



EuroEconomica

Health Capital and Economic Growth in Cameroon

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Abstract: From the macroeconomic statistics of the WHO, datum EDS and the World Bank data base, effects of the health on the growth in Cameroon are estimated thanks to a model of the production function. The macroeconomic estimations allowed to establish that the evolution of the variables of the human resources was not favourable to the increase of the GDP (Gross Domestic Product), in particular the contribution of the hand of work and the variable of the healthy living that is the life expectancy.

Keywords: health capital; economic growth; labour force; life expectancy

JEL Classification: F43

1. Introduction

The conception of health in a macroeconomic approach is multidimensional and very complex in nature. It is simultaneously defined by quality of life, a level of physical and moral well-being and the absence of disease. It also refers to the “reflexive capacity of the human being”. Thus, health is both an objective, clinically measurable state and a relative state that refers to our knowledge, freedoms, and expectations about our capacities to function and our desired state of health (Evans, 1996).

The World Health Organisation’s Commission on Macroeconomics and Health (WHO 2001) makes a revealing observation: countries with a high level of human development had growth rates of 2.3% per year on average between 1990 and 1998, compared to 1.9% for countries with a medium level of human development. In contrast, low human development countries had a growth rate close to zero.

The United Nations’ Sustainable Development for Health commitments call for “*meeting primary health care needs, especially in rural areas*”, and “*protecting vulnerable groups*”. However, the World Bank Report 2007 observes that *throughout the world, the poor experience high mortality, malnutrition and limited access to reproductive health and other basic health services. In addition, disease pushes millions of people into poverty every year.*

The economic literature on the relationship between health and economic growth¹, however, yields equally controversial results: the first category shows a positive and significant impact of improved health on economic growth (Gallup & Sachs (2001); Bloom et al. (2004); Weil (2008); Sala-i-Martin (2005); Jamison et al. (2005); Li and Huang (2008); Aghion et al (2008)). The second category shows

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that there is a link but not a significant one between these two variables (Hartwig 2009). In the same vein, Bhargava et al (2001) noted that better health has a greater effect on wages in low-income countries than in high-income countries. In contrast, the third category leads to the paradoxical conclusion of a negative relationship between health and growth (Acemoglu and Jonson 2007), i.e. better health rather negatively affects economic growth.

Some authors, on the other hand, have approached this question by considering health as a variable that can contribute to entering a “*poverty trap*” (see for example Sala-i-Martin, 2005) in poor countries. Several studies have sought to test the relevance of this hypothesis in a macroeconomic framework, preferring the term “*underdevelopment trap*” (Berthélemy, 2006) or “*human development trap*” (Mayer-Foulkes, 2003).

The instability of economic growth does not facilitate social progress and development (Barro and Lee, 1994). The low growth rate also does not allow for a return to public spending on essential social sectors such as education, health and infrastructure. Emerging countries, especially those in Asia, which were at the same level of development as Sub-Saharan African countries in the 1950s, escaped poverty once they started to perform well on the main human capital indicators of health and education (Berthélemy 2007, Tchouassi 2017). In contrast, these indicators have not progressed sufficiently in Africa and the backwardness has worsened since the 1980s following the rise of the HIV/AIDS epidemic. Human capital has been clearly shown to differentiate the development trajectory between emerging countries and poor countries since the 1950s.

In the case of Cameroon, economic growth has not improved significantly, although statistics show a slight recovery since 1994. African statistics do show a concordance between health indicators and the level of GDP. The limited accessibility to health care for half the population and the lack of advanced care strategies result in a modest use of health services. The proportion of births attended by qualified health personnel is 58%, with variations of 95% in the country’s cities of Yaoundé/Douala and 48% in rural areas. Life expectancy at birth in the last two decades has been capped at 54 years. In terms of government efforts, in the face of an unfavourable economic situation, there has been a rationing of expenditure in the social sector. Despite the good will of the public authorities, there is evidence of a low priority given to human development, following the poor economic situation of the 1980s and 1990s and the funding constraints faced by the public sector. Investment in human capital is often presented as a necessary condition for the economy to take off (Tchouassi 2017).

In the traditional growth model of Solow (1956), which was for a long time one of the references of development models³ before the development of endogenous growth models, health capital does not participate in growth. Solow considers a simplified model of growth in a closed economy:

$$Y_t = C_t + S_t$$

$sY_t = S_t$, avec s le taux d’épargne qui est exogène

$$K_{t+1} = K_t(1 - \delta) + I_t$$

$$L_t = L_0 (1 + n)^t$$

$$Y_t = a(1 + \gamma)^t K_t^\alpha L_t^{1-\alpha}, 0 < \alpha < 1$$

Avec $I_t = S_t$

where, Y_t represents production, C_t consumption, S_t savings, K_t capital stock, I_t investment and L_t labour respectively in period t . Labour grows at an exogenous rate n . The factors of production increase at a rate. For the resolution of the model, it is assumed that:

$$\forall t, K_{t+1} = (1 + \delta)^t K_t + sa \quad K_t^\alpha L_t^{1-\alpha} (1 + \gamma)^t$$

Thus, the balanced growth rate is given by g .

$$(1+g) = (1 + n) (1 + \gamma)^t (1/(1 - \alpha))$$

On the balanced growth path, we have

$$K_t^* = K^s (1 + g)^t, \forall t, \text{ avec } K^s = \left(\frac{sa}{g + \delta} \right)^{\frac{1}{1-\alpha}} L_0.$$

