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Analysis

Economic Growth and Cancer Incidence

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ABSTRACT

Why do we observe increasing rates of new cancer cases? Is the increasing burden of cancer mainly the outcome of higher life expectancy and better life conditions brought about by economic development? To what extent do environmental degradation and changes in life-styles play a relevant role? To answer these questions, we empirically assessed the relationship between per capita income and new cancer cases (incidence) by using cross-sectional data from 122 countries.

We found that the incidence rate of all-sites cancer increases linearly with per capita income, even after controlling for population ageing, improvement in cancer detection, and omitted spatially correlated variables. If higher incidence rates in developed countries were merely due to those factors, and not also to life-styles and environmental degradation, we would have found a flat or even an inverted-U pattern between per capita income and cancer incidence.

The regression analysis was applied also to the eight most common site-specific cancers. This confirmed the existing evidence on the different patterns in rich and poor countries, explained the pattern of the estimated relationship for aggregate cancers, and gave some other interesting insights.

1. Introduction

Cancer incidence (yearly new cases of cancer) is increasing and predicted to grow fast. The term 'Cancer epidemic' has become frequently used, not only by the media (e.g. Servan-Schreiber, 2008), but also by academic journals and by the World Health Organization.[1] The problem is particularly alarming in lower- and middle-income countries (see, e.g., Boyle and Levin, 2008; GLOBOCAN, 2012; Stewart and Wild, 2014; Vineis and Wild, 2014; Ferlay et al., 2015; Torre et al., 2015). For some rich countries, incidence rates are stabilizing (or slightly decreasing), however at very high levels. In the USA, this has been the case since the mid 1990s (Siegel et al., 2016).

Although data availability on cancer has increased significantly in the last years,[2] the relationship between cancer incidence and economic development remains largely unexplored, with just a few exceptions, namely: Beaulieu et al. (2009), Bray et al. (2012), Fidler et al. (2016).[3] The first is a report by "The Economist" Intelligence Unit on the health and economic burden of cancer. As a supplementary result, in one of its appendices, the report shows the outcome of a multiple regression analysis aimed at understanding cross-country variations in both estimated cancer incidence rates for 2009, and in fatality rates for 2002. Regressors included p.c. income, per cent of population aged 65 +, and regional dummies. The authors found a positive association of higher cancer incidence rates with both age and higher per capita income countries, which they attributed to the belief of "underreporting of cancer cases in developing countries" (Beaulieu et al., 2009, 62).

Bray et al. (2012) and Fidler et al. (2016) grouped countries according to the four levels (low, medium, high, and very high) of the Human Development Index (HDI) and compared incidence and mortality rates across groups. Both articles brought support in favour of the so-called "cancer-transition", according to which the demographic transition and economic development are changing the composition of the different types of cancers, with a shift from cancers linked to infections to those associated with non-infectious risk factors and possibly associated with the "western" lifestyle.

The above-mentioned papers are in line with the health literature, briefly summarised in the next section. The general idea is that increasing cancer incidence rates might be the outcome of economic development, which delivered not only higher life expectancy and improved cancer detection and statistical reporting, but also environmental degradation and "bad" life-styles.

The aim of our research was to empirically investigate the macro

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[1] In April 2015, the Lancet Oncology and The Lancet launched a joint campaign against cancer "to inform strategies to control the global cancer epidemic" (see http://www.thelancet.com/campaigns/cancer). In 2005 the term 'epidemic' was used in the 58th resolution of the WH assembly, see http://www.who.int/mediacentre/news/releases/2005/pr_wha05/en/

[2] For an assessment of the status of population-based cancer registries worldwide see Bray et al. (2015).

[3] The differences between the present research and the previously mentioned studies will be discussed in Section 5.

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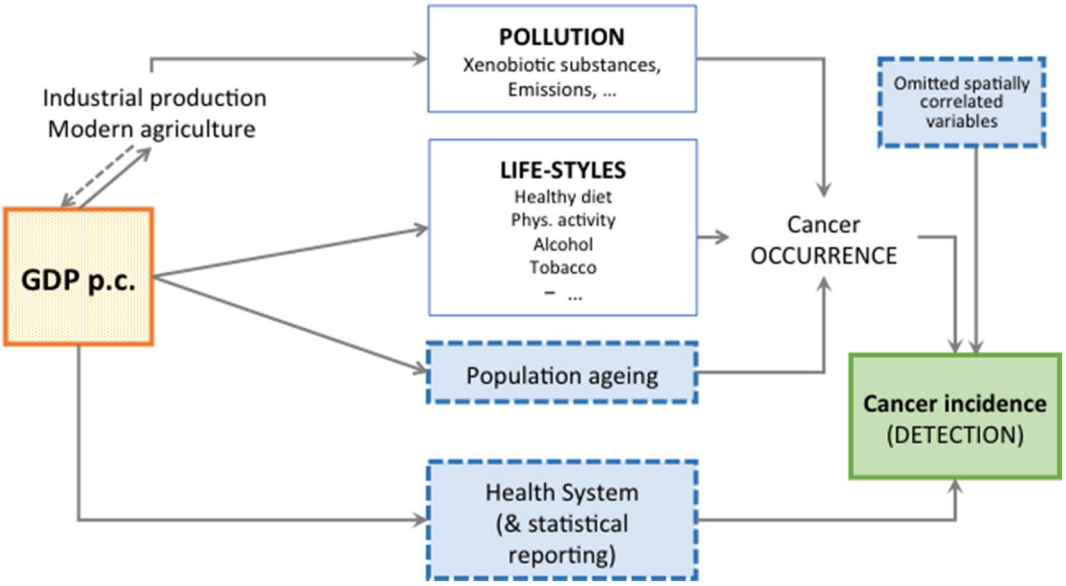

