## TESTING THE NEOCLASSICAL GROWTH MODEL: A CAUSALITY APPROACH

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# ABSTRACT

In this paper we propose a method to discriminate among exogenous and endogenous growth models analyzing the causality relationship from accumulation rates in human and physical capital to growth and income per capita. We claim that the convergence property of the exogenous growth model can be interpreted as an error correction mechanism which implies the existence of long run causality from accumulation rates to income. In a sample of OECD countries, our results overwhelmingly reject the null hypothesis of causality in a variety of model specifications. These results provide strong and robust evidence against the adjustment mechanism built in the constant returns growth model, and indicate that the endogenous growth framework would be more appropriate to understand the long run experience of OECD countries during the sample period.

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## I. Introduction.

The empirical growth literature has recently flourished mainly around the convergence regression. As it stands, this approach relies on the long run causality between savings and income per capita levels. In the Solow-Swan model, augmented with human capital, the steady state income level is determined by the rate of both human and physical capital accumulation, as well as by the rate of growth of efficient labour plus some technological parameters. Indeed, the expected close association between these variables is found in the data and has been taken as a consistent empirical fact (Levine and Renelt (1992)). This link is also dictated by common sense. It is difficult to imagine a growth mechanism that does not work through the increase of capital in one way or another.

Whereas the association between investment and growth is a property of most models of economic growth, causality from the former to the later is mainly an implication of the exogenous growth model. In this model there is a long run relationship among accumulation rates and income per capita such that the economy returns to that path if it happens to be away from it. In this case, accumulation rates should improve the forecast of future growth rates based on a simple autorregressive specification. In this paper we propose a method to discriminate among exogenous and endogenous growth models along these lines. We claim that the convergence property can be interpreted as an error correction mechanism which implies the existence of long run causality from investment and human capital accumulation to income. The lack of

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causality in a fully specified model should be taken as strong evidence against the dynamics of income implied by the exogenous growth model.

The evidence of a positive association among growth and investment has been taken as consistent with the constant returns technology. However, correlation does not imply causality even in a statistical sense. In this paper we investigate the causality among physical and human capital and growth within the OECD from 1960 to 1990. Our results overwhelmingly reject the null hypothesis of causality from the accumulation rates to income and growth in a variety of model specifications. Controlling for individual effects, this is so both in levels and in first differences and irrespective of the inclusion of other regressors in the equation. In a few cases there significant causality but with a counterintuitive negative sign. In is contrast, there is some evidence of causality from income to human and physical capital investment. Contrary to what some authors have suggested<sup>1</sup>, the failure of investment to cause income does not undermine the crucial role of capital accumulation in the growth process. As we read them, these results provide strong and robust evidence against the adjustment mechanism built in the constant returns growth model, and suggest that the endogenous growth framework would be more appropriate to understand the long run experience of OECD countries during the sample period.

See Blömstrom, Lipsey and Zejan (1993) for instance.

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The rest of the paper is organized as follows. In section II, we discuss the relevant econometric issues involved in testing causality when regressors are non stationary and might be cointegrated. Given the structure of our data set, we also discuss alternative methods to deal with the, most likely, presence of country specific effects. In section III, we analyze the relationship among growth and investment using alternative estimation methods. In section IV we apply some of this methods to test for causality between growth and human capital accumulation. Section VI concludes with some final remarks.

### II. Exogenous versus endogenous growth models: some econometric issues.

### 2.1. Exogenous growth and causality.

The good performance of accumulation rates in growth equations, picking up the potential income level or steady state of each economy has been presented as evidence in favour of both the exogenous growth and the endogenous growth models (Romer (1994)). There is however a way in which this correlation can be used to discriminate among these two approaches. The argument can be stated in—very simple terms. Built in the constant returns growth model there is an adjustment mechanism. The level of accumulation rates drive the long run level of attainable income per capita, so that when this is not on its long run path it tends to it at a positive speed. To predict future income levels we need to know both where the economy has been in the past as well as where it is heading towards in the future. According

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to this, past income levels should not be enough to predict future ones, and the observed physical and human capital accumulation rates (to the extent that they proxy their long run value) should improve this forecast. In the endogenous growth models, in which the level of income is not tied down by the accumulation rates, this does not need to be the case. In fact, growth rates are driven by the structural parameters of each economy (the same that drive the accumulation rates) and the autorregressive forecasts of future income levels cannot be improved upon.

The convergence regression has been derived by Barro and Sala i Martin (1992) as a log linear approximation of the adjustment process to the steady state. It can be generalized to allow for adjustment costs and other lags as:

$$\Delta \mathbf{y}_{t} = \pi \left[ \mathbf{y}_{t-1} - \beta \mathbf{X}_{t-1}^{*} \right] + \sum_{i} a_{i} \Delta \mathbf{y}_{t-i} + \sum_{i} \mathbf{B}_{i} \Delta \mathbf{X}_{t-i}^{*} + \mathbf{u}_{t}$$
(1)

where y represents income per capita and  $X^*$  is a vector of accumulation rates (investment, schooling and population growth) at their steady state levels. The main hypothesis to test in (1) is the significance of  $\pi$  which would discriminate among the exogenous and the endogenous growth models. Actually, a test of this kind is already provided in the standard convergence equation in the coefficient of lagged income. However, the convergence hypothesis can also be viewed as the adjustment process around a cointegration relationship, and the convergence equation as a non complete specified error correction model<sup>2</sup>. The distinctive feature of the exogenous growth model is not then that capital accumulation affects growth rates, but that current accumulation rates should improve the forecast of future income levels based on the past history of this variable, i.e. that there is long run causality from accumulation rates to income.

In the rest of the paper we focus on the causality structure among income and capital accumulation rates. It should be noticed that the difference among exogenous and endogenous growth models relies upon the significance of  $\pi$  rather than in that of **B**<sub>i</sub>. To discriminate among these two hypothesis we could estimate the cointegration relationship among y and X<sup>\*</sup>. However, tests of cointegration have low power, and we know very little about their behaviour in pooled samples as the one we deal with here. Instead we try a more general approach, testing the causality from X<sup>\*</sup> to both income per capita and its rate of growth in a more general model, mixing long run and short run effects. If causality is rejected in both cases we could safely conclude that variables in X<sup>\*</sup> do not help to predict future income levels so that there is not a long run equilibrium relationship as the one implied by the Solow-Swan model. Although what the model predicts is that all three accumulation rates determine the steady state of the level of income, we shall consider each of them in turn<sup>3</sup>. Nevertheless in all cases we also

Similarly, Quah (1994) has suggested the interpretation of the absolute convergence regression as a test of unit root in the process generating income per capita levels.
 With the exception of the rate of growth of population which we leave

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With the exception of the rate of growth of population which we leave out of the analysis at this stage.

report causality tests of each variable in models including the others components of the  $\mathbf{X}^*$  vector.

