The effect of the quality of institutions on the relationship between public health expenditure and the infant mortality rate: evidence from the PVAR (X) and PSTR approaches

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Abstract: Infant mortality is a major health problem in developing countries. It is an important indicator of a country's public health as it goes hand in hand with socio-economic conditions and many others. Public health spending has been committed to reducing this scourge. This has led to the completion of numerous studies which have yielded mixed results. The main objective of this study is to test the effect of public health expenditure (% GDP) on the infant mortality rate, taking into account the role that institutional quality can play. To achieve this, we use two approaches which are the autoregressive vector panel model with exogenous variables (PVAR (X)) and the smooth threshold regression model (PSTR) on annual data covering the period 2002-2016 and covering 37 African countries. Sub-Saharan. Our main results through the PVAR (X) reveal that in the absence of institutional variables, public health expenditure has a negative and significant effect on the infant mortality rate, whereas, in the presence of the various institutional variables, this effect is still negative but is no longer significant. Our results show that the presence of institutional variables, the weight of public health expenditure in explaining the infant mortality rate. In addition, our results show through the PSTR that there is a certain level of institutional qualities that these countries must achieve for public health expenditure to positively affect infant mortality rates. These thresholds oscillate for all the institutional variables around 7%. Taking institutional variables into account will help reduce infant mortality in Sub-Saharan African countries.

Keywords: Health expenditure, Infant mortality rate, Quality of institutions, PVAR model (X)), PSTR model.

JEL Classification: H51 ; J12 ; B52 ; C33 ; C23.

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1. Introduction

Infant mortality remains an important indicator of a country's public health insofar as it goes hand in hand with certain variables such as socio-economic conditions and public health practices, the quality and access to medical care, maternal health, to name but a few. The analysis of these various variables mentioned above presents infant mortality as the epicentre of the main economic policies of States and international organizations. Thus, the UNICEF report (2017) underlines that nearly 50 million children under the age of 5 have been saved since 2000, which testifies to the seriousness of the commitment made by governments and development partners to end preventable child deaths. In addition, the Sustainable Development Goals (SDGs, 2015), the World Bank (2017), and the World Health Organization (WHO, 2017) integrate the issue of child mortality in most of their development assistance programs. However, according to joint stillbirth estimates released by UNICEF and WHO in 2020, nearly 2 million babies are stillborn each year. In addition, according to UN statistics (2017), 15,000 children died before reaching their fifth birthday, 46% of them in the first 28 days of their life, or 7,000 newborns. According to the World Bank (2018), two regions alone accounted for more than 80% of deaths of children under five: Sub-Saharan Africa (54%) and South Asia (28%). Half of the deaths recorded were recorded in 5 countries: India (24%), Pakistan (10%), Nigeria (9%), the Democratic Republic of the Congo (4%), and Ethiopia (3%). At the current rate, further efforts must be made to prevent more newborns from dying at birth or in the days following childbirth. Therefore, studies are looking at the factors that can explain infant mortality.

Studies support that the evolution of this scourge is closely linked to the health and social conditions of the country. Thus, according to the 2020 reports from UNICEF and WHO, while the deaths of children under 5 had reached a low of 5.2 million in 2019, disruptions in child health services and maternal health caused by the COVID-19 pandemic threaten millions of more lives. In addition, the UNICEF report points out that nearly 68% of the 77 countries surveyed reported that children's medical examinations and immunization services were disrupted to a greater or lesser extent. In addition, 63% reported experiencing disturbances in antenatal exams and 59% in postnatal care. Similarly, the WHO report reveals that 52% of the 105 countries surveyed reported disruption in health services for sick children and 51% in services to manage malnutrition. However, these types of interventions are vital in stopping the deaths of newborns and children. By way of illustration, according to the same WHO report, women cared for by midwives qualified according to international standards have a 16% lower risk of losing their baby and 24% lower risk of giving birth prematurely. In addition, in the theory of exogenous growth, health during childhood constitutes an important element of socio-economic development. This situation has sparked a research interest for economists on the factors of infant mortality. These factors are among, others the level of education of the parents, the environment of residence, but also and above all the public expenditure on health.

The debate over the relationship between public health spending and infant mortality has been the subject of recent studies with mixed results. The first part of the literature emphasizes that public health expenditure does not affect infant mortality. For others, this effect is limited, and the third category of studies has shown that public health expenditure affects only specific variables other than infant mortality. Thus, Musgrove (1996) shows that public health expenditure does not affect infant mortality. In the same vein, Filmer and Pritchett (1997), pointed out that variables such as income, income inequality, women's education, and the degree of ethnolinguistic fragmentation are the explanatory factors of infant mortality in a country, unlike public health spending. The second section of the literature presents a positive relationship between public health spending and infant mortality (Baldacci et al., 2004; Berger and Messer, 2002). On the other hand, some economists prove the

contrary (see Filmer and Pritchett, 1999). In addition to previous work, Baldacci et al. (2003) show in their study that the results depend on the types of data and the estimation methods used. Some studies show that the contribution of public health expenditure to the reduction of infant and maternal mortality is even less statistically significant (Filmer et al., 1998; Musgrove, 1996). As for Berger and Messer (2002), they underline in their studies that an increase in the share of public health expenditure leads to an increase in mortality rates while Khaleghian and Gupta (2005) show that public health expenditure plays a primary role in the poor in low-income countries compared to high-income countries. Other economists such as Harttgen and Misselhorn (2006) show that access to health care infrastructure significantly reduces child mortality and socio-economic variables are the main determinants of child mortality (Nolte and McKee, 2004; Stleger, 2001).

However, these previous studies are subject to discussion. They seek to test only the effect of public health expenditure on infant mortality. Moreover, it is not entirely accepted that the relationship between these variables is negative as one would expect. Empirically, the nature of this relationship remains ambiguous. Faced with such an observation, it seems inevitable to identify the variable which, when present, allows public health expenditure to reduce the infant mortality rate. Thus, recent literature places particular emphasis on institutional variables. However, except for the work of Dhrifi (2020) with the method of generalized moments (GMM) in the Blundell-Bond system (1998), very few studies have focused on Sub-Saharan Africa, yet this part of the world remains the most affected by this scourge. In addition, this work has only assessed the role played by the quality of institutions in the relationship between public health expenditure and infant mortality and these studies have ignored the issue of a threshold of institutional variables in this relationship. This article, therefore, aims to revive the forgotten interest in the quality of institutions in the relationship between public health expenditure and Africa.

