ABSTRACT

This study investigated the relationship among health expenditure, HIV prevalence and expansion of productive economic activities in West Africa using sub-regional aggregate time series data from 1992 to 2020. The augmented Solow growth model was adopted to enable us to understand and maximize both the theoretical and empirical anatomy of HIV prevalence as it relates with the expansion of productive economic activities and disaggregated health expenditure in West Africa in the context of a growth theory and to conveniently apply the Hsiao causality framework in the form of a vector autoregressive model. The augmented Solow model theoretically made it possible for us to uniquely identify and specify six (6) bivariate hypotheses-supported models as sustainable fulcrums for the empirical application and validation of the Hsiao causality test. Health expenditure was disaggregated into total, public, household out-of-pocket, and non-household private health expenditures to specifically determine the impact of the relationship between the expansion of productive economic
activities and HIV prevalence on each of these components of health expenditure. The results show that the total health expenditure-led growth and private health expenditure-led growth hypotheses were not validated for the sub-region. However, the growth-led total, growth-led public, and growth-led private health expenditure hypotheses are validated for the sub-region. Also, HIV prevalence-led total, HIV prevalence-led public, and HIV prevalence-led private health expenditure hypotheses are validated for West Africa. The study recommended that for sustainable inflows of total, public and private health expenditure into the sub-region, the expansion of productive and growth-enhancing economic activities and HIV mitigation policies should incorporate health expenditure policies both in the long, and short-run periods.

**Keywords:** Hsiao causality, HIV Prevalence, Health Expenditure, Economic Growth, Solow Model.

**JEL CLASSIFICATION:**C11, C18, I10, I150

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**INTRODUCTION**

The nature of the hypothesis-led inter-connectivity, joint movement and co-variation between either health expenditure (henceforth $H_{exp}$) and expansion of productive and growth-enhancing economic activities (henceforth GDP) or HIV prevalence ($PHIV$) and $H_{exp}$ has been an issue of continuous discourse anchored on the seminal study of Mushkin (1962) since it evolved. Concerning the interconnectivity and co-variation between $H_{exp}$ and GDP, four hypotheses-led interconnectivities and co-variations have been identified and validated in the literature: Health-led growth hypothesis, growth-led health hypothesis, feedback hypothesis and a neutral hypothesis. The health expenditure-led growth hypothesis is widely discussed in both health, economic and econometric literature (Ishioro, 2022a; and Ishioro and Maku, 2022). The health expenditure-led growth posits that health is a major stimulant and determinant of GDP; hence, direct interconnectivity and co-variation have been postulated between $H_{exp}$ and GDP. Proponents of the direct inter-connectivity and co-variation between $H_{exp}$ and GDP hypothesized that, sustained $H_{exp}$ enhances productivity (through better health status of labour) which ultimately promotes GDP. Hence, to these proponents, $H_{exp}$ *Hsiao Granger causes GDP*, *ceteris paribus* (Piabuo and Tieguhong, 2017).

The major deficiency of this empirical position is that, this linkage originated from the general consensus that was predicated on perceived and somewhat unverified causal linkage between the two series as there seems to be no evidence-based or empirically-supported findings on this view (Alhowaish, 2014). Either a bidirectional or a one-way causal link from growth of the economy to growth in $H_{exp}$ has been validated in the literature (Alhowaish, 2014). Regardless of the genre of interconnectivity existing between the series, the conclusion derived from different studies are undoubtedly mixed, with most of the studies concentrating on the developed and industrialized economies (Boussalem, Boussalem and Taiba, 2014). But sub-region-specific studies focusing on West Africa are either relatively scarce or non-existent.

The main intent of our re-search is to delve into the interconnectivity between healthcare spending and GDP in West Africa (henceforth W.AFRICA) on one hand and, conversely between $PHIV$ and $H_{exp}$. Specifically, this research paper attempts to empirically answer the following questions: Is there any interconnectivity between healthcare spending and GDP, and...
between \textit{PHIV} and the components of \textit{H exp} in W.AFRICA? And, if an interconnectivity exists among these variables, is it causal in the long-run? what is the nature and direction of causality among these variables? In other words, this study examined the extent to which \textit{H exp} is a determinant of GDP and mitigation of the \textit{PHIV} epidemic for the West African sub-region? Or to what degree has the tempo of \textit{PHIV} impinged on the performances of GDP and \textit{H exp} in the sub-region?

Finally, Kwak (2009) has noted that there are no recent literature that simultaneously included and investigated both public health expenditure, \textit{PbH exp}, and private health expenditures within the framework of the Mankiw, Romer and Weil (1992) version of the Solow growth model (Henceforth SGM). But our study went beyond including both components of \textit{H exp} to include public and the disaggregated \textit{Pr H exp} (as household out-of-pocket private health (denoted as \textit{OoHexp}) and non-household private health expenditures (denoted as \textit{Pr H exp})).

The other of aspects of this paper is organized as follows: Section two contains the literature review; section three focuses on the theoretical foundation of the augmented neoclassical model adopted in this study; section four is on the materials and methods including in specific terms the sources and nature of data and estimation techniques while section five discusses the results obtained in this study with the conclusion as a complementary section.

**LITERATURE REVIEW**

This section has been sub-divided into three sub-sections: \textit{H exp} versus GDP and SGM; \textit{H exp} versus GDP and, \textit{H exp} versus health outcomes.

**Health Expenditure Versus Economic Growth in the Augmented Solow Growth Model**

Heshmati (2018) applied the augmented SGM to the study of \textit{H exp} for the Organization for Economic Cooperation and Development (OECD) countries. The growth model was adopted as the pedestal for the explanation of the variations in output and expenditure per capita across the OECD countries in tandem with Heshmati (2001). In order to effectively examine the causal linkage between output and \textit{H exp}, the study specified two VAR models for output and \textit{H exp} indicators. Annual time series data on population, degree of openness, welfare index, different definitions of GDP (output), exchange rate, investment, and private and public consumption that spanned the period 1950 to 1992 were extracted from the Penn World Tables. The period 1950 to 1992 was divided into sub-groups as follows: most recent to less recent sub-groupings (1990-1992;1985-1989;1980-1984;1975-1979 and 1970-1974).

Advances in the literature have also shown that more explanatory variables can be suitably and justifiably augmented into the traditional SGM. For instance, Kwak (2009) noted some of the versions of the model including: Lucas (1988) and, Nonneman and Vanhoudt (1996) that expanded the SGM by including research and development; Ram (2007, 1997) augmented distance to the equator and IQ into the model; Knowles and Owen (1995) added varied definitions of education and health to the growth model; Temple (1998) augmented investment in equipment into the model; Yoo (2003) augmented technology and investments in information technology into the model; Ishioro (2019) augmented the model by adding energy to it. Aurangzeb (2003) expanded the model by adding \textit{H exp}. Others include Yoo and Yang(2004); Kwak (2009) added \textit{PbH exp}, and \textit{Pr H exp}, to the SGM.
Filho, Silva and Diniz (2005) identified some limitations of the SGM: first, the share of income (implicit in the model) as derived or originating from capital (as estimates) does not synchronize with those provided from statistical data. But the study confirmed that this limitation was resolved by Lucas (1988) by expanding the theoretical conceptualization of capital to include health and other explanatory variables. This is one of the justifications for the various versions of the model including the current study. Second, the traditional SGM is fraught with the problem of convergence. But Filho, Silva and Diniz (2005) suggested that to solve the problem of convergence, recent advances in econometrics such as unit root test should be applied to the variables of the augmented SGM. This is our further justification for applying different unit root testing techniques.

Bhattacharya and Qiao (2005) adopted the overlapping generations model in studying the impact of an infinite sequence of a two-period and infinitely-lived government and generations on health with two major health systems (private and public health systems) with expenditures (disaggregated as $PrH_{exp}$ and $PbH_{exp}$). The study provided the complementarity between $PbH_{exp}$ and $PrH_{exp}$, by assuming that economic agents may privately invest (through either $Ooh_{exp}$ and $PrH_{exp}$) in their well-being and longevity. The study concluded that privately-financed health expenditures are more productive only when complemented with publicly financed health investments.