A number of studies have recently tackled the issue of causality among growth and some other variables using multi-country data<sup>4</sup>. They all rely in one way or another in the notion of Granger causality, which tells us that we can improve the predictions of one variable y taking into account past values of another variable x, which presumably *causes* y. Assuming that both x and y are stationary, a more precise definition can be made in terms of the mean square error (*MSE*): variable x causes y if the *MSE* of the prediction of y when we consider past values of x is smaller than when we excluded x from our information set. Using the standard notation of a bivariate VAR(p), we represent the process as:

$$\Pi(L) \begin{bmatrix} y_t \\ x_t \end{bmatrix} = \begin{bmatrix} \alpha_0 \\ \alpha_0 \end{bmatrix} + \begin{bmatrix} u_t \\ \varepsilon_1 \end{bmatrix}$$

where  $\Pi(L)$  is a (2×2) matrix of polynomials of lag operators,  $(u_1, \varepsilon_1)$ , a bivariate white noise process and  $(\alpha_0, \alpha_0)$  a vector of constants. Writing (2) in a more extensive way we have:

(2)

<sup>&</sup>lt;sup>4</sup> See Conte and Darrat (1988), LaCivita and Frederiksen (1991), Blömstrom, Lipsey and Zejan (1993) and Carrol and Weil (1993) among others.

$$\begin{bmatrix} 1-\alpha_1(L)-&-\alpha_p(L)&-\beta_1(L)-&-\beta_p(L)\\ -\alpha_1'(L)-&-\alpha_p'(L)&1-\beta_1'(L)+&-\beta_p'(L) \end{bmatrix} \begin{bmatrix} y_1\\ x_1 \end{bmatrix} = \begin{bmatrix} \alpha_0\\ \alpha_0 \end{bmatrix} + \begin{bmatrix} u_1\\ \varepsilon_1 \end{bmatrix}$$

(3)

When  $(\alpha'_{1}(L) + ... + \alpha'_{p}(L)) = 0$  and  $\beta_{i} \neq 0$  at least for some *i*, past values of *y* do not have any influence in the present value of *x* but not vice versa, therefore we have *unidirectional causality* running from *x* to *y*. The Granger causality test consists in regressing  $y_{t}$  on  $y_{t-1}, ..., y_{p}, x_{t-1}, ..., x_{t-p}$  and testing the joint significance of the lagged values of *x* included in the regression. Because this test has to be well specified, we have to obtain white noise residuals in this regression that can be very sensitive to the lag structure, otherwise the test result is misleading.

## 2.2. Causality tests with non stationary variables.

When x and y are stationary, the F-test of the p restrictions converges asymptotically to a  $\chi^2$  with p degrees of freedom. However if the VAR system includes non stationary variables this test has a non standard asymptotic distribution, which depends on the existence of a cointegrating vector among the variables (Sims, Stock and Watson (1990)). As most of growth variables are assumed to be non stationary, in this section we explore two alternative situations in our VAR system which depends on the presence of a cointegrating vector. We also present the procedure proposed by Dolado and Lütkepohl (1994) which leads to a Wald test with standard  $\chi^2$  asymptotic distribution without the specification of the cointegrating vector. Let us consider that x and y are integrated of order one, I(1), with cointegrating vector  $(1,\alpha)$ . In this case, following Sims, Stock and Watson (1990), causality can be tested in the following model:

$$y_{t} = a_{o} + g_{1}(y_{t-1} - \alpha x_{t-1}) + \mu_{1}(1 - L)y_{t-1} + \dots + \mu_{p}(1 - L)y_{t-p} + \pi_{1}(1 - L)x_{t-1} + \dots + \pi_{p}(1 - L)x_{t-p} + d_{1}y_{t-1} + u_{t}$$
(4)

Notice that now, most of the regressors included in (4) are I(0). Hence, we can use the *F*-test for restrictions on the parameters  $g_1$ ,  $\pi_1, \ldots, \pi_p$  which has standard asymptotic distribution. In particular, when we cannot reject  $g_1=0$ , i.e. the error correction mechanism is not significant, there is not long run causality from x to y. If  $\pi_i=0$  for all *i*, there is not short run causality from x to y.

If there is not a cointegrating vector, we can still rewrite the first equation in (3) in terms of I(0) and I(1) regressors:

$$y_{t} = \alpha_{o} + (\alpha_{1} + ... + \alpha_{2})y_{t-1} - (\alpha_{2} + ... + \alpha_{p})\Delta y_{t-1} - ... - \alpha_{p}\Delta y_{t-p} + (\beta_{1} + ... + \beta_{2})x_{t-1} - (\beta_{2} + ... + \beta_{p})\Delta x_{t-1} - ... - \beta_{p}\Delta x_{t-p} + u_{t}$$
(5)

That can be reparameterized defining  $\alpha_i^* = \sum_{i=j+1}^p \alpha_i \ y \ \beta_i^{*} = \sum_{i=j+1}^p \beta_i^{*}$ ; then we have:

$$y_{t} = \alpha_{o} + \Sigma_{i}^{p} \alpha_{i} y_{t-1} + \Sigma_{i}^{p} \beta_{i}^{*} x_{t-1} - \Sigma_{j} \alpha_{j}^{*} \Delta y_{t-j} - \Sigma_{j} \beta_{j}^{**} \Delta x_{t-j} + u_{t}$$
(6)

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Again, we can test for *long run* and *short run causality*. However, while the  $\beta_j^{**}$ 's are coefficients of I(0) regressors and, therefore, the test of the joint significance of  $\Delta x_{t-1}, \dots, \Delta x_{t-p}$  follows asymptotically a  $\chi^2(p)$ , testing the significance of  $x_{t-1}$  implies a restriction on the coefficient of a I(1) regressor so that the *F*-test has a non standard asymptotic distribution.

More recently, Dolado and Lütkepohl (1994) have put forward a procedure to avoid the specification of the cointegrating vector and still have an asymptotically standard distribution for the F-test. Their method consists in a direct estimation of the VAR process by least squares, with the variable in levels, fitting a VAR whose order exceeds the true order of the process (i.e. adding an extra lag if variables are I(1)). Although there is a loss in efficiency since the system is overparameterized, tests based on the estimated coefficients have a standard  $\chi^2$  asymptotic distribution.

# 2.3. Causality tests with panel data

So far we have discussed the properties of causality tests taking only into consideration the time series properties of the data. However, the cross section structure of our multi-country data set must be carefully handled in order to avoid imposing too many restrictions. Pooling data, without taking into account the presence of country specific effects, is a usual procedure in the empirical growth literature that, nonetheless can lead to misleading results. Indeed, there is some evidence that suggests that the correlation between growth, investment and schooling is not that strong, once country

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specific effects are allowed for (Andrés and Bosca (1994) and Cohen (1993)). Some authors have simply proposed to test causality for each individual country since the estimated parameters in the pooled sample are at most consistent estimates of the average across countries (Conte and Darrat (1988), LaCivita and Frederiksen (1991)), and could even be strongly biased (Pesaran and Smith 1993). Since our time dimension is relatively large (over 30 years) in this paper we start by assuming homogeneous slopes across countries and allowing for country specific time invariant effects. When necessary we resort to causality tests for individual countries.