Four important contributions can be drawn from this study compared to the work of Dhrifi (2020). First, we use the six institutional variables from Kaufmann et al. (2010). The six aggregate indicators are based on over 30 underlying data sources reporting the perceptions of governance of many survey respondents Second, we analyze the countries of sub-Saharan Africa because it is an area very affected by this scourge. Third, we do not create an institutional quality-public health expenditure interaction variable to capture the role of institutions in the public health expenditure-infant mortality relationship, rather we use a panel VAR (X) model. This model, used very little in the empirical literature, is interesting insofar as the gradual addition of each institutional variable can modify the nature of the relationship between public health expenditure and infant mortality. Fourth, we do not determine the thresholds of institutional variables using the first order condition of the estimated model, instead, we will determine the thresholds of these institutional variables using the PSTR model presented by González et al. (2017).

The paper is structured as follows: section 2, discloses the empirical model and the theoretical framework, section 3 describes the data from the study, section 4 presents the econometric methods, in section 5, are discussed the outcomes of our and the conclusion of our study will be done in section 6.

2. Empirical model and theoretical framework

The analysis of the relationship between public expenditure on public health and the infant mortality rate requires the use of an econometric model in light of the literature. Thus, we opt for a standard model presented most of the time in previous studies (see Berthelemy and Seban, 2009; Baldacci et

al., 2004; Berger and Messer, 2002). This model explains the Infant Mortality Rate by public health expenditure and control variables. This model is written as follows:

$$TMI_{it} = \alpha_i + \beta' X_{it} + \varepsilon_{it} \tag{1}$$

Where α_i is the country-specific effect distributed independently and constant for all countries, represents the country and t denotes the study period. ε_{it} is the error term assumed to be independently distributed, TMI_{it} is the endogenous variable denoting infant mortality under 5 (per 1000 live births). This variable is used to measure the proportion of deaths among infants and children under 5 years old. X_{it} is the vector of the explanatory K variables that are likely to affect TMI variable. Since we want to assess the effect of public health expenditure on the infant mortality rate considering the institutional quality, equation (1) can be rewritten as follows:

$$TMI_{it} = \alpha_i + \alpha_1 DEP _ SANT_{it} + \alpha_2 INST_{it} + \beta' X_{it} + \varepsilon_{it}$$
(2)

 DEP_SANT_{it} represents public health expenditure (% GDP); $INST_{it}$ denotes the institutional variables of Kaufmann et al. (2010); X_{it} represents all the variables identified in health economics as the determinants of TMI. These variables are, among others, the literacy rate of women, medical density (defined by doctors as doctors per thousand inhabitants), technological progress measured by research and development expenditure (% of GDP), access to healthcare. Water is measured by the proportion of households using tap water, protected wells, and boreholes, the growth rate of GDP per capita, urbanization measured by the urban population as a proportion of the total population, and CO2 which designates the environmental variable.

3. Data of the survey

The data used in this paper covers the period 1996-2019. The sample is made up of 93 countries in Sub-Saharan Africa. The sample size and period of our study are limited by the availability of data on the control variables. These annual data are collected from the World Health Organization (WHO), the International Country Risk Guide (ICRG), and World Development Indicators (WDI).

4. Methodologies

This section is a detailed description of the methodological steps of the study.

4.1. Pesaran's Test of Cross Sectional Dependence

Consider the standard panel-data model

$$y_{it} = \alpha_i + \beta_i' X_{it} + u_{it} \tag{3}$$

Where X_{it} is a $k \times 1$ vector of regressors, β_i are defined on a compact set and are allowed to vary across *i*, and α_i are time-invariant individual nuisance parameters. Under the null hypothesis, u_{it} is assumed to be independent and identically distributed over periods and across cross-sectional units. Under the alternative, u_{it} may be correlated but the assumption of no serial correlation remains. In the context of seemingly unrelated regression estimation, Breusch and Pagan (1980) proposed an LM statistic, which is valid for fixed N and $T \rightarrow \infty$ and is given by,

$$CD_{LM} = T \sum_{i=1}^{N-1} \sum_{j=i+1}^{N} \hat{\rho}_{ij}$$
(4)

where $\hat{\rho}_{ij}$ is the sample estimate of the pair-wise correlation of the residuals. CD_{LM} is asymptotically distributed as χ^2 with N(N-1)/2 degrees of freedom under the null hypothesis. However, this test is likely to exhibit substantial size distortions when N is large and T is finite. A situation that is commonly met in empirical applications, mainly because the CD_{LM} statistic is not correctly centered for finite T and the bias is likely to get worse with N large. Pesaran (2004) has proposed the following alternative

$$CD = \sqrt{\frac{2T}{N(N-1)}} \left(\sum_{i=1}^{N-1} \sum_{j=i+1}^{N} \hat{\rho}_{ij} \right)$$
(5)

and showed that under the null hypothesis of no cross-sectional dependence $CD \Rightarrow N(0,1)$ for T sufficiently large, as $N \to \infty$; where here ' \Rightarrow ' denotes 'converge to'. The CD statistic has mean at exactly zero for fixed values of T and N, under a wide range of panel-data models, including homogeneous/heterogeneous dynamic models and nonstationary models.

4.2. Unit root tests in panel

In this subsection, we present the second generation unit root tests of Pesaran (2003 and 2007).