Given that $K > 0$, we show that $\frac{K_t}{(1 + g)^t} \rightarrow K^s$

In this model, economic growth is described as resulting solely from *exogenous variables*: the increase in the active population and a factor of technical progress. But this technical progress, which Solow considers as the engine of growth, is not explicitly modelled. His model gives greater importance to the output per capita y .

$$Y = F(K, L)$$

$$\frac{Y}{L} = F\left(\frac{K}{L}, 1\right)$$

$$\Rightarrow y = f(k)$$

Thus, output per worker increases as capital per worker increases.

$$y = f(k) \quad f'(k) > 0$$

The dynamics of the economy do not come from the individual and collective behaviour of economic agents, nor from the attitude of institutional actors. Under these conditions, the growth mechanism is reduced to a purely mechanical macroeconomic phenomenon not based on private knowledge systems or human capacities. Human capital in its individual health component is rarely considered in this modelling.

The neo-classical analysis of long-term growth predicts a convergence of output per unit of labour, conditioned by the rates of investment in physical and human capital as well as by the rate of growth of the active population. Indeed, the studies inspired by Solow's model predicted in most cases a general catching up of the living standards of the richest industrialised countries, including the developing countries, in only three or four decades. These predictions have been proved, as a large part of the world has so far been excluded from this catching-up process (Kousnetzoff, 2001).

Economic growth, which is often referred to as the “*engine of economic development*”, can only be sustained in an economy with a well-skilled workforce that also enjoys an adequate level of health.

During the 1970s and until the mid-1980s, macroeconomic theoretical research in the explanation of growth focused essentially on cyclical fluctuations and short economic cycles (Barro & Sala-i-Martin,

1995). Finally, in the middle of the 1980s, under the initial impetus of Romer (1986) and Lucas (1988), the neo-classical theory of long-term growth underwent a profound renewal with the appearance of endogenous growth models.

Studies show that healthy workers are also less prone to absenteeism, whether due to their own health problems or those of their families (Sachs 2002). Considering the neoclassical assumption that each worker should receive a wage equivalent to his or her marginal productivity, it is logical that illness that affects health status also affects labour income. Thus, for several authors, illness and disability reduce hourly wages by substantial amounts; this is particularly detrimental in developing countries where a high proportion of the workforce is engaged in manual work. Even when it does not prevent them from working, illness reduces the productivity of individuals, shortens the period of activity, and increases the number of days lost due to illness (World Bank, 1993). Barro (1996) shows in the same perspective that a 10% increase in life expectancy could lead to a 0.4% increase in future growth.

The increase in labour productivity, induced by the improvement of individuals' capabilities (Sen 1985), generates an increase in national income. Several analyses by economists have also tried to formalise the relationship between health and economic growth within the framework of the endogenous growth model.

These authors start from a growth model inspired by Lucas and completed by Grossman's health demand model. They make their contribution from two different approaches to the relationship between growth and health. First, they establish the link between these two variables based on the work of Baumol (1967) who explains the evolution of health expenditure and its consequences for growth. Secondly, they assume that health plays a major role in growth. Thus, the health sector can compete with the education sector in the context of scarce resources. The model leads to maximising a utility function of the following form:

$$U = \int_0^{\infty} e^{-\rho t} \left(\frac{(C/L)^{1-\sigma} - 1}{1-\sigma} \right) L dt$$

with: C : consumption and L : all available labour in the economy assumed constant, σ : degree of risk aversion related to consumption, also called intertemporal elasticity of substitution, ρ : discount rate. The production function is:

$$Y = (A(1-h))^{\alpha} \cdot K^{1-\alpha}$$

At stationary equilibrium, the growth rate of the economy is obtained by solving the Model.

$$g = \mu_A = \frac{1}{\sigma}(\delta - \rho) \quad \text{et} \quad h = \mu_A / \delta = \frac{1}{\sigma} \left(1 - \frac{\rho}{\delta} \right)$$

The conclusions of the model are well known since the Lucas model (1988): the growth rate g increases as a function of the accumulation of knowledge (δ productivity of the accumulation process) as well as the value of the intertemporal elasticity of the agents and the latter reflects their willingness to postpone

their consumption in the future to invest and reap the benefits later on. An increase in growth also requires an increase in the share of labour allocated to the accumulation of knowledge.

Subsequently, Van Zon and Musken (1997) will take up Grossman's model with the health production equation:

$$\dot{s} = \delta_s \left(\frac{vsL}{L} \right)^\beta - \Delta s = \delta_s (vs)^\beta - \Delta s$$

Given s : the average health status of the population, these two authors assume that the volume of work provided by a worker is proportional to s . Δ corresponds to the rate of exogenous depreciation of health over time as in Grossman. v is the share of the health sector in total employment, vsL is the total number of inputs that generate health services. For simplicity, the authors assume that the production of health services is proportional to the sum of the inputs (an assumption that simplifies the resolution of the model). This proportionality is represented by δ_s . In addition, the assumption that the change in the average health status of the population is not proportional to the resources allocated to health services per capita is retained. This means that $0 < \beta < 1$.

