$

$$\Delta Y_t = \alpha_1 + \alpha_2 t + \phi Y_{t-1} + u_t \quad (4)$$

The hypothesis of each case of the stationarity test includes:

Null hypothesis:  $H_0 : \phi = 0$  :

The null hypothesis states that there is a unit root or the series is not stationary.

Alternative hypothesis:  $H_A : \phi < 0$  :

The alternative hypothesis states that the time series is stationary around a deterministic trend or there is no unit root. But if the assumption of uncorrelated error terms is violated, the ADF extends the above Dickey and Fuller unit root models as shown below:

$$\Delta Y_t = \alpha_1 + \alpha_2 t + \phi Y_{t-1} + \sum_{i=1}^n \beta_i \Delta Y_{t-i} + u_t \quad (5)$$

Where  $u_t$  represents pure white noise error term.

$$\begin{aligned} \Delta Y_{t-1} &= (Y_{t-1} - Y_{t-2}), \text{ and} \\ \Delta Y_{t-1} &= (Y_{t-2} - Y_{t-3}) \dots \end{aligned} \quad (6)$$

The rule of thumb for the determination of stationarity (or non-stationarity ) is that, if the ADF statistic is smaller than the McKinnon critical values or if the ADF statistic is negative and has smaller value than the McKinnon critical values that are negative, then we reject the null hypothesis of the presence of unit root.

**Step Two: State the null hypothesis of the Wiener-Granger causality test.** The general null hypothesis is stated as:

$$H_0 : \alpha_i = 0 \forall i = 1, 2, 3, \dots, n$$

The general null hypothesis which represents equations(9),(12),(14),(16),(18),(20),(22),(24),(26),(28),(30) and (32) states that the lagged values of HIV prevalence do not belong to the regression.

**Step Three: Determine the appropriate lag length.** EvIEWS 8 provides automatic lag selection method and as such, only a complementary effort is needed.

**Step Four: Determine the nature of correlation (non-correlation) between the error terms.** In line with [12]; we assume that the error terms are uncorrelated; each error term has the classical structure(satisfied the assumption of absence of autocorrelation and of the presence of homoskedasticity). The VAR system covariance matrix is stated as:

$$\Omega = \begin{pmatrix} \alpha_{11} & \alpha_{12} \\ \alpha_{21} & \alpha_{22} \end{pmatrix} \otimes I = \sum \otimes I \quad (7)$$

where the covariance matrix connotes that there is within system of equation correlation.

**Step Five: Specify the Bivariate or Bilateral Wiener-Granger causality models.** Generally, the Wiener-Granger bivariate causality regression equations are stated as:

$$X_t = \phi_1 + \gamma_t X_{t-1} + \sigma_1 Y_{t-1} + u_{1t} \quad (8a)$$

$$Y_t = \phi_2 + \Theta_t Y_{t-1} + \alpha_1 X_{t-1} + u_{2t} \quad (8b)$$

But in this study, the bivariate models are specified according to the following categories: economic, education, demographic and health categories.

### 2.2.2 Granger causality of HIV prevalence and economic indicators

#### Bivariate Model of HIV prevalence and Income:

$$HIV_t = \sum_{i=1}^l \sigma_i PGDP_{t-i} + \sum_{j=1}^l \Phi_j HIV_{t-j} + u_{1t} \quad (9)$$

$$PGDP_t = \sum_{i=1}^l \psi_i PGDP_{t-i} + \sum_{j=1}^l \delta_j HIV_{t-j} + u_{2t} \quad (10)$$

where  $HIV_t$  represents current HIV prevalence rate.  $PGDP_t$  is current real per capita income (a proxy for income).

Equation(9) implies that the current HIV prevalence ( $HIV_t$ ) is related to the previous values of HIV prevalence ( $HIV_{t-i}$ ) and the past values of real GDP per capita ( $PGDP_{t-i}$ ). Equation(10) posits that the current real GDP per capita ( $PGDP_t$ ) is related to the past values of both real GDP per capita ( $PGDP_{t-i}$ ) and HIV prevalence ( $HIV_{t-i}$ ). Equations (9) and (10) implies that sex is a normal good with own price, and the income of the demanders as major determinants of its demand. The higher the income of the demander, the more the sex demanded (perhaps including risky sex). Therefore, as income increases, the demand for risky sex is expected to increase including the ability to keep "pockets of concubines" and sex partners. On the other hand, [1] has noted that HIV/AIDS can facilitate the increase in income through increased mortality and its impact on the denominator factor of GDP per capita.

