Dismay and disparities – economic development and cancer incidence

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Abstract: The distribution and determinants of cancerous diseases in specified populations attempts to prevent and control cancer-related public health issues, and is an essential step in epidemiological studies. During economic growth, every society undergoes several substantial structural changes in healthcare demand and supply. In this paper, we discuss the relationship between economic growth and cancer incidences. The purposes is to describe and measure the influence of an increasing per capita income on the overall incidence of cancer. By using worldwide cross-sectional data from 162 countries, regression results with crude and age-standardised rates, allows us to measure the elasticity of cancer incidences with respect to per capita income and to decompose the elasticity coefficient into two components: age-effect and lifestyle-effect. In this article we sketch a macroeconomic theory of cancer incidence. We introduce some basic hypotheses about how demand-side economic structural changes may affect the evolution of cancer incidence. Finally, we try to develop a basic framework in order to explain how economic structural changes on the demand-side can affect the evolution of cancer incidence.

Keywords: cancer incidence; economic growth; structural change; per-capita income; lifestyle effect; age effect.

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1 Introduction

Quantifying cancer occurrences in a given population is therefore an essential step in epidemiological studies. During economic growth, however, every society undergoes several substantial structural changes in healthcare demand and supply. These changes modify both composition and priority of society's health problems. Cancers: "…that once were rare and considered the diseases of western countries … are now frequently diagnosed in less developed or economically transitioning countries and their rates are on the rise" (Jemal et al., 2010).

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 resource allocation in planning health services. In this article, we sketch a macroeconomic theory of cancer incidence. We introduce some basic hypotheses about how demand-side economic structural changes may affect the evolution of cancer incidence. The purpose of this study is to simply highlight some theoretical insights to be considered for further research. This is in order to start developing an economic theory of cancer incidence.

Quality of life is a multidimensional construct with many outcomes of many different variables. Some of these variables, however, show a positive and significant correlation with per capita income, usually measured by the ratio of real gross national income (GNI) to population. This is why the real GNI per capita is often used as the first and basic indicator of standard of living. Health is a fundamental dimension of quality of life. In fact, almost all indices of economic and social well-being contain at least one variable for measuring health conditions. There are some important exceptions, but in the long-run, higher values of real per capita income usually correspond to better hygiene and sanitary conditions. During growth, however, each society undergoes several important changes

in both demand and supply of healthcare. Therefore, the process of economic growth modifies the composition and order of importance of the main population which modifies the hierarchy of health needs within a population.

This paper analyses one of the foremost of these changes with society's health problems and focuses on the relationship between economic growth and cancer incidence. In particular, we explore this relationship at the macroeconomic level, using worldwide cross-sectional data from 162 countries in 2012. First, we attempt to collect some empirical regularity concerning how an increasing real per capita income influences the overall incidence of cancer. Second, we use these results to introduce some basic hypotheses about how the demand-side economic structural changes can affect the evolution of cancer incidence in a given population.

Understanding and measuring how cancer incidence evolves during economic growth can be useful for forecasting the economic impact of cancer and for governing the process of resource allocation in planning health services. However, it is necessary to emphasise that this is not a study about social and economic factors causing cancerous conditions. This paper simply highlights some basic empirical regularities and theoretical insights to be considered for further research in order to develop an economic theory of cancer incidence.

The remainder of the paper is organised as follows. Firstly, an introduction of the three essential measures of cancer frequency. Secondly, a discussion to confirm the value of quantitative analysis. Thirdly, a review of cancer incidence theory within a structural economic dynamic framework. Finally, the paper concludes with some suggestions for further research.

2 Cancer risk factor and cancer incidence

In order to describe and measure the frequency of cancerous diseases, epidemiology utilises three main 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 one year). Prevalence is a stock variable. It indicates the number of cancer cases in a specific population at a given point in time (Last, 2001).

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, current cases or deaths by the corresponding number of people in the population-at-risk. On the other hand, an age-standardised rate (ASR) (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 gender and age) that include only the people who are potentially susceptible to develop one or the group of cancerous diseases under consideration. The age-adjusted rates are calculated to allow comparison between populations with different age structures (results are usually presented as annual rates per 100,000 persons-at-risk) and they are particularly useful in making international comparisons.

Where raw data are regularly collected by local cancer registry, these basic measures of cancer frequency can be computed for each type of cancer, usually classified according to the International Classification of Diseases (ICD) or for all cancerous diseases as a whole (WHO, 2015). In the 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.

The term cancer refers to a broad group of diseases in which normal cells of a specific tissue changes and begin to 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 not one single factor to cause a 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.

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's 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. Furthermore, as in other non-communicable diseases, there is a delay between the illness onset and the exposure to risk factors that is 'today's incidence rate affected by yesterday's exposure and today's exposure affecting tomorrow's incidence rate'.