This approach leads to writing the growth model with health as:

$$U = \int_0^\infty e^{-\rho t} \left(\frac{(C/L)^{1-\sigma} - 1}{1-\sigma} \right) L dt$$

$$Y = (A(1-h-v)sL)^\alpha \cdot K^{1-\alpha}$$

$$\dot{K} = Y - C$$

$$\dot{A} = \delta_{hsA}$$

$$s = z_0 v^{z_1}$$

The steady state resolution gives the growth rate which is a function of the health factors:

$$g = \mu_A = \frac{1}{\sigma} \left(\delta_A \cdot (1-\beta) \cdot \bar{s} - \rho \right)$$

Average health depends positively on the efficiency of the health system and negatively on the rate of depreciation of health capital. The share of labour allocated to the health service depends only on the productivity characteristics of the health sector. Moreover, in equilibrium, when $v=\beta$, the growth rate of the system which is a function of v is maximum. This allows our authors to demonstrate that health increases utility only through its contribution to growth.

In a second approach, Van Zon and Musken (1997) take up an approach relatively close to that of Piatecki and Ulmann (1995), this time considering health as an argument of the utility function and

therefore as a source of well-being for individuals and society. The new utility function becomes:

$$U = \int_0^{\infty} e^{-\rho t} \left(\frac{\left(\left(\frac{s}{\bar{s}} \right)^{\omega} (C/L) \right)^{1-\sigma}}{1-\sigma} \right) L dt$$

The authors assume that $\omega \geq 0$ with decreasing marginal utility of health for $\omega < 1$. Since the average level of health s/\bar{s} is determined based on the results of the previous model, if $\omega = 0$ we obtain the same model.

The graphical resolution of the model shows that health is produced under conditions of diminishing returns while knowledge is produced under conditions of increasing returns. If health is not included in the utility function directly, the health sector has an optimal size that is compatible with optimal growth. In this case, health is a pure complement to growth from the supply side. Therefore, any reallocation of labour from the health sector to the knowledge- generating sector should slow down growth. On the other hand, an increase in the demand for care, whether due to an increase in health preference or to population ageing, will have a negative effect on growth. Several other models are in line with this approach to the macroeconomic explanation of growth by the public health sector.

This model adopts a Keynesian approach by formalising instead the influence of health sector employment on economic growth and improved health status. Both authors choose a representative household utility function which is as follows:

$$U = \int_0^{\infty} e^{-\rho t} \left(\frac{C^{1-\sigma}}{1-\sigma} + \frac{S^{1-\omega}}{1-\omega} \right) dt$$

With C : the consumption and S the health of the representative household, σ : the degree of risk aversion related to consumption, ω : the degree of risk aversion related to health, ρ : the discount rate.

Consumption and health in the model are considered as two substitutable goods, contrary to the generally accepted assumption of complementarity. Indeed, beyond the simple assumption of rationality, the analysis in health economics pragmatically assumes that it is impossible to consume when the level of health status is zero (death). Just as consuming nothing undoubtedly leads to a person's death⁵.

In each period, the output Y is divided between investment (I) and consumption (C):

$$Y = C + I.$$

The representative firm produces the single good of the economy according to a cobb-Douglas type production function with S , the health factor; L , the labour devoted to the production of the good and K the physical capital:

$$Y = A K^{\alpha} L^{\beta} S^{\gamma}$$

Finally, the production function of health is defined as a function of the share of labour devoted to health,

i.e. $(1-L)$ and a technological parameter δ . The following equation represents the variation of health S

$$\dot{S} = (1-L)S$$

The model considers two decision variables in the formalisation: consumption C and time spent on production L . These variables are determined in such a way as to maximise the programme. This is due to an assumption that the consumer forms his preferences based on a consumption-health partition.

On the balanced growth path, K , S and C grow at a constant rate and with constant N .

This growth rate is defined as μ_K , μ_S et μ_C , and respectively. Then:

$$\mu_K = \frac{\gamma}{1-\alpha} \mu_S$$

We find in the case of constant returns to scale, i.e., $\alpha + \beta + \gamma = 1$ and $\gamma < 1 - \alpha$ that the growth of the economy is lower than the growth of the health state. In the case of increasing returns to scale, the growth of the economy can also be less than, equal to or greater than the growth of the health state. In Africa, the costs of illness are borne mainly by individual agents because of the lack of adequate health coverage. All of which further drains savings, which are already excessively low, and ultimately slows the process of capital accumulation (Theodore 2001, MacFarlan and Sgherri 2001).

Ulmann (1999) integrates into the endogenous growth model proposed by Lucas (1988), the health demand model introduced by Grossman (1972) to test the relationship between health, human capital accumulation and economic growth. This approach thus extends Lucas' analysis by incorporating health as a determinant of the quality of human capital. The intertemporal allocation choice of individuals involves a trade-off not only between consumption and investment in human capital, but also on the allocation of resources for health care and services (Ulmann 2003). While Grossman's model assumes a single homogeneous health service, this specification distinguishes between health enhancing activities and care. The programme to be maximised is therefore the following:

$$\text{Max } U = \int u(c_t, s_t, b_t) e^{-\rho t} dt$$

where c_t represents per capita consumption; s_t the per capita health capital stock; b_t the share of resources allocated to health; ρ the subjective discount rate.

$$\dot{k}^* = (1 - \tau_b)F[k_t, (1 - h_{h,t} - h_{s,t} - \varepsilon(s_t, b_t))h_t] - \delta k_{t,k} - c_t - \pi_t$$

$$\dot{h}^* = (\mu h_{h,t} - \delta_h)h_t$$

$$\dot{s}^* = G(h_{h,t}, s_t) - \delta_h s_t$$

where k_t represents the physical capital stock, h the human capital stock, h_{ht} and h_{st} the share of human capital allocated to human capital accumulation and health improvement respectively. δ_k , δ_h , δ_s are respectively the depreciation rates of physical capital, human capital and health stock. $\varepsilon(s, b)$ is the share of human capital not used due to illness. π represents health insurance and τ represents supplementary

insurance, i.e. the fraction of expenditure remaining to be paid by the insured (covered or not by a supplementary insurance).

