Author(s): Ferretti, Fabrizio, Jones, Simon and McIntosh, Bryan

Title: Long-Run Macroeconomic Determinants of Cancer Incidence.

Publication year: 2013

Journal title: International Journal of Statistics in Medical Research.

Publisher: Lifescience Global

Link to original published version: http://dx.doi.org/10.6000/1929-6029.2013.02.04.4

Citation: Ferretti, F., Jones, S. and McIntosh, B. (2013) Long-Run Macroeconomic Determinants of Cancer Incidence. International Journal of Statistics in Medical Research. 2 (4) 275-288.

Long-Run Macroeconomic Determinants of Cancer Incidence

Fabrizio Ferretti[1], Simon Jones[2] and Bryan McIntosh[3,*]

[1]*Department of Communication Sciences and Business Economics, University of Modena and Reggio Emilia, Italy*

[2]*Department of Health Care Management and Policy, University of Surrey, UK*

[3]*Department of Business and Economics, Richmond University, UK*

Abstract: *Background*: Understanding how cancer incidence evolves during economic growth is useful for forecasting the economic impact of cancerous diseases, and for governing the process of resources allocation in planning health services. We analyse the relationship between economic growth and cancer incidence in order to describe and measure the influence of an increasing real per capita income on the overall rate of cancer incidence.

Method: We test the relationship between real per capita income and the overall rate of cancer incidence with a cross-sectional analysis, using data from the World Bank and the World Health Organization databases, for 165 countries in 2008. We measure the elasticity of cancer incidence with respect to per capita income, and we decompose the elasticities coefficients into two components: age-effect and lifestyle-effect.

Results: An Engel's model, in a double-log quadratic specification, explains about half of the variations in the age-standardised rates and nearly two thirds of the variations in the incidence crude rates. All the elasticities of the crude rates are positive, but less than one. The income elasticity of the age-standardised rates are negative in lower income countries, and positive (around 0.25 and 0.32) in upper middle and high income countries, respectively.

Conclusions: These results are used to develop a basic framework in order to explain how demand-side economic structural changes may affect the long run evolution of cancer incidence. At theoretical level, a *J*-Curve is a possible general model to represents, other things being equal, how economic growth influence cancer incidence.

1. INTRODUCTION

The importance of economic growth on population's health conditions is difficult to overstate. At macroeconomic level, both theory [1] and empirical evidence [2] indicate that there is a positive causal relation between per capita income and some fundamental measures of health performance (*e.g.*, life expectancy and infant mortality, among others). Overall, it seems that 'wealthier nations are healthier nations' [3].

During economic growth, however, every economy undergoes several substantial structural changes in healthcare demand and supply. Thereby, the process of economic growth modifies both composition and priority of society's health problems. In particular, cancers and others non-communicable diseases, that once were considered the diseases of high income countries, are now frequently diagnosed in developing economies [4]. Understanding how cancer incidence evolves during economic growth is increasingly useful for forecasting the economic impact of cancerous diseases, and for governing the process of resources allocation in planning health services [5]. However, there has been a scarcity of research about the long-run macroeconomic determinants of cancer frequency [6].

This paper analyses the relationship between economic growth and cancer incidence at macroeconomic level, using worldwide cross-sectional data for 165 countries in 2008. First, we attempt to collect some empirical regularities concerning how an increasing real per capita income influences the overall rate of cancer of incidence. Second, we use these results to introduce some basic hypotheses about how economic structural changes may affect the evolution of cancer incidence. We emphasise that this is not a study about social and economic factors causing cancerous diseases, and the paper does not provide a complete account of the role of economic growth on cancer frequency. We simply highlights some basic empirical regularities and theoretical insights to be considered for further research, in order to start developing an economic theory of cancer incidence.

The remainder of the paper is organized as follows. Section 2 briefly introduces the essential measures of cancer frequency. Section 3 summarizes some basic concepts of cancer aetiology. Sections 4 and 5 are

*Address correspondence to this author at the Department of Business and Economics, Richmond The American International University in London, Richmond Hill Campus, Queen's Road, Richmond-upon-Thames, Surrey, TW10 6JP, England; Tel: 02073688602; Mobile: 07919833754; E-mail: bryan.mcintosh@richmond.ac.uk

devoted to quantitative analysis. Section 6 contains a sketch of a theory of cancer incidence, within a simple structural economic dynamics framework. Finally, various objections can be raised to this work, and we discuss many of them in Section 7, that concludes the paper.

2. CANCER EPIDEMIOLOGY: BRIEF CONCEPTS

Cancer epidemiology studies the distribution and determinants of cancerous diseases in specified populations, and applies this knowledge to prevent and control cancer-related public health problems. Quantifying cancers occurrence in a given population is therefore an essential step in epidemiological studies [7].

In order to describe and measure cancers frequency, epidemiology utilises, among others, three mains indicators: incidence, prevalence and mortality. Incidence and mortality are flow variables. They indicate the number of new cancer cases and the number of deaths due to cancer, respectively, which occur in a specific population, over a given period (usually 1 year). Prevalence is a stock variable. It indicates the number of cancer cases in a specific population at a given point in time (such as at the end of a given year). As in other stock–flow relationships, incidence, mortality and prevalence are closely related. Specifically, for a given average duration of the disease, prevalence is a function of incidence and mortality [8].

Data on incidence, prevalence and mortality are usually expressed as absolute numbers or as rates. Rates can be crude or age-standardised. A crude rate (*cr*) is calculated by dividing the absolute number of new cases, cases or deaths by the corresponding number of people in the population-at-risk. On the other hand, an age-standardised rate (*sr*) is a weighted average of the age-specific crude rates, where the weights are the proportion of people in the corresponding age groups of a specific standard population. Since cancer is not a single disease, but a collection of diverse yet related diseases, the population-at-risk is a subset of the total population under study (usually defined by sex and age) that include only the people who are potentially susceptible to develop one or the group of cancerous disease under consideration. The age-adjusted rates are calculated to allow comparison between populations with different age structures, and they are particularly useful in making international comparisons. In this

case, the most frequently used standard population is the world standard population [9] and the results are usually presented as annual rates per 100,000 persons-at-risk [10].

Where raw data are regularly collected by local cancer registry, these basic measures of cancer frequency can be computed for each type of cancers, usually classified according to the International Classification of Diseases (ICD), or for all cancerous diseases as a whole [11]. In this latter case, epidemiologists usually refer to the overall prevalence rate as a measure of society's cancer burden. In the same way, since incidence is regarded as a useful approximation to the average risk of developing any type of cancer, the overall incidence rate is considered as an index of the level of cancer risk factors that exist in a given society, during a given period. Finally, the overall mortality rate provides an approximation to the average risk of dying from some type of cancer.

3. CANCER RISK FACTORS AND CANCER INCIDENCE

The term cancer refers to a broad group of diseases in which normal cells of a specific tissue change and start to do not function properly. In particular, mutated cells do not respond to regular cell cycle control signals and begin to grow and divide in an uncontrolled way. This population of abnormal cells is able to invade and destroy other nearby tissues and also to spread to other parts of the body, causing severe illness and death.

