INCORPORATING THE ENVIRONMENTAL CONTEXT IN THE STUDY OF CANCER

ISSUES AND IMPLICATIONS

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Summary

The analysis in Section 2 of this chapter presents several lines of evidence that implicate the environment in cancer causation; specifically, findings from wildlife studies, cancer trend reports, immigrant studies, childhood cancer studies and twin studies are reviewed. Having established the general evidentiary basis for the cancer-environment linkage, in Section 3 we turn to a discussion of the current methodological difficulties in incorporating the environmental context in the study of cancer. The particular focus here is on exposure assessment - a key methodological limitation in studying the cancer-environment linkage. It is reasonable to expect that cancer cases arising from point source environmental exposure will tend to cluster geographically. For this reason the role of exposure assessment and other methodological issues in the context of cancer cluster investigations are considered. The case of the Woburn Massachusetts leukaemia cluster is reviewed to illustrate some of the pertinent issues involved. In Section 4, we move to a general discussion of the implications of cancer risk assessment methodologies for cancer policy and intervention. In light of the observational evidence concerning the cancer-environment link (Section 2), as well as the uncertainties involved in assessing the risks associated with environmental carcinogens (Section 3), it is suggested that the precautionary principle be adopted as a guiding principle for cancer policy and intervention. The precautionary principle calls for protective action, even when the evidence of harm remains inconclusive and the adoption of this principle seems warranted under the present technical and policy circumstances. The final section ends the chapter with some recommendations and concluding remarks.

P. Nicolopoulou-Stamati et al. (eds), Cancer as an Environmental Disease, 11-38.

1. Introduction

The 'environment' can be broadly defined as including the surroundings constituted by the natural, built, and social dimensions. Today, natural environments (including air, water, and soil) are often plagued with environmental pollutants that are potentially carcinogenic. The toxic effects of these pollutants are difficult to document because they generally result from low-level chronic exposure to a multiplicity of carcinogens through a variety of environmental routes. The analysis of data concerning the ***body burden*** - the total sum of carcinogens in the body - becomes quite complicated because it must cover all routes of entry (i.e. inhalation, ingestion and dermal absorption) as well as all sources of carcinogens (i.e. food, air, water).

Unlike natural environments, built environments include the human-made infrastructure, as well as the products of industrialisation - in particular, the numerous synthetic and radioactive substances produced and introduced into the environment after the Second World War. Concern over the health impacts of such substances has prompted the formation of environmental movement organisations that have brought to the forefront issues pertaining to the social environment, that is, the institutional arrangements within society (specifically, the relationship between industry, government and the citizenry). The social dimension is an important consideration because it draws attention to the social, political and economic ramifications associated with cancer risk management, especially issues such as risk distribution and the question of voluntary exposures due to lifestyle choices versus imposed exposures due to industrial activity and policy decisions. The importance of all three dimensions of the environment in the study of cancer is well-illustrated by the following historical study.

The linkage between cancer and the environment was noted as early as 1755 when Sir Percivall Pott recognised the association between scrotum cancer (then referred to as 'soot wart') and exposure to soot amongst chimney sweeps (the active agent in the soot was later identified as benzo[a]pyrene, now classified as Group 2A by IARC). Although commonly regarded as the first documented discovery linking an occupational environment to cancer, it is important to recognise that lifestyle played a determining role as indicated by the fact that 'scrotum cancer was not a world-wide phenomenon, but that which mainly adhered to British chimney sweeps' (Butlin, 1892a: 1341). Further investigation into this matter led Butlin to the following conclusions more than a century after Pott's initial discovery:

> '[C]himney sweeps in Great Britain typically wore loose clothing which was often torn leaving parts of their bodies accessible to dirt when sweeping chimneys, they lived in soot-filled homes in which two or more generations of the business of sweep was conducted and they seldom washed the whole body more than once or at most twice a week, unlike chimney sweeps in other regions of the world.'
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> (Butlin, 1892b: 5)

This historical case clearly indicates that the cancer-environment relationship is quite complex and very much influenced by the interaction of a multiplicity of factors such as occupation, lifestyle and socioeconomic status. More recently, this research tradition involving the avoidable causes of cancer is found in the classical analysis by Doll and Peto (1981) and the present chapter builds on some of their general research focus.