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 ionising radiation and chronic infection from some viruses are the main factors which play an important role in causing cancers. However, people can still get skin cancer if wearing sun cream even if superficially protected from ionising radiation they are still exposed

By affecting the individual and giving them a chance to acquire this disease, all non-congenital cancer risk factors, taken as a whole, is a leading force that contributes to determine the overall rate of cancer incidence in a given population. A distinctive feature of these external cancer risk factors is that, at least partially, they are avoidable. Each

combination of behaviours and situations are associated with a low or a high risk to developing any type of cancer, reflecting on a given healthy or unhealthy lifestyle. Therefore, the population exposure to cancer risk factors change 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 regulations of environmental pollution and workplace conditions).

3 International evidence

Economic growth can affect cancer incidence, prevalence and mortality in various ways. In this paper we focus on the influence of a long-term increase in real per capita income on the overall incidence rate of all types of cancer.

Table 1 Variables and summary statistics

Description	*Variable*	*Mean*	*Std. dev.*	*Min*	*Max*
World group (n = 162)					
Per capita gross national income	Y	8,166.90	8,937.60	500.00	37,270.00
Total (female and male), incidence crude rate	TCR	183.83	139.25	44.70	507.99
Female, incidence crude rate	FCR	178.45	124.30	58.90	496.40
Female, incidence age-standardised rate	FASR	163.03	53.30	77.90	308.70
Male, incidence crude rate	MCR	190.24	156.56	36.00	585.80
Male, incidence age-standardised rate	MASR	187.67	80.30	64.90	406.60
Western group (n = 41)					
Per capita gross national income	Y	18,515.00	10,325.70	1,620.00	37,270.00
Total (female and male), incidence crude rate	TCR	388.02	85.18	198.09	507.99
Female, incidence crude rate	FCR	359.98	78.71	196.70	496.40
Female, incidence age-standardised rate	FASR	224.04	40.51	160.60	308.70
Male, incidence crude rate	MCR	419.35	98.78	196.00	585.80
Male, incidence age-standardised rate	MASR	290.26	47.89	206.50	406.60

Notes: Y, current international dollars, purchasing power parity (PPP), 2012 (WB, 2014).
CR and ASR, crude and ARSs for all types of cancers: all sites but non-melanoma skin (C00-C96, but C44, International Classification of Diseases – ICD).
Incidence rates per 100,000 persons, 2012 (IARC, 2004).
Countries in the 'Western group': Albania, Austria, Belarus, Belgium, Bosnia and Herzegovina, Bulgaria, Croatia, Czech rep. Denmark, Estonia, Finland, France, Germany, Greece, Hungary, Iceland, Ireland, Italy, Latvia, Lithuania, Malta Netherlands, Norway, Poland, Portugal, Rep. Moldova, Romania, Russian Fed., Serbia and Mont., Slovakia Slovenia, Spain. Sweden, Switzerland, TFYR Macedonia, the UK, Ukraine, Australia, Canada, New Zealand, and the USA.

Table 2 Regression results, World Group

	Dependent variable	*Regressor*	*Coefficient*	*T-ratio*	*Prob.*	*R-bar squared*	*Heteroskedasticy* CHSQ LM version*	*Functional form** CHSQ LM version*	*n*
Total (female and male) crude rate									
1	TCR	Constant	75.52	9.67		0.72	0.920 [0.337]	9.063 [0.003]	162
		Y	0.132	20.52					
2	TCR	Constant	53.13	5.04		0.74	1.184 [0.276]	0.235 [0.627]	162
		Y	0.020	8.70					
		Y^2	–2.27E–07	–3.06					
3	LogTCR	Constant	0.286	2.45	0.01	0.61	0.010 [0.918]	21.66 [0.000]	162
		LogY	0.508	16.09					
Female crude rate									
4	FCR	Constant	81.43	11.79		0.73	1.206 [0.272]	7.730 [0.005]	162
		Y	0.012	20.80					
5	FCR	Constant	63.15	6.74		0.74	1.859 [0.173]	0.035 [0.850]	162
		Y	0.017	8.52					
		Y^2	–1.86E–07	–2.82					
6	LogFCR	Constant	0.463	4.38		0.68	0.145 [0.703]	22.52 [0.000]	162
		LogY	0.462	16.19					
Male crude rate									
7	MCR	Constant	68.76	7.77		0.72	2.610 [0.106]	9.75 [0.002]	162
		Y	0.148	20.33					
8	MCR	Constant	42.48	3.57		0.73	3.000 [0.083]	0.617 [0.432]	162
		Y	0.023	8.78					
		Y^2	–2.67E–07	–3.19					

Notes: *Based on the regression of squared residuals on squared fitted values. **Ramsey's RESET test using the squares of the fitted values.