Although all cancerous diseases begin in cells, with some kind of damage in genetic material, there is no one single factor to cause an healthy cell to become cancerous. Cancer is likely to be influenced by many variables. Different types of cancer usually share some basic causes, and at the same time each type of cancer has its own specific determinants. The transformation from a normal cell into a cancer cell is indeed a multistage and complex process. According to a large literature on cancer aetiology, however, this process is the result of the interaction between the inborn genetic characteristics of each individual and numerous external causes, that can be gathered and classified into three main categories: biological, chemical and physical carcinogens agents [12].

Genetic characteristics, along with external carcinogens agents, determine a set of cancer risk factors. A cancer risk factor is anything that may

increase an individual probability of developing some type of cancer. A risk factor itself does not necessarily cause the disease. Nevertheless, the frequency of cancers in a specific population is associated, *ceteris paribus*, with the intensity and the duration of people's exposures to one or more risk factors.

Specifically, the subset of the external carcinogens agents is strictly related to the general environmental and socio-economic conditions, as well as population habits and customs. Epidemiological studies suggest a long list of behaviours and situations associated with an increased cancer incidence. Tobacco and excessive alcohol consumption, qualitative and quantitative unhealthy nutrition, chemical contamination of food, air and water, lack of physical activity, unprotected exposure to ultraviolet and ionizing radiation, and chronic infection from some viruses are the main factors able to play an important role in causing cancers [13].

By affecting the individual chance to become ill, all non-congenital cancer risk factors, taken as a whole, are a leading force that contributes to determine the overall rate of cancer incidence in a given population. But, a distinctive feature of these external cancer risk factors is that, at least partially, they are avoidable. Each combination of behaviours and situations associated with a low or a high risk to developing any type of cancer, reflects a given healthy or unhealthy lifestyle. Therefore, the population exposure to cancer risk factors changes when people modify their habits and customs, both directly *via* individual choices (such as variations in dietary components and eating patterns) and/or indirectly by means of collective choices (such as changes in regulation of environmental pollution and workplace conditions).

4. METHODS AND DATA

4.1. An Engel Function for Cancer Incidence

Abstracting from the complexity of the causal interactions between different carcinogens agents and the process of cancer initiation and progression, at a macroeconomic level the relationship between cancer incidence and lifestyle-related factors may be described by a simple and single equation model, like:

$$isr = f(q; \alpha) \tag{1}$$

where the age-standardized rate of incidence for all type of cancers (*isr*) in a given population depends on the people's exposure to external cancer risk factors

(*q*), for a stated level of not avoidable agents due to individuals' genetic characteristics (α).

In equation 1), *q* is a catchall variable that stands for all the behaviours and situations that characterize people habits and customs and it serves as a proxy for measuring the average population exposure to lifestyle cancer risk factors. One may think at *q* as a bundle of goods (such as foods) and/or bads (such as environmental pollutions), in which each item is described by the set of its healthy related attributes [14]. For instance, the safety and nutritional characteristics of foods that reflect a poor or a healthy eating habits. The whole set of these attributes determines a more or a less cancer risk prone lifestyle.

In the short run, changes in relative prices may have some influence on *q*, but its main composition is likely to be about constant. On the other hand, in the long run the average population exposure to external cancer risk factors tends to undergo dramatic structural changes. In particular, as real per capita income increases there are successive income threshold levels where people shift their behaviours and start following a new lifestyle [15]. In each stage of development, the population consumption pattern follows a hierarchy of needs and wants (determined by many biological, cultural and social factors), so that as the average income rises, increases in consumption tends to concentrate on a particular group of goods with specific characteristics, and this group change, sometimes gradually and sometimes abruptly, from one level of real per capita income to another [16].

This is a well-known generalisation of the so-called 'Engel's law' [17]. It simply states that the proportion of income spent on each type of goods changes as real average income increases, because people modify their preferences, by means of both individual and collective choices, along a distinct hierarchy of needs[1] [18]. Put differently, the science and technology evolution that goes with economic growth, along with an increasing average purchasing power, deeply modifies people habits and customs. These changes in lifestyles causing transformations of the set of

---

[1]In a narrow meaning, an Engel's curve 'is the function describing how a consumer's expenditures on some good or service relates to the consumer's total resources, holding prices fixed, so $q_i = g_i(y, z)$, where q_i is the quantity consumed of good *i*, *y* is income, wealth, or total expenditures on goods and services, and *z* is a vector of other characteristics of the consumer, such as age and household composition' [18]. In Engel's function, *q* may measure the physical quantity consumed, or typically the aggregate expenditure for a group of goods or services.

Table 1:   Variables and Descriptive Statistics

Description	Variable	Mean	Std. Dev.	Min	Max
Gross National Income Per Capita	*y*	11,874.96	13,205.43	286.66	64,943.33
Both sex, Incidence Crude Rate	*icr*	192.6	161.4	44.6	589.7
Both sex, Incidence Age-Standardized Rate	*isr*	162.9	67.2	72.2	326.1
Male, Incidence Crude Rate	*micr*	188.2	143.7	45.4	590.6
Male, Incidence Age-Standardized Rate	*misr*	155.8	55.7	69.3	325.3
Female, Incidence Crude Rate	*ficr*	200.9	183.0	33.4	648.0
Female, Incidence Age-Standardized Rate	*fisr*	175.8	86.4	68.6	360.6

Gross National Income per capita in PPP, current international $.
Three years average (2006, 2007 and 2008). Source: World Development Indicators, World Bank.
Crude and Age-Standardized rates per 100.000 persons. All ages and all types of cancers.
Estimated cancer incidence for 2008 (all sites, but non melanoma skin: C00-C96, but C44).
Source: Globocan 2.0 - International Agency for Research on Cancer, World Health Organization.

attributes that enter the bundle of health-related goods (and bads) faced by the population and therefore they have a strong effects on population health-related consumption patterns.

An aggregate Engel's function, in which the average people exposure to external cancer risk factors (*q*) depends on the population real average income (*y*):

$$q = g(y) \tag{2}$$

albeit very simple, may be a useful tool to capture the influences of economic growth on cancer incidence. In effect, replacing *q* in equation 1) by its expression from equation 2), gives:

$$isr = f[g(y); \alpha] \tag{3}$$

a relationship between real per capita income and the age-standardized rate of incidence, for a given level of the not avoidable cancer risk factors.

4.2. Data on Cancer Incidence and Per Capita Income

This paper focuses on the influence of economic growth on cancer incidence. In particular, we test the relationship between real per capita income and the overall rate of cancer incidence with a cross-sectional analysis, using data from the World Bank and the World Health Organization (WHO) databases, for 165 countries in 2008.

Specifically, real per capita income (*y*) is measured by the ratio of *GNI* to population and it is expressed in current international dollars, using purchasing power parity (*PPP*) exchange rates [19]. While, cancer incidence (*isr*) is measured by the age-standardised

rate of all types of cancer – 'all sites, but non melanoma skin', according to the International Classification of Diseases – provided by the International Agency for Research on Cancer (IARC) within the Globocan project [20].

