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Review

The multitude and diversity of environmental carcinogens

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Abstract

We have recently proposed that lifestyle-related factors, screening and aging cannot fully account for the present overall growing incidence of cancer. In order to propose the concept that in addition to lifestyle related factors, exogenous environmental factors may play a more important role in carcinogenesis than it is expected, and may therefore account for the growing incidence of cancer, we overview herein environmental factors, rated as certainly or potentially carcinogenic by the International Agency for Research on Cancer (IARC).

We thus analyze the carcinogenic effect of microorganisms (including viruses), radiations (including radioactivity, UV and pulsed electromagnetic fields) and xenochemicals. Chemicals related to environmental pollution appear to be of critical importance, since they can induce occupational cancers as well as other cancers. Of major concerns are: outdoor air pollution by carbon particles associated with polycyclic aromatic hydrocarbons; indoor air pollution by environmental tobacco smoke, formaldehyde and volatile organic compounds such as benzene and 1,3 butadiene, which may particularly affect children, and food pollution by food additives and by carcinogenic contaminants such as nitrates, pesticides, dioxins and other organochlorines. In addition, carcinogenic metals and metalloids, pharmaceutical medicines and cosmetics may be involved. Although the risk fraction attributable to environmental factors is still unknown, this long list of carcinogenic and especially mutagenic factors supports our working hypothesis according to which numerous cancers may in fact be caused by the recent modification of our environment.

Keywords: Air-pollution; Cancer dioxins; Environment; Food-additives; Food-contaminants; Nitrates; Pesticides; Radiations; Viruses

We have previously shown that improvement in screening detection and longer life expectancy cannot account alone for the recently observed growing incidence of cancer in high income countries (Belpomme et al., 2007). In addition, decrease of tobacco smoking and of alcohol consumption in some European countries and in North

Abbreviations: AIDS: acquired immune deficiency syndrome; ALL: acute lymphoblastic leukemia; ATL: adult T cell leukemia; BL: Burkitt's lymphoma; CMR: carcinogenic; mutagenic and reprotoxic; CNS: central nervous system; DDT: 1, 1, 1-trichloro-2, 2-bis(*p*-chlorophenyl) ethane; DEHP: di(2-ethylhexyl)phthalate; DNA: deoxyribonucleic acid; EBV: epstein-barr virus; EMF: electro magnetic fields; ELF: extremely low frequency; ETS: environmental tobacco smoke; HBV: hepatitis-B virus; HCB: hexachlorobenzene; HCC: hepatocellular carcinoma; HCV: hepatitis-C virus; HD: Hodgkin's disease; HHV: human herpes virus; HIV: human immunodeficiency virus; HPV: human papilloma virus; HTLV-1: human T-cell lymphotropic virus type 1; IARC: International Agency for research on cancer; KS: Kaposi sarcoma; MRI: magnetic resonance imaging; NHL: non-Hodgkin lymphoma; NOC: N-Nitroso compounds; PAH: polycyclic aromatic hydrocarbons; PCB: polychlorinated biphenyls; PCE: perchloroethylene; POP: persistent organic pollutants; PVC: polyvinyl chloride; RNA: ribonucleic acid; SM: somatic mutation; TCDD: 2, 3, 7, 8-tetrachlorodibenzo-*p*-dioxin; TCE: trichloroethylene; US-EPA: US Environmental Protection Agency; UV: ultraviolet; VLF: very low frequency; VOC: volatile organic compounds

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America allows us to consider that these factors cannot account for the overall growing incidence of cancer. Moreover, although changes in diet and hormone use are recognized cancer risk factors, they cannot be considered *per se* as cancer initiating factors, because they mainly act as promoters or cocarcinogens rather than as mutagens. This suggests that the formation of endogenous mutagens which are thought to be associated with lifestyle-related factors may in fact play a role far less important that it is usually speculated. By contrast, we defined environmental factors as physical, chemical and biological agents present in the surroundings of individuals. In order to explain the currently increasing cancer incidence and to justify the hypothesis according to which it could mainly be related to environmental factors, we examine environmental carcinogens, in particular mutagens and analyse their potential role in inducing cancer. In this paper, we intend to show that many cancer types are induced by multiple and diverse exogenous environmental carcinogens, so that in many cases, cancer may be considered not only as a lifestyle related-disease, but also as an environmental one. We analyse herein microorganisms, radiations and xenochemicals, which have been rated as certainly or potentially carcinogenic by national and international organizations.