**Health Expenditure versus Health Outcomes.**

The reverberation of robust and consistent health spending on health outcomes especially chronic and infectious diseases such as HIV/AIDS although discussed in some reasonable details in the literature is still controversial. Most of the studies on this issue focused on the mortality dimension of the discourse without considering the fact that mortality does not take into account the quality of life of those on whom enormous spending on health is made (Becchetti, Conzo and Salustri, 2017).

In a general dimension; Becchetti, Conzo and Salustri (2017) examined the impact of health spending on the number of diseases (health outcomes) using both individual-specific and regional data drawn from a large sample of European countries for citizens aged 50 years and above. The Countries covered include: Austria, Germany, Sweden, Netherland, Spain, Italy, France, Denmark, Greece, Switzerland, Belgium, Czech Republic and Poland. The study adopted the synthetic outcome approach (that uses the first difference in the reported number of chronic diseases as health outcomes). Seventeen major chronic diseases representing proxies for health outcomes were modeled. It was established by the study that a non-positive but statistically significant interconnectivity exist between $H_{exp}$ and health outcomes.

More specifically, Hsiao and Emdin (2014) investigated the interconnectivity between development assistance in support of health and, health and economic outcomes for the period 1990-2010. Low income, low middle income and upper middle income countries with population larger than 200,000 as at 2010 were selected for the longitudinal Panel analysis adopted by the study. The Generalized Method of Moment (GMM) was the estimation technique applied by the study. The GMM was applied by the study in order to circumvent the drawbacks associated with the traditional OLS estimation technique. The results affirmed that PHIV-targeted development assistance was significant in reversing the toll of the PHIV...
pandemic in W.AFRICA during the period studied. The core of the finding emanating from this study is that, donor funding was very potent in the fight against \textit{PHIV}.

**THE MODEL**

The Harrod-Domar growth model (hereafter HD model) hypothesized that, in any economy, capital accumulation is a potent instrument of GDP. That is, the rate of growth of any economy is proportional to the rate of capital accumulation at a given level of technology at a point in time (Filho, Silva and Diniz, 2005). However, Solow (1956) (hereafter Solow) extended the HD model and developed an exogenous growth theory often referred to as the Neoclassical growth model with the aim of analyzing the nature and determinants of GDP. The difference between the HD model and the SGM is that Solow (1956) extended the HD model by including and modifying the assumption of labor as a factor of production (and treated as inputs). The SGM is presented below:

\[ GDP_t = F(L_t, K_t) \]  

Equation (1) shows that there exists a single final good \( GDP_t \) produced using the standard neoclassical production function denoted as equation (1). The final good thus produced can either be consumed immediately after production or be accumulated for investment or future consumption. However, for the purpose of analytical convenience, we re-specify equation (1) as:

\[ GDP_t = F(P_t, K_t) \]  

\( GDP_t \) represents Output
\( L_t \) represents labour input at time \( t \)
\( K_t \) represents physical capital at time \( t \)
\( P_t \) represents current population of the country

**The Health Expenditure-augmented Solow Model**

Fundamentally, the health expenditure-augmented SGM posits that \( H_{exp} \) fosters GDP because in the framework of the SGM, health is treated as a form of capital input into the growth-producing process (Knowles and Owen, 1995; Heshmati, 2001; Novignon, Olakojo and Novignon, 2012). Furthermore, investment deployed to health can increase the accumulation of both human and physical capital thereby engendering GDP and economic development. Investment on health as represented by \( H_{exp} \) makes the population healthy and increase their income earning capacity therefore enhancing the growth of the economy (Donadelli, Paradiso and Liveri, 2018). Thus, equation (2) is re-expressed as:

\[ GDP_t = F(H_{exp}, P_t, K_t) \]  

\( H_{exp} \) represents health care expenditure

Introducing the innovation or knowledge parameter into equation (3), we obtained equation (4) after the pragmatic specification and practice of McDonald and Roberts (1996; 2002; 2004), Aurangzeb (2003) and Heshmati (2018) as:

\[ GDP_t = H_{exp}^\alpha, K_j^\beta, (A_t P_t)^{\gamma - \delta} \]  

The intensive form of equation (4) is expressed as:

\[ gd_{p_t} = h_{exp}^\alpha, k^\beta. \]
The PHIV augmented SGM is expressed as:

\[ GDP_t = H \exp{\beta_i} K^j PHIV^{\gamma_i} (A_P)_{t}^{1-\alpha-j-\gamma} \]  

(6)

PHIV represents current HIV prevalence rate at time t.

Augmenting \( H \exp \) into the SGM as either a function of \( PHIV \) or other growth-enhancing or distorting variables has been eulogized by various studies including Cuddington (1993); Dixon, McDonald and Roberts (2001); Heshmati (2018), and Ishioro (2018). Most studies adopted different variations and definitions of \( H \exp \) but ours follows the pragmatic delineation of Ishioro (2018). Therefore, the health expenditure component (\( H \exp \)) of the augmented SGM specified in equation (4) and (5) can be disaggregated according to the sources of funding with PHIV as growth-distorting variable as follows:

\[ GDP_t = Pr \exp{\theta_i} PbH \exp{\beta_i} TH \exp{\theta_i} Ooohexp{\theta_i} K^j PHIV^{\gamma_i} (A_P)_{t}^{1-\varphi-\delta-\omega-j-\gamma} \]  

(7)

\( Pr \exp \) is private non-household health expenditure at time t.

\( PbH \exp \) is public health expenditure at time t.

\( TH \exp \) is Total health expenditure at time t.

\( Ooohexp \) is out-of-pocket is household health expenditure at time t.

\( PbH \exp \) and \( Pr \exp \) were augmented into the SGM because it has been argued by Aurangzeb (2003) that the degree of absorption of innovation and technological progress increases with the degree of openness and reception to external health inflows. Furthermore, \( Pr \exp \) was augmented into the model because the economic agent may embark on self health or own health protection by privately investing in their own health (Liu and Nelson, 2005; Bhattacharya and Qiao, 2005).

Furthermore, both \( PbH \exp \), \( Pr \exp \) and \( TH \exp \) are included in the SGM to show the unavoidable complementarity that is needed between the components of \( H \exp \) as private undertakings and investments to improve the quality of the health services without commensurate public sector involvement might amount to no progress (Dow, Philipson and Sala-i-Martins, 1999; Bhattacharya and Qiao, 2005). Therefore, equation (7) is disaggregated according to the right hand side series to obtain equation 7(a)-7(d).

\[ GDP_t = Pr \exp{\theta_i} K^j PHIV^{\gamma_i} (A_P)_{t}^{1-\varphi-j-\gamma} \]  

(7a)

\[ GDP_t = PbH \exp{\beta_i} K^j PHIV^{\gamma_i} (A_P)_{t}^{1-\beta-j-\gamma} \]  

(7b)

\[ GDP_t = TH \exp{\delta_i} K^j PHIV^{\gamma_i} (A_P)_{t}^{1-\beta-j-\gamma} \]  

(7c)

\[ GDP_t = Ooohexp{\theta_i} K^j PHIV^{\gamma_i} (A_P)_{t}^{1-\theta-j-\gamma} \]  

(7d)

The fractions of output saved (often referred to as saving rate) are distributed to the accumulation of K, \( H \exp \) whether private (\( Pr \exp \)) in which household out-of-pocket private health expenditure (\( Ooohexp \)) and non-household private health expenditure.
(PrH exp,) are found, public (PbH exp,) or total (TH exp,) and for mitigating PHIV. Equation (7a)-(7d) posit that GDP is determined by H exp (whose components are: TH exp,, Ooohexp, Pr H exp,, PbH exp,) capital accumulation and PHIV.