Especially in low and middle income countries, economic growth leads to remarkable rises in the average life expectation at birth. In the same countries, furthermore, changes in lifestyle due to an increasing purchasing power may be different between female and male population. Both, life expectation at birth and gender affect cancer incidence. Thus, even thought our analysis is at aggregate level, it is useful to measure cancer incidence with the crude rate (*icr*) together with *isr*, and also with both crude and age standardised rates computed separately for male (*micr* and *misr*) and female (*ficr* and *fisr*) population. Table **1** contains a short description and some basic descriptive statistics of all variables (the full database is available from the authors)[2].

5. RESULTS

5.1. International Evidence of Income Elasticity of Cancer Incidence

The simple scatter plots depicted in Figure **1**, where variables are measured in natural logarithms, show for each country the pairs of observations on per capita income and the crude (Figure **1a**) and age-standardised rates (Figure **1b**) of cancer incidence in

---

[2]The complete database contains 169 observation. However, there are four very small oil countries (namely, Brunei Darussalam, Kuwait, Qatar and United Arab Emirates) that both visual inspection and influence statistics quite clearly indicate as outlier.
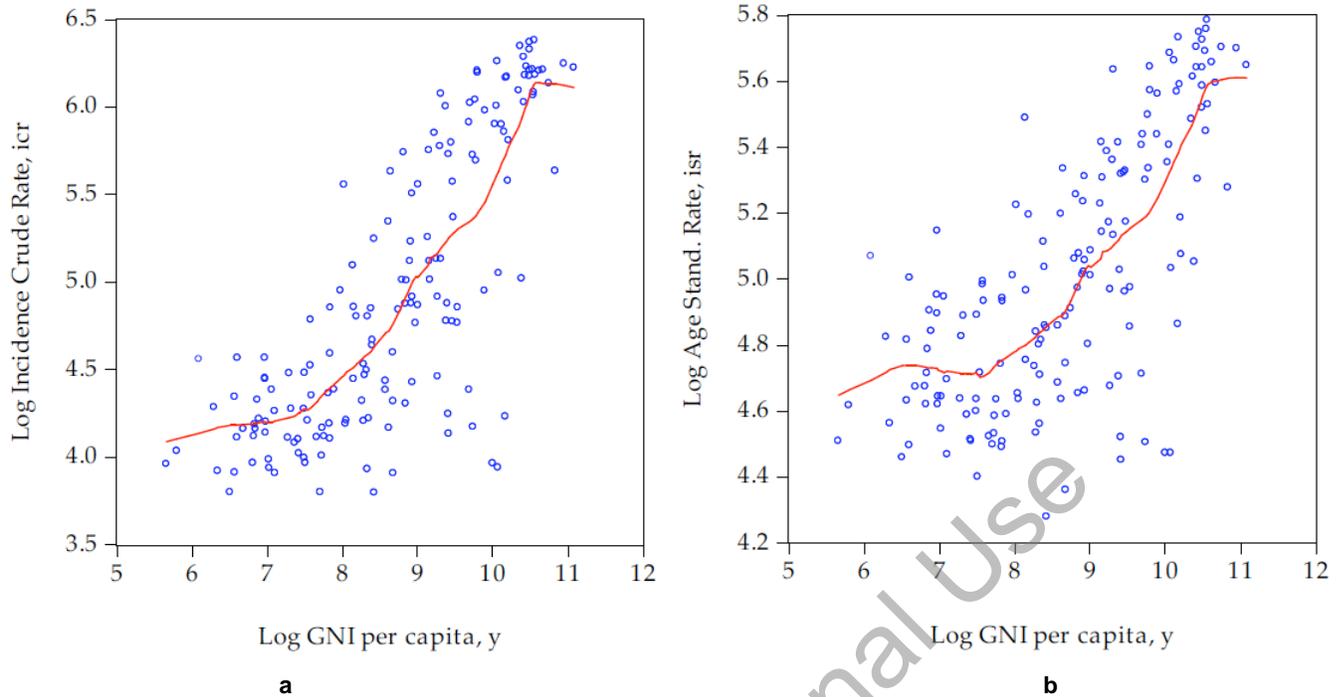
Figures 1: a and **b**. GNI per capita and cancer incidence, in both sex (smooth line indicates the Nearest Neighbor Fit, Lowess function, span = 0.3).

the whole (female and male) population. Both graphs seem to suggest a strong influence of real per capita income on the average risk of developing some type of cancer. Indeed, the correlation coefficients between $\log(y)$ and $\log(icr)$ or $\log(isr)$ are, respectively, 0.78 and 0.69 (Table **1A** in Appendix). The same pattern of relationship, with only slightly differences, appears if one plots both the crude and the age standardised rates against real per capita income, but separately for male and female populations, as shown in Figure **1A** in Appendix.

A straightforward procedure for quantifying the 'sensitivity' of cancer incidence with respect to the process of economic growth, is to estimates a double-log model, with the log of incidence rate as dependent variable and the log of per capita income as explanatory variable, that is: $\log(ir_i) = \beta_0 + \beta_1 \times \log(y_i)$. Indeed, in the double-log model the estimated slope parameter (β_1) is itself a coefficient of elasticity (η), that measures the relative change in the dependent variable for a given relative change in the explanatory variable [21].

A constant elasticity function, however, is not able to capture the complex interactions between economic growth and the population health conditions. Both, the direction and the extent of the influence of the growth process on the people exposure to external cancer risk

factors are likely to be remarkable different at different stages of social and economic development. In order to model the full range of possible influences, it is preferable to utilize a more flexible specification, such as a combined logarithmic and polynomial functional form, as follows:

$$\log(ir_i) = \beta_0 + \beta_1 \times \log(y_i) + \beta_2 \times [\log(y_i)]^2 + \varepsilon_i \qquad (3)$$

where ε is the stochastic error term. This is a double-log quadratic regression model that allows a non constant elasticity. Specifically, the income elasticity of cancer incidence, η_{IR}:

$$\eta_{IR} = (dir/dy) \times (y/ir) = \beta_1 + 2 \times \beta_2 \times \log(y_i) \qquad (4)$$

may be either negative or positive, and in turn when η_{IR} has a positive sign it may be less or greater than one [22].

An attempt to develop a quantitative assessment of the influences of economic growth on cancer incidence, using model in equation 3), is summarised in Table **2**. Although, the use of natural logs contributes to moderate potential problems due to heteroskedasticity, all equations are estimated using the White's coefficient covariance matrix in order to obtain heteroskedasticity robust standard errors [23]. The goodness of fit is fairly high in all equations. Movements in real per capita income are able to