Table 2 Regression results, World Group (continued)

	Dependent variable	*Regressor*	*Coefficient*	*T-ratio*	*Prob.*	*R-bar squared*	*Heteroskedastic* CHSQ LM version*	*Functional form** CHSQ LM version*	*n*
	Male crude rate								
9	LogMCR	Constant	0.095	0.75	0.45	0.62	0.392 [0.531]	21.17 [0.000]	162
		LogY	0.558	16.21					
	Female age-standardised rate								
10	FASR	Constant	127.46	32.74		0.53	0.013 [0.907]	0.830 [0.362]	162
		Y	0.043	13.52					
11	FASR	Constant	124.08	23.00		0.53	0.005 [0.940]	0.117 [0.732]	162
		Y	0.0054	4.56					
		Y^2	–3.43E–08	–0.90	0.37				
12	LogFASR	Constant	1.515	22.43		0.38	0.607 [0.436]	18.28 [0.000]	162
		LogY	0.184	10.06					
	Male age-standardised rate								
13	MASR	Constant	132.48	23.41		0.56	0.087 [0.768]	10.63 [0.001]	162
		Y	0.0067	14.43					
14	MASR	Constant	114.93	15.13		0.59	0.003 [0.958]	0.465 [0.495]	162
		Y	0.012	7.28					
		Y^2	–1.78E–07	–3.34					
15	LogMASR	Constant	1.232	14.75		0.47	6.039 [0.014]	12.98 [0.000]	162
		LogY	0.273	12.08					

Notes: *Based on the regression of squared residuals on squared fitted values. **Ramsey's RESET test using the squares of the fitted values.

Table 3 Regression results, Western Group

	Dependent variable	*Regressor*	*Coefficient*	*T-ratio*	*Prob.*	*R-bar squared*	*Heteroskedastic*y* CHSQ LM version*	*Functional form** CHSQ LM version*	*n*
Total (female and male) crude rate									
1	TCR	Constant	268.50	15.44		0.60	0.233 [0.629]	6.305 [0.012]	41
		Y	0.0064	7.85					
2	TCR	Constant	208.45	7.44		0.65	0.039 [0.842]	1.976 [0.160]	41
		Y	0.0156	4.36					
		Y^2	–2.48E–07	–2.62	0.01				
3	LogTCR	Constant	1.449	12.48		0.70	0.615 [0.433]	0.103 [0.748]	41
		LogY	0.270	9.75					
Female crude rate									
4	FCR	Constant	246.55	16.06		0.64	0.194 [0.659]	3.942 [0.047]	41
		Y	0.0061	8.43					
5	FCR	Constant	204.61	8.00		0.66	0.043 [0.834]	1.177 [0.278]	41
		Y	0.0120	3.83					
		Y^2	–1.73E–07	–2.01	0.05				
6	LogFCR	Constant	1.438	12.62		0.70	1.116 [0.291]	0.122 [0.727]	41
		LogY	0.265	9.75					
Male crude rate									
7	MCR	Constant	288.21	13.24		0.54	0.028 [0.867]	6.902 [0.009]	41
		Y	0.0071	6.87					
8	MCR	Constant	209.54	6.03		0.60	0.906 [0.341]	1.353 [0.245]	41
		Y	0.0192	4.30					
		Y^2	–3.25E–07	–2.77	0.01				

Notes: *Based on the regression of squared residuals on squared fitted values. **Ramsey's RESET test using the squares of the fitted values.

Table 3 Regression results, Western Group (continued)

	Dependent variable	*Regressor*	*Coefficient*	*T-ratio*	*Prob.*	*R-bar squared*	*Heteroskedasticy* CHSQ LM version*	*Functional form** CHSQ LM version*	*n*
	Male crude rate								
9	LogMCR	Constant	1.440	10.75		0.65	0.112 [0.737]	0.327 [0.567]	41
		LogY	0.280	8.75					
	Female age-standardized rate								
10	FASR	Constant	169.13	19.45		0.56	0.013 [0.907]	0.018 [0.892]	41
		Y	0.0029	7.21					
11	FASR	Constant	170.75	11.21		0.55	0.023 [0.878]	0.030 [0.862]	41
		Y	0.0027	1.39	0.17				
		Y^2	6.71E–09	0.13	0.89				
12	LogFASR	Constant	1.598	13.92		0.51	0.542 [0.462]	3.968 [0.046]	41
		LogY	0.178	6.50					
	Male age-standardized rate								
13	MASR	Constant	239.14	18.95		0.34	0.014 [0.906]	1.253 [0.263]	41
		Y	0.0027	4.62					
14	MASR	Constant	219.72	10.09		0.34	0.219 [0.640]	3.911 [0.048]	41
		Y	0.0057	2.05	0.04				
		Y^2	–8.02E–08	–1.09	0.28				
15	LogMASR	Constant	1.847	16.37		0.41	0.055 [0.814]	0.015 [0.969]	41
		LogY	0.146	5.42					

Notes: *Based on the regression of squared residuals on squared fitted values. **Ramsey's RESET test using the squares of the fitted values.

Using data from the World Bank (WB) and the World Health Organization (WHO), we developed an empirical analysis of the relationship between economic growth and cancer incidence. For this purpose, we use cross-sectional data on per capita income and cancer incidence in 201 from 16 countries including the WB and WHO statistical databases, and for a subset of these 16 countries, which consist of a more homogeneous group of 41 countries, that are characterised by a 'Western lifestyle'. In this subset there are 36 European countries plus Australia, Canada, New Zealand and the USA (from now, we simply refer to the whole set as the world group and to the subset as the Western group).