The percentage of cancer that is attributed to the environment varies between 2 per cent (Trichopoulos *et al.*, 1996) and 80 per cent (Schneiderman, 1978: 559). Such extreme differences reflect the polarities and debates that exist within the field of cancer risk assessment, with those supporting estimates at the lower end accusing those supporting higher-end estimates of being 'alarmists', while charges of being 'nay-sayers' and 'pro-industry' flow in the opposite direction. To some extent, efforts to fix an exact percentage may be futile, simply because the number of variables, assumptions, data-types and definitions that need to be considered in making such an estimate is formidable. As such, a 'weight of evidence' approach may be more appropriate. In this light, we consider several lines of evidence that build on some of the classical approaches of Carson (1962), Doll and Peto (1981) and Schneiderman (1978), in addition to our own emphasis.

2. Evidence for the Environmental Basis of Cancer

The first line of evidence discussed below examines studies on cancers in the wildlife setting. Due to the varying susceptibilities of different animal species, an increasing number of cancer cases in particular wildlife species may be the first hint of the pervasiveness and impacts of carcinogens in the general environment. Secondly, the examination of cancer trends may reveal important information about the environmental basis of cancer. If cancer was attributed solely to genetic factors, then dramatic fluctuations in cancer rates could not occur over short periods of time nor 'would they be sharply increasing in some cancers and not in others' (Schniederman, 1978: 559). A third important line of evidence involves findings from immigrant studies. If genetics/ethnicity factors played a determinant role in cancer risk, then immigrants would retain the cancer incidence of their homelands. On the other hand, if the cancer rate of immigrants tended to approximate those of the host country, then this would suggest that environmental factors play an important role in cancer incidence (Steingraber, 1998: 58). Fourth, childhood cancer rates are noteworthy because they minimise the confounding effects associated with a long latency period. It is argued that although low-level environmental exposures may not affect adults, they may have adverse effects on children. Increased cancer rates amongst children may therefore signal the presence of environmental carcinogens. Indeed, studies have shown that removal of environmental carcinogens may have an immediate effect, as illustrated by the immediate decrease in different types of childhood cancer after the cessation of atmospheric nuclear weapons testing (Mangano *et al.*, 2002: 29). Finally,

the findings from twin studies perhaps represent the most compelling evidence of the role of the environment in cancer causation. As will be discussed, since the genetic makeup of identical twins is virtually the same, the occurrence of differential cancer rates between twins cannot logically be ascribed to genetic factors, thus pointing to the importance of environmental factors in the onset of cancer.

2.1. Wildlife Studies

Silent Spring by Rachel Carson (1962) was perhaps the first and most influential document that dealt with the issue of how environmental carcinogens, particularly pesticides, may affect a disturbingly large number of different species ranging from robins to caddis fly larvae, to quail, salmon, cats and ultimately humans. Increased frequencies of unusual disease outcomes in wildlife may represent the first warning signs of the health impacts of environmental contaminants on humans (Colborn *et al.*, 1997) because 'wild animals living in contaminated habitats are exposed to low levels of ever-changing combinations of chemicals throughout their lifetimes,' just as humans are (Steingraber, 1998: 142). The enhanced sensitivity of animals to particular environmental contaminants has been known for some time as, 'over a century ago, coal miners carried caged canaries into underground mines to alert them to the presence of carbon monoxide gas' (Manuel, 1996: 934). Similarly, diseased wildlife may also signal the widespread presence of carcinogenic agents and/or pollution in the general environment which may in turn endanger human health; the three examples that follow illustrate this.