Table 2: Regression Results

Dependent variable	Regressor	Coefficient	Std. Error	*t*-Statistic	Prob.	Adj. *R*-sq.	*n*
Log(*icr*)	Constant	7.375	1.155	6.384	<0.0001	0.65	165
	Log(*y*)	-1.104	0.279	-3.949	0.0001		
	[Log(*y*)]^2	0.093	0.016	5.623	<0.0001		
Log(*isr*)	Constant	7.267	0.764	9.514	<0.0001	0.54	165
	Log(*y*)	-0.767	0.181	-4.228	<0.0001		
	[Log(*y*)]^2	0.057	0.010	5.421	<0.0001		
Log(*ficr*)	Constant	7.643	1.072	7.129	<0.0001	0.64	165
	Log(*y*)	-1.105	0.259	-4.265	<0.0001		
	[Log(*y*)]^2	0.089	0.015	5.892	<0.0001		
Log(*fisr*)	Constant	7.679	0.756	10.156	<0.0001	0.47	165
	Log(*y*)	-0.822	0.179	-4.586	<0.0001		
	[Log(*y*)]^2	0.058	0.010	5.545	<0.0001		
Log(*micr*)	Constant	6.884	1.306	5.271	<0.0001	0.66	165
	Log(*y*)	-1.068	0.315	-3.386	0.0009		
	[Log(*y*)]^2	0.095	0.018	5.087	<0.0001		
Log(*misr*)	Constant	6.689	0.839	7.964	<0.0001	0.56	165
	Log(*y*)	-0.679	0.199	-3.399	0.0008		
	[Log(*y*)]^2	0.055	0.011	4.760	<0.0001		

explain about half of the variations in the age-standardised rates and nearly two thirds of the variations in the crude rates of cancer incidence. All estimated regression coefficients are strongly statistically significant (*p*-values are always less than 0.01). Moreover, the decomposition of total population by sex does not alter the main outcomes. There is only a slight reduction in the goodness of fit for the regression using the age-standardised rate within the female population.

These results confirm the intuitive finding from the visual inspection of Figure **1**. The process of economic growth plays a crucial role on the determination of the rates of cancer incidence. More specifically, the significance of coefficient β_2 in all regressions indicates that the elasticity of cancer incidence with respect to income is not likely to be constant as development proceeds and the real average income rises. The estimated regression coefficients β_1 and β_2, along with equation 4), allow us to compute the income elasticity of cancer incidence: that is, the percentage change in the rate of cancer incidence when real per capita income changes by 1 percent. The results of these calculations are collected in Table **3**, where countries

Table 3: Elasticities of Cancer Incidence and Development Stage*

	Low income GNI per capita ($1,025 or less)	Lower middle income GNI per capita ($1,026 to $4,035)	Average	Upper middle income GNI per capita ($4,036 to $12,475)	High income GNI per capita ($12,476 or more)	Average
icr	0.127	0.326	0.226	0.545	0.780	0.663
isr	-0.008	0.115	0.053	0.250	0.395	0.323
ficr	0.087	0.279	0.183	0.492	0.719	0.605
fisr	-0.056	0.068	0.006	0.205	0.351	0.278
micr	0.129	0.326	0.227	0.545	0.780	0.663
misr	0.058	0.117	0.117	0.308	0.449	0.378

*World Bank's classification by GNI per capita (World Bank, 2012).

are classified in four main groups, according to the World Bank ranking of economies by their *GNI* per capita [24].

All the elasticities of the crude rates are positive but less than one. They are, on average, around 0.2 in the low and middle income countries and about 0.6 in the more developed economies. Furthermore, it is worthy to notice that coefficients concerning the age-standardised rates are about a half and one third of those computed for the crude rates in richer and poorer countries, respectively. In words, there are both a *gross* and a *net* effect of economic growth on cancer incidence. The former includes the positive influence of an increasing real per capita income on the average duration of life, while the latter measures the reactivity of cancer incidence to economic growth due only to changes in health related population lifestyles. Figure **2** provides an idea of the evolution of both *gross* and *net* effects of economic growth on cancer incidence in the total population at different development stages.

Only the income elasticity of the age-standardised rates (η_{ISR}) are a correct measure of the magnitude of the influence of economic growth on population exposure to external cancer risk factors. These elasticity coefficients are both negative and positive. Negative, or around zero, values of η_{ISR} are found in low and lower middle income countries. The reactivity of cancer incidence to per capita income increases in richer countries, however it remains rather inelastic,

around 0.25 and 0.32 in upper middle and high income societies, respectively. Finally, an interesting result is the difference between η_{ISR} in male and female population, especially in the poorer countries.

5.2. The Delay Between Onset and Exposure and the Inter-Country Variability

In brief, cancer incidence depends on the population exposure to external cancer risk factors which, in turn, depends on the level of development, *ceteris paribus*. Changes in income, therefore, lead to changes in lifestyle, and thus to changes in new cancer cases.

But, as in other non-communicable diseases (like, for example, the cardio-vascular diseases), there is a delay between the illness onset and the exposure to risk factors, that is 'today's incidence rate is affected by yesterday's exposure, and today's exposure will affect tomorrow's incidence rate'. To capture this temporal lag, we rewrite the econometric model as follows:

$$isr_t = f\left[g(y_{t-n});\ \alpha\right] \rightarrow \log(isr_{it}) = \beta_0 + \beta_1 \times \log(y_{it-n}) + \beta_2 \times [\log(y_{it-n})]^2 + \varepsilon_i \tag{4}$$

where isr_t is the rate of cancer incidence in year *t* and y_{t-n} is the per capita income *n* years before *t*. We estimate equation 4), using data on *y* in 1990 (before 1990 the sample become too small, and strongly biased towards the developed countries).
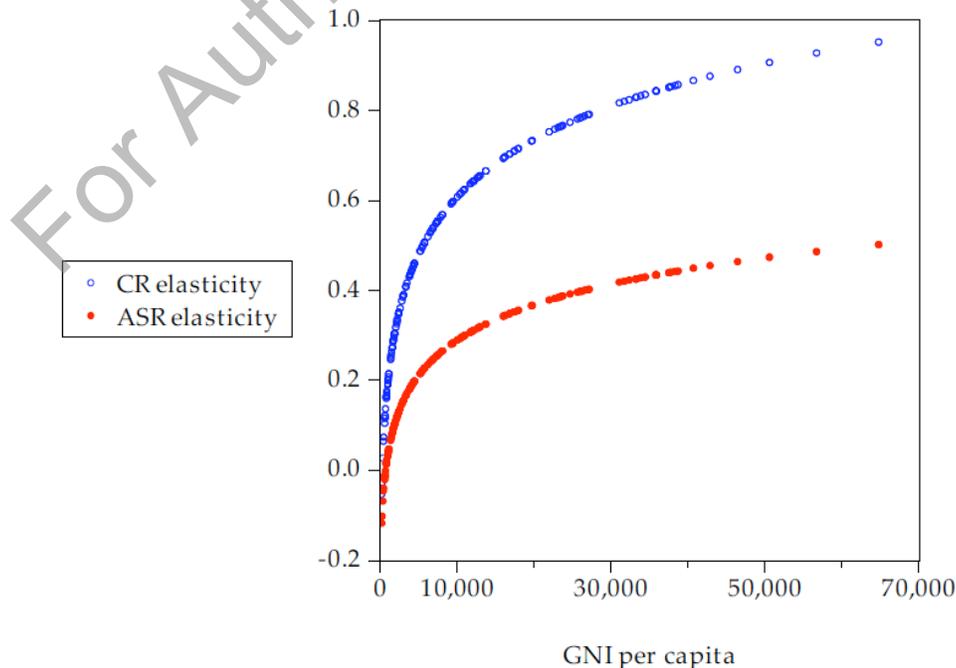
Figure 2: Income elasticity of CR and ASR, in both sex.