More particularly, real per capita income is measured by the ratio of GNI to population and it is expressed in current international dollars, using purchasing power parity (PPP) rates (WB, 2014). Cancer incidence is measured by the crude and the ARSs of all types of cancer ("all sites, excluding non-melanoma skin", according to the ICD classification) provided by the WHO within *Globocan project* (Bray et al. 2004). In fact, International Agency for Research on Cancer (IARC) publishes free software with worldwide estimation of cancer incidence, prevalence and mortality. For cancer incidence, these data include absolute numbers of new cancer cases and crude and ARSs, in both the male and female population.

Table 1 contains a short description and some basic descriptive statistics of all variables for the two groups of countries (the complete database is available from the author).

As a useful starting point to highlight this issue, in Figures 1 and 2, the crude rate of incidence of all types of cancers within the total population (TCR) is plotted against the per capita income (Y) for all 162 countries and for the subset of 41 countries. Both scatter plots seem to suggest a strong influence of real per capita income on the average risk of developing some type of cancer.

Figure 1 Economic growth and cancer incidence (see online version for colours)

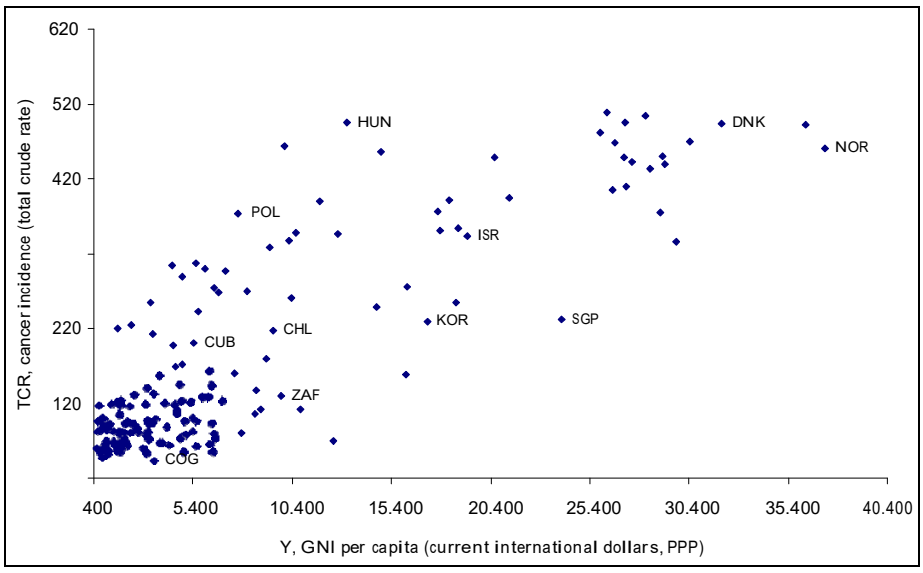
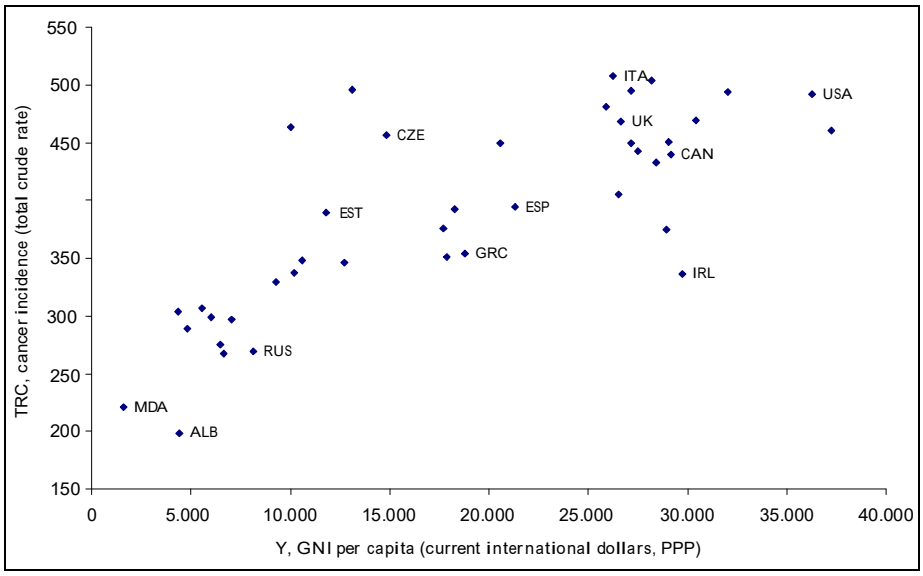


Figure 2 Economic growth and cancer incidence (see online version for colours)



In order to measure the magnitude of this influence, Tables 2 and 3 show the results of ordinary least square (OLS) estimations of the relationship TCR = *f*(Y) in three different specifications: linear, quadratic and double-log. The goodness of fit is fairly high in all equations. The crude rate shows a strong positive and significant relationship with per capita income, but it tends to increase less than proportionally with respect to Y. In other words, during economic growth the crude rate of cancer incidence increases, but it evolves drawing approximately the early stage of an inverted U-shaped curve. Indeed, the quadratic and double-log forms are the two specifications that best fit data for the world and Western groups, respectively.

This analysis, however, neglects the powerful influence of gender and age on cancer incidence. Thus, it is necessary to repeat OLS estimation using crude and ASR, in both the male and female populations (again in linear, quadratic and double-log specifications).