An increased rate of cancer deaths amongst beluga whales in the St. Lawrence estuary (which drains the North American Great Lakes system) was found to be related to pollutants; more specifically polycyclic aromatic hydrocarbons (PAHs), originating from the nearby aluminium smelters. (Fox, 2001; Watanabe, 2000; Steingraber, 1998; Colburn *et al.*, 1997). 'The human population living in the proximity of this beluga habitat is affected by rates higher than those found in people in the rest of Quebec and Canada, and some of these cancers have been epidemiologically related to PAHs' (Martineau *et al.*, 2002: 285). Such increases of cancer within a particular community are also known as a cancer cluster, as further discussed in Section 3.3.

A second example includes the relationship that was found between liver cancer in wild fish and PAHs that entered the Black River in Lorain County, Ohio from storm, sewer and road run-off (US Fish and Wildlife, 2000; Baumann and Harshbarger, 1995). Similar findings have been documented in other seaways (Pinkey *et al.*, 2000; Harshbarger and Clark, 1990; McMahon *et al.*, 1990).

A third example pertains to the high levels of Persistent Organic Pollutants (POPs), particularly hexachlorocyclohexane (HCH - one substance within the class of POPs; IARC, 2000 Group 2A carcinogen), found in Arctic wildlife. The accumulation of POPs in the Arctic region readily occurs because of the particular chemical and

physical properties of these substances, such as the capability to undergo long cycles of volatilisation followed by condensation (McGinn, 2000). Since HCH is fat-soluble, it tends to bioaccumulate in the fat of Arctic mammals that are at the top of the food chain, such as polar bears, seals and human beings. In fact, Canadian Inuit mothers have been found to have some of the highest HCH body burdens in the world (NRTEE, 2001). Furthermore, HCH tends to concentrate in the mothers' milk because of the high fat content, thereby posing a threat to infants (NRTEE, 2001).

2.2. Growing Cancer Trends

In the current world population of 6,157,400,560 (The World Fact Book, 2001) it is estimated that 20 million people have cancer (WHO, 2002: iii). The annual world-wide cancer incidence is estimated to be over 10 million new cases, while 6 million people world-wide succumb to this disease each year. Two decades ago, the respective figures were 6 million and 4 million (Tomatis *et al.*, 1990). Such findings are particularly alarming in light of how 'in the middle of the nineteenth century, cancer deaths accounted for only 1.3 per cent of all deaths' (Logan, 1982: 8), while 'today cancer is the second-leading cause of mortality in the developed world and fourth in the developing world - accounting for 12 per cent of all deaths world-wide' (WHO, 2002: 17). Furthermore, in approximately 20 years' time, it is projected that the annual cancer mortality will increase from 6 to 10 million (WHO, 2002: 17).

Cancer incidence for specific sites varies considerably between different world regions and by gender. For instance, North American females are found to have the highest age standardised rate of breast cancer incidence at 90.41, compared to Middle Africa at 13.46 (Ferlay *et al.*, 2001). In the case of esophageal cancer, males in East Asia experience much higher rates (21.79) than their female counterparts (8.92) and males in Western Africa (1.08). There is no real consistency between sites, country or gender; it is only known that certain countries are more prone to specific types of cancer(s) (see Table 1), thus indicating that lifestyle and environmental factors must play some role alongside genetic factors in cancer causation. In this connection, Taubes (1995: 165) notes 'the fact that no single cancer affects every population at the same rate suggests that factors external to the human body cause 70 per cent to 90 per cent of all cancers'.

It should also be noted that relatively rapid changes in trends in cancer incidence rates cannot be accounted for by genetic changes alone, thus providing supporting evidence for the role of environmental and lifestyle factors in influencing cancer. The most notable illustration of this involves changes in lung cancer incidence trends that tend to reflect changes in cultural norms related to smoking. Specifically, this refers to the dramatic increase in male lung cancer incidence after the approximately 20-year latency period that elapsed following both World Wars, as well as the increase in female lung cancer incidence following the Second World War (during which time changes in gender roles led to increased smoking in females as an increasing number of