Using the intertemporal optimisation programme defined above, we study the steady- state relationships between health, human capital accumulation and economic growth. We can thus study the case where health is an investment in the sense that it does not affect the utility of consumption. In the stationary state:

$$\begin{aligned} \mu(1 - h_s - \varepsilon(s, b)) &= \rho + \delta_h + \sigma g \\ G_s(h_s, b) - \varepsilon_s(s, b)G_h(h_s, b) &= \varphi + \delta_s - (1 - \sigma)g \\ \left(\frac{c}{k}\right) &= \frac{1-b}{\alpha(1-\tau b)}(\varphi + \delta_k + \sigma g) - \delta_k - g \\ \left(\frac{h_n}{k}\right)^* &= \left(\frac{1}{\alpha} \frac{\varphi + \delta_k + \sigma}{1-\tau b}\right)^{\frac{1}{1-\alpha}} \end{aligned}$$

The theoretical predictions of the model assume that:

- Any change in behaviour or economic policy that alters health has effects on growth (g), through equations (11) and (12) and effects on the economy in level, through equations (13) and (14);
- A population with a lower subjective discount rate (i.e. a high concern for the future) enjoys better health and faster economic growth below a certain high threshold of health $status_G$;
- Behavioural changes (stopping smoking, eating a balanced diet, following preventive health programmes, etc.) that reduce the rate of depreciation of health capital have similar effects.

Macroeconomic modelling of the effect of an “epidemiological trap” on growth.

Couderc et al (2006) have developed an econometric model to show the effect of an “epidemiological trap” with the case of AIDS on economic growth. The simplified model is as follows:

$$Y = K^\alpha (h(\varepsilon, X^-)L)^\alpha$$

With h , the level of individual health capital of workers. $h'_\varepsilon < 0$ as illness reduces the health capital of workers. It is assumed that $L = [1 - \varepsilon(1 - g(X^-))]N$ represents the number of workers in an economy with N agents. The prevalence of disease affecting the country negatively affects the L/N ratio of workers. The use of health care by households contributes significantly to the restoration of their health capital, even if the marginal efficiency of care is decreasing (as such, the distribution function of this efficiency of care g is concave and upper bounded, with

$$g' > 0, g'' < 0 \quad g(0)=0 \text{ and } g(+\infty) = g^* < 1 \quad h = X^- \cdot l(\varepsilon^-)$$

The effect of disease on the quantity of labour force is measured by $\varepsilon(1 - g)$ representing the ratio of labour force to total population. The qualitative impact is estimated by the human capital. Health expenditure is expressed by the relation $X = \lambda y$ with y the per capita income, $y = Y/N$. The accumulation

function of health capital is formalised by the quantity: where $l(\varepsilon)$ is the impact of the disease on health status l' . Health care contributes to human capital accumulation through the positive externalities of health expenditure $h'_x > 0$.

The capital stock can be written under the assumption of profit maximisation of firms as follows:

$$K = \frac{\alpha(1-\mu)}{r} Y$$

With the corporate tax rate. The growth rate of GDP per capita is translated into the following final relationship:

$$1+G = \frac{Y}{Y^-} = \lambda l(\varepsilon^-) (1 - \varepsilon(1 - g(\lambda Y^-))) \left(\frac{\alpha(1-\mu)}{r} \right)^{\frac{\alpha}{1-\alpha}}$$

The growth rate G of the economy is thus a function of GDP per capita, and its values are situated between a ceiling and a \underline{G} floor. Thus, across a certain threshold, it can be shown that, depending on the magnitude of the prevalence of certain diseases in an economy and the subsequent health expenditure, a depressive growth scenario can occur.