The simple decomposition of crude rate by gender does not alter the previous results (as shown in Tables 2 and 3). Instead, using ARSs there is a slight reduction in the goodness of fit. Changes in per capita income, however, continue to explain an important part of the change in cancer incidence. This effect is not due to the positive influence of economic growth on the average duration of life.

In particular, a straight line best describes the relationship between real per capita income and cancer incidence within the female population in both groups of data, while quadratic and double-log forms provide a reasonably close approximation of data for the male population in countries within the world and Western groups, respectively. All this seems to confirm a relevant negative effect of economic growth on the presence of cancer risk factors in different populations.

3.1 Income elasticity of cancer incidence

These results suggest that economic growth has a negative influence on cancer incidence, but they also indicate that in some cases cancer incidence tends to rise less than proportionally with respect to the increase in real per capita income. Moreover, the negative effect of economic growth on the overall rate of cancer incidence seems to be only partially related to the implications of an ageing population. Results of our regressions on crude and ARSs indicate that there are both *gross* and *net* effects of economic growth on cancer incidence, respectively.

Regression models using the double-log specifications allow us to easily calculate the elasticity of cancer incidence with respect to real per capita income. These coefficients provide an idea of the dimensions of both *gross* and *net* effects of economic growth on cancer incidence.

Table 4 Elasticties and income turning points

Variable	*World elasticity*	*World Y*, PPP (2012)*	*Western elasticity*	*Western Y*, PPP (2012)*
1 TCR	0.508	44.053	0.270	31.452
2 FCR	0.462	45.773	0.265	34.642
3 MCR	0.558	42.697	0.280	29.548
Mean CR	0.510	44.235	0.273	32.095
4 FASR	0.184	--	0.178	--
5 MASR	0.273	33.632	0.146	35.532
Mean ASR	0.229	--	0.162	--
All observations mean	0.397	41.539	0.228	32.793
Std. dev.	0.151	4.845	0.058	2.431

As shown in Table 4, all elasticities are positive but less than one. On average, the coefficient is about 0.4 for countries within the world group and 0.2 for those within the Western group. Differences in elasticity between these two groups are mainly due to the effect of economic growth on the average life expectations at birth. As a matter of fact, using the ARSs, cancer incidence increases on average by 0.23% and 0.16% in the world and Western groups, respectively, for each 1% increase in real per capita income.

Finally, using regression results from the quadratic specification, we can calculate the average hypothetical threshold level of Y (that is, the real per capita income beyond which cancer incidence starts diminishing, Y*). As shown in Table 4, in a long-run perspective, the process of economic growth may tend to exhaust the strong *net* negative effect on cancer incidence. However, our results within the male population indicate that this threshold level of real per capita income is extremely high (about $33,000 and $35,000 for the world and Western groups, respectively).

3.2 Economic growth, structural change and cancer incidence

Abstracting from the complexity of the causal interactions between different carcinogen 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 model, like: isr = f(q; α), where the ARS of incidence for all types of cancer (isr) in a given population, at time tn, depend on the people's exposure to external cancer risk factors (q), at time t0, for a stated level of unavoidable agents due to the individuals' genetic characteristics (α).

Here q is a catchall variable that stands for all the behaviours and situations that characterise people's habits and customs and it serves as a proxy for measuring the average population exposure to lifestyle cancer risk factors. One may think q as a bundle of low risk (such as foods) and/or high risk (such as environmental pollutions), in which each item is described by the set of its healthy related attributes for instance, the safety and nutritional characteristics of foods that reflect a poor or healthy eating habits. The whole set of these attributes determines 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 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. 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 changes, sometimes gradually and sometimes abruptly, from one level of real per capita income to another.

'Engel's law', states that the proportion of income spent on each type of goods changes as real average income increases. This is due to people modifying their preferences, by means of both individual and collective choices, along a distinct hierarchy of needs. Put differently, an increasing purchasing power deeply modifies people habits and customs. These changes in lifestyles cause transformations of the set of attributes that enter the bundle of health-related low risk (and high risk) faced by the population, and therefore they have 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) like: q = g(y), albeit very simple, may be a useful tool to capture the influences of economic growth on cancer incidence. In effect, replacing q in isr = f(q; α) by its expression from q = g(y), gives: isr = f [g(y); α]. That is a relation between real per capita income, at time t0, and the ARS of incidence at time tn, for a given level of the unavoidable cancer risk factors, α.

At the microeconomic level, many studies have examined how personal income and wealth can influence individual exposure to cancer risk factors. On the contrary, macroeconomic analysis of this issue seems to be a relatively underdeveloped area of research. Previous studies in this field have highlighted that the evolution of cancer incidence in a growing economy is a very complex subject that should be approached in an interdisciplinary framework (Ukraintseva and Yashin, 2005). To contribute to this

aim, here, we develop a very basic economic model that can provide some insights into building a more realistic and complex theory (Bosanquet and Sikora, 2006).

As in other stock-flow relationships, a given average duration of the disease prevalence is a function of the incidence and mortality rates. In the following discussion, this kind of stock–flow relationship allows us to simply focus on cancer incidence and mortality, in order to develop an elementary framework where, *ceteris paribus*, changes in mortality and incidence rates during economic growth are primarily due to structural changes operating on the supply and demand sides of the economy, respectively.