Under these assumptions, the basic model can be written as in (7),

$$y_{it} = \alpha_{o} + \alpha_{i} + \alpha_{1}y_{it-1} + \alpha_{2}y_{it-1} + \dots + \alpha_{p}y_{it-p} + \beta_{1}x_{it-1} + \beta_{2}x_{it-2} + \dots + \beta_{p}x_{it-p} + u_{it}$$
(7)

This equation includes a time invariant individual effect ( $\alpha_i$ ). If all the regressors were stationary, as long as time dimension is relatively large, we could estimate this equation including an individual dummy with the variable in levels. However, the properties of the tests for individual effects when the variables are non stationary are less well known. Quah (1993) has explored the implication of the cross section variation upon the unit root regression. He concludes that the estimated coefficient has an asymptotic distribution which is neither the normal distribution nor the Dickey-Fuller one. In fact, for a given time dimension T, increasing N

drives the distribution of the estimated coefficient towards the normal<sup>5</sup>. This suggests that that cross section variation mitigates the problem of non stationarity along the time series dimension. If both variables in eq.(7) are I(1), tests based on the estimated coefficients presumably do not have a standard  $\chi^2$  asymptotic distribution, although now the problem will be less severe. Following Dolado and Lütkepohl (1994) we can estimate eq. (7) in levels with an individual dummy as well as an additional lag for y and x.

Another way of testing for causality in eq. (7) has been proposed by Holtz-Eakin, Newey and Rosen (1988), although this method is only operative with the standard panel data structure in which only the cross-section dimension is large. Taking first differences, equation (7) becomes<sup>6</sup>:

$$\Delta y_{it} = \alpha_1 \Delta y_{it-1} + \alpha_2 \Delta y_{it-1} + \ldots + \alpha_p \Delta y_{it-p} + \beta_1 \Delta x_{it-1} + \beta_2 \Delta x_{it-2} + \ldots + \beta_p \Delta x_{it-p} + u_{it} - u_{it-1}$$

Holtz-Eakin, Newey and Rosen proposed a two-step estimation of equation (8). First, we need to estimate (8) as a system of T-p-1 cross section equations using instrumental variables to obtain a consistent estimation of the residuals. All p+1 lags are valid instruments and for this reason the set of

(8)

<sup>According to the Monte Carlo results presented by Quah, when N=T=25 the critical value for a probability no greater than 2.5% is -2.60, above the -1.96 for the normal distribution but below the Dickey-Fuller case.
Notice that, as Nickell (1981) has shown, the usual transformation of variables in equation (7), as deviation with respect to individuals time means, produces inconsistent estimates due to the presence of lagged endogenous variables.</sup> 

instruments is not the same for each equation. With the estimated residuals and the matrix of instruments we can compute a covariance matrix of the errors terms which is used in the second step to estimate all the parameters in (8) by simultaneous GLS methods<sup>7</sup>. Despite the efficiency gains, this method becomes rather cumbersome when T becomes relatively large. An alternative is to reduce the number of instruments, but then the efficiency gains are more limited.

As the authors point out, this method yields an straightforward test of the constraints across equation in  $\alpha$ 's and β's coefficients. This is particularly useful if one wishes to test the maintained assumption of parameter stability both in the constant term or in the slopes. However, the investigation on the stability of (7) and (8) is beyond the scope of this paper. We shall rather exploit the panel structure of our data set estimating equation (8) by instrumental variables, using a constant set of instruments (Anderson and Hsiao (1982)). As an additional advantage, taking first differences of equation (7) renders all I(1) variables stationary.

#### III. Investment and growth.

As we have discussed earlier, it is possible to conduct causality tests in different ways. In this section we discuss the relationship among investment

<sup>&</sup>lt;sup>7</sup> In fact, we could also estimate equation (8) by the generalized method of moments (GMM), as Arellano and Bond (1991) have proposed.

and growth using three alternative specifications<sup>8</sup>. Following Dolado and Lütkepohl (1994) we can estimate equation (7), with and without individual effects, adding additional lags to the variables. The problem with this approach is that we ignore the true order of the VAR process. In this case, an iterative process could be followed, whereby equation (7) is estimated with p lags to test the significance of the p-1 coefficient. If this coefficient is not significant only p-1 lags should be included, testing the significance of the p-2 coefficient. This process goes on until the p-j coefficient is significant in an equation with p-j+1 lags. In practice most of the lags of investment turned out to be non significant in our model, so that we have followed a conservative strategy choosing a long enough laglength as to remove the effects of cyclical fluctuations. Alternatively, we have estimate equation (8) following Anderson and Hsiao's approach. Finally, we also present the results of applying some of these methods to each country individually.

Tests of causality running from investment to per capita GDP are presented in Tables 1 and 2. In Table 1 we present the results of estimating (7) and (8) with different methods and/or model specifications, with the *level* of

<sup>&</sup>lt;sup>8</sup> Most of the data used in this paper have been used in Andrés, Doménech and Molinas (1994), where there is a brief description and an analysis of the variables.

GDP per capita on the left hand side<sup>9</sup>. There is overwhelming evidence against the hypothesis of positive causality. This is so irrespective of the inclusion or not of individual effects (cols. 2 to 5 versus col. 1) and of the presence of other regressors such as the rate of human capital accumulation and the rate of growth of population (as in cols. 3 and 5). Alternative estimation methods (OLS in cols 1 to 3 and GMM in cols. 4 and 5) yield also much the same outcome. In fact, the only specifications in which causality cannot be rejected (cols. 2 and 3) yields a negative sign for the sum of the lagged coefficients of investment, implying a negative long run influence of investment on income per capita, which is somewhat counterintuitive and it is not a prediction of the neoclassical growth model.

In Table 2 the dependent variable is the growth rate of income per capita, as it appears on the left hand side in convergence regressions<sup>10</sup>. The model in col. 1 includes neither individual effects nor additional regressors, and causality is strongly rejected. The same happens in the model in col. 2, which includes country dummies and is estimated in levels by OLS; in this case, the point estimate of the impact of investment on growth is negative. Including additional *steady* state regressors<sup>11</sup>, as in column 3, does not

All specifications include six lags of the RHS and LHS variables.

- <sup>10</sup> We have tried alternative lag lengths without significant differences among them. We report models with six lags. In all cases, the fifth lag was significant, indicating that the corresponding test has a standard distribution.
- <sup>11</sup> By these we mean the lagged income per capita level, the rate of human capital accumulation and the rate of population growth. It should be

change the result either. In columns 4 to 6, the model is estimated in first differences by GMM. Again, causality is strongly rejected regardless of whether other regressors are included (col. 4) or not (col. 6). When we remove non significant additional regressors, as in col. 5, causality cannot be rejected, although the sign of the effect turns out to be negative and quite big.