1. Viruses and other microorganisms

It is estimated that oncogenic viruses are involved worldwide in about 16% of neoplasia (Pisani et al., 1997), while it would range from less than 10% in high-income countries to 25% in Africa (Parkin et al., 2001; Talbot and Crawford, 2004). However, because in Western countries, there are some cancer types for which a viral origin is suspected, although not yet proved, it is likely that the percentage of 5–10% is underestimated. Three groups of double-stranded DNA viruses and two groups of diploid RNA viruses have been shown to be associated with human cancers (Table 1). In Western developed countries, human papilloma virus (HPV) in particular HPV type 16 (HPV-16) and hepatitis-B virus (HBV) are the most frequent oncogenic DNA viruses. They have been recognized as being associated with cervical cancer and hepatocellular carcinoma (HCC), respectively (IARC, 1995a, 1997a). These two viruses contribute differently to carcinogenesis: HPV-16 is directly mutagenic by inducing the viral genes E6 and E7 (Song et al., 1999), while HBV is thought to be indirectly mutagenic by generating reactive oxygen species through chronic inflammation induction (Blumberg et al., 1975; Hagen et al., 1994; Jackson and Loeb, 2001). Epstein-Barr Virus (EBV) has been related to Hodgkin's disease (HD) as well as to non-Hodgkin lymphoma (NHL) occurring in immunosuppressed patients (IARC, 1997a; Griffin, 2000). In non Western countries, in addition to the abovementioned cancers, Burkitt's lymphoma (BL) and nasopharyngeal carcinoma have been shown to be caused by EBV, and Kaposi Sarcoma (KS), to be associated with HIV and the Human herpes virus type 8 (HHV-8) (De The, 1995; IARC 1996a, 1997a; Pagano et al., 2004). In Western countries, BL is an uncommon disease. This may be due to the fact that in endemic area, in addition to natural promoters such as extracts of a commonly used plant, *Euphorbia tirucalli*, malaria as well as arbovirus infections are mosquitoes- so climate-dependent cofactors (Brooks et al., 1999; Van Den Bosch, 2004).

RNA tumor viruses must also be considered. They include the Hepatitis C virus (HCV) and the unique retrovirus presently known to be oncogenic in humans, the human T-cell lymphotropic virus type 1 (HTLV-1) which is the causal agent of a very rare T-cell type leukemia (IARC, 1996a; Talbot and Crawford, 2004). Also, these two oncogenic viruses contribute differently to carcinogenesis. HTLV-1 is directly mutagenic, while HCV, as HBV, is thought to produce oxidative stress in infected cells and thus to act indirectly through chronic inflammation (De Maria et al., 1996; Koike et al., 2002).

However, whatever their direct or indirect mechanisms of action, most of these human DNA or RNA viruses are oncogenic, meaning that they can initiate carcinogenesis by inducing mutations. They must be therefore distinguished from other retroviruses such as the human immunodeficiency viruses (HIV) (Gallo and Montagnier, 2003), which