More particularly, on the supply side, as real GNI per capita income increases, better medical and surgical treatments become available, and notably, better techniques for early diagnosis. All these technical changes can dramatically reduce cancer mortality. This is why, all other things being equal, for a given incidence rate, economic growth implies a notable increase in prevalence rates (Capocaccia et al., 2002). In contrast, on the demand side of the economy, long-run increases in real per capita income tend to raise the average life expectations at birth. Since the average risk of developing any type of cancer is strongly influenced by age, economic growth may lead to an increase in the overall incidence rate of cancerous diseases.

In this paper we focus only on the demand side effects of economic growth on cancer incidence. Economic and social structural changes that characterise the processes of economic growth deeply modify the population's habits and lifestyles. Studies on cancer aetiology point out the multifactorial nature of these types of diseases and the great importance of habits and lifestyles as risk factors (Nasca, 2007). As a result, economic growth tends to modify population exposure to cancer risk factors (such as, nutritional and environmental risk factors).

Considering these structural changes from the demand side of the economy, an Engel's function may be a simple, but very useful tool for analysing how cancer incidence changes during economic growth. From this perspective, exposure to cancer risk factors can be thought as the consequence of the characteristics of goods and services that enter the average consumption bundle demanded by the representative consumer at each stage of economic growth.

If there is something that we positively know about the expansion of per capita demand when real income increases, it is that per capita demand for each commodity usually does not increase proportionally (Pasinetti, 1981). This is a well-known generalisation of Engel's law: it simply states that the proportion of income spent on each type of goods and services changes as real per capita income increases because consumers increase consumption along a hierarchy of needs. Therefore, during economic growth the composition of the average consumption bundle demanded by the representative consumer changes continually over time.

For our purposes, it is useful to think of a consumption bundle of goods and services (QAC) that reflects a lifestyle characterised by a low risk of developing any type of cancerous diseases (i.e., an *anti-cancer lifestyle, noting this would not reduce risk of genetically linked cancers – cancer-risk reducing lifestyle*). In the presence of a hierarchy of needs (determined by biological, cultural and social factors), even the demand for the QAC consumption bundle does not increase proportionally. With regard to how the demand for an anti-cancer lifestyle consumption bundle increases in a growing economy, it seems reasonable to introduce some basic hypotheses (Figure 3). In the early stages of economic growth, the demand for a healthy lifestyle is likely to be close to zero and it

may tend to increase less than proportional with respect to real per capita income (Y). However, in the subsequent stages of growth, real per capita income increases demand and income may be linearly related. Finally, after real per capita income reaches a threshold level (Y″), the demand for an anti-cancer lifestyle may tend to increase more than proportionally.

Figure 3 Engel's curve

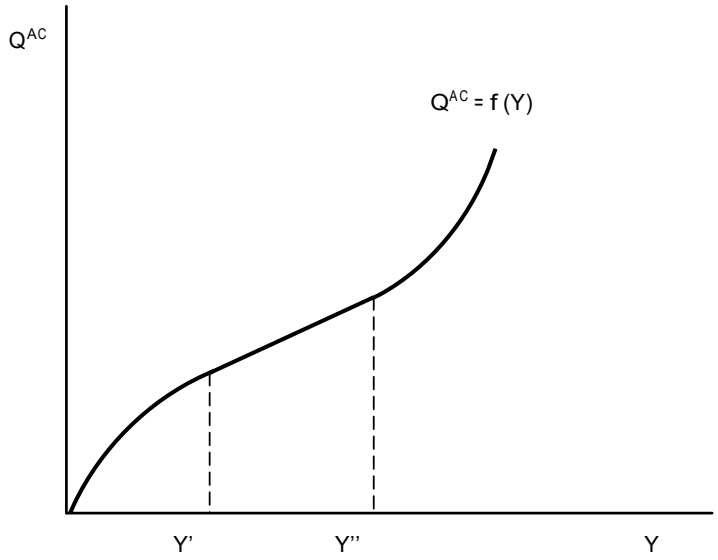
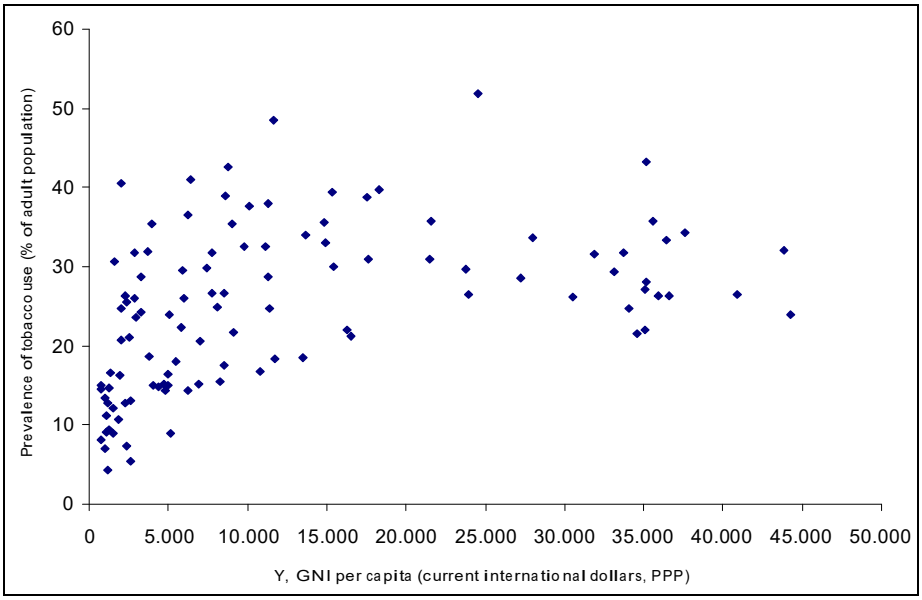