The picture that comes out of these results is disappointing for the exogenous growth interpretation of the link among investment and income per capita. Neither in levels nor in growth rates the GDP in the OECD seems to be caused by the rate of investment<sup>12</sup>. Other authors report similar results although with different econometric methods and a rather different economic interpretation. Blömstrom, Lipsey and Zejan (1993) find no evidence of causality from investment to growth in cross section and pooling regressions on five years averages, even after controlling for specific country effects<sup>13</sup>. In a related work Carroll and Weil (1993) find that savings do not

noticed that once the lagged income per capita is included, the test of causality can be understood as running from the level of investment to the *level* of income again, and hence the results should be consistent with those in table 1.

<sup>&</sup>lt;sup>12</sup> Actually, we have also tried longer lag structures. Only when lag fifteen and beyond are included, there appears to be causality running from investment to growth. However, in this case too the sum of all lags included is negative when individual effects are included.

<sup>&</sup>lt;sup>13</sup> They do so dividing the variables by their average over the sample period.

cause growth for the OECD sample or if they do the coefficient of growth on lagged savings turns out to be negative. The interpretation in both cases is against the so called 'mechanical link' (Carrol and Weil, 1993), running from investment to growth.

The investment rate appears as the single most relevant determinant of growth rates in the recent growth literature. In their comprehensive study Levine and Renelt (1992) find a robust correlation among these two variables irrespective of the conditioning information set. How can we account for this correlation in the light of the lack of causality?. One possibility is that the positive sign in regression models simply reflects a simultaneity bias. Accelerator models of investment can give rise to a positive causation running from income to investment. Similarly, as Carroll and Weil (1993) claim, the life cycle theory of consumption would predict a negative impact of current income on the future savings rate (and hence investment) as a result of forward looking consumers feeling wealthier. These authors find in fact the opposite happening; current GDP causes future savings rates with a positive sign. Blömstrom, Lipsey and Zejan (1993) come up with similar results; the correlation among current growth and future investment rates is positive and stronger than the correlation with current and past investment.

In table 3 we have tested this direction of causality, from growth to investment, applying the same econometric methods as in tables 1 and 2. Regressions in columns 1 to 3 are estimated by OLS in levels, whereas those in columns 4 and 5 are estimated by GMM in first differences; all equations

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include six lags. Models in columns 1, 2 and 4 do not include any additional regressor, whereas in columns 3 and 5 the causality regressions are augmented with the same steady state proxies we discussed earlier. The corresponding  $\chi^2$  statistics show that the null of no causality can be safely rejected in most specifications at the 5 per cent level of significance. When the model is estimated by GMM and excluding other regressors (as in col. 4) the null can only be rejected at the 10 per cent level. In all cases, though the sign of the correlation among current growth and future investment/GDP ratio is positive and quite strong (not far from a unit long run elasticity in four of five cases), which is consistent with the results by Carrol and Weil, and with the importance of anticipated demand growth in investment decisions.

This feedback from growth to investment does not invalidate conventional growth equations. It only means that the positive correlation can be contaminated by a simultaneity bias. However, when the investment rate is instrumented (as in Andrés, Doménech and Molinas (1994) amongst others) it stills retains its strong positive correlation with growth. This correlation can also be explained by the absence of individual effects in the regressions. In general, the sum of coefficients of lagged investment in growth equations in table 2 changes sign once individual effects are removed. In column 1, when country specific dummies are not included this sign is positive (though non significant) and becomes negative in the specifications in which these effects are either included or removed through differencing (the only exception being column 4). Blömstrom, Lipsey and

Zejan get a similar result when the variables are divided by their sample averages as to remove country specific effects.

this possibility we have tried a number of cross section To asses regressions to explain both the individual effects as well as their impact in standard cross section convergence regressions. The individual effects of a causality regression from investment to growth are stuck together in the variable INDIV. In table 4, columns 1 and 2, we try to explain these individual effects using the average investment/GDP ratio for each country during the sample period, the average growth rate and the initial income per capita. The correlation among the individual effects and average investment is quite strong, irrespective of whether we include other regressors or not, with a t statistic near 2.50. Individual effects are then strongly linked not only to the initial conditions of each economy but also to its average growth and investment rates. It might be guessed that it is precisely the absence of this effects in standard convergence regressions what could be behind the high significance of investment rates, and indeed of some other regressors such as the initial per capita income, in these models<sup>14</sup>. The models in columns 3 and 4 suggest that this might well be the case. We first present (col. 3) a standard cross section convergence model including the

<sup>&</sup>lt;sup>14</sup> We have also checked our procedure in two samples of pooled data consisting in 5 and 10 years averages of the variables of interest. In all specifications persistence is significant. Lagged investment seems to cause growth when we estimate eq. (7), that is, when we do not control for individual effects. However, the investment rate is not significant in specifications where individual effects are removed as in eq. (8).

average investment rate as a proxy for the steady state savings rate. The fit is reasonably good and we get the usual parameter signs and significance levels. The average growth rate is positively correlated with investment and negatively so with the initial income level; the implicit convergence rate is 1.1 per cent and strongly significant. When we enlarge the conditioning set to include the DINVID variable (col. 4) things look quite different. The goodness of fit improves dramatically (to achieve an  $R^2$  of 0.98)<sup>15</sup>, and the convergence rate gets closer to the widely accepted 2 per cent value. Most important though is the dramatic change in the role of investment in this equation; once the possibility of different intercepts is allowed for, investment is no longer significant. Furthermore, if any its influence upon growth seems to be negative<sup>16</sup>, contrary to what is commonly accepted.

This result casts some doubts as far as the interpretation of convergence regressions is concerned, and can be given an explanation alongside Cohen's (1993) interpretation of the role of human capital accumulation. Country specificities explain why some countries save (and invest) more than others as well as why some countries grow faster than others. If these country specific effects are omitted, there is a biased correlation among the variables included in the regression. Faster growing countries are also the

<sup>&</sup>lt;sup>15</sup> Which is well above the R<sup>2</sup>'s usually obtained in this kind of regressions, even including other steady state proxies such as population growth and human capital accumulation (see Mankiw, Romer and Weil (1992) or Andrés, Doménech and Molinas (1994) amongst others).

<sup>&</sup>lt;sup>16</sup> With a t statistic of -1.75.

ones with higher investment. However, we should not jump too quickly into the seemingly natural policy conclusion. These economies do not grow faster because they invest a large share of their current output. Rather, these economies grow faster and invest more than the others because some idiosyncratic features encourage to do so. These unknown factors (market organization, public sector efficiency, financial development, inflation control, etc.) are the ones we should look before we can put forward any policy recommendations.

We shall turn to this issue in the next section, where we shall analyze the role of human capital accumulation. However, before doing so is worth pursuing the issue of country specificities in more detail. Some authors have argued that pooling time series regressions might impose too many restrictions on the parameter set, that can be avoided when the time series dimension is sufficiently large (Pesaran and Smith (1993)). Unless a rather restrictive set of assumptions is satisfied, the estimated coefficients may be severely biased and bear little or non resemblance with the true average parameters we are interested in. When this assumptions are not met, pooling regressions are useless, and we must resort to individual country models. As far as causality among growth variables is concerned, different countries may undergo very different experiences too. This has led some authors to suggest that individual country analysis can be more informative<sup>17</sup>.