4.2.1. Pesaran's second generation unit root tests (2003 and 2007)

According to Pesaran (2003 and 2007), let y_{it} be the observation on the *ith* cross-section unit at time t and suppose that it is generated according to the simple dynamic linear heterogeneous panel data model

$$y_{it} = (1 - \phi_i) \mu_i + \phi_i y_{i,t-1} + u_{it}, \quad i = 1, \dots, N; \ t = 1, \dots, T$$
(6)

where initial value, y_{it} , has a given density function with a finite mean and variance, and the error term, y_{it} , has the single-factor structure

$$u_{it} = \gamma_i f_t + \varepsilon_{it} \tag{7}$$

in which f_t is the unobserved common effect, and ε_{it} is the individual-specific (idiosyncratic) error. It is convenient to write (6) and (7) as

$$\Delta y_{it} = \alpha_i + \beta_i y_{i,t-1} + \gamma_i f_t + \varepsilon_{it}$$
(8)

Where, $\alpha_i = (1 - \phi_i) \mu_i$, $\beta_i = -(1 - \phi_i)$ and $\Delta y_{it} = y_{it} - y_{i,t-1}$. The unit root hypothesis of interest, $\phi_i = 1$, can now be expressed as

$$H_0: \beta_i = 0 \text{ for all } i \tag{9}$$

against the possibly heterogeneous alternatives,

$$H_1: \beta_i < 0, \ i = 1, 2, \dots, N_1; \ \beta_i = 0, \ i = N_1 + 1, N + 2, \dots, N$$
(10)

Otherwise, we shall assume that N_1/N , is stationary, nonzero and tends to the fixed value δ such that $0 < \delta \le 1$ as $N \to \infty$. As noted in Im et al. (2003), this condition is necessary for the consistency of the panel unit root tests. We shall also make the following assumptions:

Assumption 1 The idiosyncratic shocks, ε_{it} , i = 1, ..., N; t = 1, ..., T are independently distributed both across *i* and *t*, have mean zero, variance σ_i^2 , and finite fourth-order moment.

Assumption 2 The common factor, f_t , is serially uncorrelated with mean zero and a constant

variance, σ_f^2 , and finite fourth-order moment. Without loss of generality σ_f^2 will be set equal to unity. **Assumption 3** ε_{it} , f_t , and γ_i are independently distributed for all *i*.

Let $\overline{\gamma} = N^{-1} \sum_{j=1}^{N} \gamma_j$ and suppose that $\overline{\gamma} \neq 0$ for a fixed N and as $N \to \infty$. Then following the line of reasoning in Pesaran (2006), the common factor f_t can be proxied by the cross-section mean of y_{it} , namely $\overline{y}_t = N^{-1} \sum_{j=1}^{N} y_{jt}$, and its lagged value(s), $\overline{y}_{t-1}, \overline{y}_{t-2}, \ldots$, for N sufficiently large. We shall therefore base our test of the unit root hypothesis, (9), on the t – ratio of the OLS estimate of $b_i(\hat{b}_i)$ in the following cross-sectionally augmented DF (CADF) regression:

$$\Delta y_{it} = a_i + b_i y_{i,t-1} + c_i \overline{y}_{t-1} + d_i \Delta \overline{y}_t + e_{it}$$

$$\tag{11}$$

Denoting this t – ratio by $t_i(N,T)$ we have

$$t_{i}(N,T) = \frac{\Delta y_{i}' \overline{M}_{w} y_{i,-1}}{\hat{\sigma}_{i} \left(y_{i,-1}' \overline{M}_{w} y_{i,-1} \right)^{1/2}}$$
(12)

Always following Pesaran (2003 and 2007), considering y_{it} defined by (8) and consider the statistics $t_i(N,T)$ defined by (12). Suppose that Assumptions 1–3 hold and $\overline{\gamma}$ tends to a finite non-zero limit as $N \to \infty$, then under (4) and as N and $T \to \infty$, $t_i(N,T)$ has the same sequential $(N \to \infty, T \to \infty)$ and joint $[(N,T)_j \to \infty]$ limit distributions, referred to as cross-sectionally augmented Dickey–Fuller (CADF) distribution given by

$$CADF_{if} = \frac{\int_{0}^{1} W_{i}(r) dW_{i}(r) - \psi_{if}' \Lambda_{if}' \kappa_{if}}{\left(\int_{0}^{1} W_{i}^{2}(r) dr - \kappa_{if}' \Lambda_{f}^{-1} \kappa_{if}\right)^{1/2}}$$
(13)

Where $\Lambda_{f} = \begin{pmatrix} 1 & \int_{0}^{1} W_{f}(r) dr \\ \int_{0}^{1} W_{f}(r) dr & \int_{0}^{1} W_{f}^{2}(r) dr \end{pmatrix}; \psi_{if} = \begin{pmatrix} W_{i}(1) \\ \int_{0}^{1} W_{f}(r) dW_{i}(r) \end{pmatrix}; \kappa_{if} = \begin{pmatrix} \int_{0}^{1} W_{i}(r) dr \\ \int_{0}^{1} W_{f}(r) W_{i}(r) dr \end{pmatrix}$

With $W_i(r)$ and $W_f(r)$ being independent standard Brownian motions. For the joint limit distribution to hold it is also required that as $(N,T)_j \rightarrow \infty$, $N/T \rightarrow k$, where k is a non-zero, finite positive constant. One possibility would be to consider a cross-sectionally augmented version of the IPS test based on

$$CIPS(N,T) = t - bar = N^{-1} \sum_{i=1}^{N} t_i(N,T)$$
(14)

where $t_i(N,T)$ is the cross-sectionally augmented Dickey–Fuller statistic for the *ith* cross-section unit given by the *t*-ratio of the coefficient of $y_{i,t-1}$ in the CADF regression defined by (11).

4.3. Optimal delay of the autoregressive vector model in panel with exogenous variables

Panel VAR analysis is predicated upon choosing the optimal lag order in both panel VAR specification and moment condition. Andrews and Lu (2001) proposed MMSC for GMM models based on Hansen's (1982) J statistic of overidentifying restrictions. Their proposed MMSC are analogous to various commonly used maximum likelihood-based model-selection criteria, namely, the Akaike information criteria (AIC) (Akaike, 1969), the Bayesian information criteria (BIC) (Schwarz 1978; Rissanen 1978; Akaike 1977), and the Hannan–Quinn information criteria (HQIC) (Hannan and Quinn, 1979).

$$MMSC_{BIC,n}(k, p, q) = J_{n}(k^{2}p, k^{2}q) - (|q| - |p|)k^{2} \ln n$$

$$MMSC_{AIC,n}(k, p, q) = J_{n}(k^{2}p, k^{2}q) - 2k^{2}(|q| - |p|)$$

$$MMSC_{HQIC,n}(k, p, q) = J_{n}(k^{2}p, k^{2}q) - Rk^{2}(|q| - |p|) \ln n \quad R > 2$$
(15)

Where $J_n(k, p, q)$ is the J statistic of overidentifying restriction for a k-variate panel VAR of order p and moment conditions based on q lags of the dependent variables with sample sizen.