The Keynesian macroeconomic assumption of the equality of savings and investment is invoked and holds as expressed in equation (8)

\[ SV_t = I_t \text{ and } \frac{SV_t}{Y_t} = \frac{I_t}{Y_t} \]  

(8)

\( SV_t \) represents saving at time \( t \)

\( I_t \) represents investment at time \( t \)

Equation (8) is further expressed in relation to the saving rate of each RHS variables as:

\[ SV_t = \frac{SV_{PrH exp} + SV_{PbH exp} + SV_{Ooohexp} + SV_{PHIV} + SV_{PHIV}}{GDP} = \frac{I_t}{GDP} = \frac{I_{PrH exp} + I_{PbH exp} + I_{Ooohexp} + I_{PHIV}}{GDP} + I^k \]  

(9)

\[ \tilde{k}_t = SV_t^k g\hat{d}_p - k_t(n_t + g_t + \delta_t) \]  

(10)

In equation (10), \( \tilde{k}_t \) is the pace at which capital is accumulated, \( SV_t^k g\hat{d}_p \) and \( SV_t^k \) represents the saving per capital and the pace at which capital is saved while \( g\hat{d}_p \) is the output per capita. Equation (10) implies that as saving per capital increases with the increase in investment per capital; it will result in the frantic expansion of physical capital per capita. But as the relative amount of people that is morbid as a result of PHIV grows without an attendant increase in capital accumulation, physical capital per capita reduces and the economy is likely to be trapped and enmeshed in the doldrums of PHIV during such periods. Heshmati (2001) and Manning (2007) observed that, akin to human capital augmentation, the SGM can be appropriately and satisfactorily augmented to accommodate investment in health in the form of H exp (Crémieux, Ouellette & Pilon, 1999). Equation (11)-(14) account for the H exp evolution in the context of the SGM model.

\[ prhexp_t = SV_{PrH exp}^t g\hat{d}_p - prhexp_t(n_t + g_t + \delta_t) \]  

(11)

In equation (11), \( prhexp_t \) represents the pace at which PrH exp is accumulated by non-governmental organizations and donor agencies while \( SV_{PrH exp}^t g\hat{d}_p \) is the non-household private health expenditure saving per morbid individual; \( SV_{PrH exp}^t \) represents the pace at which the non-household private health expenditure is saved and \( g\hat{d}_p \) is the income or output per capita. Equation (11) posits that as \( PrH exp \) increases and investment in the health of the relative amount of people suffering from HIV/AIDS increases, mortality reduces while output generated increases, resulting in the growth of output per capita. However, as new infections of the disease increase and the relative amount of people who are HIV positive grows each year, negative impact is exerted on \( PrH exp \), which makes previous period \( prhexp \) inadequate for
the current population. This implies that savings and investment increase \( prh_{exp} \) while the growth of the relative amount of people who are HIV infected reduce \( prh_{exp} \). But when \( prh_{exp} \) is larger than the amount of new \( prh_{exp} \) needed to compensate for the growth of HIV positive persons in the affected economies; then the rate of accumulation of \( prh_{exp} \) is positive which implies that \( (\hat{ph}_{exp}) \) i.e. \( prh_{exp} \) per capita at time \( t \) is increasing.

\[
Ooh_{exp} = SV_{\hat{oo}h_{exp}} \hat{gdp}_{t} - ooh_{exp}(n_{t} + g_{t} + \delta_{t})
\]  

(12)

In equation (12), \( Ooh_{exp} \) is the pace at which household out-of-pocket private health expenditure is accumulated by either individuals, households and/or other private economic agent while \( SV_{\hat{oo}h_{exp}} \hat{gdp}_{t} \) is the household out-of-pocket private health expenditure saving per morbid economic agent in the economy. \( SV_{\hat{oo}h_{exp}} \) is the pace at which the household out-of-pocket private health expenditure is saved and \( \hat{gdp}_{t} \) is the income per capita. Equation (12) hypothesizes that as household out-of-pocket private health expenditure and investment in the health of the relative amount of people suffering from HIV/AIDS increases, HIV-related deaths reduce while output generated increases resulting in the growth of output per capita. However, as more persons are infected and the relative amount of people who are HIV positive grow each year, negative expenditure-pressures are exerted on household out-of-pocket private health expenditure making previous period \( Ooh_{exp} \) inadequate for the current population. This implies that savings and investment increase \( oh_{exp} \) while the growth of the relative amount of people who are HIV infected reduce \( Ooh_{exp} \). But when \( Ooh_{exp} \) is larger (smaller) than the amount of new \( Ooh_{exp} \) needed to compensate for the growth of HIV positive persons; then the rate of accumulation of \( Ooh_{exp} \) is positive (negative) which implies that \( (\hat{oh}_{exp}) \) i.e. \( Ooh_{exp} \) per capita at time \( t \) is increasing (decreasing).

\[
\hat{ph}_{exp} = SV_{\hat{ph}_{exp}} \hat{gdp}_{t} - \hat{ph}_{exp}(n_{t} + g_{t} + \delta_{t})
\]  

(13)

In equation (13), \( \hat{ph}_{exp} \) represents the pace at which \( Ph_{exp} \) is accumulated by either government and/or her agencies while \( SV_{\hat{ph}_{exp}} \) and \( \hat{gdp}_{t} \) are the \( Ph_{exp} \) per morbid individual in the economy and the pace at which \( \hat{ph}_{exp} \) is saved, and income per capita. Equation (13) hypothesizes that as \( Ph_{exp} \) and investment in the health of the relative amount of people suffering from HIV/AIDS increases, HIV-related deaths reduce while output generated increases resulting in the growth of output per capita. However, as more persons are infected and become morbid as a result of infectious diseases and the relative amount of people who are HIV positive grows each year, negative expenditure-pressures are exerted on \( Ph_{exp} \) making previous period expenditure \( (Ph_{exp}) \) inadequate for the current population. This implies that savings and investment increase current \( Ph_{exp} \) while the growth of the relative amount of people who are HIV infected reduces it. But when \( Ph_{exp} \) is expanding and becomes larger (smaller) than the amount of new \( Ph_{exp} \) needed to compensate for
growth of HIV positive persons then the rate of accumulation of \( PbH \text{ exp} \) is positive (negative) which implies that \( \dot{ph}h_{exp} \), i.e. \( PbH \text{ exp} \), per capita at time \( t \) is increasing (decreasing).

\[
\text{th} \text{exp}_t = SV_{t}^{\text{thexp}} g \hat{d}p_t - th \text{exp}_t(n_t + g_t + \delta_t) \tag{14}
\]

In equation (14), \( \text{thexp} \) is defined as the current cumulative health expenditure. \( \text{thexp} \) is described as the pace at which \( \text{thexp} \) is accumulated by both internal and external governments and/or their agencies and the private sectors including donor agencies (Ishioro, 2019; WHO, 2019). But the \( \text{thexp} \) accruing to each morbid individual in the economy and the pace at which it is either saved or invested is described as \( SV_{t}^{\text{thexp}} \) while \( gd\hat{p}_t \) represents income per capita. Equation (14) posits that as \( \text{thexp} \) and investment in the health of the relative amount of people suffering from either HIV/AIDS or other opportunistic diseases increases, HIV-induced mortality in the economy reduces while output generated increases resulting in the expansion of output per capita. However, if more persons are infected and affected by the disease and become morbid, the relative amount of people who are HIV positive grows each year and as a result negative health expenditure-pressures are exerted on \( \text{thexp} \) making the total health expenditure (\( \text{thexp} \)) accumulated during the previous period inadequate for the current population. This means that saving and investment enhance current performance of \( \text{thexp} \) while the expansion of the relative amount of people who are HIV-positive reduces it. But when \( \text{thexp} \) is consistently increasing and eventually becomes larger than the amount of new \( \text{thexp} \) needed to compensate for the growth of HIV positive persons in the economy then, its rate of accumulation becomes positive implying that \( \text{thexp} \), i.e. current total health expenditure (\( \text{thexp} \)) per capita at time \( t \) will increase. However, if \( \text{thexp} \) is consistently decreasing and becomes smaller than the new \( \text{THexp} \) needed to compensate for the increase in the number of HIV positive persons then its rate of accumulation becomes negative meaning that \( \text{thexp} \), i.e. \( \text{thexp} \) per capita at time \( t \) is dwindling.