<sup>&</sup>lt;sup>17</sup> See Conte and Darrat (1988) for the causality among public sector size and growth, and LaCivita and Frederiksen (1991) who focus in the relationship among growth and defense spending, for instance.

In table 5 we present causality regressions for every individual country in the OECD, estimated using similar methods as those in tables 1 to 3. Instead of the full set of parameter estimates and their statistics, we just present results focusing on the existence of a significant statistical causality running from the investment rate towards growth, as well as on its sign. The overall picture we get from table 5 is one of a huge disparity among individual countries that might well account for the aggregate results we have obtained so far. Following Dolado and Lütkepohl (1994), the regression in column 1 has been carried out in levels, and does not include any additional regressor other than lags of investment and growth. Fourteen out of the twenty four countries show a significant improvement of growth rates forecasts following the inclusion of investment rates; among these, just eight display a positive long run effect. Besides, no clear pattern emerges from them in relation with the wealth ranking within the OECD. Rich countries (such as Japan, Germany and Switzerland) appear in the causality group alongside with poor ones (Spain, Portugal or Turkey). The correlation between the relative income and the sign of the long run elasticity is not clear-cut either; negative causality characterizes very different countries (Canada, Denmark, Ireland and Spain for instance) as much as positive causation does (Australia, Germany and Portugal).

Including accumulation rates as additional regressors changes the picture somewhat but does not make it less puzzling. Now the null of non causality is overwhelmingly rejected in both models. In the model in levels (col. 2)

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non causality is rejected in 18 out of the 24 cases. The sign of causality is nevertheless unclear; it is negative in 14 countries and positive in the remaining ones. No clear pattern in relation with the income ranking arises either. We find a negative correlation between growth and lagged investment, both among high income (such as US, Canada or Sweden) and low income (Ireland, Portugal, for instance) countries. Similarly, positive correlations can be found all across the board, without any clear link with either growth rates or income levels. This ambiguity carries over the model in first differences (col. 3), in which causality is the norm, although no sign predominates among the OECD countries.

The overall picture we get from tables 1 to 5 is disappointing for the neoclassical exogenous growth models in several respects. First, they show that the link among investment and growth is, at the very least, far more complicated than is usually assumed. The 'mechanical link' running from investment to growth is not found in the data. The forecast of future growth rates cannot be improved upon by taking in account current investment rates. Second, some of the correlations found in cross section or pooling models of multi-country data sets seem non robust once country specific effects are taken into account. The cross section correlation among growth and average investment rates on the OECD vanishes (and even changes sign) once individual effects are included in the regression. Furthermore, when causality regressions are run for every individual country the diversity of results calls into question the validity of pooling models, in which slopes are assumed to be homogeneous across countries. Finally and most important,

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these results provide strong evidence against the error correction model implied by the dynamics of the canonical exogenous growth models.

#### IV. Human capital and growth.

The contribution of human capital to growth is one of the most promising and consistent findings of the new growth literature, both theoretical and empirical. Lucas (1988) has shown how the time devoted to enhance human capital formation can be one of the 'engines of growth' and can hold some of the clues to explain the huge differences in wealth among world economies. In the context of exogenous growth models, Mankiw, Romer and Weil (1992) first showed that augmenting the production function with human capital as an accumulable factor improved the fit of convergence regressions and yielded very sensible parameter values. Since then, several authors have found one proxy or another of human capital to be positively correlated with growth rates<sup>18</sup>. The role of human capital in the exogenous growth model is similar to the one expected for physical capital, i.e. the higher the proportion devoted to investment in skills the faster the economy will grow in the future. More recently Cohen (1993) has disputed the robustness of this empirical link and found that differences in schooling rates might be picking up the effect of omitted structural differences across countries.

<sup>18</sup> For the OECD sample see Andrés, Doménech and Molinas (1994).

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Once these differences are taken into account, the correlation among growth and schooling no longer holds.

In this section we look at the correlation between growth and human capital in more detail. In particular we focus in the causality from several proxies of human capital to growth under alternative econometric specifications, similar to those in the previous section.

In principle, in the neoclassical model we could include the fraction of total output devoted to accumulate human capital in the characterization of the steady state. However, such variable is not available for all OECD countries in different benchmark years covering our sample period. We only have a component of this variable in several years: public spending devoted to education as percentage of GDP. The problem with this measure is that OECD countries present a wide diversity in the finance of their education systems, so this variable is a poor proxy of the variable of interest for our purposes.

As a better proxy for human capital accumulation, Mankiw, Romer and Weil (1992) proposed the use of enrolment rates in secondary education. However, this variable is also not exempt of criticisms, because most of OECD countries have enrolment rates in secondary education near 100% at the end of the period we analyze. Thus, it can be more convenient to use enrolment rates in higher education as a proxy of human capital accumulation. Again, this measure has its own shortcomings too, since not all higher studies have

the same impact in future possibilities of production (Mulligan and Sala i Martín (1993)).

Recently, Barro and Lee (1993) have constructed a data set of human capital variables in which includes all OECD countries with the exception of Luxembourg. From this set we have selected three alternative variables to enrolment rates in secondary and higher education. The three variables correspond to total, secondary and higher years of schooling of total population over age 25. These are all stock measures, and it is usually assumed that economies with higher level in these variables have higher preferences in human capital accumulation. The data of these variables is available at 5 years intervals from 1960 to 1985. Enrolment rates from UNESCO are available at shorter intervals, but to ensure comparability we have used a pooled sample consisting in five years averages for all variables with the exception of human capital and initial income per capita which refer to the initial year in each interval.

The basic results of the test for causality from human capital to growth are displayed in table 6. In all cases we present five different models. These results appear outstandingly robust to alternative specifications as well as to the choice of the proxy for human capital. Positive causality can be rejected in almost all specifications we have tried. Non significant positive correlation among current growth and lagged human capital accumulation dominates the scene. The third model in each case is of particular interest since it recalls very much the format of standard convergence regressions. In all cases, the initial income, the investment rate and population growth appear strongly significant and correctly signed. This is most surprising since it is in this type of equations in which many authors have found a significant role of human capital on growth. We have also carried out causality tests from human capital to the *level* of GDP per capita (table 7). The null of no causality could just be rejected in seven out of the seventy five specifications tried<sup>19</sup>; in all these seven, though, the sign of causality was positive.

These results cast additional doubts on the interpretation of augmented convergence regressions, and they seem inconsistent with the positive correlation found among growth and human capital accumulation in most of them. The possibility of reverse causation is appealing, since investment in education is bound to be fostered by economic prosperity<sup>20</sup>. The main results are summarized in table 7. Unlike the case of physical capital we cannot find evidence of causality running from the rate of growth of income per capita to human capital. When the level of GDP per capita appears in the right hand side of the regression, the results are rather mixed and positive causality cannot be rejected in a small but significant number of cases<sup>21</sup>.

<sup>&</sup>lt;sup>19</sup> These specifications include five models for each of the five human capital indicators. Each model was estimated with three different lag structures.