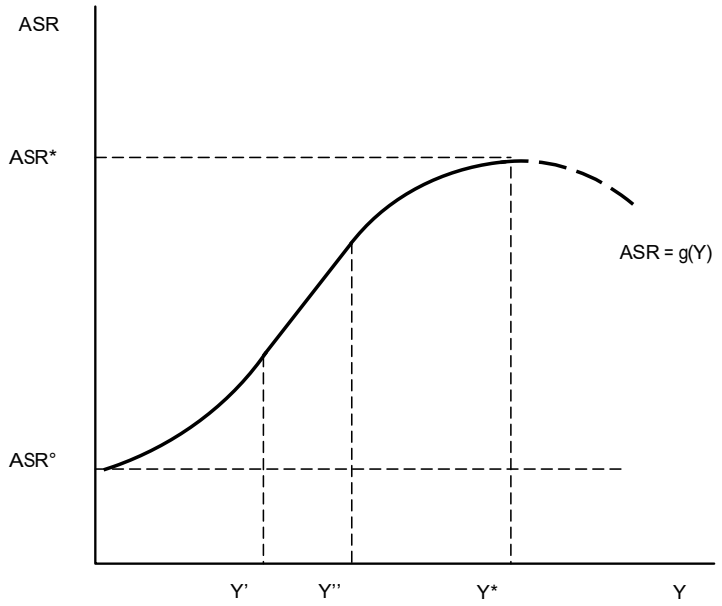
Figure 4 Economic growth and tobacco consumption (118 countries) (see online version for colours)



Source: WHO (2005)

Epidemiological data on tobacco consumption, for example, seems to support this hypothesis. In Figure 4, the age-standardised prevalence of current tobacco smoking among adults is plotted against the real per capita income, for a set of 118 countries (WHO, 2017). The scatter plot confirms that anti-cancer lifestyle behaves like a luxury good. At lower per capita income, economic growth pushes up tobacco consumption. Prevalence rate of current tobacco smoking is positive related to economic growth until about $15,000. After this threshold level, tobacco becomes an inferior good and the age-standardised prevalence of current tobacco smoking starts declining.

Figure 5 Kuznets' curve



From the Engel's curve depicted in Figure 3, we can derive a function that describes how, *ceteris paribus*, the overall cancer incidence evolves in a growing economy. Figure 5 shows a possible general form of this relationship, in which cancer incidence is measured by the ASR and economic growth is measured by the real per capita income (Y). It is interesting to note that the relationship assumes the form of a type of Kuznets curve (Kuznets, 1955). Increases in real per capita income have a more-than-proportional negative effect on the overall cancer incidence only in the early stages of economic growth. During growth, as a result of the expansion of demand for the anti-cancer lifestyle consumption bundle, this more-than-proportional relationship tends to disappear. In particular, if a healthy lifestyle is a luxury good, after the early stages of economic growth the overall incidence rate will increase, but less than proportional with respect to Y (there also can be an interval of the growth process where the ARS of cancer incidence rises approximately linearly with per capita income).

Furthermore, the relationship between overall cancer incidence and real per capita income has a positive intercept on the y-axis and a turning point. In particular, ASR° measures the autonomous component of the incidence rate (namely, the component that is independent of income because it is weakly influenced by exposure to risk factors,

such as in the type of cancer with an important genetic and/or infective aetiology) and Y* is the threshold level of per capita income beyond which cancer incidence starts diminishing.

4 Impact of life expectancy with increase in economic wealth

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 side of the economy, respectively.

On the supply side, 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 the population. These medical improvements are able to dramatically reduce the mortality of cancers. Improvements in healthcare provision across all diseases will impact on cancer prevalence as people live longer and survive other diseases such as cardiac failure, their risk of developing cancer increases, i.e., they survive long enough to get it rather than dying at a younger age from something else. Arguably, this is why in each society, all 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 affect today's incidence rates.

On the demand side, it is useful to denote with qLOW and qHIGH two specific combinations of the risk-increasing and risk-reducing aspects that reflects a lifestyle characterised by low and high risk of developing any type of cancer, respectively. Specifically, qLOW indicates a set of behaviours and situations associated with a minimum level of the average population exposure to the external cancer risk factors, and vice versa for qHIGH. It seems reasonable to think at qLOW (that is, to think at 'an anti-cancer lifestyle') as a luxury good, with an income elasticity greater than one, and at qHIGH 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 a good).