\[
ph_{iv_t} = SV_t^{phiv} g \hat{d}p_t - ph_{iv_t}(n_t + g_t + \delta_t) \tag{15}
\]

In equation (15), \( ph_{iv_t} \) represents the current pace at which the HIV epidemic affects economic agents (individuals, households or firms) and has been described in this study as the prevalence rate of HIV. \( SV_t^{phiv} \) can be loosely described as the pace at which either health expenditure, income or health enervating resources are accumulated for the purpose of HIV mitigation or care of HIV-morbid / HIV-positive economic agents. \( SV_t^{phiv} \) represents health expenditure per HIV-positive individual. However, health expenditure per HIV-positive individual has been generally categorized in the context of this study into: non-household private health expenditure (\( PrH \text{exp}_t \)) per HIV positive individual or household; household out-of-pocket private health
expenditure (\(PbH\ exp,\)) per HIV positive individual or household; \(PbH\ exp\), per HIV positive individual or household; and \(TH\ exp\), per HIV positive individual or household. \(g\hat{d}p\), represents income per HIV positive individual. Equation (15) posits that as the HIV epidemic heightens, the proportion of HIV positive individuals and affected households / firms increase thereby making previously accumulated saving or investment in \(H\ exp\) inadequate for current \(PHIV\) rate (\(phiv\)) \cite{Islam1995}.

\[
\hat{k} = \left[ \frac{\left( sv^k \right)^{1-\beta-\gamma-j-\varphi} \left( sv^{prhexp} \right)^{\phi} \left( sv^{pbhexp} \right)^{\beta} \left( sv^{oohexp} \right)^{\Theta} \left( sv^{phiv} \right)^{\gamma}}{n + g + \delta} \right]^{\frac{1}{1-\beta-\gamma-\varphi-j}}
\]

Equation (16) shows that the stock of capital (\(k\)) converges to a steady state value of accumulated capital (\(k^*\)). In equation(16), \(k^*\) is positively related to \(H\ exp\) (\(PbH\ exp,\ Ooh\ exp,\ PrH\ exp,\ and\ TH\ exp,\)) but negatively related to the growth rate of population and depreciation.

\[
prhexp^* = \left[ \frac{\left( sv^{prhexp} \right)^{1-\beta-\gamma-j-\varphi} \left( sv^{pbhexp} \right)^{\phi} \left( sv^{phiv} \right)^{\gamma}}{n + g + \delta} \right]^{\frac{1}{1-\beta-\gamma-\varphi-j}}
\]

\[
pbhexp^* = \left[ \frac{\left( sv^{pbhexp} \right)^{1-\beta-\gamma-j-\varphi} \left( sv^{prhexp} \right)^{\phi} \left( sv^{phiv} \right)^{\gamma}}{n + g + \delta} \right]^{\frac{1}{1-\beta-\gamma-\varphi-j}}
\]

\[
oohexp^* = \left[ \frac{\left( sv^{oohexp} \right)^{1-\beta-\gamma-j-\varphi} \left( sv^{pbhexp} \right)^{\phi} \left( sv^{phiv} \right)^{\gamma}}{n + g + \delta} \right]^{\frac{1}{1-\beta-\gamma-\varphi-j}}
\]

\[
thexp^* = \left[ \frac{\left( sv^{thexp} \right)^{1-\beta-\gamma-j-\varphi} \left( sv^{oohexp} \right)^{\phi} \left( sv^{phiv} \right)^{\gamma}}{n + g + \delta} \right]^{\frac{1}{1-\beta-\gamma-\varphi-j}}
\]

Equation (17)-(19) connote that either \(prhexp^*\), \(pbhexp^*\), \(oohexp^*\) or \(thexp^*\) would converge to its steady state value of accumulated \(H\ exp\) (denoted as either \(prhexp^*\), \(pbhexp^*\), \(oohexp^*\) or \(thexp^*\)). Based on the complementarity clause emphasized by Dow, Philipson and Sala-i-Martins (1999) and, Bhattacharya and Qiao (2005), as either \(prhexp^*\), \(pbhexp^*\), \(oohexp^*\) or \(thexp^*\) would be positively related to other components of \(H\ exp\) and PHIV. We specify the PHIV intensive equation as:
\[ \text{phiv} = \left[ \frac{(sv^{\text{phiv}})^{(1-\phi-\beta-\gamma-\phi-\theta-\phi)}}{(sv^{\text{phiv}})^{(1-\phi-\beta-\gamma-\phi-\theta-\phi)}} (sv^{\text{phiv}})^{(1-\phi-\beta-\gamma-\phi-\theta-\phi)}} (sv^{\text{phiv}})^{(1-\phi-\beta-\gamma-\phi-\theta-\phi)}} (sv^{\text{phiv}})^{(1-\phi-\beta-\gamma-\phi-\theta-\phi)}} (sv^{\text{phiv}})^{(1-\phi-\beta-\gamma-\phi-\theta-\phi)}} (sv^{\text{phiv}})^{(1-\phi-\beta-\gamma-\phi-\theta-\phi)}} (n + g + \delta) \right]^{1/(1-\phi-\beta-\gamma-\phi-\theta-\phi)}} 

(21)

The definition found in equation (22) has been popularly applied by McDonald and Roberts (1996, 1999, 2002 and 2004), and Manning (2007).

\[
\ln y_i^* = \beta_i + \gamma_i \ln(\text{GDP}) + \rho_i \ln(\text{HEX}) + \sigma_i \ln(\text{PHIV}) + \epsilon_i
\]

(22)

Equation (22) implies that in the context of augmented SGM the steady state path for the log of GDP or income per capita follows a specific linear time trend (Heshmati, 2001). But the slope of the linear time trend implicit in equation (22) is determined exogenously using the rate of technical progress (Heshmati, 2001). The intercept of the model represents the impacts of the pace at which the population of the economy grows and the saving and investments deployed to physical, health and human capital respectively. The saving and investment deployed to physical, health and human capital are designed to boost the level of income per capita. The high growth of labour or population reduces income per capita.

**Hsiao Causality in the Framework of the Augmented Solow Growth Model.**

Following Heshmati (2001) and Ishioro (2018), we specify the restricted (variable-specific) and unrestricted (generalized) versions of the Hsiao causality test as expressed in equation 23(a-c) and 24(a-d).

**Variable-specific equations are:**

\[
\ln \text{GDP}_t = z_0 + \sum_{j=1}^{m} (L)z_{ij} \ln \text{GDP}_{t-1} + \epsilon_{it}
\]

(23a)

\[
\ln \text{HEX}_t = \sigma_0 + \sum_{j=1}^{m} (L)\sigma_{ij} \ln \text{HEX}_{t-1} + \epsilon_{it}
\]

(23b)

\[
\text{PHIV}_t = \sigma_0 + \sum_{i=1}^{m} (L)\sigma_{ij} \text{PHIV}_{t-1} + \epsilon_{it}
\]

(23c)

**Generalized equations are:**

Generalized GDP versus $\text{HEX}$, and, $\text{HEX}$ versus GDP equations

\[
\log \text{GDP}_t = z_0 + \sum_{i=1}^{m} z_{ij} \log \text{GDP}_{t-1} + \sum_{j=1}^{N} z_{2j} \log \text{HEX}_{t-j} + \phi_{2t}
\]

(24a)

\[
\log \text{HEX}_t = \sigma_0 + \sum_{i=1}^{m} \sigma_{ij} \log \text{HEX}_{t-1} + \sum_{j=1}^{N} \sigma_{2j} \log \text{GDP}_{t-j} + \phi_{2t}
\]

(24b)

Equations (24a) and (24b) are the Hsiao bivariate regression models showing the indicators adopted in modelling the causality between the health expenditure vector (HEX) and GDP.
Hexp is the vector representing \( PrHexp \), \( Oohexp \), \( PbHexp \) and \( THexp \) in our econometric model.