Fig. 1. From income to cancer incidence: major links.

level relationship between cancer incidence rates and per capita income. For this purpose, we tested some reduced models that looked only at the ends of the complicated causal chains. Such an approach has been followed by the so-called Environmental Kuznets Curve (EKC) literature that has been investigating the relationship between economic growth and the environment for more than 25 years (e.g., Stern, 2004; Dinda, 2004; Luzzati, 2015). While the EKC literature focused on anthropic pressures, e.g. emissions, here we focused on one possible outcome of pressures, that is, cancer occurrence.

The paper is structured as follows. The Section 2 outlines the links between cancer and economic development, from which we derived the conceptual model for our empirical analysis (Fig. 1). The Section 3 describes data and methods. In the Section 4 results are presented and discussed. The last section gives our conclusions.

2. Cancer and its Possible Links With Economic Development

This section firstly summarises what we know about cancer genesis, and then why economic development can play a major role in cancer occurrence. The dominant theory explaining cancer is the so-called Somatic Mutation Theory (SMT) (Nowell, 1976; Hanahan and Weinberg, 2000, 2011) according to which "random mutations in the genes which control proliferation or apoptosis are responsible for cancer" (Bertram, 2001, p. 170). Hence, cancer is due to stochastic (relevant) mutations that occur in oncogenes and tumour suppressor genes (Lodish et al., 2000). The older a person, the higher is the number of accumulated stochastic mutations, which ultimately leads to higher probability of cancer occurrence.

Recently, SMT has been criticised on the basis of theoretical reasons and experimental and epidemiological evidence. Hence, other theories of carcinogenesis have begun to gain ground. They shift the focus from single cells to the entire tissue and attribute a prominent role to altered environments (epigenetic signals) for regulating gene expression, rather than to stochastic mutations of DNA (see e.g. Burgio and Migliore, 2015). For instance, Tissue Organization Field Theory (TOFT) (see e.g. Baker, 2015), which is better seen as integrative rather than alternative to SMT (Bedessem and Ruphy, 2015), looks promising for understanding the role of low-dose foetal exposure to ubiquitous and long lived chemical pollutants, namely the endocrine-disrupting chemicals (EDCs).[4] These chemicals, by mimicking physiologic hormone signalling molecules, perturbate tightly regulated intercellular signalling pathways. This leads to subtle architectural changes in tissue organization that increase the risk of cancer development (Howard and Staats, 2013).

Overall, cancer is increasingly seen as the disruption of a complex equilibrium, that is, the outcome of an evolutionary process in which random genetic mutations have to face the selection of environmental pressure; moreover, intrinsic epigenetic plasticity, clonal evolution and high cellular adaptability are also crucial (Greaves, 2014). Hence, cancer is acknowledged as stemming from many interacting factors, that is, from mutations in oncogenes and tumour suppressor genes, from genetic inheritance,[5] work and living environment, and lifestyles (see e.g. Belpomme et al., 2007a, b; Stewart and Wild, 2014).

Many studies have investigated the differential contribution to cancer incidence of non-genetic risk factors (e.g. Danaei et al., 2005) and of environmental factors (e.g., Alavanja et al., 2003, Boffetta, 2006, Mannucci et al., 2015, Stare and Jozefowicz, 2008). The confluence of diverse types of evidence increasingly indicates the relevance of involuntary exposure to environmental contaminants, which affect particularly the "developing foetus, the developing child and adolescent" (Newby and Howard 2005, 57). For instance, there is evidence of decrease in the average age of cancer onset (e.g. Newby et al., 2007) and increase in childhood cancers (e.g. Steliarova-Foucher et al., 2004), which are also attributed to environmental factors (Stewart and Wild, 2014; Norman et al., 2014). Historical evidence supports the idea that cancer is a disease of industrialization/wealth since "in preindustrial societies, the death rate in infancy was high, but if adolescence was reached then […] the chances of living a reasonable life span in good health were high and unlikely to end in the development of cancer" (Howard and Staats, 2013). It is not under dispute that economic and technological progress led to the introduction of a complex mixture of persistent xeno-chemicals and other pollutants that have been recognised as carcinogenic.

Aggregate quantifications of the environmental risk factors have been proposed in a wide–ranging report by the World Health Organization that surveys the findings on the environmental risk factors (Prüss-Üstün et al., 2016). According to this report, household and

[4] A useful introduction to EDCs is Gore et al. (2014).

[5] The heritable factors have an important, but not exclusive, role. For instance, using data from Swedish, Danish and Finnish twin registries, it has been reported (Lichtenstein et al., 2000) that genetic influence on the incidence of cancer explains no more that 42% of the variance in incidence rate, depending on the cancer site.