#### Bivariate Model of HIV prevalence and economic growth:

$$GDPC_t = \sum_{i=1}^l \alpha_i GDPC_{t-i} + \sum_{j=1}^l \beta_j HIV_{t-j} + u_{1t} \quad (11)$$

$$HIV_t = \sum_{i=1}^l \phi_i GDPC_{t-i} + \sum_{j=1}^l \eta_j HIV_{t-j} + u_{2t} \quad (12)$$

where GDPC represents economic growth (this is defined as the growth rate of per capita). Equation(11) postulates that in the era of HIV prevalence, current performance of economic growth ( $GDPC_t$ ) is related to the past values of economic growth ( $GDPC_{t-i}$ ) and the previous prevalence rates of HIV ( $HIV_{t-i}$ ). It has been hypothesized that previous prevalence rates of HIV can act as distortion or inhibition to current economic performance i.e. inhibiting and depleting the numerator component of GDP per capita. Also, previous prevalence rate of HIV can affect current economic performance through its effects on the denominator factor of GDP per capita (through HIV-induced mortality and morbidity). Equation(12) hypothesizes that the current prevalence rate of HIV ( $HIV_t$ ) is a function of the previous performance of economic growth ( $GDPC_{t-i}$ ) and the past prevalence rates of HIV ( $HIV_{t-i}$ ). This means that those who are currently infected were infected by those who were previously infected, that is, current HIV prevalence rates are the multiplier effects of previous HIV prevalence rates. Indeed, equation(12) connotes that current HIV prevalence rates are linked to either the worsening economic performance

of previous periods( $GDP_{t-i}$ ),that is, poverty-induced supply of risky sex or the economic prosperity of previous periods, that is, prosperity-induced demand for risky sex.

**Bivariate Model of HIV prevalence and economy-wide savings:**

$$GDS_t = \sum_{i=1}^l \alpha_i GDS_{t-i} + \sum_{j=1}^l \beta_j HIV_{t-j} + u_{1t} \tag{13}$$

$$HIV_t = \sum_{i=1}^l \phi_i GDS_{t-i} + \sum_{j=1}^l \eta_j HIV_{t-j} + u_{2t} \tag{14}$$

where GDS is Gross Domestic Savings (defined as economy-wide savings).

Equation(13) and (14) posits that in the era of HIV prevalence(epidemic),current economy-wide savings ( $GDS_t$ ) is related to the previous values of economy-wide savings( $GDS_{t-i}$ ) and the past values of HIV prevalence rates ( $HIV_{t-i}$ ).This means that previous HIV prevalence lead to current dis-accumulation of capital(savings).Equations (13) and (14) hypothesize the link between HIV prevalence and savings. Although, the link between HIV/AIDS and savings is controversial. Most studies including [2] and [23] found a negative causal link between HIV/AIDS and savings. But other studies argued that, the relationship between HIV/AIDS and savings is determined by the theoretical perspective adopted [20].

**The bivariate Model of HIV prevalence and the growth of export:**

$$GEXP_t = \sum_{i=1}^l \Omega_i GEXP_{t-i} + \sum_{j=1}^l \Theta_j HIV_{t-j} + u_{1t} \tag{15}$$

$$HIV_t = \sum_{i=1}^l \nu_i GEXP_{t-i} + \sum_{j=1}^l \delta_j HIV_{t-j} + u_{2t} \tag{16}$$

$GEXP_t$ represents the current growth of export. Equation(15) posits that the current growth of export ( $GEXP_t$ ) is related to the past values of both growth of export ( $GEXP_{t-i}$ ) and HIV prevalence( $HIV_{t-i}$ ).Equation(16) implies that the current HIV prevalence ( $HIV_t$ ) is related to the previous values of HIV prevalence ( $HIV_{t-i}$ ) and the growth of export ( $GEXP_{t-i}$ ).

**2.2.3 HIV and Education Human Capital**

**The bivariate model of HIV prevalence and gross primary education.**

$$GPRS_t = \sum_{i=1}^l \mu_i GPRS_{t-i} + \sum_{j=1}^l \varpi_j HIV_{t-j} + u_{1t} \tag{17}$$

$$HIV_t = \sum_{i=1}^l \phi_i GPRS_{t-i} + \sum_{j=1}^l \chi_j HIV_{t-j} + u_{2t} \tag{18}$$

$GPRS_t$ represents Gross Primary School Enrolment rates. Equation(17) posits that the current Gross Primary School Enrolment rates ( $GPRS_t$ ) is related to the past values of both Gross Primary School Enrolment rates ( $GPRS_{t-i}$ ) and HIV prevalence( $HIV_{t-i}$ ).Equation(18) implies that the current HIV prevalence ( $HIV_t$ ) is related to the previous values of HIV prevalence ( $HIV_{t-i}$ ) and the past values Gross Primary School Enrolment rates ( $GPRS_{t-i}$ ).