The generalized \( H \) \( \exp \) versus \( PHIV \) and; \( PHIV \) versus \( H \) \( \exp \) equations are specified as:

\[
\log H_{it} = \sigma_0 + \sum_{i=1}^{m_i} \sigma_i H_{i-j} + \sum_{j=1}^{N} \sigma_2 PHIV_{i-j} + \Phi_{2i}
\]

\[
PHIV_{i} = \sigma_0 + \sum_{i=1}^{m_i} \sigma_i PHIV_{i-j} + \sum_{j=1}^{N} \sigma_2 H_{i-j} + \Theta_{2i}
\]

(Equation 24c) and (24d) represent the bivariate regression of \( PHIV \) and \( H \) \( \exp \). Also, \( H \) \( \exp \) is as denoted in equation (24a) and (24b) respectively.

A résumé of the link between the Hsiao causality test and the augmented SGM is presented in table 1. The equations presented in the table are suggestive of the foundation/building blocks for the specification of our Hsiao models.

**Table 1**

<table>
<thead>
<tr>
<th>S/NO</th>
<th>AUGMENTED SOLOW GROWTH EQUATION</th>
<th>HYPOTHESIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>( pr\text{exp}<em>t = SV</em>{i}^{pr\text{exp}} gdp_t - pr\text{exp}_t (n_i + g_i + \delta_i) )</td>
<td>Non-household Private Health expenditure-led HIV prevalence Hypothesis</td>
</tr>
<tr>
<td></td>
<td>( phiv_t = SV_{i}^{phiv} gdp_t - phiv_t (n_i + g_i + \delta_i) )</td>
<td>HIV Prevalence-led Private Health spending Hypothesis</td>
</tr>
<tr>
<td>2</td>
<td>( Oohexp_t = SV_{i}^{oohexp} gdp_t - oohexp_t (n_i + g_i + \delta_i) )</td>
<td>Household Out-of-Pocket Health expenditure-led HIV prevalence Hypothesis</td>
</tr>
<tr>
<td></td>
<td>( phiv_t = SV_{i}^{phiv} gdp_t - phiv_t (n_i + g_i + \delta_i) )</td>
<td>HIV Prevalence-led Out-of-Pocket Health spending Hypothesis</td>
</tr>
<tr>
<td>3</td>
<td>( pbhexp_t = SV_{i}^{pbhexp} gdp_t - pbhexp_t (n_i + g_i + \delta_i) )</td>
<td>Public Health expenditure-led HIV prevalence Hypothesis</td>
</tr>
<tr>
<td></td>
<td>( phiv_t = SV_{i}^{phiv} gdp_t - phiv_t (n_i + g_i + \delta_i) )</td>
<td>HIV Prevalence-led Public Health spending Hypothesis</td>
</tr>
<tr>
<td>4</td>
<td>( \text{the}<em>t = SV</em>{i}^{the} gdp_t - \text{the}_t (n_i + g_i + \delta_i) )</td>
<td>Total Health expenditure-led HIV prevalence Hypothesis</td>
</tr>
<tr>
<td></td>
<td>( phiv_t = SV_{i}^{phiv} gdp_t - phiv_t (n_i + g_i + \delta_i) )</td>
<td>HIV Prevalence-led Total Health spending Hypothesis</td>
</tr>
<tr>
<td>5</td>
<td>( \text{Growth-led Private Health Expenditure/ Growth-led HIV Prevalence Hypothesis} )</td>
<td></td>
</tr>
<tr>
<td></td>
<td>( GDP_t = PrH exp_t \theta K^{j-i} PHIV_j (A_{P_t})^{(i-j-\gamma)} )</td>
<td>Growth-led Private Health Expenditure and Growth-led HIV Prevalence Hypothesis</td>
</tr>
<tr>
<td>6</td>
<td>( \text{Growth-led Out-of-Pocket Health Expenditure/ Growth-led HIV Prevalence Hypothesis} )</td>
<td></td>
</tr>
<tr>
<td></td>
<td>( GDP_t = Oohexp_t \theta K^{j-i} PHIV_j (A_{P_t})^{(i-j-\gamma)} )</td>
<td>Growth-led Out-of-Pocket Health Expenditure and Growth-led HIV Prevalence Hypothesis</td>
</tr>
</tbody>
</table>
Health expenditure (henceforth $H\ exp$) has been hypothesized to be a function of GDP (represented as real \textit{per capita} gross domestic product (GDP)), Ishioro (2018). It has further been argued that the existence of a bilateral interconnectivity between $H\ exp$ and GDP is a validation of both economic growth-led $H\ exp$ and $H\ exp$-led economic growth hypotheses. The bilateral interconnectivity can also exist between disaggregated $H\ exp$ and GDP. The foundation of this conclusion is based on the premise that the health of the population is seen as an input into the aggregate production function. Health expenditure-led growth hypothesis, by definition, connotes the performance of $H\ exp$ (s) hypothesized to be a function of resources derived and generated from GDP (including income or wealth). The economic growth-led health expenditure hypothesis posits that income derived from economic growth is a function of $H\ exp$ (s) because $H\ exp$ is a determinant of both labour supply and productivity and human capital accumulation. Arising from the \textit{law of transitivity}, if $H\ exp$ is regarded as an investment in human capital accumulation and, human capital is regarded also as \textit{“engine”} of GDP then an increase in $H\ exp$ translates to higher GDP and enhancement of income. Increase in $H\ exp$ makes higher labor supply and productivity possible eventually leading to higher income.

**MATERIALS AND METHODS**

**Sources and Description of Data**

The data used in this study are from the World Bank World Development Indicators (WDI) for the period 1992 to 2020. The indicators adopted in this study are presented in table 2:

<table>
<thead>
<tr>
<th>S/NO</th>
<th>Variable</th>
<th>Description of Variable</th>
<th>Source of Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>$GDP$</td>
<td>Economic growth</td>
<td>WDI, 2021</td>
</tr>
<tr>
<td>2</td>
<td>$TH\ exp$</td>
<td>Total health expenditure</td>
<td>WDI, 2021</td>
</tr>
<tr>
<td>3</td>
<td>$PbH \ exp$</td>
<td>Public health expenditure</td>
<td>WDI, 2021</td>
</tr>
<tr>
<td>4</td>
<td>$Pr\ H\ exp$</td>
<td>Non-Household Private health expenditure</td>
<td>WDI, 2021</td>
</tr>
<tr>
<td>5</td>
<td>$Oohexp$</td>
<td>Household Out-of-Pocket private health expenditure</td>
<td>WDI, 2021</td>
</tr>
<tr>
<td>6</td>
<td>$PHIV$</td>
<td>HIV prevalence rate</td>
<td>WDI, 2021 and UNAIDS 2021</td>
</tr>
</tbody>
</table>


NOTE: WDI is World Bank World Development Indicators

We have used the region-specific aggregate data because of the empirically validated observations of Heshamati (2018) that, results obtained from the SGM are usually sensitive to the characteristics of country-specific data.

**Estimation Techniques**

The estimation techniques adopted in this study are the unit root, cointegration and the Hsiao Causality tests.
Unit Root and Cointegration Tests
Building on the existing pragmatic tradition of time series analysis such as Osinska (2008); Hong, Liu and Wang (2009), Pearl (2000); Hiemstra and Jones (1994); Cheung and Ng, (1996), and Pierce and Haugh (1977); we have evaluated the order of integration of the variables used in this study by applying the unit root and cointegration tests. The lag selected in the test of the order of integration is chosen to ensure that the residuals of our series are free from serial correlation (Aurangzeb, 2003; Ishioro, 2020a, 2020b, 2022c, 2022e). The Augmented Dickey Fuller (ADF) [Dickey and Fuller, 1979, 1981; Ishioro 2015a and 2015b], Phillips-Perron (PP) [Phillips and Perron, 1988] and Kwiatkowski, Phillips, Schmidt and Shin (KPSS) [Kwiatkowski-Phillips-Schmidt-Shin, 1992] unit root tests were adopted in this study. The hypothesis of the ADF and PP tests is specified following (Ishioro, 2017, 2018, 2019, 2022a) as:

$H_0$: Indicator is not stationary

$H_a$: Indicator is stationary

The rule of thumb for interpreting the results of the ADF and PP tests is: the null hypothesis of non-stationarity (stationarity) is not accepted (accepted) if the value of the value is less than 5 percent (greater than 5 percent).