Table 4: Regression Results with 1990 GNI Per Capita

Dependent variable	Regressor	Coefficient	Std. Error	*t*-Statistic	Prob.	Adj. *R*-sq.	*n*
Log(*isr*)	Constant	7.318	0.903	8.104	<0.0001	0.55	140
	Log(y_{90})	-0.828	0.230	-3.591	0.0005		
	[Log(y_{90})]^2	0.066	0.014	4.583	<0.0001		
Log(*fisr*)	Constant	8.011	0.874	9.165	<0.0001	0.49	140
	Log(y_{90})	-0.957	0.224	-4.279	<0.0001		
	[Log(y_{90})]^2	0.070	0.014	5.065	<0.0001		
Log(*misr*)	Constant	6.394	1.042	6.131	<0.0001	0.58	140
	Log(y_{90})	-0.649	0.265	-2.450	0.0155		
	[Log(y_{90})]^2	0.058	0.016	3.570	<0.0001		

The temporal lag effect is a crucial issue in every study that examines the relation between the exposure to particular external risk factor and the onset of a specific type of cancer (for instance, between tobacco consumption and the insurgence of lung cancer), with time series data. Nevertheless, in our macroeconomic analysis that relates cancer incidence to per capita income, using cross sectional data, the delay between *y* and *isr* tends to show an important quantitative role, but only a minor effect on the characteristics of the relationship. In Table **4** are collected regression results for the age-standardised rates as dependent variables and the *GNI* per capita in 1990 as explanatory variable. The sample now includes 140 observations (all countries of the full database for which the World Bank provides data on *y* measured in *PPP* terms for 1990). A list of countries included in the two samples are compiled in Table **2A** in Appendix. There are no significant differences between output of regressions with lagged and non-lagged income. On average, the goodness of fit is now slightly higher, and all coefficients remains strongly significant (except for the linear component in equation for the male population).

However, the delay between average people exposure and the illness onset affects the income elasticity of cancer incidence, as shown in Table **5**. On average, all coefficients are now slightly greater than those with non-lagged *y*. But, the main characteristics of the relationship still remain the same. Anyway, the differences between η_{IR} computed with y_{t-n} and *y* are a useful indirect measure of the importance of temporal lag effect of economic growth on cancer incidence.

Finally, in our worldwide samples there is great variability. Countries differ not only in terms of their income per capita but also, and perhaps mainly, in

ethnic, cultural and other socio-economic characteristics. A basic strategy to deal with this problem is the use of one or more dummy variables. We make a first attempt to reduce inter-country variability by creating a new variable, *w*. In particular, *w* is an intercept dummy variable, that assumes value 1 if the country is characterised by a 'western lifestyle', and 0 otherwise[3]. Regression results, using *GNI* per capita in 1990, and with a simplified double-log model, are collected in Table **6**. The variable *w* is highly significant in all equations. Thus, the relation between income per capita and cancer incidence shifts upward when *w* equals 1. This evidence allow us to make a distinction between a movement along the '*y-ir* curve', and a shift in the '*y-ir* curve'. The former is due to a change (that is, an increase) in per capita income, *ceteris paribus*. On the other hand, changes in variables included in the set of the cancer risk factors cause an upward (or a downward) shift in the aggregate Engel's function between *y* and *ir*.

6. DISCUSSION

6.1. On the Macroeconomic Determinants of Cancer Incidence

In modern theories of economic growth, technical change has a key role in explaining the determinants of population's standard of living [25]. In particular, when inventions and innovations relax and change the technological constraints, the economic system undergoes a complex process of transformational growth [26]. More specifically, on the one side, technical change means a flow of both new production

---

[3]We include in this subset all European countries, plus Australia, Canada, New Zealand and the United States of America.

Table 5:   **Elasticities of Cancer Incidence and Development Stage in 1990**

	Low income GNI per capita ($1,025 or less)	Lower middle income GNI per capita ($1,026 to $4,035)	Average	Upper middle income GNI per capita ($4,036 to $12,475)	High income GNI per capita ($12,476 or more)	Average
isr	0.018	0.173	0.095	0.324	0.463	0.393
fisr	-0.049	0.117	0.034	0.280	0.429	0.354
misr	0.104	0.242	0.173	0.377	0.500	0.438

Table 6:   **Regression Results with Dummy Variable (1990 GNI Per Capita)**

Dependent variable	Regressor	Coefficient	Std. Error	*t*-Statistic	Prob.	Adj. *R*-sq.	*n*
Log(*isr*)	Constant	3.804	0.180	21.11	<0.0001	0.63	140
	Log(y_{90})	0.138	0.024	5.608	<0.0001		
	w	0.423	0.063	6.706	<0.0001		
Log(*fisr*)	Constant	4.113	0.165	24.84	<0.0001	0.54	140
	Log(y_{90})	0.098	0.022	4.373	<0.0001		
	w	0.370	0.060	6.156	<0.0001		
Log(*misr*)	Constant	3.433	0.209	16.35	<0.0001	0.67	140
	Log(y_{90})	0.186	0.028	6.518	<0.0001		
	w	0.485	0.071	6.749	<0.0001		

techniques and new (or better) goods available to producers and consumers. On the other side, it means an increasing productivity of resources and therefore a higher and higher amount of wages and profits that goes to workers and capitalists or, more generally, an increasing trend in real average income [27].

Technical change, and therefore economic growth, affects cancer incidence, prevalence and mortality in various ways. In particular, at macroeconomic level, changes in cancer frequency are primarily due to some relevant structural changes operating on the supply and demand sides of the economy, respectively (as shown in Figure **3**).

Let us first consider the production effects. As the growth process progresses, better medical and surgical treatments, and notably, better techniques for early diagnosis become available (and usually affordable) to a large proportion of population. These medical improvements are able to dramatically reduce the mortality of cancers. This is why in each society, other things being equal, for a given incidence rate economic growth implies a notable increase in prevalence rates. However the supply-side influences of economic growth may also be negative. In fact, the new products and production processes discovered in the past

sometimes reveal harmful effects, and therefore affects today incidence rates.

Let us now consider the consumption effects. We denote with q_{LOW} and q_{HIGH} two specific combinations of bads and goods that reflects a lifestyle characterised by a low and a high risk of developing any type of cancer, respectively. Specifically, q_{LOW} indicates a set of behaviours and situations associated with a minumum level of the average population exposure to the external cancer risk factors, and *vice versa* for q_{HIGH}. It seems reasonable to think at q_{LOW} (that is, to think at 'an anti-cancer lifestyle') as a sort of luxury good, with an income elasticity coefficient greater than one, and at q_{HIGH} as a normal (or inferior) good, that is a good with an income elasticity positive, but always less than one (or negative, in the case of inferior good)[4].