4.4. The autoregressive vector panel model with exogenous variables (PVAR (X))

We consider a k-variate homogeneous panel VAR of order p with panel-specific fixed effects represented by the following system of linear equations,

$$Y_{it} = Y_{it-1}A_1 + Y_{it-2}A_2 + \dots + Y_{it-p+1}A_{p-1} + Y_{it-p}A_p + X_{it}B + u_i + e_{it}$$

$$i \in \{1, 2, \dots, N\}, \ t \in \{1, 2, \dots, T\}$$
 (16)

Where Y_{it} is a $(1 \times k)$ vector of dependent variables, X_{it} is a $(1 \times l)$ vector of exogenous covariates, and \mathbf{u}_i and \mathbf{e}_{it} are $(1 \times k)$ vectors of dependent variable-specific panel fixed effects and idiosyncratic errors, respectively. The $(k \times k)$ matrices $\mathbf{A}_1, \mathbf{A}_2, \dots, \mathbf{A}_{p-1}, \mathbf{A}_p$ and the $(l \times k)$ matrix B are parameters to be estimated. We assume that the innovations have the following characteristics: $E(\mathbf{e}_{it})=0, E(\mathbf{e}'_{it}\mathbf{e}_{it})=\sum_{i}$, and $E(\mathbf{e}'_{it}\mathbf{e}_{is})=0$ for all t > s.

Like Holtz-Eakin and al. (1988), we assume that the cross-sectional units share the same underlying data generating process, with the reduced-form parameters $A_1, A_2, ..., A_{p-1}, A_p$ and B to be common among them. The parameters above may be estimated jointly with the fixed effects or, alternatively, with ordinary least squares (OLS) but, with the presence of lagged dependent variables in the right-hand side of the system of equations, estimates would be biased even with large N (Nickell, 1981). Various estimators based on GMM have been proposed to calculate consistent estimates of the above equation, especially in fixed T and large N settings (Kiviet, 1995; Bun and Carree, 2006). With our assumption that errors are serially uncorrelated, the model in first difference (FD) may be consistently estimated equation by equation by instrumenting lagged differences with differences and levels of Y_{ir} from earlier

periods as proposed by Anderson and Hsiao (1982). This estimator, however, poses some problems so Arellano and Bover (1995) proposed forward orthogonal deviation (FOD) as an alter-native transformation, which does not share the weaknesses of the FD transformation. The estimators by Anderson and Hsiao (1982) and by Arellano and Bover (1995), as well as other dynamic panel GMM estimators using similar moment restrictions, like those by Arellano and Bond (1991) and by Blundell and Bond (1998).

In the time-series VAR, it is common to test each variable for stationarity using unit-root tests. This is also relevant in GMM estimation of linear dynamic panel models. As noted by Blundell and Bond (1998) in the univariate case, the GMM estimators suffer from the weak instruments problem when the variable being modeled is near unit root (Bond, 2002). The moment conditions become completely irrelevant when unit root is present. While equation-by-equation GMM estimation yields consistent estimates of panel VAR, fitting the model as a system of equations may result in efficiency gains (Holtz-Eakin and al, 1988). Suppose the common set of $L \ge kp + l$ instruments isgiven by the row vector Z_{it} , where $X_{it} \in Z_{it}$, and equations are indexed by a number in superscript. Consider the following transformed panel VAR model based on (16) but represented in a more compact form,

$$Y_{it}^{*} = \tilde{Y}_{it}^{*} \mathbf{A} + e_{it} = \begin{bmatrix} y_{it}^{1*} & y_{it}^{2*} & \dots & y_{it}^{k-1*} & y_{it}^{k*} \end{bmatrix}$$

$$\tilde{Y}_{it}^{*} = \begin{bmatrix} Y_{it-1}^{*} & Y_{it-2}^{*} & \dots & Y_{it-p+1}^{*} & Y_{it-p}^{*} & X_{it}^{*} \end{bmatrix}$$

$$e_{it}^{*} = \begin{bmatrix} e_{it}^{1*} & e_{it}^{2*} & \dots & e_{it}^{k-1*} & e_{it}^{k*} \end{bmatrix}; \mathbf{A}' = \begin{bmatrix} \mathbf{A}_{1}' & \mathbf{A}_{2}' & \dots & \mathbf{A}_{p-1}' & \mathbf{A}_{p}' & \mathbf{B}' \end{bmatrix}$$
(17)

where the asterisk denotes some transformation of the original variable. If we denote the original variable, then the FD transformation implies that $m_{it}^* = m_{it} - m_{it-1}$, while for the forward orthogonal deviation, $m_{it}^* = (m_{it} - \overline{m}_{it})\sqrt{T_{it}/(T_{it}+1)}$, where T_{it} is the number of available future observations for panel *i* at time *t* and m_{it} is the average of all available future observations. Suppose we stack observations over panels then over time. The GMM estimator is given by

$$\mathbf{A} = \left(\tilde{Y}^{*'} Z \hat{W} Z' \tilde{Y}^{*}\right)^{-1} \left(\tilde{Y}^{*'} Z \hat{W} Z' Y^{*}\right)$$
(18)

Where \hat{W} is an $(L \times L)$ weighting matrix assumed to be nonsingular, symmetric, and positive semi definite. Assuming that E(Z'e) = 0 and $rank E(\tilde{Y}_{it}^{*'}Z) = kp + l$, the GMM estimator is consistent. The weighting matrix \hat{W} may be selected to maximize efficiency (Hansen, 1982).