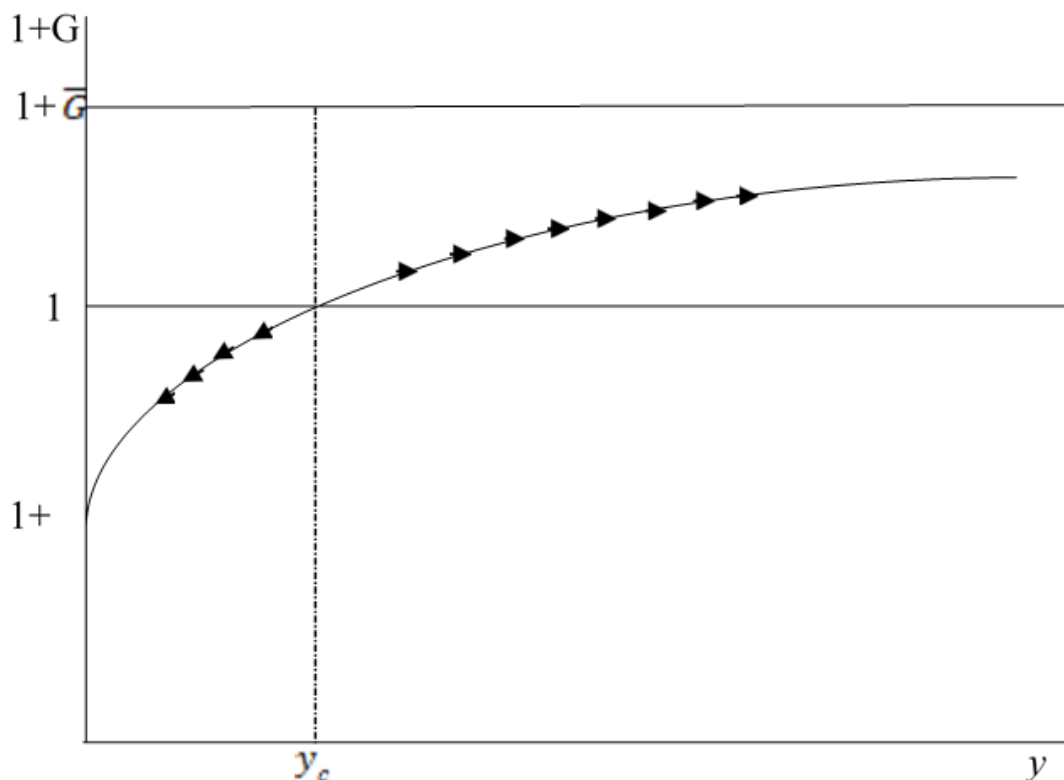
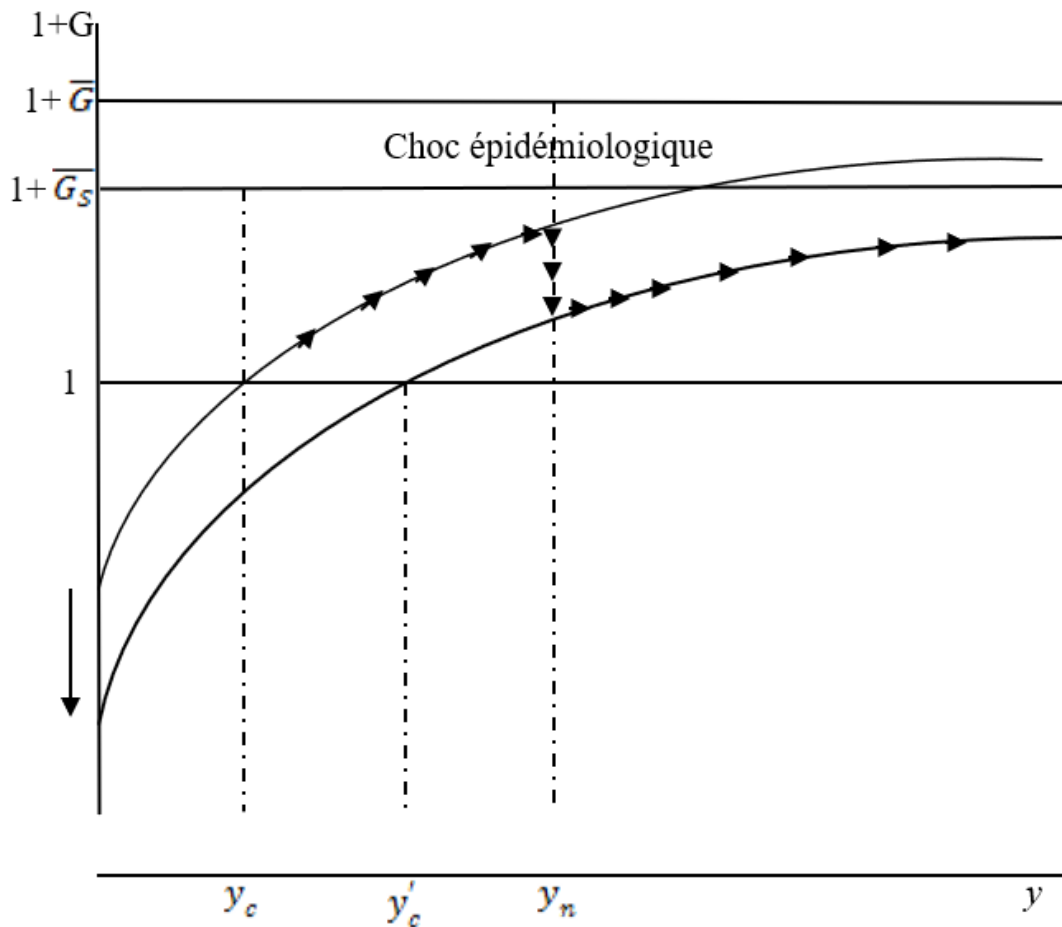


Figure 3.3. The threshold effect of health on growth

Indeed, for a health expenditure rate λ associated with the prevalence rate there is a threshold Y_c below which the growth rate will become negative. Above this threshold, the economy benefits from a positive GDP growth dynamic.



Graphique 3.4. Choc Épidémique Dépressif.

A major epidemiological shock, as was the case for AIDS in Africa in the 1990s will be responsible for a major depression in year n . This is indeed the situation currently observed with the COVID19 pandemic affecting the world since January 2020. In the worst case, the epidemic could even make the growth rate of the product totally negative (Couderc et al. 2006).

The econometric growth model with the health capital factor.