The hypothesis of the KPSS unit root test is stated as:

$H_0$: Indicator is stationary

$H_a$: Indicator is not stationary

The rule of thumb for interpreting the results of the KPSS test is: the null hypothesis of stationarity (non-stationarity) is not accepted (accepted) if the probability value is less than 5 percent (greater than 5 percent).

The ADF test is complemented with the PP and KPSS unit root tests because the ADF has been adjudged as sensitive to certain econometric conditions (such as independence and homogeneity assumptions). Filho, Silva and Diniz (2004) presented a procedure for applying econometric tests to the explanatory variables of the SGM as follows: first, include suitable measure of human capital; and second, test for the unit root and third, apply suitable cointegration test.

Hsiao Granger Causality Test
Filho, Silva and Diniz (2005) submitted that the SGM is usually fraught with the problem of accurate interpretation of the empirical results obtained from its estimates especially when applied in the context of the cross-section regressions. To solve this problem we innovatively applied the Hsiao Granger causality test (whose results are relatively easy to interpret)[Amiri and Lindel, 2016]. This was made possible because Hsiao (1979) provided a Final Prediction Error-based (henceforth FPE) condition for applying and testing the Granger causality test structure of selected series. The major advantage of the Hsiao Granger causality is that, it is not a statistical test but an estimation procedure that depends on FPE in order to ascertain the optimal lag length. As a result, it is insensitive to inferential problems when applied to integrated series (Amiri and Lindel, 2016; Maku and Ishioro, 2023; Maku, Ishioro and Asagba, 2023). The Hsiao causality procedure that begins with statement of the null hypothesis is briefly explained below.
For \( H \exp \) versus GDP unrestricted models, the set of null hypothesis is that: \( H \exp \) does not \textit{Hsiao Granger cause} GDP and; GDP does not \textit{Hsiao Granger cause} \( H \exp \).

Also, for \( H \exp \) versus \( PHIV \), the set of null hypothesis is that: \( H \exp \) does not \textit{Hsiao Granger cause} \( PHIV \) and, \( PHIV \) does not \textit{Hsiao Granger cause} \( H \exp \) (See Granger, 1969, 1988; Granger and Newbold, 1986; Ishioro, 2022a, 2022b, and Ishioro and Maku, 2022).

\[
FPE(m) = [(T + m + 1)(T - m + 1)^{-1}], \text{SES}(m), T^{-1}
\]

(25)

where \( T \) represents the number of observations found in the sample size; \( m \) represents the optimal lag length of the series (GDP), SES is the sum of squares error. Furthermore,

\[
FPE(m*, n*) = [(T - m* - n* - 1)^{-1}(T + m* + 1)], T^{-1}[\text{SES}(m*, n*)]
\]

(26)

Where \( T \) is the number of observation in the sample, \( m* \) is the optimal lag length of GDP. \( n* \) represents optimal lag length of either \( H \exp \) or \( PHIV \), and SES is the sum of squares error.

**Rule of Thumb for Testing the Hsiao Hypotheses:**

If \( FPE_{Y}(m*) < FPE_{Y}(m*, n*) \); if \( FPE(m*, n*) \) is greater than \( FPE(m*) \) or if \( FPE(m*) \) is less than \( FPE(m*, n*) \), then the null hypothesis of Hsiao non-causality is not rejected implying that either \( PHIV \) or \( H \exp \) "does not Hsiao Granger cause" GDP.

**BUT** if \( FPE_{Y}(m*) > FPE_{Y}(m*, n*) \) that is, if \( FPE(m*, n*) \) is less than \( FPE(m*) \) or if \( FPE(m*) \) is larger than \( FPE(m*, n*) \), then the null hypothesis of non-causality is rejected implying that either \( PHIV \) or \( H \exp \) "Hsiao Granger causes" GDP.

Furthermore, we specify the restricted (variable-specific) and unrestricted (generalized) Hsiao Granger causality equations.

**ANALYSIS OF EMPIRICAL RESULTS**

**Results of Augmented Dickey Fuller Test**

Table 3

<table>
<thead>
<tr>
<th>Series</th>
<th>GDP</th>
<th>( Pr \ H \exp )</th>
<th>( Oohexp )</th>
<th>( PbH \exp )</th>
<th>( TH \exp )</th>
<th>( PHIV )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level</td>
<td>-3.211</td>
<td>-3.067</td>
<td>-1.030</td>
<td>-1.805</td>
<td>-0.879</td>
<td>-5.220*</td>
</tr>
<tr>
<td>1st Difference</td>
<td>-7.385*</td>
<td>-8.363*</td>
<td>-3.758**</td>
<td>-2.878**</td>
<td>-1.739***</td>
<td>-0.482</td>
</tr>
</tbody>
</table>

**Results of Phillips-Perron Unit Root Test for West African Region**

| Level           | -3.244** | -3.067           | -1.043       | -1.059         | -1.109       | -5.827*      |
| 1st Difference  | -7.405*  | -20.745*         | -3.816**     | -7.635*        | -7.292*      | -0.884       |

**Results of KPSS Unit Root Test for West African Region**

| Level           | 0.325**  | 0.145**          | 0.188**      | 0.174**        | 0.172**      | 0.179**      |
| 1st Difference  | 0.032    | 0.418**          | 0.103        | 0.158**        | 0.080        | 0.182        |

Source: Author's Computation

NOTE:***,** and * represent significance at 1, 5 and 10 percent levels.

The results of the ADF unit root test (with intercept and trend or without trend) in relation to our selected variables: GDP, \( TH \exp \), \( PbH \exp \), \( Pr \ H \exp \), \( Oohexp \) & \( PHIV \) indicated that all
the variables were non–stationary at levels (with or without trend). However, these became stationary after the first difference. In the econometric literature, it has been acknowledged that if two or more variables are non stationary individually, and the first difference of all the variables is stationary then the linear combination of any of these variables will not be spurious (if the residual has no unit root). This implies that, since our variables were stationary at first difference, cointegration exists between the variables (Ishioro, 2022a; 2022b; 2022d).

**Johansen Co-integration test**

<table>
<thead>
<tr>
<th>Hypothesized No of CE(s)</th>
<th>Trace Statistic</th>
<th>Maximal. Eigen Value Statistic</th>
<th>0.05 Critical Value</th>
<th>Prob.</th>
<th>Max. Eigen Value</th>
<th>0.05 Critical Value</th>
<th>Prob</th>
</tr>
</thead>
<tbody>
<tr>
<td>None*</td>
<td>0.966</td>
<td>247.950</td>
<td>95.753</td>
<td>0.000</td>
<td>98.673</td>
<td>40.077</td>
<td>0.000</td>
</tr>
<tr>
<td>At Most* 1</td>
<td>0.895</td>
<td>149.277</td>
<td>69.818</td>
<td>0.000</td>
<td>65.577</td>
<td>33.876</td>
<td>0.000</td>
</tr>
<tr>
<td>At Most* 2</td>
<td>0.796</td>
<td>83.699</td>
<td>47.856</td>
<td>0.000</td>
<td>46.114</td>
<td>27.584</td>
<td>0.000</td>
</tr>
<tr>
<td>At Most* 3</td>
<td>0.601</td>
<td>37.585</td>
<td>29.797</td>
<td>0.005</td>
<td>26.667</td>
<td>21.131</td>
<td>0.007</td>
</tr>
<tr>
<td>At Most 4</td>
<td>0.242</td>
<td>10.918</td>
<td>15.494</td>
<td>0.216</td>
<td>8.070</td>
<td>14.264</td>
<td>0.371</td>
</tr>
<tr>
<td>At Most 5</td>
<td>0.093</td>
<td>2.847</td>
<td>3.841</td>
<td>0.091</td>
<td>2.847</td>
<td>3.841</td>
<td>0.091</td>
</tr>
</tbody>
</table>

Source: Ishioro(2022)

The results of the cointegration test displayed in table 4 are presented based on both the trace and maximal Eigen value statistics. The values of both statistics established 4 cointegrating equations (which is a confirmation of the presence and/or existence of a long-run cointegrating interconnectivity among the variables in this study). The policy implication of this is that, the design and implementation of $H\text{exp}$ ($PHIV$ and GDP) policies would have long-run implications for the growth performance and $PHIV$ mitigation (performance of $H\text{exp}$ (s)) in the West African region. Furthermore, it means that the implementation of either PHIV or expansion of productive economic activities policies in the sub-region without considering the effects on $H\text{exp}$ (s) would hamper the long-run performance of $Oohexp$ and non-household private expenditure, $PbH\text{exp}$ and $TH\text{exp}$.  