4.5. VAR stability

Without loss of generality, we drop the exogenous variables in our notation and focus on the autoregressive structure of the panel VAR in (16). Lütkepohl (2005) and Hamilton (1994) both show that a VAR model is stable if all moduli of the companion matrix \overline{A} are strictly less than one, where the companion matrix is formed by

$$\overline{A} = \begin{bmatrix} A_1 & A_2 & \cdots & A_p & A_{p-1} \\ I_k & O_k & \cdots & O_k & O_k \\ O_k & I_k & \cdots & O_k & O_k \\ \vdots & \vdots & \ddots & \vdots & \vdots \\ O_k & O_k & \cdots & I_k & O_k \end{bmatrix}$$
(19)

Stability implies that the panel VAR is invertible and has an infinite-order vector moving-average (VMA) representation.

4.6. Variance decomposition

The h-step ahead forecast error can be expressed as

$$Y_{it+h} - E(Y_{it+h}) = \sum_{i=0}^{h-1} e_{i(t+h-i)} \Phi_i$$
(20)

where Y_{it+h} is the observed vector at time t+h and $E(Y_{it+h})$ is the *h*-step ahead predicted vector made at time *t*. As with IRFs, we orthogonalize the shocks using the matrix P to isolate each variable's contribution to the forecast-error variance. The orthogonalized shocks $e_{it}P^{-1}$ have a covariance matrix I_k , which allows straightforward decomposition of the forecast-error variance. More specifically, the contribution of a variable *m* to the *h*-step ahead forecast-error variance of variable n may be calculated as

$$\sum_{i=0}^{h-1} \theta_{mn}^2 = \sum_{i=1}^{h-1} (i'_n P \Phi'_i i_m)^2$$
(21)

where \mathbf{i}_s is the sth column of \mathbf{I}_k . In application, the contributions are often normalized relative to the h-step ahead forecast-error variance of variable n

$$\sum_{i=0}^{h-1} \theta_n^2 = \sum_{i=1}^{h-1} \mathbf{i}'_n \Phi_i' \Sigma \Phi_i \mathbf{i}_n$$
(22)

Like those of IRFs, confidence intervals may be derived analytically or estimated using various resampling techniques.

4.7. Impulse–response

The simple IRF Φ_i may be computed by rewriting the model as an infinite VMA, where Φ_i are the VMA parameters.

$$\Phi_{i} = \begin{cases} \mathbf{I}_{k} & i = 0\\ \sum_{j=1}^{i} \Phi_{t-j} \mathbf{A}_{j} & i = 1, 2, \dots \end{cases}$$
(23)

However, the simple IRFs have no causal interpretation. Because the innovations e_{it} are correlated contemporaneously, a shock on one variable is likely to be accompanied by shocks in other variables.

4.8 Panel Smooth Threshold regression (PSTR) model

González and al. (2017) defined the basic PSTR model with two extreme regimes as

$$y_{it} = \mu_i + \lambda_t + \beta_0' x_{it} + \beta_1' x_{it} g(q_{it}; \gamma, c) + u_{it}$$
(24)

for i = 1, ..., N and t = 1, ..., T, where N and T denote the cross-sectional and time dimensions of the panel, respectively. The dependent variable y_{it} is a scalar, x_{it} is a k-dimensional vector of time-

varying exogenous variables, μ_i and λ_i represent fixed individual effects and time effects, respectively, and u_{it} are the errors. Furthermore, the regressors x_{it} are assumed exogenous. The transition function $g(q_{it};\gamma,c)$ in (24) is a continuous function of the observable variable q_{it} and is normalized to be bounded between zero and one. These two extreme values are associated with regression coefficients β_0 and $\beta_0 + \beta_1$. More generally, the value of the transition variable q_{it} determines the value of $g(q_{it};\gamma,c)$, and thus the effective regression coefficients $(\beta_0 + \beta_1 g(q_{it};\gamma,c)))$ for individual *i* at time *t*. We follow Teräsvirta (1994, 1998), by using the logistic specification

$$g\left(q_{it};\gamma,c\right) = \left(1 + \exp\left(-\lambda \prod_{j=1}^{m} \left(q_{it} - c_{j}\right)\right)\right)^{-1} \text{ with } \gamma > 0 \text{ and } c_{1} < c_{2} < \dots < c_{m}$$
(25)

where $c = (c_1, ..., c_m)'$ is an m-dimensional vector of location parameters, the slope parameter γ determines the smoothness of the transitions. The restrictions $\gamma > 0$ and $c_1 < \cdots < c_m$ are imposed for identification purposes. In practice it is usually sufficient to consider m = 1 or m = 2, as these values allow for commonly encountered types of variation in the parameters. For m = 1, the model implies that the two extreme regimes are associated with low and high values of q_{it} with a monotonic transition of the coefficients from β_0 to $\beta_0 + \beta_1$ as q_{it} increases, where the change is centred around c_1 . For m = 2, the transition function has its minimum at $(c_1 + c_2)/2$ and attains the maximum value one both at low and high values of q_{it} .

5. Results and interpretations of estimations

In the section, we present and interpret the major of results of this article.

5.1. Results and interpretations of dependency and unit roots tests

Table 1 below shows the results of the Pesaran (2004) dependency test. Examination of this table indicates a significant probability at the 5% threshold (p-value less than 5%). Thus, we reject the hypothesis, so there is dependence between individuals (countries).

Tuble 1. Result of the intermutvidual dependence test of 1 esurum (2004)							
Variable	CD-test	p-value	Corr	abs(corr)			
ols_res	-2.38	0.017 **	-0.024	0.474			

Table 1: Result of the interindividual dependence to	est of	Pesaran	(2004)
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Note: (**) indicates the rejection of the null hypothesis at the 5% threshold

The result obtained from Table 1 leads us to perform the second generation unit root tests developed by Pesaran (2003, 2007). The results of these tests are shown in Table 2.