Table 1
Human viruses with recognized oncogenic potential

Virus family	Virus	Human tumors	Non-malignant disease	Experimental tumors in animals
Papovaviridae	Human papillomavirus	Cervical cancer, Anogenital cancer, Skin cancer	Warts	
Herpesviridae	Epstein-Barr virus	Nasopharyngeal carcinoma, Burkitt's lymphoma, Hodgkin's lymphoma,	Infectious mononucleosis	Lymphoma in primates
Retroviridae	Kaposi's sarcoma-associated herpesvirus	Kaposi's sarcoma, primary effusion lymphoma,	Multicentric, Castleman's disease	Not known
	HTLV-1	Adult T-cell Leukaemia (ATL)	Tropical spastic paraparesis	ATL in rabbits
	HIV	B-cell lymphoma, Kaposi's sarcoma	AIDS	
Hepadnaviridae	Hepatitis B virus	Liver cancer	Hepatitis, Cirrhosis	Not known
Flaviviridae	Hepatitis C virus	Liver cancer	Hepatitis, Cirrhosis	Not known

Adapted from Talbot and Crawford (2004).

are not *per se* mutagenic, but which have been classified as carcinogenic by IARC. They have been shown in epidemiological studies to be strongly linked with the occurrence of cancer, because they can promote the initiating effect of oncogenic viruses through immunosuppression induction (IARC, 1996a). In addition to a possible effect favoring chemically induced carcinogenesis, HIV infection accounts for the occurrence of AIDS-associated viral neoplasia (Frisch et al., 2001). Indeed, a variety of cancers of viral or presumed viral origin, including leukemia, lymphoma, soft tissue sarcoma and other solid tumors have been reported in HIV-infected people. This is the case in HIV-infected adults and children, be they AIDS-bearing or not. So, estimates of cancer incidence in HIV-infected children range from 1 to 4 per 1000 children per year, representing a 10–40-fold increased risk (Pollock et al., 2003).

In fact, in addition to the abovementioned well-established human virus-associated cancers, there are strong biological and epidemiological arguments for an infectious viral etiology of other cancers. This is the case for childhood acute leukemia, particularly for the common acute lymphoblastic leukemia (ALL), for which the virus presumably involved has not yet been isolated although it is strongly suspected (Belpomme et al., 1969a, b; Guibout et al., 1977; Greaves, 2002). Indeed, several additional observations have contributed to the hypothesis that a virus is potentially involved in ALL. In fact, given the biological diversity of leukemia, it is unlikely that it is the single cause (see Belson et al., 2007 for review). In the *population mixing theory*, it has been postulated that an extremely infectious but not highly pathogenic virus introduced into a previously unexposed rural community, may trigger clusters of ALL cases, due to the existence of an associated immune deficiency (Kinlen 1988; Kinlen et al., 1991; Alexander et al., 1997; Boutou et al., 2002). However, an alternative explanation based on a "*delayed infection*" hypothesis leads to consider the possibility of a "two hit" mechanism, involving an abnormal response to an initial common infection followed by a critical second event, promoting the ALL development (Greaves and Alexander, 1993).

A viral origin has also been suggested for some non-EBV-related lymphoma (McNally et al., 2002; Karimi and Yarmohammadi, 2003; Talbot and Crawford, 2004; Harris, 2004) and for some pediatric and adult brain tumors (McNally et al., 2002), for which a polyoma virus as it is the case in animals (Krynska et al., 1999) has been suspected in humans (Weggen et al., 2000). Moreover, there are experimental data suggesting that some human soft tissue sarcomas (Eilber and Morton 1970) and some breast carcinomas could be caused by oncogenic retroviruses. For example it has been shown that human milk from women at high risk for breast cancer (Moore et al., 1971), cancer human breast tissues (Schlom et al., 1971; Vaidya et al., 1974) and cultures of normal (Furmanski et al., 1974) and cancer human breast cells (McGrath and Jones, 1978; Keydar et al., 1984) contain virus particles associated with reverse transcriptase activity that are morphologically similar to the mouse mammary tumor virus (MMTV). Consequently these particles have been designated as the human homologue of the MMTV (HHMMTV) (Callahan et al., 1982; Miyazaki et al., 1997; Wang et al., 1998; Soble et al., 1999). Alternatively, it has been shown that HPV type 16 (Hennig et al., 1999), HPV type 33 (Yu et al., 1999) and EBV (Bonnet et al., 1999) may also be involved in the genesis of some human breast cancers and may act as cofactors in association with diet, oestrogens and other hormones in genetically susceptible women (Lawson et al., 2001).

