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HIV/AIDS-GDP Nexus? Evidence from panel-data for African countries

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Abstract

To test potential bilateral causalities relation between HIV-AIDS mortality and GDP, we propose a simple Granger noncausality test for heterogeneous panel data models. 44 African countries are selected for annual pooled data from 1990 to 2009. Results are presented for the heterogeneous noncausality hypothesis (HENC), which tests, for each cross-section unit, the nullity of all the coefficients of the lagged explanatory variable. Bilateral causality relation is observed for 5 countries out of 44 (11% of the countries in our data set). We have 18 countries of unidirectional causality, which 14 are from HIV mortality rate to GDP (43% from total), and 4 are from GDP to HIV mortality rate (9% from total). These results alert for the risk of epidemic trap, initiated first by the deleterious effect of HIV-Aids on countries income.

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1. Introduction

Since Mushkin (1962) and Grossman (1972), health is regarded as an important determinant of human capital and hence a factor of productivity. A basic element of Grossman's demand-for-health model is that health provides utility not only directly but also indirectly, since it is a key input into many production processes. Drawing on the theory above and cross-sectional international data, Knowles & Owen (1995) show a strong and robust relationship between health capital and per capita income.

Econometric analysis of economic growth is fraught with methodological difficulties that may cast doubts on the validity of the results. These are related to data and model specification. Two well-known general problems concerning model specification are the possibilities of omitted variable bias and endogeneity. The omitted variable bias arises if these absent variables are correlated with the variables included in the model, e.g., health and growth. As an example: omitted variables such as political and social factors may be correlated with both health and growth which means that it is important to control for such factors in the estimation in order to isolate the effect of health. The endogeneity bias may arise since investments in health itself can be a function of other variables, including economic growth, i.e., in the present paper, our attention is the effect of HIV/AIDS on growth but causality might run the other direction (reversed causality): many studies have shown that economic development is a key determinant of health outcomes and especially of HIV-AIDS exposure (see for ex. Volberding P et al. 2007).

On the theoretical side, there exists a large literature dealing with "poverty trap due to epidemics". A poverty trap is defined as any self-reinforcing mechanism that causes poverty to persist (Azariadis and Stachurski 2005 or Sala-i-Martin 2005, for the specific context of epidemics). As to the special case of the HIV-Aids crisis, research papers explain this persistence through the channel of human capital (the 'quality' of the human resource (Bell et al. 2003; Couderc et Ventelou 2005) or through the channel of fertility (the 'quantity' of the human resource, Boucekkine et al. 2009; Kalemli-Ozcan 2002). In all cases, the relationship between Aids rates and the GDP is plausibly bi-directional. This has led the empirical literature to test the impact of HIV-Aids on economic growth with a special effort to control the endogeneity of HIV-Aids rates (Mac Donald and Roberts 2006; Bloom and Mahal 1997). Generally the econometric modelling proceeds asymmetrically: it starts from a standard growth equation, adds HIV-Aids rates in it, and uses "instruments" to circumvent possible retroaction. They do not necessarily uses the time dimension in data (if any), nor panel data methods. In the present work, we adopted a more agnostic view, with no absolutely no hierarchy between the two causalities examined, and using exclusively panel data methods. We also introduce the most recent updating of data.

If causality is simultaneous bilateral, then a misspecification problem occurs. Therefore, it is critical to determine the direction of the causality relationship in advance. The best way to do this is to apply the Granger-causality test, or rather Granger noncausality test. This test is the most effective and practical way to test the null hypothesis of non-causality between two variables observed in time (Chamberlain 1982; Florens and

Mouchart 1982). Precisely we propose here a Granger noncausality test for heterogeneous panel data models. This panel data test allows us to take into account both dimensions of the heterogeneity in this context: the heterogeneity of the causal relationships AND the heterogeneity of the data generating-process (Hurlin 2004a). This adaptation of the simple Granger test allows detecting possible variations in the way the bidirectional causality really occurs across the different countries.

2. Data

The data of GDP per capita (US current) are derived from World Bank's World Development Indicators, 2011, and the Prevalence of HIV among adults aged 15 to 49 (%) are chosen from World Health Organization[1]. Large number of previous studies used HIV observation of WDI (available in World Bank Data), but our study is unique to use WHO data set in Prevalence of HIV among adults aged 15 to 49 (%) index. 44 countries are chosen in total for annual pooled data from 1990 to 2009 for the reason of availability in Africa. For the list of countries included in our data, see Table II.

3. Methods

Because of its suitability to our data sets, in which we have a short panel in time with a large number of cross-section units, we apply the approach proposed by Hurlin and Venet (2001), Hurlin (2004a, 2004b), used for example by Hoffman et al. (2005); Hansen and Rand (2004) or Erdil and Yetkiner (2009), treating the autoregressive coefficients and regression coefficient slopes as constants in time, but different across countries. In short panels, the fixed effects (FE) estimator of the coefficients of lagged endogenous variables is biased and inconsistent (Nickell 1981). Also the Maximum Likelihood estimators for the dynamic FE models remain biased with the introduction of exogenous variables when cross-section units is small (Hurlin and Venet 2001). Kiviet (1995) suggests an analytical expression for this and Judson and Owen (1999) provide Monte Carlo evidence showing that the FE estimator's bias decreases with cross-section observations. Thus, for our observation, we have decided to use the FE estimator since the bias may not be large.