Variables	CADF test	CIPS test	Decision criteria
DEP_SANTE	-2.645**	-2.555*	I (0)
 ТХ_РІВНВТ	-3.273***	-3.282***	I (0)
CO2	-2.262*	-1.619**	I (0)
TX_MINF	-2.308*	-2.323 ***	I (0)
VOICE_ACCOUNT	-2.232*	-1.474*	I (0)
STAB_POL	-2.761**	-2.946***	I (0)
REGUL_QUALITY	-2.893***	-2.998***	I (0)
EFF_GOUV	-2.721**	-2.781**	I (0)
CONTROL_CORRUP	-2.516*	-1.751***	I (0)
RULE_LAW	-2.201*	-1.847***	I (0)

Note: (*), (**) and (***) indicate that the variable is stationary if the CADF test and the CIPS test, the statistics of t-bar and of CIPS in absolute value are higher than those of the thresholds of 1%, 5%, 10%.

The analysis of Table 2 reveals that all the variables in the panel are stationary in level. This result shows that we can use the VAR panel model for our regressions. As a prelude to the estimations of the VAR panel model, let us determine the optimal delay.

5.2. Results of determining the number of delays and estimates

The results of the optimal delay number and estimates are shown in Tables 3 and 4 below. **Table 3:** Determination of the optimal number of lags of the PVAR model

Lags	MBIC	MAIC	MQIC	
1	-211.6752*	-23.82701*	-98.44189*	
2	-156.4552	-31.22307	-80.96633	
3	-65.67245	-3.056404	-27.92803	

Note: (*) indicates the minimum value of each information criterion

Analysis of the table reveals that all the minimum values of the identification criteria are observed when the delay is 1. Therefore, we proceed to the model estimates, Table 3 shows the results of the estimates. **Table 4:** Result of the model estimates

Without institutional variables							
TX_MINF DEP_SANTE TX_PIBHBT CO2							
TX_MINF	0.972***	-0.038**	0.178**	-0.001***			
DEP_SANTE	-0.076**	0.626***	0.896	-0.004			
TX_PIBHBT	-0.002	0.001	0.333***	0.001			
CO2	CO2 2.360* -6.562*** -6.692						
With as exogenous variable VOICE_ACCOUNT							
	TX_MINF	DEP_SANTE	TX_PIBHBT	CO2			
TX_MINF	0.966***	0.179	0.222	-0.001			
DEP_SANTE	-0.037	0.934***	0.599	-0.001			
ТХ_РІВНВТ	-0.001	0.005	0.350***	-0.001			
CO2	-0.819	-2.898	18.484	0.758***			
VOICE_ACCOUNT	0.225	-0.131	9.668	-0.065			
With as exogenous variable STAB_POL							

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	TX_MINF	DEP_SANTE	TX_PIBHBT	CO2
TX_MINF	0.964***	0.023	0.140	-0.001
DEP_SANTE	-0.036	0.933***	0.660	-0.001
TX_PIBHBT	-0.001	0.005	0.347***	-0.001
CO2	-0.992	-2.804**	11.070	0.808***
STAB_POL	-0.024	-0.267	-0.981	-0.004
	With as exoge	nous variable REGUL_	QUALITY	
	TX_MINF	DEP_SANTE	TX_PIBHBT	CO2
TX_MINF	0.962***	0.019	0.137	-0.001
DEP_SANTE	-0.039	0.933***	0.688	-0.002
TX_PIBHBT	-0.001	0.005	0.349***	-0.001
CO2	-0.954	-2.794**	10.789	0.813***
REGUL_QUALITY	-0.145	-0.011	1.192	-0.018
	With as exe	ogenous variable EFF_	GOUV	
	TX_MINF	DEP_SANTE	TX_PIBHBT	CO2
TX_MINF	0.964***	0.020	0.126	-0.001
DEP_SANTE	-0.034	0.938***	0.659	-0.001
TX_PIBHBT	-0.001	0.006	0.346***	-0.001
CO2	-0.001	-2.466	10.861 0.7	
EFF_GOUV	0.105	0.341	-0.240	-0.018
	With as exogen	ous variable CONTRO	L_CORRUP	
	TX_MINF	DEP_SANTE	TX_PIBHBT	CO2
TX_MINF	0.963***	0.034	-0.437	-0.002
DEP_SANTE	-0.037	0.976***	-0.938	-0.005
TX_PIBHBT	-0.001	0.010	0.155	-0.001
CO2	-1.061	-0.429	-77.737	0.570
CONTROL_CORRUP	-0.053	1.798	-67.454	-0.180
	With as exc	ogenous variable RULE	_LAW	
	TX_MINF	DEP_SANTE	TX_PIBHBT	CO2
TX_MINF	0.965***	0.020	0.108	-0.001
DEP_SANTE	-0.036	0.933***	0.662	-0.001
ТХ_РІВНВТ	0.001	0.006	0.341***	-0.001
CO2	-0.930	-2.777***	10.409	0.804***
RULE_LAW	0.151	0.050	-1.687	-0.008

Note: (*), (**) and (***) respectively indicate the significance thresholds of 10%, 5% and 1%.

Table 4 presents the results of the estimation of the model estimated using the dynamic GMM method of the two-step system. The dependent variable is the infant mortality rate (TX_MINF) and our variables of interest are the public health expenditure (DEP_SANTE) and the six institutional variables of Kauffman et al. (2010).

The results of the estimates without the institutional variables show some significant relationships. Indeed, we notice that the coefficient of the DEP_SANTE variable is significant at the 5% level and has a negative impact on the infant mortality rate. Thus, a 1% increase in public health expenditure leads to a decrease in the infant mortality rate of 0.076 points. This result is consistent for the TX_MINF. Indeed, an increase of 1% of TX_MINF lowers DEP_SANTE of 0.038%. This confirms the hypothesis that

public health expenditure has a positive, and statistically significant effect on infant mortality. It also means that children's health status depends on the rate of public health expenditure, which suggests that an increase in public health expenditure implies greater access to health care and services that help reduce rates. Mortality. This result confirms other studies which have found a positive relationship between public health expenditure and infant mortality (Berger and Messer, 2002). On the other hand, the coefficient of the variable DEP_SANTE is not significant in the explanation of TX_PIBHBT and CO2.