It can therefore be assumed that infection-related cancers are probably more frequent in high-income countries than it is generally recognized, all the more so that, in addition to viruses, some microflora bacteria of the gastrointestinal tract, including *Helicobacter pylori* for gastric neoplasia (IARC, 1994a; Ando et al., 2006), some parasites such as *Opisthorchis viverrini* for gallbladder cancers, *Schistosoma haematobium* for bladder cancer and some oncogenic toxins from mycosis-induced moulds inducing aflatoxins-related HCC (IARC, 1994b) and more generally, several types of inflammation (Jackson and Loeb, 2001) have been clearly recognized as risk factors of carcinogenesis.

2. Radiations

Overall, it may be speculated that radiation-induced cancers may represent up to 10% of total cases. In fact there is no clear assessment of this population attributable risk, meaning that this estimate needs to be refined. For ionizing radiation, estimates of the magnitude of the risk in relation to dose have long been based on studies of the incidence of cancers following medical irradiations or exposure to radiation from the atomic bombs at Hiroshima and Nagasaki (UNSCEAR, 2000).

Radiation-induced cancers are a stochastic late effect of ionizing or non-ionizing radiation. They include some leukemia and lymphoma, thyroid cancers, skin cancers, some sarcomas and some lung and breast carcinomas (Wakeford, 2004). Studies of radiation-exposed populations were initially based on occupational exposures and included those concerning radiologists, underground miners and radium dial painters. This began to change with the study of the survivors of atomic bombs at Hiroshima and Nagasaki. In the latest follow-up of atomic bomb survivors total malignancies were increased (Preston et al., 2004). Moreover, studies of patients treated by radiotherapy (Belpomme et al., 1974) and of populations specifically exposed for occupational reasons were carried out in an attempt at defining a causal relationship. These studies presently focus on radiation technologists, uranium miners and nuclear industry workers (UNSCEAR, 2000).

Findings based on the study of lung cancer deaths associated with exposure to radon and improved understanding regarding

the molecular basis of radon-induced cancers have provided support for incriminating low radon levels in the home environment as a cause of approximately 10% of lung cancers (Lubin and Boice, 1997; Darby et al., 2005). Exposure to radon and radon decay products at home and/or at workplace are the most widely found sources of exposure to ionizing radiation (Akerblom, 1999). In contrast, contribution of human nuclear activities to radiation exposure of the general population, although generally estimated to be small and within the variations in background radioactivity are nevertheless of serious concern (UNSCEAR, 1988; Lubin et al., 1994). Recent scientific concern appeared due to the numerous atmospheric nuclear explosions and the incidental exposure following some nuclear accident at power plants that occurred since the last world war. Indeed, artificial radioactivity adds to natural radioactivity in proportions that depend on localization and type of emission, geographic distribution of radioactive substances and type of radioelements. Moreover, dose-response curves for the incidence of cancer, plotting the incidence as a function of dose are extremely complex. Radiation-induced cancers in humans depend on several variables, most of which are not possible to correct for in epidemiologic studies and as for low dose chemicals low doses of ionizing radiation cannot be considered insignificant for risks of somatic and heritable mutations (Prasad et al., 2004). Among other parameters to be considered are the synergistic interactions of radiation with other physical, chemical or biological carcinogens. Studies of cancer risks from nuclear power plant accidents are thus a challenge. Nevertheless, an increased incidence of total malignancies after the Chernobyl radioactive fallout was reported in Sweden (Tondel et al., 2006).