For Mankiw-Romer-Weil (1992), by integrating the health variable into the neoclassical production function, we have:

$$Y_t = K_t^\alpha H_t^\beta (A_t L_t)^{1-\alpha-\beta}$$

Where Y_t represents production, K represents physical capital, H represents human capital, A represents technical progress and L represents labour. The evolution of the economy is given by: $\dot{k}^* =$

$$s_k y_t - (n_i + g + \delta)k_t$$

$$h_t^* = s_h y_t - (n_i + g + \delta)h_t$$

$$y = \frac{Y}{AL}, \quad k = \frac{K}{AL} \quad \text{et} \quad h = \frac{H}{AL}$$

The authors assumed that the same production function applies for physical and human capital. However, in their estimates they restricted the notion of human capital to education, thus ignoring the health component. The work of Knowles and Owen (1996) will, in an ‘augmented’ Mankiw-Romer-Weil model, try to take this aspect of the problem into account⁷. The human capital stock thus has two components: the education stock (noted *E*) and the health stock (noted *X*). The output at time *t* is given by :

$$Y_t = K_{it}^\alpha E_{it}^\beta X_{it}^\psi (A_{it} L_{it})^{1-\alpha-\beta-\psi}$$

Their estimates lead to conclusive results and confirm a strong correlation between the health stock and income per worker. To test the influence of health inequalities between men and women on economic development, Russo (2005) uses this ‘augmented’ Mankiw-Romer-Weil model. The particularity of his study is that he disaggregates human capital by dissociating the health stock of women from that of men. He uses the following specification:

$$Y_{it} = K_{it}^\alpha E_{it}^\beta XF_{it}^\psi XM_{it}^\omega (A_{it} L_{it})^{1-\alpha-\beta-\psi.\omega}$$

where *Y* is output, *K* is the stock of physical capital, *E* is the stock of education, *XF* is the female health stock, *XM* is the male health stock, *A* is technology, *L* is labour in country *i* at time *j* (Knowles and Owen 1997). Knowles and Owen’s studies show that women in developing countries face significantly different health risks than men, and face different constraints in addressing their problems. Women often lack the power and social status to access economic resources. Because of their different position in society, women are usually poorer than men and often depend on them economically. However, health inequalities are not solely related to gender.

Health inequalities, low health care coverage and difficulties in accessing health care in poor countries could explain the level of health capital indicators.

Unfavourable evolution of some health capital indicators in Cameroon

The evolution of life expectancy at birth remains poor, although it is an indicator of the health system.

Table 3.4: Evolution of health capital in Cameroon.

Years	1990	1991	1992	1993	1994	1995	1996	1997	1998
Life expectancy	54	54	53	53	53	52	52	51	50
Years	1999	2000	2001	2002	2003	2004	2005	2006	2007
Life expectancy	50	50	50	50	50	51	52	52	53

Source: WHO Statistics 2010.

- Life expectancy has rather deteriorated from 1990 to 2000;

- The improvement since 2004 does not allow to reach the 1990 level in 2007;
- The maximum rate of 54 years is much lower than the rates in developed countries, which are between 70 and 80 years.

Below the world average, the evolution of life expectancy remained very marginal in sub-Saharan Africa between 1960 and 1990 compared to other regions of the world, and the decline in this health indicator in the 1990s is attributed primarily to the rise of the HIV/AIDS epidemic. Other diseases, even though they are more curable, such as malaria, tuberculosis, etc., are still ranked among the main causes of death in Cameroon and are dragging down life expectancy. Improvements in nutrition, education⁹, health technologies (Cutler et al. 2006), the capacity of institutions to obtain and use information, and the ability of society to use this knowledge for effective health and social action (Deaton, 2006), have resulted in better health status for the same level of wealth.

Table 4.4. Trends in the under-five mortality rate (per 1000 births).

Years	1990	1991	1992	1993	1994	1995	1996	1997	1998
Under-5 mortality rate (per 1000 births)	145	145	146	147	147	147	148	148	148

Years	1999	2000	2001	2002	2003	2004	2005	2006	2007
Under-5 mortality rate (per 1000 births)	148	143	138	134	129	125	120	117	115

Sources: WHO Statistics (2010).

The infant mortality rate has not fallen significantly for almost two decades in Cameroon. However, this infant mortality is very much linked to the organisation of the health system (access to care and the prevention system), especially as this mortality is attributed to an essentially avoidable morbidity. As mentioned above, there is a link between these health capital indicators and economic growth.

Health, as a state specific to the individual but also as a sector of activity, generates complex and numerous links with the economy and therefore growth (Ulmann 1999). The impact of health capital on economic indicators has been the subject of numerous studies since the 1990s and 2000s in both developed and developing countries.

Equation of the model to be estimated.

According to the approach adopted by Bloom et al (2003), the effects of health on growth are evaluated from the production function that we will present here and life expectancy is retained as an indicator of health capital.

$$Y_t = AK_t^\alpha L_t^\beta e^{\theta_1 s_t + \theta_2 exp_t + \theta_3 exp_t^2 + \theta_4 h_t}$$

Where Y_t represents the growth rate of GDP in period t ; A the total productivity of factors; K_t the physical capital in period t ; L_t the labour force; the human capital is here a factor combining both the average level of schooling s , the average of the professional experience of the workers, the professional experience squared and the health capital h whose proxy is the life expectancy.

Given the non-linear nature of this specification, the logarithmic form of the production function can be considered.

$$y_t = a_t + \alpha k_t + \beta l_t + \phi_1 s_t + \phi_2 exp_t + \phi_3 exp_t^2 + \phi_4 h_t$$

Here y_t , k_t and l_t represent the logarithm of Y_t , K_t and L_t respectively. Total factor productivity is not observed and can be considered in the error term of the estimated equation.

$$a_t = a_t^* + v_t \quad \text{with} \quad v_t = \rho v_{t-1} + \varepsilon_t$$

Where a_t is total factor productivity. If we consider the variation, the product equation becomes:

$$\Delta y_t = \Delta a_t + \alpha \Delta k_t + \beta \Delta l_t + \phi_1 \Delta s_t + \phi_2 \Delta exp_t + \phi_3 \Delta exp_t^2 + \phi_4 \Delta h_t + \Delta v_t$$

Where Δv_t is the error term.