<sup>&</sup>lt;sup>20</sup> Galor and Zeira (1993) present a model in which growth and human capital investment interact each other.

<sup>&</sup>lt;sup>21</sup> These results are available on request from the authors, but they are not presented here to save space.

The lack of causality from schooling and other human capital proxies to growth might be due to the poor quality of the proxy used. However, to the extent that all-different indices yield so similar results one can be quite confident in the robustness of the lack of causality discussed above. The results in this section are consistent with those by Cohen (1993), and indicate that the 'mechanical link' from investment to growth is not supported by the data in the case of human capital either. The comments we made in the previous section in relation with investment in physical capital also apply here. The correlations found in the context of the exogenous growth model might admit an alternative explanation not inconsistent with the endogenous growth literature. The positive correlation between growth and *current* investment in human capital (when the effects of the increased efficiency in the labour force has not shown up yet) might well be picking up the effect of shocks that increase both growth and the incentive to accumulate capital at the same time.

## V. Conclusions and final remarks.

The adjustment mechanism of income towards its steady state is a distinctive feature of the exogenous growth models with diminishing marginal product of accumulable factors. The steady state accumulation rates cause income in a statistical sense in the long run, since the past history of this variable is not enough to predict its future behaviour. In the endogenous growth models, the level of income is not tied down by the accumulation rates and the autorregressive forecasts of future income levels cannot be improved upon the inclusion of those rates.

In this paper we have reported a consistent and robust lack of causality from investment and other accumulation rates to income per capita and growth. The use of a variety of specifications and estimation techniques leads us to conclude that this causality in a statistical sense is absent both in levels and in rates of change, irrespective of whether other regressors are included in the equation, when individual effects are taken into account. When this causality appears, it presents the unexpected sign. If any causality is stronger from growth and/or income per capita levels to human and physical capital investment. These results are in sharp contrast with the interpretation of the standard correlation among growth and *current* accumulation rates found in the literature. The association among growth and accumulation vanishes once lagged growth rates and country specific time invariant effects are allowed in. Taken as a whole, these results are not consistent with the predictions of the exogenous growth models and hence,

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they suggest that the explanatory power of the endogenous growth literature cannot be dismissed.

The lack of causality from investment to growth found in this paper should not lead us to conclude that investment (both human and capital) are not the engines of growth. Indeed, it is hard to think of any growth mechanism that does not work through the accumulation of these and other factors of production. All what these results tell us, is that the evidence in favour of the adjustment mechanism of the exogenous growth model, built in the convergence equations, is far less convincing than what is usually meant. Our results suggest the absence of long run and short run causality from investment to income per capita. A more elaborate test of this model should exploit the long run implications of the steady state property as a cointegration relationship.

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	Causality	<b>Table</b> from <i>ln(l</i>	1 I/GDP) to	lny	
	1	2	3	4	5
Lags included:	6	6	6	6	6
Wald test over the		•			
exclusion of:	6 lags	6 lags	6 lags	6 lags	6 lags
$\chi^2$ statistic	11.2	15.7	14.8	2.8	3.7
Significance (%)	8.1	1:5	2.2	83.2	72.1
		• · ·			
Wald test over the					
exclusion of:	5 lags	5 lags	5 lags	5 lags	5 lags
$\chi^2$ statistic	2.5	13.6	13.9	0.9	2.1
Significance (%)	77.6	1.8	1.7	97.3	83.9
$\Sigma$ coefficients of					
variable I/GDP	0.008	-0.036	-0.031	-0.014	-0.028
$\log(n+\phi+\delta)^{i}$			-0.044		-0.090
			(2.19)		(2.38)
			()		(2.00)
$\log(s_{h}^{i})$			0.009		-0.070
			(1.21)		(1.57)
Number of Obervations	624	624	624	600	600
Estimation Method	OLS	OLS	OLS	GMM	GMM
Indiv. effect included?	no	yes	yes	removed	removed
Equation estimated:	(7)	(7)	(7)	(8)	(8)

TABLE1-3.OUT

	1	2	3	4	5	6
ags included:	6	6	6	6	6	6
Wald test over the						
exclusion of:	6 lags	6 lags	6 lags	6 lags	6 lags	6 lags
$\chi^2$ statistic	9.6	4.0	11.5	2.1	16.8	17.6
Significance (%)	14.2	68.3	7.4	90.7	1.0	0.9
Wald test over the						
exclusion of:	5 lags	5 lags	5 lags	5 lags	5 lags	5 lags
$\chi^2$ statistic	5.0	2.3	10.5	0.6	16.0	16.2
Significance (%)	42.0	80.3	6.3	97.3	0.7	0.6
coefficients of ariable I/GDP	0.003	-0.002	-0.030	0.070	-0.339	-0.333
	0.003	-0.002	-0.030	0.070	-0.339	-0.333
$\log(V_{t-1}^{i})$		1 <b></b>	-0.070	<u> </u>	-0.131	-0.187
-00(-1)			(5.78)		(3.18)	(2.92)
$\log(n+\phi+\delta)$	· ·	<del></del>	-0.049	<del></del> .		-0.021
			(2.41)			(1.70)
$\log(s_{h}^{i})$		*	-0.009	·==.		0.038
- • ••			(1.03)		т. Г	(1.09)
Jumber of Obervations	600	600	600	576	576	576
Estimation Method	OLS	OLS	OLS	GMM	GMM	GM
ndiv. effect included?	no	yes	yes	removed	removed	remove
Equation estimated:	(7)	(7)	(7)	(8)	(8)	(8)

Table 2Causality from ln(1/GDP) to  $\Delta lny$ 

TABLE1-3.OUT

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	Causanty	$110 \text{ m} \Delta m$	y to <i>ln(1/G</i>	UP)	
	1	2	3	4	5
Lags included:	6	6	6	6	6
Wald test over the					
exclusion of:	6 lags	6 lags	6 lags	6 lags	6 lags
$\chi^2$ statistic	38.2	46.4	34.6	9.98	12.2
Significance (%)	0.0	0.0	0.0	12.5	5.7
				· ·	2
Wald test over the					
exclusion of:	5 lags	5 lags	5 lags	5 lags	5 lags
$\chi^2$ statistic	37.4	45.1	33.9	9.9	12.2
Significance (%)	0.0	0.0	0.0	7.8	3.2
$\Sigma$ coefficients of		_	·		
variable I/GDP	0.419	1.11	0.697	1.65	0.73
					$\mathcal{L}^{(1)} = \mathcal{L}^{(1)}$
$\log(y_{t-1}^1)$			-0.07		-0.186
			(2.09)		(1.21)
$\log(n+\phi+\delta)^{1}$			0.082		-0.052
$\log(n+\phi+0)$			(2.23)		(0.69)
			(2.23)		(0.09)
$\log(s_{h}^{i})$		^	0.006	<del></del> .	0.026
- ·			(0.32)		(0.29)
$\chi^2$ (H <sub>o</sub> : $\Sigma$ coeff. of					
variable I/GDP)	5.75	0.16	0.85	0.14	0.04
significance level	0.02	68. 6	35.5	70.9	83.5
Number of Obervations	600	600	600	576	576
Estimation Method	OLS	OLS	OLS	GMM	GMM
Indiv. effect included?	no	yes	yes	removed	removed
Equation estimated:	(7)	(7)	(7)	(8)	(8)