Regarding the coefficient of the variable GDP growth rate per capita (TX_PIBHBT), the results show that it is negative (-0.002) but not significant in the explanation of TX_MINF. This result is counter intuitive insofar as a high TX_PIBHBT should reduce the TX_MINF, solve the problems of food insecurity, poor buildings and equipment, lack of adequate social infrastructure, improve public health infrastructure such as than water and sanitation, better nutrition. This result can be explained by the fact that the inhabitants of this part of the world have low enough income to meet health needs. Our results, therefore, contradict those of Pritchett and Summers (1996). However, this result is opposed to that of TX_MINF in the explanation of TX_PIBHBT because an increase of 1% of TX_MIN results in an increase of 0.178% of TX_PIBHBT and at the threshold of 5%. This result is explained by the fact that the state of health is a primary factor of productivity, an increase in the TX_MINF will encourage States to prevent the causes of death and enormous resources will be committed to medical research with salaries. Students. Also, households without any income-generating activities will engage in entrepreneurship. All these actions will help raise per capita income. This result is in line with that of DEP_SANTE and CO2.

For the environmental variable (CO2), the coefficient is positively significant at the 10% threshold with a coefficient of 2.360 which means that an increase of 1% in the emission rate of this gas leads to an increase of 2.360% of TX_MINF. This result is explained by the fact that the health of the newborn being very weak, a polluted environment can cause poor breathing and lead to the death of the latter. This result is in line with that of Dhrifi (2018). On the other hand, this result is different from that of TX_MINF in the explanation of CO2 because an increase of 1% of TX_MINF leads to a decrease of 0.001% of CO2 and at the threshold of 1%. This result can be explained by the fact that while pollution rhymes with economic growth, achieving it requires a healthy workforce. Therefore, faced with an increase in TX_MINF, resources will be more likely to reduce it, which will have as a corollary a reduction in CO2 emissions. Also, the same reasoning is struck in the explanation of CO2 on the variable DEP_SANTE insofar as a 1% increase in CO2 reduces DEP_SANTE by 6.562% and to the threshold of 1%.

By considering the estimates in the presence of each institutional variable, the results obtained show us that an increase in the coefficients of the variable DEP_SANTE reduces the TX_MINF without however being significant in the explanation of the TX_MINF. We do note that the presence of institutional variables can attenuate the effect of the relationship between public health expenditure on the infant mortality rate. The non-significance of the coefficients means that institutions in Sub-Saharan Africa deserve to be improved because an improvement in them can be the main factor of the state of health. This confirms the hypothesis that good institutions can drastically reduce TX_MINF by improving public health expenditure allocations. Our work corroborates with that of de Dhrifi (2020). We first proceed to the verification of the stability of the PVAR (X) model, to the decomposition of the variance and to the impulse analysis.

5.3. PVAR Model Stability Result (X)

The results of the stability test show in Table 4 that all the eigenvalues are less than 1 in modulus. At the level of graph 1, we see that all the points are inside the circle. This shows that the model is quite stable.

Table 5: Result of the stability of the eigenvectors							
Eigenvalue	Modulus						
Without institutional variables							
0.971597	0.971597						
0.7236843	0.7236843						
0.4139187	0.4139187						
0.2952145	0.2952145						
With as exogenous varial	ble VOICE_ACCOUNT						
0.9613379	0.9618709						
0.9613379	0.9618709						
0.7359187	0.7359187						
0.349917	0.349917						
With as exogenous va	riable STAB_POL						
0.9631074	0.9635132						
0.9631074	0.9635132						
0.7821423	0.7821423						
0.3428169	0.3428169						
With as exogenous varia	ble REGUL_QUALITY						
0.9666113	0.9670139						
0.9666113	0.9670139						
0.7778634	0.7778634						
0.3456258	0.3456258						
With as exogenous va	riable EFF_GOUV						
0.9656676	0.9660137						
0.9656676	0.9660137						
0.7646821	0.7646821						
0.3424399	0.3424399						
With as exogenous variab	le CONTROL_CORRUP						
0.9750735	0.9754371						
0.9750735	0.9754371						
0.6329412	0.6329412						
0.081213	0.081213						
With as exogenous va	riable RULE_LAW						
0.964533	0.9649164						
0.964533	0.9649164						
0.7773775	0.7773775						
0.3375025	0.3375025						

 Table 5: Result of the stability of the eigenvectors

Without institutional variables



With the exogenous variable STAB_POL



With the exogenous variable EFF_GOUV



With the exogenous variable RULE_LAW



With the exogenous variable VOICE_ACCOUNT



With the exogenous variable REGUL_QUALITY



With the exogenous variable CONTROL_CORRUP



Graph 1: VA stability

5.4. Result of variance decomposition

The results of the variance decomposition are reported in Table 6. **Table 6:** Result of the decomposition of the variance

		CONTRIBUTIONS		
	Withou	it institutional variable	es	
	TX_MINF	DEP_SANTE	TX_PIBHBT	CO2
TX_MINF	57.96% 27.13% 0.09%		0.09%	14.82%
DEP_SANTE	0.38%	92.91%	0.02% 6.	
ТХ_РІВНВТ	1.06%	5.61%	92.61%	0.72%
CO2	0.68%	6.50%	0.57%	92.26%
	With as exoge	nous variable VOICE_A	CCOUNT	
	TX_MINF	DEP_SANTE	TX_PIBHBT	CO2
TX_MINF	78.59%	20.08%	0.01%	1.31%
DEP_SANTE	0.51%	92.89%	1.06%	5.54%
TX_PIBHBT	0.12%	6.32%	92%	1.56%
CO2	0.24%	0.78%	5%	93.97%
	With as ex	ogenous variable STAB	_POL	
	TX_MINF	DEP_SANTE	TX_PIBHBT	CO2
TX_MINF	79.64%	17.64%	0.03%	2.68%
DEP_SANTE	0.29%	93.40	0.37%	5.92%
ТХ_РІВНВТ	0.07%	8.91%	90.46%	0.55%
CO2	0.04%	3.27%	0.43%	96.24%
	With as exoge	nous variable REGUL_C	QUALITY	
	TX_MINF	DEP_SANTE	TX_PIBHBT	CO2
TX_MINF	78.74%	19.01%	0.04%	2.21%
DEP_SANTE	0.74%	92.88%	0.43%	5.94%
ТХ_РІВНВТ	0.25%	9.46%	89.75%	0.53%
CO2	0.13%	6.14%	0.80%	92.91%
	With as ex	ogenous variable EFF_G	OUV	
	TX_MINF	DEP_SANTE	TX_PIBHBT	CO2
TX_MINF	81.18%	16.71%	0.01%	2.09%
DEP_SANTE	0.53%	94.96%	0.49%	4%
ТХ_РІВНВТ	0.12%	9.32%	90.04%	0.51%
CO2	0.06%	5.85%	0.87%	93.20%
	With as exogen	ous variable CONTROL	_CORRUP	
	TX_MINF	DEP_SANTE	TX_PIBHBT	CO2
TX_MINF	82.24%	12.41%	2.82%	2.52%
DEP_SANTE	1.44%	96.86%	0.93%	0.75%
TX_PIBHBT	3.46%	15.11%	76.79	4.63%
CO2	2.54%	34.88%	20.63%	41.96%
	With as ex	ogenous variable RULE_	LAW	
	TX_MINF	DEP_SANTE	TX_PIBHBT	CO2