## 2.2.4 HIV and Demographic Indicators in Kenya

### Bivariate Model of HIV prevalence and population growth

$$PPG_t = \sum_{i=1}^l \varphi_i PPG_{t-i} + \sum_{j=1}^l \varpi_j HIV_{t-j} + u_{1t} \quad (19)$$

$$HIV_t = \sum_{i=1}^l \theta_i PPG_{t-i} + \sum_{j=1}^l \rho_j HIV_{t-j} + u_{2t} \quad (20)$$

PPG represents population growth.

Equation(19) posits that the current population growth rates ( $PPG_t$ ) is related to the past values of both population growth ( $PPG_{t-i}$ ) and HIV prevalence rates( $HIV_{t-i}$ ).Equation(20) implies that the current HIV prevalence ( $HIV_t$ ) is related to the previous values of HIV prevalence ( $HIV_{t-i}$ ) and the past values of population growth rates ( $PPG_{t-i}$ ).Equations (19) and (20) explain the causal linkage between HIV/AIDS and population growth. This relationship has been hypothesized to be negative with causality running from HIV/AIDS to population growth. On the other hand, positive causation is expected from population growth (birth and migration) to HIV/AIDS ([24]; [2]).

### Bivariate Model of HIV prevalence and Urbanization:

$$UPP_t = \sum_{i=1}^l \gamma_i UPP_{t-i} + \sum_{j=1}^l \varpi_j HIV_{t-j} + u_{1t} \quad (21)$$

$$HIV_t = \sum_{i=1}^l \delta_i UPP_{t-i} + \sum_{j=1}^l \ell_j HIV_{t-j} + u_{2t} \quad (22)$$

UPP is urbanization (this is defined as the proportion of the population that is urban).

It has been hypothesized that urbanization causes HIV/AIDS and vice versa; [24] observed that there is a higher concentration of HIV infected persons in the urban areas than in the rural areas of Kenya. This is the theoretical basis of the bivariate regression models specified as equation (21) and (22) respectively.

### Bivariate Model of HIV prevalence and the proportion of population that is rural:

$$RRP_t = \sum_{i=1}^l \alpha_i RRP_{t-i} + \sum_{j=1}^l \varpi_j HIV_{t-j} + u_{1t} \quad (23)$$

$$HIV_t = \sum_{i=1}^l \delta_i RRP_{t-i} + \sum_{j=1}^l \ell_j HIV_{t-j} + u_{2t} \quad (24)$$

$RRP_t$  represents the proportion of the population that is rural. Equation(23) postulates that the current value of the proportion of the population that is rural ( $RRP_t$ ) is related to the past values of both proportion of the population that is rural ( $RRP_{t-i}$ ) and HIV prevalence ( $HIV_{t-i}$ ).Equation(24) posits that the current HIV prevalence rate ( $HIV_t$ ) is related to the previous values of HIV prevalence ( $HIV_{t-i}$ ) and proportion of the population that is rural ( $RRP_{t-i}$ ).

**Bivariate Model of HIV prevalence and the proportion of population that is under 15:**

$$P15_t = \sum_{i=1}^l \infty_i P15_{t-i} + \sum_{j=1}^l \kappa_j HIV_{t-j} + u_{1t} \quad (25)$$

$$HIV_t = \sum_{i=1}^l \phi_i P15_{t-i} + \sum_{j=1}^l \varsigma_j HIV_{t-j} + u_{2t} \quad (26)$$

$P15_t$  is the proportion of the population that is under the age of 15. Equations (25) and (26) hypothesize that early sexual debut of the Young persons in Kenya and in most African countries is a major determinant of the spread of HIV/AIDS and rapid infection of the proportion of the population under the age of 15. Also, through the morbidity and eventual mortality, HIV/AIDS acts as a determinant of the performance in terms of longevity and mortality) of the proportion of population that is under 15 years in Kenya. [24] argued that the HIV pandemic is mostly affecting the younger generation with younger women more vulnerable because they are more sexually active at that age.