The dependent variable of the model is the variation of GDP from period to period. The study period considered here is from 1990 to 2015. The variables that can explain this variation in the product are, among others:

- the change in the capital stock in period t ;
- variation in labour;
- the change in the average level of education;
- variation in the level of professional experience;
- the change in health capital approximated by life expectancy at birth and infant mortality rate.

To avoid the correlation between these two health capital indicators, the model is estimated twice, considering only one of these variables at each estimation. A correlation between the explanatory variables and the error term would then lead to biased estimates. To correct this endogeneity problem, we used the instrumental variables technique. The instruments used are the level of education and the rate of inflation because these two variables are *a priori* correlated with the infant and child mortality rate or life expectancy but have very little effect on the health capital of the year considered. Let us recall that by considering the logarithmic form, the model to be estimated is the following

$$\Delta \log(\text{pib})_t = a + b \Delta \log(\text{pib})_{t-1} + b \Delta \log(\text{fbcf})_t + c \log(\text{labor})_t + d \log(\text{esp})_t + d \log(\text{dept})_t + d \log(\text{ouv})_t + \varepsilon_t$$

Before making these estimates, several econometric tests were carried out to avoid spurious regression.

Statistical tests appropriate to the model.

To avoid the statistical problems mentioned above, the model was subjected to several tests.

Stationarity test

The Augmented Dickey-Fuller Test (ADF)

The Augmented Dickey-Fuller (ADF) test is an improvement on the simple Dickey-Fuller test. Indeed,

the construction of the latter is based on basic models with the assumption that the errors of the latter are white noise. The Augmented Dickey-Fuller test (1981) which integrates this possibility by considering an AR representation.

The basic models used to construct this new test are as follows:

$$\left\{ \begin{array}{l} \Delta X_t = \varphi X_{t-1} + \sum_{j=2}^p \gamma_j \Delta X_{t-j+1} + \varepsilon_t [1''] \\ \Delta X_t = \varphi X_{t-1} + \sum_{j=2}^p \gamma_j \Delta X_{t-j+1} + \mu + \varepsilon_t [2''] \\ \Delta X_t = \varphi X_{t-1} + \sum_{j=2}^p \gamma_j \Delta X_{t-j+1} + \alpha + \beta t + \varepsilon_t [3''] \end{array} \right.$$

Here, the number of lags p to clear the error can be obtained by minimising information criteria. It can also be obtained by systematic regressions of the model for a sufficiently high value of p , then by successive regressions for decreasing values of p until a significant p^{ime} delay is obtained (Bourbonnais, 2004). Dickey and Fuller have tabulated the critical values of this test under the null hypothesis. The decision rule remains the same as before:

- if the value of the test statistic is less than the tabulated critical value, the hypothesis of stationarity of the series is accepted;
- if the value of the test statistic is greater than the critical value, the hypothesis of stationarity of the series is rejected.

It is this last test that we will use to study the stationarity of our variables.

The test procedure

In practice, the Augmented Dickey-Fuller test is implemented in three sequential steps (Mignon and Lardic, 2002).

Stage 1

The significance of the trend in the model is tested [3'']. If it is significant, we are interested in the null hypothesis of non-stationarity of the chronicle. There are two possible scenarios:

- the null hypothesis is accepted. Here, the column is non-stationary; it is differentiated and the procedure is repeated on the differentiated series;
- the null hypothesis is rejected. The series under study is then stationary and the test stops there.

If the test reveals a non-significant trend, proceed to step 2.

Step 2

The significance of the constant in the model is tested [2'']. If it is significant, we are interested in the null hypothesis of non-stationarity of the column. Two cases are then possible:

- the null hypothesis is accepted. Here, the column is non-stationary; it is differentiated and the

procedure is repeated on the differentiated series;

- the null hypothesis is rejected. The series under study is stationary and the test stops there.

In the case where the test reveals that the constant is insignificant, proceed to step 3.

Step 3

The null hypothesis of unit root in the model is tested^[17].

- if the null hypothesis is accepted, the column is non-stationary; it is differentiated and the procedure is repeated on the differentiated series;
- the null hypothesis is rejected. The series under study is stationary and the test stops there.

Our variables are all stationary of order 1.

Test of normality of residuals

The assumption of normality of the error terms is essential in the implementation of a VAR model. Indeed, it is based on this assumption that the statistical distributions of the estimators from the model are established. Because of its simplicity, the Jarque-Bera test is very often used to test this hypothesis. The assumptions of this test are as follows:

- H_0 : the residues are normal;
- H_1 : the residuals do not follow a normal distribution.

The test statistic is as follows:

$$JB = \frac{T - k}{6} \left[S^2 + \frac{1}{4}(K - 3)^2 \right]$$

Where : $k =$ number of estimated parameters $S =$ Skewness coefficient

$K =$ Kurtosis coefficient

The decision rule here is to accept the normality hypothesis when the probability of the test is higher than the threshold considered and to reject it otherwise. The implementation of this test gives us the results presented in Table 5 below. Thus, at the 5% threshold, we accept the normality hypothesis of the residuals of our VAR model.