## **Table 3** Causality from $\Delta lny$ to ln(I/GDP)

TABLE1-3.OUT

	Regression Sample ave	s with ind trages from			- · ·
		1	2	3	4
Dependent Variable:		DINDIV	DINDIV	∆ln y	∆ln y
List of regressors:			·		
Constant		0.16 (2.16)	0.02 (1.37)	0.22 (0.48)	-0.16 (1.255
$\log(y_{60}^{i})$			0.11 (40.6)	-0.38 (4.31)	-1.03 (34.6)
log(I/Y) <sup>i</sup>		0.06 (2.49)	0.011 (2.44)	0.45 (2.88)	-0.08 (1.75)
INDIV					9.20 (30.2)
$log(y_{90}^{i}-y_{60}^{i})$			0.10 (21.2)	- 	
₿ <sup>2</sup>		0.055	0.991	0.642	0.981
σ Number of observation Implicit speed of	5:	0.037 24	0.004 24	0.148 24	0.035 24
COnvergence:		-	• • •	1.1%	2.3%

Table 4Regressions with individual effectsSample averages from 1960 to 1990

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# Causality tests across countries

Table 5

		riables in levels	Variab leve + addition	els	Variabl first diffe + addition	erences
	Causality	sign	Causality	sign	Causality	sign
Australia	yes	+	yes		yes	-
Austria	no	-	yes	+	no	+
Belgium	no	-	no	• •	no	+
Canada	yes	-	yes	+	no	
Switzerland	yes	+	yes	+	yes	+
Germany	yes	+ .	yes	-	no	. =
Denmark	yes	-	yes	+	yes	+
Spain	yes	-	yes	+,	yes	+
Finland	no	+	no	. <del>-</del>	yes	-
France	no	-	yes	+	yes	+
United Kingdom	no	-	yes	-	no	+
Greece	no		yes	+ .	yes	+
Ireland	yes	-	yes	-	yes	-
Iceland	yes	+	yes	+	no	-
Italy	yes	+	yes	-	yes	-
Japan	yes	-	no	-	yes	
Luxembourg	no	-	no	-	no	+
Netherlands	yes	+	no	+	no	+
Norway	yes	+ .	yes	`+	no	+
New Zealand	no	_ `	no	-	no	-
Portugal	yes	. +	yes	-	yes	-
Sweeden	no	-	yes	-	yes	-
Turkey	yes	-	yes	+	yes	. +
United States	no	+	yes	-	yes	. –
		•		· ·		
· · · · · · ·						
Number of countries:					· · · · · · · · ·	
a) in which:					. · · · · · · · · · · · · · · · · · · ·	
Э causality А causality	14 10	-	18 6	-	14 10	- -
b) with a					**	
positive relationship		10	-	10	· -	12
negative relationship	-	14		14	-	12

TABLE5.OUT

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	1	2	3	4	5			6	7	8	9	10		
	. I		rates in 3 ce: UNES				Enrolment rates in 2nd. level (Source UNESCO)							
ags included:	2	2	2	2	2		÷ .	2	2	2	2	2		
Vald test over the														
xclusion of:	2 lags	2 lags	2 lags	2 lags	2 lags			2 lags	2 lags	2 lags	2 lags	2 lags		
$\chi^2$ statistic	4.63	3.73	2.53	0.17	0.51			2.34	2.18	2.47	0.83	1.83		
Significance (%)	9.9	15.1	28.3	92.0	77.4			30.9	33.7	29.1	66.0	40.1		
Vald test over the														
xclusion of:	1 lag	1 lag	1 lag	1 lag	1 lag			1 lag	1 lag	1 lag	1 lag	1 lag		
$\chi^2$ statistic	2.0	0.0	0.14	0.12	0.21			2.02	2.17	0.46	0.81	1.75		
Significance (%)	15.4	99.6	70.8	73.2	64.4			15.5	14.1	49.7	37.8	18.6		
ign of causality	(-)	(-),	(+)	(-)	(+)			(-)	(-)	(+)	(-)	· (-)		
igh of causanty	()	().												
og (y <sub>t-1</sub> )			-0.02		-0.18					-0.02		-0.16		
6 Ot-IV			(3.98)		(6.12)					(4.10)		(6.16)		
			0.02		-0.01	. "				0.02		-0.01		
og (I/GDP)			(5.07)		(0.47)		۰.		· •	(4.47)		(0.23)		
			(3.67)		(0117)							(0125)		
$\log (n + \phi + \delta)^{i}$			-0.20		0.01					-0.16		0.01		
Jg (Π+ψ+0)	1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 -		(3.18)	1.00	(0.15)					(3.19)		(0.53)		
	·		(2010)		<b>\</b> /					<b>( )</b>				
								· · · · · · · · · · · · · · · · · · ·						
ime dum. included?	no	yes OLS	yes OLS	yes GMM	yes GMM			no OLS	yes OLS	yes OLS	yes GMM	yes GMN		
stimation Method quation estimated:	OLS (7)	0LS (7)	(7)	(8)	(8)			(7)	(7)	(7)	(8)	(8)		

Table 6Causality from ln ( $S_h$ ) to  $\Delta ln$  (y)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14 1	15
		Average y (Source: Bas				Average years of schooling in 2nd. (Source: Barro & Lee (1994))							ars of sch arro & Le		
ags included:	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
Wald test over the exclusion of: $\chi^2$ statistic Significance (%)	2   1.4 49.1	2 2.42	2 lags 0.47 79.1	2 lags 0.10 95.2	2 lags 0.61 73.7	2 lags 3.58 16.7	2 lags 4.27 12.8	2 lags 0.72 69.6	2 lags 0.08 96.1	2 lags 0.98 61.2	2 lags 4.66 9.72	2 lags 4.59 10.1	2 lags 0.12 94.1	2 lags 0.04 97.6	2 lags 1.04 59.2
Vald test over the xclusion of: $\chi^2$ statistic Significance (%)	1 1 0.1 69.1		i lag 0.22 63.6	1 lag 006 80.2	i lag 0.69 44.1	1 lag 0.13 72.2	1 lag 0.34 56.0	1 lag 0.10 75.6	l lag 0.04 83.7	1 lag 0.92 33.7	l lag 1.29 25.6	1 lag 0.45 50.4	l lag 0.07 78.6	1 lag 0.00 99.7	1 lag 0.73 39.2
ign of causality	(-)	(-)	(+)	(-)	(+)	(-)	(-)	(+)	(+)	(+)	(-)	(-)	(+)	(-)	(+)
og (y <sub>t-1</sub> )			-0.017 (3.66)		-0.171 (5.96)			-0.018 (4.14)		-0.173 (6.05)			-0.016 (4.02)		-0.173 (6.20)
og (I/GDP)			0.021 (4.29)		-0.012 (0.57)			0.022 (4.36)		-0.008 (0.38)		-	0.022 (4.71)		-0.011 (0.51)
$\log((n+\phi+\delta)^i)$			-0.166 (3.07)		0.019 (0.57)			-0.184 (2.93)		0.016 (0.57)			-0.175 (2.86)		0.004 (0.17)
												. * . *			
Fime dum. included? Estimation Method Equation estimated:	no OL: (7)		yes OLS (7)	yes GMM (8)	yes GMM (8)	no OLS (7)	yes OLS (7)	yes OLS (7)	yes GMM (8)	yes GMM (8)	no OLS (7)	yes OLS (7)	yes OLS (7)	yes GMM (8)	yes GMM (8)