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

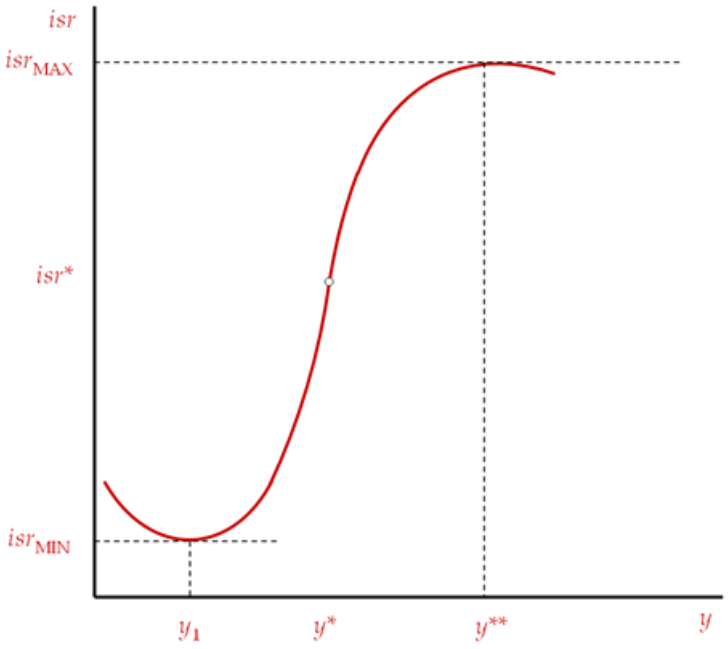
At less developed stages of social and economic conditions the process of growth usually pushes populations towards an unhealthy 'western lifestyle', such as smoking and consumption of calorie-dense food. Furthermore, in these circumstances economic growth is often driven by an industrialisation process based upon high polluting production methods, that typically takes place in unsafe and harmful working environments. 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.

5 Conclusions

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

At theoretical level, some kind of J-curve is a possible general model to represent, all other things being equal, how economic growth influences cancer incidence in a given homogeneous population. This complex relationship may be captured by some basic hypotheses, as illustrated in Figure 6, where cancer incidence is measured by the ARS of all types of cancer (isr) at time tn and economic growth is measured by the real per capita income (y) at time t0.

Figure 6 Cancer J curve (see online version for colours)



At very low-income levels, there is often a high incidence of cancers related to some biological (i.e., infectious) agents. Until y1, 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, isrMIN 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 y1, cancer incidence will rise with economic growth. More specifically, there is an early range of development stages (from y1 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 a lifestyle that reduces the risk of cancer, 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 its maximum. Finally, when the development stages pass this threshold level, the overall rate of age-standardised cancer incidence might start decreasing.

Describing and measuring the relationship between cancer incidence and real per capita income constitutes the first step in understanding how the process of economic growth affects population exposure to cancer causing factors. In fact, real per capita income is not an accurate and adequate measure of a country's level of development and it is not possible to summarise in Y a set of economic, social and health features. Further research is needed to include more variables, for example, those referring to personal income distribution, cultural habits and customs, general sanitary conditions and health policies. It is also necessary to utilise epidemiological data for each type of cancer within more homogeneous genetic populations.

This paper, however, 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.

References

Bosanquet, N. and Sikora, K. (2006) *The Economics of Cancer Care*, Cambridge University Press, London.

Bray, F., Ferlay, J., Parkin, D. and Pisani, P. (2004) *Globocan Software 2002*, IARC Press, Lyon.

Capocaccia, R., Colonna, M., Corazzieri, I., De Angelis, R., Francisi, S., Micheli, A. and Mugno, E. (2002) 'Measuring cancer prevalence in Europe', *Annals of Oncology*, Vol. 13, pp.831–839.

International Agency for Research on Cancer (IARC) (2004) *Cancer Mondial*, IARC, Lyon.

Jemal, A., Center, M., DeSantis, C. and Ward, E.M. (2010) 'Global patterns of cancer incidence and mortality rates and trends', *Cancer Epidemiology, Biomarkers & Prevention*, Vol. 19, No. 8, pp.1893–1907.

Kuznets, S. (1955) 'Economic growth and income inequality', *American Economic Review*, Vol. 45, No. 1, pp.1–28.

Last, J. (2001) *Dictionary of Epidemiology*, Oxford University Press, Oxford.

Nasca, P. (2007) *Fundamentals of Cancer Epidemiology*, Jones & Bartlett Publishing, Sudbury, MA.

Pasinetti, L. (1984) *Structural Change and Economic Growth*, Cambridge University Press, Cambridge.

Ukraintseva, S. and Yashin, A. (2005) *Economic Progress as Cancer Risk Factor*, M. Planck Institute, Berlin.

World Bank (WB) (2014) *World Economic Indicators*, World Bank Press, Washington DC.

World Health Organization (WHO) (2015) *International Classification of Diseases*, WHO Press, Geneva.

World Health Organization (WHO) (2017) *World Health Statistics*, WHO Press, Geneva.