In general, because of the existence of a hierarchy of needs, one observes that the demand for a luxury good, at aggregate level, tends to remain weak until

---

[4]Epidemiological data on tobacco and alcohol consumption, for example, seems to support this hypothesis, both are necessities (or inferior) goods in most of the developed countries and luxuries in a majority of developing countries [28-29].
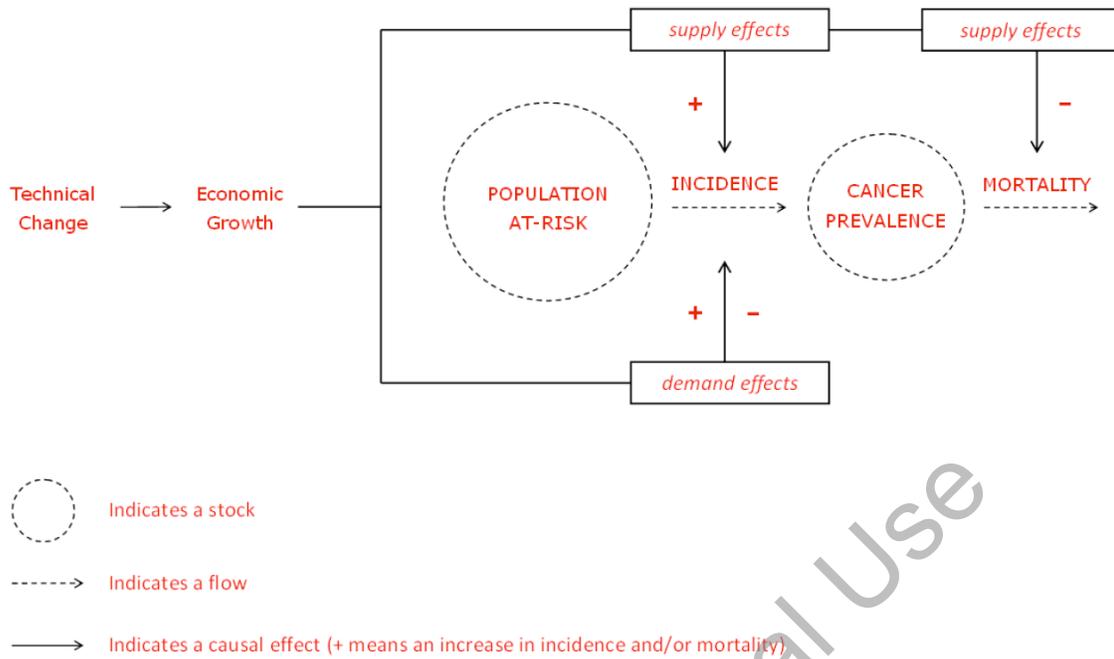
Figure 3: Negative and positive effects of economic growth on cancer incidence and mortality.

real average income reaches a threshold critical level, and after that it starts to increasing sharply. By affecting the demand for q_{LOW} and q_{HIGH}, economic growth modifies the average composition of q, positively (*i.e.*, towards q_{LOW}) or negatively (*i.e.*, towards q_{HIGH}) and in turn it changes the average population exposure to external cancer risk factors.

At less developed stages of social and economic conditions the process of growth usually pushes populations towards an unhealthy 'western lifestyle', such as smoking and consumption of calorie-dense food. Furthermore, in these circumstance economic growth is often driven by an industrialization process based upon high polluting production methods, that typically take place in unsafe and harmful working environment. As growth progresses and the average income overcomes a threshold level, changes in both individual and collective preferences lead to an increase in demand for an healthy lifestyle. As a result, the effects of economic growth on cancer incidence gradually turn from negative to positive. Finally, all these complex supply and demand side influences on incidence and mortally ends in determining the extent of cancer prevalence.

6.2. A *J*-Curve Hypothesis

At theoretical level, some kind of *J*-Curve is a possible general model to represents, other things being equal, how economic growth influence cancer incidence in a given homogeneous population. This complex relationship may be captured by some basic hypotheses, as illustrated in Figure **4**, where cancer incidence is measured by the age-standardised rate of all types of cancer (*isr*) at time *t* and economic growth is measured by the real per capita income (*y*) at time t_n. At very low income levels, there is often a high incidence of cancers related to some biological (*i.e.*, infectious) agents. Until y_1, the positive effects of economic growth on general hygiene and sanitary conditions lead to a decrease in the future overall rate of cancer incidence. However, there will be a threshold minimum level that measures the autonomous component of the incidence rate (that is, isr_{MIN} is independent of income, because it is weakly influenced by exposure to external risk factors, such as in the type of cancers with an important genetic aetiology).

Beyond y_1, cancer incidence will rise with economic growth. More specifically, there is an early range of development stages (from y_1 to y^*) in which increases in real per capita income have a more-than-proportional, negative, effect on the overall rate of cancer incidence. When average income became greater than y^* as a result of the expansion of demand for the anti-cancer lifestyle this more-than-proportional relationship tends to disappear. Cancer incidence will continue to rise, but less than proportional with respect to *y*. Economic growth returns to exert a positive effect on population exposure to external cancer risk factors only after y^{**}, where *isr* reaches his maximum. Finally, when the development stage pass this threshold level,
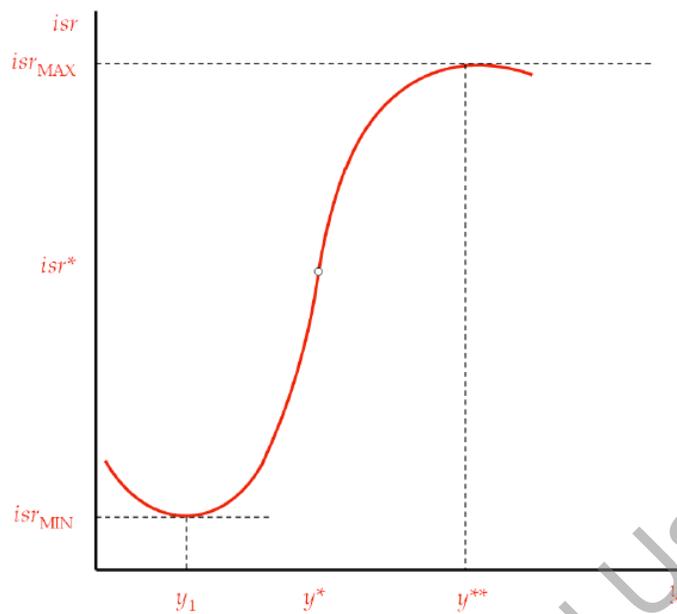
Figure 4: A *J*-Curve model for per capita income and cancer incidence in a given population (*isr* is measured at time *t*, and *y* is measured at time t_n).

the overall rate of age-standardized cancer incidence might start decreasing.

7. CONCLUSIONS

This paper simply highlights some basic empirical regularities and theoretical insights that may be useful in developing an economic theory of the evolution of cancer incidence in a growing economy. Measuring and describing the relationship between cancer incidence and per capita income, however, constitutes only a first step in understanding how the process of economic growth may affect a population's exposure to cancer causing factors.

Furthermore, a number of important limitations need to be considered. First, incidence data are usually derived from population-based cancer registries, and thus there is a problem of data reliability in poorest countries, where the low level of development makes the information collection process more complicated. Second, there are well-known detection biases that make cancer more likely to appear incident in countries with an efficient health system. As a result, in developing countries the income elasticity of cancer incidence may be higher than our estimates. Third, the relation between economic growth and cancer incidence should be investigated through longitudinal studies. Long-run data on cancer frequency, however, are available only for a small set of high developed countries. Fourth, in our study we use a polynomial models, because the aim of the paper is simply to collect some 'stylized facts' about cancer incidence and

economic growth. But, in order to forecast the impact of economic growth on cancer incidence this model may not be flexible enough, and some nonparametric models could work better.