**2.2.5 HIV prevalence and Health indicators in Kenya**

**Bivariate Model of HIV prevalence and Tuberculosis:**

$$TBC_t = \sum_{i=1}^l \xi_i TBC_{t-i} + \sum_{j=1}^l \pi_j HIV_{t-j} + u_{1t} \quad (27)$$

$$HIV_t = \sum_{i=1}^l \wp_i TBC_{t-i} + \sum_{j=1}^l \beta_j HIV_{t-j} + u_{2t} \quad (28)$$

$TBC_t$  is tuberculosis (defined as the current incidence of tuberculosis). Equation(27) posits that the current incidence of tuberculosis ( $TBC_t$ ) is related to the past values of both incidence of tuberculosis ( $TBC_{t-1}$ ) and HIV prevalence( $HIV_{t-1}$ ). Equation(28) implies that the current rates of HIV prevalence ( $HIV_t$ ) is related to the previous own values ( $HIV_{t-1}$ ) and incidence of tuberculosis ( $TBC_{t-1}$ ).

**Bivariate Model of HIV prevalence and life expectancy at birth:**

$$LEP_t = \sum_{i=1}^l \eta_i LEP_{t-i} + \sum_{j=1}^l \omega_j HIV_{t-j} + u_{1t} \quad (29)$$

$$HIV_t = \sum_{i=1}^l \varsigma_i LEP_{t-i} + \sum_{j=1}^l \vartheta_j HIV_{t-j} + u_{2t} \quad (30)$$

$LEP_t$  represents current life Expectancy at Birth. Equation (29) surmises that the current performance life expectancy at birth ( $LEP_t$ ) is related to the past values of both life expectancy ( $LEP_{t-1}$ ) and HIV prevalence( $HIV_{t-1}$ ). Equation(30) surmises that the current rate of HIV prevalence ( $HIV_t$ ) is related to the previous values of HIV prevalence ( $HIV_{t-1}$ ) and under-five mortality( $LEP_{t-1}$ ).

**Bivariate Model of HIV prevalence and under-five mortality rate**

$$UFM_t = \sum_{i=1}^l \alpha_i UFM_{t-i} + \sum_{j=1}^l \psi_j HIV_{t-j} + u_{1t} \quad (31)$$

$$HIV_t = \sum_{i=1}^l \lambda_i UFM_{t-i} + \sum_{j=1}^l \Phi_j HIV_{t-j} + u_{2t} \quad (32)$$

UFM represents under-five mortality rates.

Equation(31) presupposes that the current rate under-five mortality ( $UFM_t$ ) is related to the past rates of both under-five mortality ( $UFM_{t-1}$ ) and HIV prevalence( $HIV_{t-1}$ ).Equation(32) presupposes that the current HIV prevalence ( $HIV_t$ ) is related to the previous periods rates of HIV prevalence ( $HIV_{t-1}$ ) and under-five mortality( $UFM_{t-1}$ ).

### 3 Problem Solving/Discussion

This section is mainly focused on the empirical results of the Augmented Dickey Fuller unit root test and the Wiener-Granger causality test. The ADF results are presented in Table 3 below.

**Tab. 3: Results of the Augmented Dickey Fuller Unit Root Test**

INDICATOR	VARIABLE	LEVEL		FIRST DIFFERENCE		DECISION
		Intercept	Intercept/ Trend	Intercept	Intercept/ Trend	
ECONOMIC	GDPC	-1.979	-3.157	-5.414*	-4.452**	GDPC has unit root at level
	GEXP	-1.2604	-1.254	-8.055*	-7.733*	GEXP has unit root at level
	PGDP	-2.204	-1.074	-2.393	-3.947**	PGDP has unit root at level
	GDS	-1.623	-2.293	-4.971*	-4.871**	GDS has unit root at level
EDUCATION	GPRS	-1.7887	-4.480	-6.370*	-6.108*	GPRS has unit root at level
DEMOGRAPHIC	PPG	-2.753	-0.175	-2.709	-4.308**	PPG has unit root at level
	P15	-1.039	-0.156	-2.006	-3.914**	P15 has unit root at level
	RRP	-2.207	-0.168	-1.279	-4.662**	RRP has unit root at level
	UPP	-2.114	-1.372	-3.416**	-5.729*	UPP has unit root at level
HEALTH	HIV	-2.244	-1.531	-4.872*	-5.727*	HIV has unit root at level
	TBC	-2.271	-1.604	-4.727*	-3.482**	TBC has unit root at level
	UFM	-3.018	-3.200	-4.968*	-4.460*	UFM has unit root at level
	LEP	-1.075	-2.822	-7.031*	-6.957*	LEP has unit root at level

Source: Author

NOTE: \* denotes significance at 1% and \*\* denotes significance at 5%.