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DEP_SANTE	0.44%	93.50%	0.42%	5.65%
TX_PIBHBT	0.13%	9.06%	90.31%	0.49%
CO2	0.04%	3.44%	0.62%	95.89%

The results of the variance decomposition bring out some interesting remarks. In fact, when institutional variables are absent in the model, variations in the forecast error of the infant mortality rate are explained by its own innovations, up to 60% against 27% by innovations in public health expenditure. So, although our estimates have shown us a link between these two variables, public health expenditure contributes little to reducing the infant mortality rate in Sub-Saharan Africa. Likewise, 92.91% of public health expenditure forecasting errors are explained by its own innovations against 0.38% by innovations in the infant mortality rate. We note a weak contribution of the infant mortality rate in the explanation of investments in terms of public expenditure on public health in Sub-Saharan Africa. However, when we consider each institutional variable individually, the variations in the forecast error of TX_MINF are explained by its own innovations to the tune of 78% to 82% against 19% to 12% by the innovations of DEP_SANTE. Moreover, regarding the forecast errors of DEP_SANTE, they are explained to the tune of 0.29% to 1.44% by its own innovations against 92% to 94% by innovations in the infant mortality rate. The presence of institutional variables modifies the percentages of innovations therefore the institutional variables play a primordial role in the relationship DEP_SANTE and TX_MINF.

5.4. Result from variance decomposition

We noticed that the graphs of the stability of the model are all identical for the institutional variables, which makes our work robust. However, this finding shows us that the impulse response functions will also be identical so we will only represent two graphs, the one without the institutional variables and another which captures the inclusion of all the institutional variables.



Graph 2 : Impulse analysis Without institutional variables

By considering graph 2, we find that a positive shock on the TX_MINF lowers the DEP_SANTE while a positive shock on the DEP_SANTE first leads to a slight drop in the TX_MINF the first two years before increasing until the end of the period. tenth year. However, this pace will often be modified with regard to graph 3.



Graph 3 : Impulse analysis with institutional variables

In fact, in the presence of institutional variables, a positive shock on the TX_MINF generates a decrease with a linear trend of DEP_SANTE while a positive shock on public expenditure on public health results in an increase with a linear trend of TX_MINF. These different analyzes show us that the institutions have a preponderant role in the relation DEP_SANTE and TX_MINF. Indeed, good institutions can provide information and advice on hygiene, good health practices. So, from what level can we say that an institution is qualified as good? The answer to this question brings us back to the threshold.

5.5. Institutional Variables Threshold Results

Table 7 presents the different thresholds for institutional variables

Table 7 : Result of the thresholds						
Variables	VOICE_ACCOUNT	STAB_POL	REGUL_QUA LITY	EFF_GOUV	CONTROL_C ORRUP	RULE_LAW
Threshold	6.95%	7.30%	7.01%	6.56%	2.33%	3.51%

Our results show that all the institutional variables have thresholds. The estimated thresholds for the track and responsibility (VOICE_ACCOUNT) and government efficiency (EFF_GOUV) variables are respectively 6.95% and 6.56%. For the variables political stability (STAB_POL) and quality of regulation (REGUL_QUALITY), the thresholds are respectively 7.30% and 7.01%. Regarding the corruption control variables (CONTROL_CORRUP) and rule and laws (RULE_LAW), the thresholds are respectively 2.33% and 3.51%. From these threshold levels, we can expect the advantages of DEP_SANTE over TX_MINF. This suggests that institutional quality can be improved governance and impose discipline on macroeconomic policies. Our work joins those of Dhrifi (2020).

6. Conclusion

The main aim of this study was to assess the effect of public health expenditure (% GDP) on the infant mortality rate, considering the role that institutional quality can play. We use two approaches which are

the autoregressive vector panel model with exogenous variables (PVAR (X)) and the Panel Smooth threshold regression (PSTR) model on annual data covering the period 2002-2016 and covering 37 African countries. Subsahdarian. At the end of our investigations, our results through the PVAR (X) reveal that institutions play an important role in the link between public health expenditure (% GDP) and the infant mortality rate because, in the absence of institutional variables, public health expenditure has a negative and significant effect on the infant mortality rate, whereas, in the presence of the various institutional variables, this effect is always negative but is no longer significant. Our results show that the presence of institutions reduces the weight of public health expenditure in explaining the infant mortality rate by half. In addition, our results show through the PSTR that there is a certain level of institutional qualities that these countries must achieve for public health expenditure (% GDP) to positively affect infant mortality rates. These threshold levels oscillate for all institutional variables around 7%. However, the results of the estimates with the PVAR (X) model without the institutional variables reveal that the infant mortality rate has a negative and significant effect on public health expenditure (% GDP). This result suggests a possible causal relationship for previous studies. Similarly, since sub-Saharan Africa is full of several regional agreements and different languages, a possible extension of this study can be made according to regional agreements or grouping according to languages in order to better compare the threshold levels and to assess the effect of institutions on the relationship between public health expenditure (% GDP) and the infant mortality rate.

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