Finally, per capita income is not an accurate and adequate measure of a country's level of development, and it is not possible to summarize with *y* a set of ethnical, cultural, economic, social and health features. Further research is needed to include more variables (as, for example, those referring to personal income distribution, cultural habits and customs, general sanitary conditions and health policies). It would be also necessary to utilise disaggregated epidemiological data for single type of cancer, and within more homogeneous genetic populations.

AUTHORS' CONTRIBUTION

Fabrizio Ferretti conceived of the study, and carried out the econometric analysis. Simon Jones designed the structure of the study, participated in its coordination, and reviewed the findings. Brian McIntosh designed the structure of the study, participated in its coordination, reviewed the findings, and helped to draft the manuscript.

APPENDIX

Table 1A: Correlation Coefficients Table

	Log(*icr*)	Log(*isr*)	Log(*micr*)	Log(*misr*)	Log(*ficr*)	Log(*fisr*)
Log(*y*)	0.78	0.69	0.79	0.73	0.77	0.63
	(16.15)	(12.48)	(16.78)	(13.74)	(15.56)	(10.45)
p-value	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

Note: *t*-statistics in parentheses.
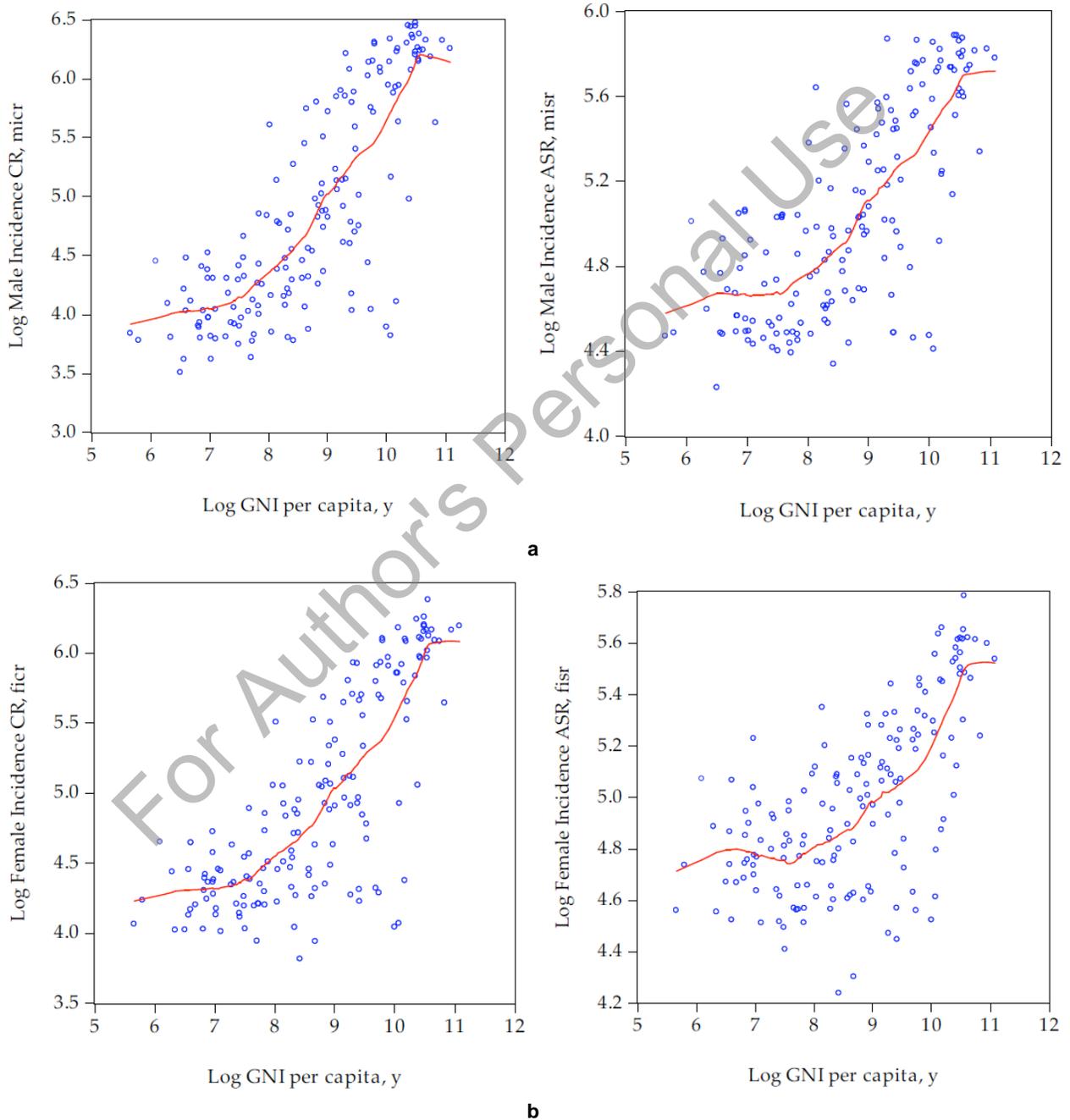
Figure 1A: a. GNI per capita and cancer incidence, in male population (smooth line indicates the Nearest Neighbor Fit, Lowess function, span = 0.3).

b. GNI per capita and cancer incidence, in female population (smooth line indicates the Nearest Neighbor Fit, Lowess function, span = 0.3).