**Table 6** (cont.) Causality from ln (S<sub>h</sub>) to  $\Delta ln$  (y)

		1	2	3	4	5		6	7	8	9	10		
		]		rates in 3 ce: UNES				]	Enrolment (Sour	rates in 2 ce UNES			•	
Causality from	· · ·				-									
lnS <sub>h</sub> to lny														
• 3 causality (1 lag)		no	no	yes	no	no		no	yes	no	no	no		
Sign of causality		(-)	(+)	(+)	(+)	(+)		(+)	(+)	(+)	(+)	(+)		
• 3 causality (2 lag)		no	no	yes	no	no		no	no	no	no	no		
Sign of causality		(+)	(+)	(+)	(+)	(+)		(+)	(+)	(+)	(-)	(-)		
• 3 causality (3 lag)		no	no	yes	no	no		no	no	no	no	no		
Sign of causality		(+)	(+)	(+)	(+)	(+)		(+)	(+)	(+)	(-)	(-)		
Causality from Iny to InS <sub>b</sub>							- -							
• J causality (1 lag)		no	no	no	no	no		no	no	no	no	no	1.1.1	
Sign of causality		(+)	(+)	(+)	(-)	(-)		(+)	(+)	(+)	(-)	(-)		·
• 3 causality (2 lag)		yes	no	no	no	no		no	no	no	no	no		
Sign of causality		(+)	(+)	(+)	(+)	(+)		(+)	(+)	(+)	(-)	(-)		
• 3 causality (3 lag)		yes	yes	yes	no	no		no	no	no	no	no		
Sign of causality		( (+)	(+)	(+)	(+)	(+)		(+)	( <b>+)</b>	(+)	( <b>-</b> )	(-)		
Causality from			· · ·								· · ·			
$\Delta lny$ to $lnS_{\rm b}$														
• 3 causality (1 lag)		no	no	no	no	no		no	no	no	no	no		
Sign of causality		(+)	(+)	(+)	(-)	(-)		(+)	(+)	(+)	(+)	(+)		. *
• 3 causality (2 lag)		no	no	no	no	no		no	no	no	no	no		
Sign of causality		(+)	(+)	(+)	(-)	· (-)		(+)	(-)	· (-)	(+)	(-)		
• 3 causality (3 lag)		no	no	no	no	no		no	no	no	no	no		÷
Sign of causality		(+)	(+)	(+)	(+)	(+)		(-)	(-)	(+)	(-)	(-)		
				H		·								
Fime dum. included?		no	yes	yes	yes	yes		no	yes	yes	yes	yes		
Estimation Method		OLS	OLS	OLS	GMM	GMM		OLS	OLS	OLS	GMM	GMM		
Equation estimated:	1.1	(7)	(7)	(7)	(8)	(8)		(7)	(7)	(7)	(8)	(8)		

 Table 7

 Causality between humana capital, GDP per capita and growth

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
		Average y Source: Bai					rage years ource: Ba		ling in 2nd (1994))	<b>l.</b>				oolin in 31 ee (1994))	
Causality from															
InS <sub>h</sub> to Iny															
• I causality (1 lag)	yes	no	no	no	no	no	no	yes	no	no	no	no	yes	no	no
Sign of causality	(+)	(+)	(+)	(-)	(-)	(+)	(+)	(+)	(-)	(+)	(+)	(+)	(+)	(-)	(+)
• 3 causality (2 lag)	no	no	no	no	no	no	no	no	no	no	no	no	no	no	no
Sign of causality	(+)	(+)	(+)	(-)	(-)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(-)	(-)
• 3 causality (3 lag)	no	no	no	no	no	no	no	no	no	no	no	no	no	no	no
Sign of causality	(+)	(+)	(+)	(-)	(-)	(+)	(+)	(+)	(-)	· (-)	(+)	(+)	(+)	(-)	(-)
Causality from Iny to InS <sub>b</sub>									1 <del>.</del>						
• 3 causality (1 lag)	yes	no	no	no	no	yes	yes	yes	no	no	yes	yes	yes	no	no
Sign of causality	(+)	(+)	(+)	(+)	(-)	(+)	(+)	· (+)	(+)	(+)	(+)	(+)	(+)	(-)	(-)
• 3 causality (2 lag)	no	no	no	no	no	no	no	no	no	no	yes	no	no	no	no
Sign of causality	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	( <b>+</b> ) <sup>2</sup>	(+)	(+)	(+)	(-)	·(-)
• 3 causality (3 lag)	no	no	no	no	no	no	no	no	no	no -	no	no	no	no	no
Sign of causality	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	( <b>+</b> )	(+)	(+)	(+)	(+)	(+)
Causality from															
$\Delta lny$ to $lnS_{\rm b}$										• .	•				e e e e e e e e e e e e e e e e e e e
• 3 causality (1 lag)	no	no	no	no	no	no	no	no	no	no	yes	yes	no	no	no
Sign of causality	(-)	(-)	(-)	(+)	(+)	· (-)	(-)	(-)	(+)	(+)	(-)	(-)	(-)	(-) <sup>1</sup>	(-)
• 3 causality (2 lag)	no	no	no	no	no	no	no	no	no	no	yes	yes	no	no	no
Sign of causality	(-)	(-)	· (-)	(-)	(-)	(-)	(-)	(-)	(-)	(-)	(-)	(-)	<b>(-)</b>	· (-)	(-)
• 3 causality (3 lag)	no	no	no	no	no	no	no	no	no	no	no	no	no	no	yes
Sign of causality	(-)	(-)	(-)	(-)	(-)	(+)	(+)	( <b>-</b> )	(-)	(-)	(-) ·	(-)	(-) <sup>1</sup>	(-)	(-)
										•			· . ·		
Time dum. included?	no	yes	yes	yes	yes	no	yes	yes	yes	yes	no	yes	yes	yes	yes
Estimation Method	OLS	OLS	OLS	GMM	GMM	OLS	OLS	OLS	GMM	GMM	OLS	OLS	OLS	GMM	GMM
Equation estimated:	(7)	(7)	(7)	(8)	(8)	(7)	(7)	(7)	(8)	(8)	(7)	(7)	(7)	(8)	(8)

 Table 7 (cont.)

 Causality between humana capital, GDP per capita and growth