Non-ionizing radiations comprise ultraviolet (UV) rays, which have been rated as carcinogenic to humans by IARC (1992). Exposure to UV is a dose-dependent risk factor (Kopf et al., 1984; Gallagher et al., 2005), which can cause skin cancers, mostly basal cell and squamous cell carcinoma (Henriksen et al., 1990; Urbach, 1991; Feychting et al., 1995; Foliart et al., 2006) and can be also involved in carcinogenesis of melanoma (IARC, 1992) and consequently, in its current growing incidence. Increase in lifestyle-related natural or artificial (sun beds) exposure to UV has been incriminated to account for the growing incidence of melanoma. However, depletion of the stratospheric ozone layer by augmenting the dose intensity of UV-B or C to the skin (Cutchis, 1974) is another factor that could also contribute to the growing incidence of skin cancers (Kelfkens et al., 1990; De Fabo, 2005).

Electromagnetic fields (EMF) of very low frequency (VLF) or extremely low frequency (ELF) have been the object of many scientific research efforts to answer the question whether they can induce cancers. For a long time, many scientists were doubtful about the role of EMF in inducing cancers. This was mainly due to the difficulty in assessing a causal link between long-term exposure to weak EMF and cancer occurrence and because although it has been observed that pulsed EMF can be clastogenic by breaking DNA (Haider et al., 1994), the mechanism whereby VLF or ELF could induce cancer is not clear. To date many studies correlating EMF to childhood acute leukemia have been reported (Ahlbom et al., 2001; IARC, 2002a). Despite differences in study design and setting, results are sufficiently consistent to believe that an increased risk of leukemia does exist in children with high exposure. Indeed, from a meta-analysis of nine studies, it has been estimated that there is a relative risk of acute leukemia of 2 for children living in area associated with an average EMF strength above 0.4 μT and that the EMF-related relative risk of childhood leukemia is 2 for about 1% of the overall children population (Ahlbom et al., 2000). These data have been confirmed by a more extensive meta-analysis (Greenland et al., 2000). Moreover, it has been recently confirmed that children living within a distance of 200 m from high voltage power lines have a relative risk increase of leukemia of 69%, while those born between 200 and 600 m have a relative increase risk of leukemia of 23%. In this study, there was a significant dose-effect dependency in relative risk in relation to the reciprocal of distance from the line (Draper et al., 2005). Other epidemiological studies revealed that EMF and mostly ELF could be also associated with brain tumors (Mack et al., 1991; Beall et al., 1996; Guenel et al., 1996; Feychting et al., 1997), breast carcinoma (Guenel et al., 1996; Feychting et al., 1997; Wakeford 2004) and melanoma (Hallberg and Johansson, 2002; Levi et al., 2006). The most visible source of ELF–EMF is power lines, in particular high voltage transmission lines, but other sources include transformers, electric train engines and more generally all types of electrical equipment. In a series of recent epidemiological studies, one of us has shown that long-term cellular or cordless phone use is also a risk factor for brain tumors (Hardell and Hansson Mild, 2006; Hardell et al., 2006a), whereas several previous studies were negative. This positive result has been recently challenged by an updated Danish cohort analysis, showing no correlation between cellular phone use and cancer risk (Schuz et al., 2006). However, results from this study are presumably limited, because there was no consideration about dose effect, i.e. the search for a correlation between duration of exposure and cancer risk. Indeed, in a recent meta-analysis of all available epidemiologic data, one of us has shown that daily prolonged use of mobile phones associated with a long-term use of it for $\geqslant$10 years give a consistent pattern of an increase risk of brain tumors including neuroma and glioma and that the risk is highest for ipsilateral exposure (Hardell et al., 2007). Moreover, it is assumed that X-rays-related medical imaging and magnetic resonance imaging (MRI) can induce a low number of cancer cases in selected populations (IARC, 2000b; Nitta et al., 2002).

Finally, it is concluded that in addition to ionizing irradiation, non-ionizing irradiation including UV and

pulsed EMF can cause cancers, in particular, skin cancers (UV) some acute leukemia and brain tumors in children (EMF). An explanation could be that EMF may be mutagenic *per se* or induce cellular epigenetic changes and thus contribute to cancer induction. Such a mechanism could be in fact complex. For example, it has been recently shown that corona ions induced by powerlines and increased chemical exposure to pollutant aerosols may be involved in carcinogenesis (Fews et al., 1999a, b).