For all the series shown in Table 3 above, the results indicated that the series are non-stationary at level(but are stationary at first difference either with intercept or with intercept and trend).Hence, we do not reject the null hypothesis that unit root is present in each of our series. Since all the series are stationary at first difference (integrated of order one); the first condition for the implementation of the Wiener-Granger causality test has been satisfied. Therefore, we implement the Wiener-Granger causality test as reported below.

#### 3.1 Results of the Bivariate Wiener-Granger Causality Tests

The results of the Wiener-Granger causality test are presented in Table 4 below. The discussions are succinctly presented in the ensuing section.

**Tab. 4: The Results of the Bivariate Wiener-Granger Causality Tests**

<b>HIV PREVALENCE VERSUS ECONOMIC INDICATORS</b>			
<b>Null Hypothesis</b>	<b>F-Statistics</b>	<b>Direction of causality</b>	<b>Decision</b>
HIV Does not Granger cause GDPC	0.36	-	Do not Reject
GDPC Does not Granger cause HIV	0.19	-	Do not Reject
HIV Does not Granger cause GEXP	0.822	-	Do not Reject
GEXP Does not Granger cause HIV	16.53*	GEXP→HIV	Reject
HIV Does not Granger cause GDS	0.69	-	Do not Reject
GDS Does not Granger cause HIV	1.90	-	Do not Reject
PGDP Does not Granger cause HIV	20.46*	PGDP→HIV	Reject
HIV Does not Granger cause PGDP	0.51	-	Do not Reject
<b>HIV PREVALENCE VERSUS EDUCATION INDICATOR</b>			
<b>Null Hypothesis</b>	<b>F-Statistics</b>	<b>Direction of causality</b>	<b>Decision</b>
HIV Does not Granger cause GPRS	3.46***	HIV→GPRS	Reject
GPRS Does not Granger cause HIV	0.299	-	Do not Reject
<b>HIV PREVALENCE VERSUS DEMOGRAPHIC INDICATORS</b>			
<b>Null Hypothesis</b>	<b>F-Statistics</b>	<b>Direction of causality</b>	<b>Decision</b>
PPG Does not Granger cause HIV	0.30	-	Do not Reject
HIV Does not Granger cause PPG	8.79*	HIV→PPG	Reject
HIV Does not Granger cause P15	18.37*	HIV→P15	Reject
P15 Does not Granger cause HIV	0.77	-	Do not Reject
HIV Does not Granger cause RRP	16.72*	HIV→RRP	Reject
RRP Does not Granger cause HIV	0.344	-	Do not Reject
HIV Does not Granger cause UPP	36.36*	UPP→HIV	Reject
UPP Does not Granger cause HIV	10.64*	HIV→UPP	Reject
<b>HIV PREVALENCE VERSUS HEALTH INDICATORS</b>			
<b>Null Hypothesis</b>	<b>F-Statistics</b>	<b>Direction of causality</b>	<b>Decision</b>
HIV Does not Granger cause TBC	3.76***	TBC→HIV	Reject
TBC Does not Granger cause HIV	9.50**	HIV→TBC	Reject
HIV Does not Granger cause LEP	11.12*	HIV→LEP	Reject
LEP Does not Granger cause HIV	0.32	-	Do not Reject
HIV Does not Granger cause UFM	12.71*	HIV→UFM	Reject
UFM Does not Granger cause HIV	0.54	-	Do not Reject

*Source: Author's computation using Eviews 8.*

**NOTE:** \* denotes significance at the 1% level, \*\* at the 5% level and \*\*\* at the 10% level. All the variables are as previously defined. Where  $\rightarrow$  denotes one-way causation.

The results of the Wiener-Granger causality test shown above suggest that there exists a two-way statistically insignificant causality between HIV prevalence and GDPC and GDS, that is, HIV/AIDS does not “Granger cause” economic growth(GDPC) and gross domestic savings(GDS); neither are these indicators determinants of the spread of HIV/AIDS in Kenya. The results prognosticate that the causal relationship between HIV prevalence and either GDPC or GDS is both statistically insignificant and empirically picayune. Therefore, we do not reject the null hypotheses that HIV prevalence does not Granger-cause economic growth and gross domestic savings in Kenya during the period under consideration. When our results were compared to the findings of [23] that found a negative relationship between HIV/AIDS and economic growth; they seem to suggest that the negative impact of the epidemic on economic growth in Kenya has reached its peak and is undergoing a downward trajectory. It also suggests that the mitigation steps taken so far have been efficient and effective. Furthermore, our results empirically suggest that the grueling consequences of the HIV epidemic on gross domestic savings have been reasonably inundated in Kenya.