Table 4.6. Normality test of the residuals

	Jarque-Bera	Df	Prob.
Joint	17.88113	10	0.0570

Source: Author's calculations.

Heteroscedasticity of errors test

The notion of heteroscedasticity refers to the non-constancy of the variance of the error. In the case of proven heteroscedasticity, the estimators of the Ordinary Least Squares method are no longer minimum variance. There are many tests for heteroscedasticity. The one we will use is the White (1980) test.

The White test is based on the existence of a relationship between the squares of the residual of a model and one (or more) level or squared explanatory variable(s). For a model of the form:

$$Y_t = \alpha_1 + X_{1t} + X_{2t} + \dots + X_{kt} + \epsilon_t,$$

The following regression is to be performed:

$$\epsilon_t^2 = \beta_1 + \beta_2 X_{2t} + \dots + \beta_k X_{kt} + \lambda_1 X_{2t}^2 + \lambda_2 X_{3t}^2 + \dots + \lambda_k X_{kt}^2 + v_t$$

If at least one of the coefficients of this regression is significant, then the null hypothesis of homoscedasticity is rejected in favour of the alternative hypothesis of heteroscedasticity (Mignon and Lardic, 2002). In practice, the probability of the test can be compared to the threshold considered. When this probability is higher than the threshold, we accept the homoscedasticity of the residuals. Otherwise, the residuals are heteroscedastic.

In our case, White’s test allows us to accept the null hypothesis of homoscedasticity of the model errors at the 5% threshold. Indeed, as shown in Table 4.7 below, the probability of the test is greater than 0.05.

Table 4.7. Heteroscedasticity Test of Residuals

Chi-sq	df	Prob.
298.6035	330	0.8920

Source: Author’s calculations

The model is estimated using the Ordinary Least Squares (OLS) method with the Eviews 5 software. The data used range from 1980 to 2015 and are taken from the World Bank CD Rom 2018. Coefficients marked with (**) are those that are significant at the 10% level.

$$\Delta \log (pib)_t = 0.04^{**} + 0.63^{**} \Delta \log (pib)_{t-1} - 0.08 \Delta \log (fbcf)_t - 10.22^{**} \log (labor)_t - 6.44^{**} \log (esp)_t - 0.05 \log (dept)_t + 0.06 \log (ouv)_t + \epsilon_t$$

Estimation of the same model by replacing the health capital variable with infant mortality yielded results that were globally insignificant and not amenable to credible interpretation.

Interpretation of the estimation results.

- According to the estimation results, the evolution of the human capital variables was not favourable to the increase in GDP, particularly the contribution of the labour force and the health capital variable of life expectancy. The explanation that one can try to give for this result is that, except for some engineers who work in the field, most of the country’s intellectuals are occupied with the bureaucracy, whose impact on economic growth is not always favourable.
- The growth rate of the previous year contributes positively to the growth of GDP in Cameroon. This result expresses the possibility for the country to return to self-sustaining and sustainable economic growth, based on the structure and/or achievements of the economy, but also relying on a favourable economic climate.

As regards the particularity of the education factor in human capital and its contribution to growth, the study of Lau, Jamison and Louat (1990) showed indeed that the elasticity of production in relation to education is appreciably lower in Africa than in the rest of the world. But it also varies according to the level of education considered. Two explanations were put forward to justify this result:

- The sociological specificities of Africa, but also the problems of management and administration of the education system noted by Orivel (1995), are at the origin of the lower efficiency of African education.
- There is a threshold effect, at about four years of schooling for primary education, below which training has little impact on productivity. Lau et al (1990) on a set of developing countries and on Brazilian states were able to highlight this phenomenon.

Indeed, the role of education on economic growth has been questioned in some empirical works. Aghion and Cohen have shown that it depends on the level of development. Jaoul (2007) shows that, unlike France, which does not show a link between higher education and economic growth, Japan and the USA show a link between these indicators.

Conclusion

In the process of evaluating the effects of health on economic growth, some authors have done so through the diffusion of the benefits of health improvement or health sector research and development on GDP. By using a synthetic indicator that usually allows the level of health of populations to be evaluated (life expectancy at birth), the results we have arrived at show that the influence of health capital on economic growth is a reality in Cameroon.

Using WHO macroeconomic statistics, DHS and World Bank data, the effects of health on growth are assessed using a production function model. The macroeconomic estimates show that the evolution of human capital variables has not been favourable to the growth of Gross Domestic Product (GDP), notably the contribution of the labour force and the health capital variable of life expectancy.

Episodes of morbidities such as malaria, tuberculosis, and other AIDS block life expectancy at around 54 years. Eventually, this short life expectancy would have a negative impact on savings, educational investment, and private investment prospects. The combination of all these factors negatively affects growth. By way of economic policy recommendations, the search for strategies to finance health care must be one of the priorities for improving the health system. A reform of the Caisse Nationale de Prévoyance Sociale (CNPS) with the aim of at least extending health coverage to all strata of Cameroonian society is a necessity. At the same time, the government must continue to promote and support mutual health insurance for the poor and those in rural areas.

Health expenditure should no longer be considered an obstacle to the promotion of economic growth. If it is not possible to make the treatment of certain diseases free of charge for the poor, the State must invest more in the prevention system (environmental sanitation, awareness-raising on life hygiene, vaccination, promotion of the use of impregnated mosquito nets, promotion of access to drinking water, etc.), as certain preventive actions are less costly than medical treatment and allow gains in life expectancy.

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