**Results of Hsiao Causality Test**

Two strands of the results of the Hsiao causality tests are reported hereunder: results for $H\text{exp}$ versus GDP and, $H\text{exp}$ versus $PHIV$.

**Hsiao Results for Health Expenditure versus Economic Growth**

The results presented in table (5) show that total and $Pr\ H\text{exp}$ do not "Hsiao Granger cause" GDP in W.AFRICA. This connotes the existence of a unidirectional causality between $TH\text{exp}$ and GDP; and $Pr\ H\text{exp}$ and GDP but with causality flowing from GDP to both $H\text{exp}$ (s). The empirical connotation of this result is that: first, the $TH\text{exp}$ and $Pr\ H\text{exp}$-led growth hypotheses are not supported by our findings. This implies that using the aggregate data for W.AFRICA for the period 1992-2020 in the context of the augmented Solow growth model, $TH\text{exp}$ and $Pr\ H\text{exp}$ are not determinants of GDP; second, economic growth-led $TH\text{exp}$, $PbH\text{exp}$ and $Pr\ H\text{exp}$ hypotheses are validated by our results and findings in this study. However, bidirectional causality exists between only $PbH\text{exp}$ and GDP. This means that the changes and variances that occurred in the performance of $TH\text{exp}$ in the sub-region were caused by the growth of the economy while the changes in GDP are not fundamentally explained by the variations in $TH\text{exp}$; implying that
Bidirectional causal linkage was found between \( \text{PHIV} \) and \( H_{\text{exp}} \) mix; a bidirectional causality was established between \( \text{PHIV} \) and \( TH_{\text{exp}} \). Bidirectional causal linkage was found between \( \text{PHIV} \) and \( PbH_{\text{exp}} \) and, \( \text{PHIV} \) and \( PrH_{\text{exp}} \) in W.AFRICA. This result suggests that the \( \text{PHIV} \) epidemic has attracted huge \( H_{\text{exp}} \) to W.AFRICA; and that \( H_{\text{exp}} \) has been very effective and pivotal to the reversal, curtailing and mitigation of the HIV pandemic in W.AFRICA.

Concerning the interconnectivity between \( \text{PHIV} \) and \( H_{\text{exp}} \) mix; a bidirectional causality was established between \( \text{PHIV} \) and \( TH_{\text{exp}} \). Bidirectional causal linkage was found between \( \text{PHIV} \) and \( PbH_{\text{exp}} \) and, \( \text{PHIV} \) and \( PrH_{\text{exp}} \) in W.AFRICA. This result suggests that the \( \text{PHIV} \) epidemic has attracted huge \( H_{\text{exp}} \) to W.AFRICA; and that \( H_{\text{exp}} \) has been very effective and pivotal to the reversal, curtailing and mitigation of the HIV pandemic in W.AFRICA.

### Table 5

**Results of Hsiao Causality Test for Health Expenditure Versus Economic Growth West Africa**

<table>
<thead>
<tr>
<th>HYPOTHESIS TESTED</th>
<th>HSIAO CRITERIA FOR EVALUATING RESULTS</th>
<th>NATURE OF CAUSALITY OBTAINED</th>
<th>DECISION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Health Expenditure-Led Growth Hypothesis</td>
<td>( 1.114 \times 10^{-2} &lt; 3.442 \times 10^{-2} ) ( [F(m^*)] ) ( [F(m^<em>n^</em>)] )</td>
<td>Neutral Causality ((TH_{\text{exp}} \rightarrow \text{GDP}))</td>
<td>Total Health Expenditure-led Economic Growth is not validated for West Africa</td>
</tr>
<tr>
<td>Growth-Led Total Health Expenditure Hypothesis</td>
<td>( 4.699 \times 10^{-3} &gt; 1.611 \times 10^{-3} ) ( [F(m^*)] ) ( [F(m^<em>n^</em>)] )</td>
<td>Unidirectional Causality ((GDP \rightarrow TH_{\text{exp}}))</td>
<td>Economic Growth-led Total Health Expenditure is validated for West Africa</td>
</tr>
<tr>
<td>Public Health Expenditure-Led Growth Hypothesis</td>
<td>( 2.178 \times 10^{-2} &gt; 1.216 \times 10^{-2} ) ( [F(m^*)] ) ( [F(m^<em>n^</em>)] )</td>
<td>Unidirectional Causality ((GDP \rightarrow PbH_{\text{exp}}))</td>
<td>Public Health Expenditure-led Economic Growth is validated for West Africa</td>
</tr>
<tr>
<td>Growth-Led Public Health Expenditure Hypothesis</td>
<td>( 4.782 \times 10^{-4} &gt; 4.016 \times 10^{-4} ) ( [F(m^*)] ) ( [F(m^<em>n^</em>)] )</td>
<td>Unidirectional Causality ((GDP \rightarrow PbH_{\text{exp}}))</td>
<td>Economic Growth-led Public Health Expenditure is validated for West Africa</td>
</tr>
<tr>
<td>Non-Household Private Health Expenditure-Led Growth Hypothesis</td>
<td>( 1.020 \times 10^{-2} &gt; 4.222 \times 10^{-2} ) ( [F(m^*)] ) ( [F(m^<em>n^</em>)] )</td>
<td>Neutral Causality ((PrH_{\text{exp}} \rightarrow GDP))</td>
<td>Non-household Private Health Expenditure-led Economic Growth is not validated for West Africa</td>
</tr>
<tr>
<td>Growth-Led Non-household Private Health Expenditure Hypothesis</td>
<td>( 7.350 \times 10^{-3} &gt; 6.215 \times 10^{-3} ) ( [F(m^*)] ) ( [F(m^<em>n^</em>)] )</td>
<td>Unidirectional Causality ((GDP \rightarrow PrH_{\text{exp}}))</td>
<td>Economic Growth-led Non-household Private Health Expenditure is validated for West Africa</td>
</tr>
<tr>
<td>Household Out-of-Pocket Private Health Expenditure-Led Growth Hypothesis</td>
<td>( 2.041 \times 10^{-2} &gt; 3.324 \times 10^{-2} ) ( [F(m^*)] ) ( [F(m^<em>n^</em>)] )</td>
<td>Neutral Causality ((Oohexp \rightarrow GDP))</td>
<td>Household Out-of-Pocket Private Health Expenditure-Led Economic is not validated for West Africa</td>
</tr>
<tr>
<td>Growth-Led Household Out-of-Pocket Private Health Expenditure Hypothesis</td>
<td>( 6.510 \times 10^{-3} &gt; 4.044 \times 10^{-3} ) ( [F(m^*)] ) ( [F(m^<em>n^</em>)] )</td>
<td>Unidirectional Causality ((GDP \rightarrow Oohexp))</td>
<td>Economic Growth-led Household out-of-Pocket Private Health Expenditure is validated for West Africa</td>
</tr>
</tbody>
</table>

**Source:** Ishioro(2022)

**NOTE:** The Hsiao Criteria for evaluating the empirical results is: if \( F(m^*) > F(m^*n^*) \) then do not reject the hypothesis but if \( F(m^*) < F(m^*n^*) \) reject the hypothesis

**Hsiao Results for Health Expenditure versus HIV Prevalence**
The results of the Hsiao test presented in table 6 validated the total health expenditure-led HIV prevalence hypothesis. This means that causality flows from TH exp to PHIV implying that TH exp Granger-caused PHIV. This further means that TH exp is a significant determinant for reversing PHIV in West Africa. Also, since TH exp is generally made up of PrH exp and PbH exp, the results suggest that private health investment in the fight against PHIV can only become a veritable tool for reversing the doldrums of the epidemic if adequately complemented by robust PbHe. This is the complementarity condition referred to in Bhattacharya and Qiao (2005). Therefore, we do not reject the total health expenditure-led PHIV hypothesis. The results also reveals a reverse (feedback) causality: there is a flow of causality from PHIV to TH exp. This means that the PHIV -led TH exp hypothesis is supported by our results and findings. Hence, we do not reject the PHIV - led THexp hypothesis.