Following Hurlin and Venet (2001)[2], we consider two covariance stationary variables, denoted by *x* and *y*, observed on *T* periods and on *N* cross-section units. In the context of Granger causality procedure, for each cross-section unit *i* from [1, N], the variable *x* is causing *y* if we are much more able to predict *y* using all available information on *y* and *x*, than if only the historical information on *y* had been used. Thus, we use a time-stationary VAR representation, used for a panel data set. For each country *i* we estimate the following model:

[1]More information on data are available at http://devdata.worldbank.org/wdi2011.htm and http://apps.who.int/gho/indicatorregistry.htm

[2]In order to explain FE method we reference some parts of the paper of Erdil and Yetkiner (2010) can be found in the special issue of *Applied Economics*.

$$y_{i,t} = \sum_{k=1}^{p} \beta_k y_{i,t-k} + \sum_{k=0}^{p} \theta_k x_{i,t-k} + u_{i,t}$$ [3]

In panel analysis, one should deal with the potential heterogeneity problem between cross-section units. The first source of heterogeneity is caused by permanent cross sectional disparities. A pooled estimation without the heterogeneous intercepts may lead to a bias of the slope estimates and can result in a fallacious inference in causality tests (Hurlin, 2004a). The second source of heterogeneity is caused by heterogeneous regression coefficients. In sum, there exist two different types of tests[4] which have to be carried out in the panel data set. Homogenous and instantaneous noncausality hypothesis (HINC) is the first one which tests whether the coefficients of the independent variable are simultaneously null for all cross-section units and all k lags.

If the HINC hypothesis is rejected, there are two possibilities. The first one is the homogenous causality hypothesis (HC) and takes place if all the independent coefficients are identical (and are non-null) over all cross-section units for all lag k. The second is heterogeneous noncausality (HENC), i.e. where some of the independent coefficients are different. If the HC hypothesis is also rejected, this means that the process is non-homogenous, and then, that heterogeneous causality relationships can be considered (Hurlin, 2004a)[5]. The last step is to test the heterogeneous noncausality hypothesis (HENC), which assesses, for each cross-section unit, the nullity of all the coefficients of the lagged explanatory variable.

4. Results

Because of short time period data we assume a maximum lag length equal to three[6]. We find no significant coefficients in including 4th lags (AR(4)) and 5th lags (AR(5)) for any coefficient in a causality test compared to an AR(1, 2, or 3) model. Table I shows values of Wald statistics for testing two types of homogenous causality hypothesis, namely HINC and HC. Results allow us to reject both of the null hypotheses at 1% level of significance which means that there is heterogeneous causal relationship between GDP and HIV. Next step is to find whether the causality is homogenous over countries or sourced from causality relations for individual countries (heterogeneous). The results confirm existence of heterogeneous causal relationships as a result of testing HC hypothesis.

Table I. Test results for homogenous causality hypotheses

44 African counties	Test	HIV→ GDP	GDP→ HIV
	HINC	1.59**	1.30*

[3] u is normally distributed with $u_{i,t}=\alpha_i+\varepsilon_{i,t}$, p is the number of lags, and $\varepsilon_{i,t}$ are *i.i.d.* $(0, \sigma 2)$.
[4] For more discussion of these two tests, see Hurlin and Venet (2001).
[5] It may still be the case that the homogeneous causality hypothesis holds for a subgroup of cross-section units -as may be tested.
[6] In a no-causality test we have to try maximum allowed lags of variables, but considering length of data and the resulting limitation in the degree of freedom of tests. On the empirical work in health economics at least 3 lags is assumed (see Devlin and Hansen 2001).

	HC	1.62**	5.99E+8**

Notes: ** and ** represent respectively rejection null hypothesis at 1% and 5% level of significance.