Amazingly, there is a unidirectional causality flowing from the growth of export (GEXP) and income (PGDP) to HIV prevalence. Thus, reject the null hypothesis. This means that the growth of export in Kenya is a determinant of the prevalence of the HIV disease. This is a major contribution of this study to knowledge. Income (PGDP) “Granger-causes” HIV/AIDS. This result supports the income-led HIV prevalence hypothesis. Also, the results seem to support the theoretical premise that sex is a normal good whose demand is determined by the income of the demander and its own price. The higher the income of the demander, the higher the risky sex demanded through the keeping of “pockets of sex partners”. From the supply nexus, income also drives the supply of risky sex; this in turn facilitates the spread of the disease. This has serious policy implications. This suggests that economic prosperity boost the demand for risky sex while poverty drives the supply of risky sex. There is no reverse causality from HIV prevalence to income (PGDP).

There is also a statistically significant unidirectional causality flowing from HIV prevalence to the education capital variable (GPRS). This is not surprising because during the epidemic, Kenya was one of the first countries that made primary education compulsory and with incentives. Therefore, as the epidemic ascends the upward trajectory, primary school enrolment was also increasing.

HIV prevalence “Granger” causes life expectancy but life expectancy (LEP) does not “Granger” cause HIV prevalence. This means that HIV prevalence has sinister impacts on the life expectancy of the population in Kenya (See [31] for details). This might be the reason why our results indicate a significant impact at the 1% level.

There is no causality from population growth (PPG) to HIV prevalence but causality runs from HIV prevalence to PPG in Kenya. Population growth (PPG) consists of mortality, fertility and migration. This implies that as the tempo of the epidemic heightens in Kenya, population growth dynamics are severely impacted upon through its impact on mortality, fertility and migration. The empirical evidences on this are overwhelming in both the economic and public health literatures [2, 24 and 32].

In Kenya, HIV prevalence “Granger causes” P15 at 1% level of significance which connotes that HIV prevalence depletes the young people in the population. A corollary explanation to this is that HIV prevalence has impacted severely and negatively on the proportion of the population that is in their “prime age”. Hence, the results further suggest that as HIV/AIDS escalates into widespread epidemic, the proportion of the population under 15 (P15) is depleted.

There exists a bidirectional causality between the incidence of tuberculosis and HIV prevalence. This validates the empirical findings in the health literature that both tuberculosis and HIV prevalence are opportunistic diseases for the incidence and prevalence of each other. Equation (27) and (28) are corroborated by our findings.

## **Conclusion and Recommendations**

This study investigated the multi-dimensional impacts of the HIV/AIDS epidemic on the growth and performance of the Kenyan economy for the period 1990 to 2013. The main objective of the study is to evaluate the type and direction of the relationship existing between HIV prevalence and selected indicators. The Wiener-Granger causality test was applied as the estimation technique in the determination of the causal linkages among the selected series. The selected indicators were categorized into demographic, economic, education and health series.

The principal results obtained from the study include the followings: First, there is bidirectional (two-way statistically significant) causality existing between HIV prevalence and the following indicators (urbanization and tuberculosis). Second, there is a one-way statistically significant causality from HIV prevalence to gross primary school enrolment, population growth, proportion of the population under the age of 15 years, proportion of the population that is rural, life expectancy at birth and under-five mortality. Hence, the study concludes that HIV prevalence Granger-causes gross primary school enrolment, life expectancy at birth, population growth, proportion of the population under the age of 15 years and proportion of the population that is rural. Also, the HIV epidemic "Granger-causes" the incidence of tuberculosis (tuberculosis meningitis, intracerebral and Mycobacterium tuberculosis), and urbanization. Third, there is a one-way causal linkage flowing from the growth of export and income to HIV prevalence. This implies that these indicators are among the determinants of the prevalence of the disease in Kenya. But there is no causality between HIV prevalence and gross domestic savings rate and economic growth in Kenya. This means that HIV epidemic has not assumed a sinister dimension in Kenya. The study recommends that in order to obtain sustainable results, government, non-governmental organizations and donor agencies should committedly and consistently intervene in the mitigation of the epidemic.

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