Table 2A: List of Countries

Sample 165 countries
Afghanistan, Albania, Algeria, Angola, Argentina, Armenia, Australia, Austria, Azerbaijan, Bahamas The, Bahrain, Bangladesh, Barbados, Belarus, Belgium, Belize, Benin, Bhutan, Bolivia, Bosnia and Herzegovina,, Botswana, Brazil, Bulgaria, Burkina Faso, Burundi, Cambodia, Cameroon, Canada, Cape Verde, Central African Republic, Chad, Chile, China, Colombia, Comoros, Congo, Dem. Rep., Congo, Rep., Costa Rica, Cote d'Ivoire, Croatia, Cyprus, Czech Republic, Denmark, Djibouti, Dominican Republic, Ecuador, Egypt, Arab Rep., El Salvador, Equatorial Guinea, Eritrea, Estonia, Ethiopia, Fiji, Finland, France, Gabon, Gambia The, Georgia, Germany, Ghana, Greece, Guatemala, Guinea, Guinea-Bissau, Guyana, Haiti, Honduras, Hungary, Iceland, India, Indonesia, Iran Islamic Rep., Iraq, Ireland, Israel, Italy, Jamaica, Japan, Jordan, Kazakhstan, Kenya, Korea, Rep., Kyrgyz Republic, Lao PDR, Latvia, Lebanon, Lesotho, Liberia, Libya, Lithuania,, Luxembourg, Macedonia (FYR), Madagascar, Malawi, Malaysia, Maldives, Mali, Malta, Mauritania, Mauritius, Mexico, Moldova, Mongolia, Montenegro, Morocco, Mozambique, Namibia, Nepal, Netherlands, New Zealand, Nicaragua, Niger, Nigeria, Norway, Oman, Pakistan, Panama, Papua New Guinea, Paraguay, Peru, Philippines, Poland, Portugal, Romania, Russian Federation, Rwanda, Samoa, Saudi Arabia, Senegal, Serbia, Sierra Leone, Singapore, Slovak Republic, Slovenia, Solomon Islands, South Africa, Spain, Sri Lanka, Sudan, Suriname, Swaziland, Sweden, Switzerland, Syrian Arab Republic, Tajikistan, Tanzania, Thailand, Timor-Leste, Togo, Trinidad and Tobago, Tunisia, Turkey, Turkmenistan, Uganda, Ukraine, United Kingdom, United States, Uruguay, Uzbekistan, Vanuatu, Venezuela (RB), Vietnam, Yemen Rep., Zambia, Zimbabwe.
Sample 140 countries (GNI per capita 1990)
Albania, Algeria, Angola, Argentina, Armenia, Australia, Austria, Bahamas The, Bangladesh, Barbados, Belarus, Belgium, Belize, Benin, Bhutan, Bolivia, Botswana, Brazil, Bulgaria, Burkina Faso, Burundi, Cameroon, Canada, Cape Verde, Central African Republic, Chad, Chile, China, Colombia, Comoros, Congo, Dem. Rep., Congo, Rep., Costa Rica, Cote d'Ivoire, Cyprus, Denmark, Dominican Republic, Ecuador, Egypt, Arab Rep., El Salvador, Equatorial Guinea, Ethiopia, Fiji, Finland, France, Gabon, Gambia The, Georgia, Germany, Ghana, Greece, Guatemala, Guinea, Guinea-Bissau, Guyana, Honduras, Hungary, Iceland, India, Indonesia, Iran Islamic Rep., Ireland, Israel, Italy, Japan, Jordan, Kenya, Korea, Rep., Kyrgyz Republic, Lao PDR, Latvia, Lebanon, Lesotho, Lithuania, Luxembourg, Macedonia (FYR), Madagascar, Malawi, Malaysia, Mali, Malta, Mauritania, Mauritius, Mexico, Moldova, Mongolia, Morocco, Mozambique, Namibia, Nepal, Netherlands, New Zealand, Nicaragua, Niger, Nigeria, Norway, Pakistan, Panama, Papua New Guinea, Paraguay, Peru, Philippines, Portugal, Romania, Russian Federation, Rwanda, Samoa, Senegal, Sierra Leone, Singapore, Slovak Republic, Solomon Islands, South Africa, Spain, Sri Lanka, Sudan, Suriname, Swaziland, Sweden, Switzerland, Syrian Arab Republic, Tajikistan, Tanzania, Thailand, Togo, Trinidad and Tobago, Tunisia, Turkey, Uganda, Ukraine, United Kingdom, United States, Uruguay, Vanuatu, Venezuela (RB), Vietnam, Yemen Rep., Zambia.

REFERENCES

[1]   Barro JR. Health and economic growth. Ann Econ Financ 2013; 14: 329-66.

[2]   World Health Organization. World health statistics. Geneve: WHO 2013.

[3]   Pritchett L, Summers LH. Wealthier is healthier. J Hum Resour 1996; 31: 841-68.
http://dx.doi.org/10.2307/146149

[4]   Jernal A, Center MM, DeSantis C, Ward EM. Global patterns of cancer incidence an mortality rates and trends**.** Cancer Epidemiol Biomarkers Prev 2010; 19: 1893-907.
http://dx.doi.org/10.1158/1055-9965.EPI-10-0437

[5]   Bray F, Moller B. Predicting the future burden of cancer. Nat Rev Cancer 2006; 6: 63-74.
http://dx.doi.org/10.1038/nrc1781

[6]   Bray F, Jernal A, Gray N, Ferlay J, Forman D. Global cancer transition according to Human development Index (2008-2030): a population-based study. Lancet Oncol 2012; 13: 790-11.
http://dx.doi.org/10.1016/S1470-2045(12)70211-5

[7]   Dos Santos Silva I. Cancer epidemiology. Principles and methods. Geneve: WHO-IARC 1999.

[8]   Bonita R, Beaglehole R, Kejellstrom T. Basic epidemiology. Geneva: WHO 2007.

[9]   World Health Organization. Rates are age-standardized to WHO's world standard population. Geneva: WHO 2001.

[10] Last JM. A dictionary of epidemiology. New York: Oxford University Press 2001.

[11] World Health Organization. International classification of Diseases. Geneve: WHO 2010.

[12] World Health Organization. Cancer facts sheet. Geneve: WHO; 2013 (http://www.who.int/media centre/factsheets/fs297/en/).

[13] Nasca PC, Harris P. Fundamentals of cancer epidemiology. Sudbury: Jones & Bartlett Publishing 2007.

[14] Lancaster KJ. A new approach to consumer theory. J Polit Econ 1966; 2: 132-57.
http://dx.doi.org/10.1086/259131

[15] Argyrous G. Endogenous demand in the theory of transformational growth. In: Setterfield M, Ed. The Economic of demand-led growth. New York: Edward Elgar 2002; pp. 237-250.

[16] Cornwall J. Modern capitalism. Its growth and transformation. London: Martin Robertson 1977.

[17] Engel E. Die lebenskosten belgischer arbeiter-familien fruher and jetzt. Int Statist Instit Bull 1895, 9: 1-74.

[18] Lewbel A. Engel curves. In: Durlauf SN, Blume LE, Eds. The new Palgrave dictionary of economics. London: Palgrave-McMillan 2008; pp. 1-7.
http://dx.doi.org/10.1057/9780230226203.0476

[19] World Bank. World development indicators. Washington: The World Bank Group 2012.

[20] Ferlay J, Shin HR, Bray F, Forman D, Mathers C, Parkin DM. Globocan 2008 v2.0. Cancer incidence and mortality worldwide. Lyon: International Agency for Research 2010.

[21] Baddeley MC, Barrowclough DV. Running Regressions. Cambridge: Cambridge University Press 2009.

[22] Wooldridge JM. Introductory econometrics. Mason: South Western-Cengage Learning 2009.

[23] Starz R. E-Views illustrated, for version 7. Irivine: Quantitative Micro Software 2010.

[24] World Bank. How we classify countries. Washington: The World Bank Group; 2013 (http://data. worldbank.org/about/country-classifications)

[25] Jones CI. Introduction to economic growth. New York: WW Norton & Company 2002.

[26] Nell EJ. The general theory of transformational growth. Cambridge: Cambridge University Press 1998.

[27] Pasinetti LL. Structural change and economic growth. Cambridge: Cambridge University Press 1984.

[28] Selvanathan S, Selvanathan EA. Consumption patterns of food tobacco and beverages: a cross-country analysis. Appl Econ 2006; 38: 1567-84.
http://dx.doi.org/10.1080/00036840500392664

[29] Cutler DM, Glaeser EL. Why do Europeans smoke more than Americans? In: Wise DA, editor. Developments in the economics of aging. Chicago: University of Chicago Press 2009; pp. 255-282.
http://dx.doi.org/10.7208/chicago/9780226903361.003.0008