Table II. Test results for heterogeneous causality hypotheses

Country	From HIV to GDP		From GDP to HIV		Direction of causality
	F-statistics	Size of effect	F-statistics	Size of effect	
Algeria	0.00	–	0.00	–	No
Angola	3.37***	-1709.26	1.48	–	HIV→y
Benin	0.36	–	0.25	–	No
Botswana	2.89***	1992.63	0.67	–	HIV→y
Burkina Faso	1.77*	-47.47	0.69	–	HIV→y
Burundi	0.40	–	0.46	–	No
Cameroon	4.13***	-319.08	2.42***	4.20E-05	Bilateral
Central African Republic	4.79***	-24.71	0.71	–	HIV→y
Chad	2.31**	-32.36	2.16***	-3.90E-04	Bilateral
Comoros	0.00	–	0.00	–	No
Côte d'Ivoire	1.71*	-104.29	0.91	–	HIV→y
Congo	1.38	–	1.55	–	No
Djibouti	2.92***	19.39	0.87	–	HIV→y
Egypt	0.00	–	0.00	–	No
Equatorial Guinea	6.34***	16041.42	1.32	–	HIV→y
Gabon	1.44	–	0.82	–	No
Gambia	1.28	–	1.94**	-4.56E-04	y→HIV
Ghana	0.21	–	0.29	–	No
Guinea	0.94	–	2.28***	1.04E-03	y→HIV
Guinea-Bissau	0.18	–	0.02	–	No
Kenya	1.85*	-90.23	3.67***	-3.37E-04	Bilateral
Lesotho	1.11	–	0.54	–	No
Liberia	1.48	–	0.56	–	No
Madagascar	0.00	–	0.00	–	No
Malawi	1.02	–	0.67	–	No
Mali	3.61***	-158.74	3.23***	-3.49E-04	Bilateral
Mauritania	0.72	–	1.20	–	No
Mauritius	4.27***	3243.45	1.47	–	HIV→y
Morocco	0.00	–	0.00	–	No
Mozambique	3.41***	27.74	5.82***	2.54E-03	Bilateral
Namibia	5.57***	140.36	0.95	–	HIV→y
Niger	0.81	–	0.57	–	No
Nigeria	0.55	–	0.49	–	No
Rwanda	0.84	–	2.27***	8.51E-04	y→HIV
Senegal	3.84***	93.51	0.83	–	HIV→y
Sierra Leone	2.83***	42.21	0.95	–	HIV→y
South Africa	3.13***	66.31	0.66	–	HIV→y
Sudan	2.06**	685.27	1.38	–	HIV→y
Swaziland	1.63	–	1.99	–	No
Togo	0.89	–	2.50***	3.41E-04	y→HIV
Tunisia	0.00	–	0.00	–	No
Uganda	1.19	–	0.89	–	No
Zambia	0.80	–	0.50	–	No
Zimbabwe	4.47***	27.47	0.60	–	HIV→y

Notes: Hurlin (2004a) critical values for Wald statistics for testing causality in micro panels is used to find the valid coefficients. ***, **, and * represent respectively 1%, 5% and 10% significance levels. Cross-section weight is used for making our observation more balanced.

Table I gives the general result for all countries pooled: bidirectional causality would be globally accepted within the HINC assumption. However, the final step is to discover the individual countries' contribution to the existence of causality. According to Table II,

bilateral causality relation is observed, one by one, for 5 countries out of 44, meaning that for around 11% of the countries in our dataset, bidirectional causality both from GDP to HIV-Aids rate and *vice versa* is relevant (Cameroun, Chad, Kenya, Mali, Mozambique). We have 18 countries showing unidirectional causality, which 14 are from HIV mortality rate to GDP (32% from total:Angola, Botswana, Burkina Faso, Central African Republic, Côte d'Ivoire, Djibouti, Equatorial Guinea, Mauritius, Namibia, Senegal, Sierra Leone, South Africa, Sudan and Zimbabwe). Also, 4 are from GDP to HIV rate only (9% from total), included Gambia, Guinea, Rwanda and Togo. No causality relation is observed in 21 directions (48% from total). An important thing: over 90% of significant coefficients in cross-section units are negative, which is close to economic theory which defines a negative relationship between HIV/AIDS and income.

5. Conclusion

This study aims to supply more substantial evidence on the endogeneity of epidemiological indicators and GDP by employing a comprehensive dataset and advanced econometric techniques. We test both the homogenous and instantaneous no-causality (HINC) and the homogenous causality (HC) hypotheses in the whole sample. The 44 models under heterogeneous non-causality hypothesis (HENC) are also estimated, since the FE method allows separate tests. We find that uni-directional Granger-causality is the leading type of causality for our sample of African countries, while it is not homogenous.

Although the analysis conducted in this paper has shed light on the interest of a one-by-one examination of the bidirectional causality between HIV/AIDS and the GDP, some practical limitations of our study are worth mentioning. First, FE models are designed to study time-variant characteristics and cannot estimate the impacts of time-invariant difference between countries, like political institutions or religion (time-invariant characteristics of the units are perfectly collinear with the entity dummies). Second, we have had, in actual fact, a weak window of opportunity to include lags (max=3), because of the short time period of observation and the cost of a decreasing degree of freedom. A further prospect for this type of study is to repeat the tests proposed here with longer time series. This will be possible whenever supranational institutions produce consistent time series data on national accounts.

In view of these provisional statistical results about bilateral causality, the occurrence of an “epidemic poverty trap” turns out to be plausible for certain African countries. Following our results, five countries indeed experiment an “epidemic poverty trap”, in which the infectious disease generates poverty, and, poverty produces –or rather increases- critical exposure to infectious diseases and their consequences (Chakraborty et al. 2010). Our contribution to the literature is also to show the leading evidence, at the present time, of a unidirectional Granger-causality running from HIV/AIDS to GDP levels (true for the larger set of African countries). This result updates the result of Bloom and Mahal (with data collected or estimated before 1996) and shows the risk of deleterious effect of the HIV crisis on GDP per capita. International aid and/or national public action should be designed in view of this risk, with priorities given to health

interventions. Healthcare investments may interrupt the downward spiral, first initiated by the disease crisis, and possibly transformed into a trapping effect.

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