Furthermore, the PbH exp-led PHIV and, PrH exp and Oohexp -led PHIV hypotheses are validated and supported by our results as there exist a one-directional causality from PbH exp and PrH exp to PHIV with feedback causations. Empirically, it means that all the components of H exp (such as TH exp, PrH exp, Oohexp and PbH exp) "Granger-cause" PHIV in West Africa during the period covered by this study. This suggests that all the components of H exp ( given as TH exp, PrH exp, Oohexp and PbH exp) are potent determinants and drivers of the mitigation of the prevalence of HIV in the West African sub-region.

Table 6
Results of Hsiao Causality Test for West Africa

<table>
<thead>
<tr>
<th>HYPOTHESIS TESTED</th>
<th>HSIAO CRITERIA FOR EVALUATING RESULTS</th>
<th>NATURE OF CAUSALITY OBTAINED</th>
<th>DECISION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Health Expenditure-Led HIV Prevalence Hypothesis</td>
<td>0.989 x 10^{-4} &gt; 0.172 x 10^{-4} \left[ \frac{F(n*)}{F(n*)} \right]</td>
<td>Unidirectional Causality (TH exp \Rightarrow PHIV)</td>
<td>Total Health Expenditure-led HIV Prevalence Hypothesis holds for West Africa</td>
</tr>
<tr>
<td>HIV Prevalence-Led Total Health Expenditure Hypothesis</td>
<td>4.614 x 10^{-2} &gt; 2.33 x 10^{-2} \left[ \frac{F(n*)}{F(n*)} \right]</td>
<td>Unidirectional Causality (PHIV \Rightarrow TH exp)</td>
<td>HIV Prevalence-led Total Health Expenditure Hypothesis holds for West Africa</td>
</tr>
<tr>
<td>Public Health Expenditure-Led HIV Prevalence Hypothesis</td>
<td>3.022 x 10^{-1} &gt; 2.871 x 10^{-1} \left[ \frac{F(n*)}{F(n*)} \right]</td>
<td>Unidirectional Causality (PbH exp \Rightarrow PHIV)</td>
<td>Public Health Expenditure-led HIV Prevalence Hypothesis holds for West Africa</td>
</tr>
<tr>
<td>HIV Prevalence-Led Public Health Expenditure Hypothesis</td>
<td>3.777 x 10^{-4} &gt; 3.059 x 10^{-4} \left[ \frac{F(n*)}{F(n*)} \right]</td>
<td>Unidirectional Causality (PHIV \Rightarrow PbH exp)</td>
<td>HIV Prevalence-led Public Health Expenditure Hypothesis holds for West Africa</td>
</tr>
<tr>
<td>Non-Household Private Health Expenditure-Led HIV Prevalence Hypothesis</td>
<td>1.700 x 10^{-3} &gt; 0.039 x 10^{-3} \left[ \frac{F(n*)}{F(n*)} \right]</td>
<td>Unidirectional Causality (PrH exp \Rightarrow PHIV)</td>
<td>Non-Household Private Health Expenditure-led HIV Prevalence Hypothesis holds for West Africa</td>
</tr>
<tr>
<td>HIV Prevalence-Led Non-household Private Health Expenditure Hypothesis</td>
<td>5.559 x 10^{-2} &gt; 4.275 x 10^{-2} \left[ \frac{F(n*)}{F(n*)} \right]</td>
<td>Unidirectional Causality (PHIV \Rightarrow PrH exp)</td>
<td>HIV Prevalence-led Non-Household Private Health Expenditure Hypothesis holds for West Africa</td>
</tr>
<tr>
<td>Household Out-of-Pocket Private Health Expenditure-Led HIV Prevalence Hypothesis</td>
<td>3.420 x 10^{-2} &gt; 1.325 x 10^{-2} \left[ \frac{F(n*)}{F(n*)} \right]</td>
<td>Unidirectional Causality (Oohexp \Rightarrow PHIV)</td>
<td>Household Out-of-Pocket Private Health Expenditure-Led HIV Prevalence Hypothesis holds for West Africa</td>
</tr>
</tbody>
</table>
Furthermore, the results show that PHIV "Granger causes" $TH_\text{exp}$, $PrH_\text{exp}$, $Oohexp$ and $PbH_\text{exp}$ in W.AFRICA. Therefore, the results supported and validated the HIV prevalence-led $TH_\text{exp}$, $PbH_\text{exp}$ and, $Oohexp$ and household private health expenditure hypotheses given that there is a one-way causal linkage flowing from PHIV to each of these components of $H_\text{exp}$. This implies that the pressure exerted by the toll of PHIV on the components of $H_\text{exp}$ is both statistically and quantitatively Hsiao-tangible and significant in the explanation of the performance, changes and variations of $TH_\text{exp}$, $PrH_\text{exp}$, $Oohexp$ and $PbH_\text{exp}$ in W.AFRICA.

**CONCLUSION**

The major pre-occupation of this study is to investigate the interconnectivity among PHIV, GDP and $H_\text{exp}$ in W.AFRICA using aggregate sub-regional data. Furthermore, for the purpose of thoroughness and empirical depth, $H_\text{exp}$ is disaggregated into three (3) components viz: $TH_\text{exp}$, $PbH_\text{exp}$ and $PrH_\text{exp}$. $PrH_\text{exp}$ was further sub-categorized into non-household private and household out-of-pocket private health expenditures. These components are individually used as $H_\text{exp}$ vectors in testing the unit root, cointegration and Hsiao causality tests among PHIV,GDP and disaggregated $H_\text{exp}$ in W.AFRICA. The results revealed that $TH_\text{exp}$ and $PrH_\text{exp}$ do not "Granger-cause" GDP in W.AFRICA; showing the existence of a one-directional causality between $TH_\text{exp}$ and GDP; and $PrH_\text{exp}$ and GDP. The results suggest that $TH_\text{exp}$ and $PrH_\text{exp}$-led growth hypotheses are not supported by our findings. This insinuates that both $TH_\text{exp}$ and $PrH_\text{exp}$ (s) are not determinants of GDP; hence economic growth-led $TH_\text{exp}$, $PbH_\text{exp}$ and $PrH_\text{exp}$ hypotheses are validated by our results. Two-way causality exists between $PbH_\text{exp}$ and GDP.

Furthermore, a bidirectional causality was established between $TH_\text{exp}$ and PHIV. This portends $TH_\text{exp}$ as a mechanism for reversing the conundrum of the HIV epidemic in W. Africa. Therefore, $TH_\text{exp}$ policies should be designed and implemented in consonance with HIV mitigation policies in view. Since $TH_\text{exp}$ is a combination of other facets of $H_\text{exp}$, we recommend that all the facets of $H_\text{exp}$ be sustainably and consistently increased in order to mitigate the contumacious reverberations of the epidemic in W. Africa.

**References**