3. Xenochemicals

The industrial revolution over the second half of the last century and its consequences in domains such as energy, transport, agriculture, food and health led to synthesize, produce and introduce into the environment, millions of man-made chemicals or substances. As a result, according to the European commission, about 100,000 chemicals have been so far marketed, since the last world war, without sufficient toxicological control. Such products can act as persistent toxic pollutants and contaminate air, soil, water and food. Many of them are carcinogenic, mutagenic and/or reprotoxic (CMR) molecules (Clapp et al., 2005) and therefore can act as mutagens, promoters or both or be cocarcinogenic, meaning that they can contribute to the genesis of cancers and therefore account for their currently growing incidence (Epstein, 1994, 2004).

3.1. Occupational cancers

Since the seminal report of Percival Pott in 1775, cancers of the scrotum were the first recognized occupational chemically-induced cancers. Today, occupational cancers are reported to represent 2–10% of all cancers, but this percentage is probably underestimated and may be as high as 15–20% in men. In 1996, the Harvard Center for Cancer Prevention (HCCP) classified 32 substances or industries as carcinogenic in humans (HCCP, 1996). Recently, 28 agents have been considered as definite occupational carcinogens in human, 27 as probable occupational carcinogens and 113 as possible occupational carcinogens (Siemiatycki et al., 2004; Clapp et al., 2005). Doll and Peto (1981) had listed only 16 in 1981. In Europe, there could be 32 million people exposed to hazardous carcinogenic chemicals at work (Kauppinen et al., 2000). Among substances, asbestos is a classical example. It is a calcium and magnesium hydrate silicate that comes from the transformation of the mineral. It was widely used owing to its fireproofing and heat-retaining properties. There is no doubt that asbestos is carcinogenic and induces occupational cancers, including mesothelioma and approximately 10% of lung cancers (IARC, 1977, 2002b). Although asbestos was recognized as carcinogenic one century ago in the UK, its legal use was forbidden in Europe just about one or two decades ago. Likewise, wood-dust-related cancers, though their occurrence in joiners or cabinetmakers is limited, are also occupational cancers, insufficiently declared (ethmoïd cancers) or even not yet declared (sinus cancers) (Hayes et al., 1986; Blot et al., 1997), although they are both acknowledged as occupational cancers by IARC (1995b). Solvents, paints, dyes, gasoline and other petroleum products can also cause occupational cancers. After the leukemogenic effect of benzene was first recognized (Goguel et al., 1967; Surralles et al., 1997), the mutagenic effect of other solvents was established. For example, trichloroethylene (TCE) has been reported to be strongly associated with kidney, liver, esophageal cancers and non-Hodgkin's lymphoma (IARC, 1995c; ASTDR, 1997; Wartenberg et al., 2000; Hansen et al., 2001; Wartenberg and Siegel Scott, 2002; Raaschou-Nielsen et al., 2003) and perchloroethylene (PCE) with oesophageal cancers (Ruder et al., 1994; Weiss, 1995). Likewise, dye products or byproducts of aromatic amines and/or aminophenol groups are strongly associated with bladder cancer (Cantor et al., 2006). Moreover mineral oils and lubricants have been associated with some types of cancers, including larynx, skin and bladder cancers (Kane et al., 1984; IARC, 1984, 1989; Hewstone, 1994; Tolbert, 1997; Mackerer et al., 2003). Phthalates are widely used since the last world war, due to their plasticizing and emulsifying properties. For this reason, they are added to polyvinyl chloride (PVC) in particular in common medical devices and cosmetics. Phthalates are the cause of repeated abortions in rats and mice (Saillenfait et al., 2003, 2006) and of sterility in man (Latini et al., 2006). In addition, di(2-ethylhexyl)phthalate (DEHP) and butyl-benzyl-phthalate have been suspected to be carcinogenic (Shea, 2003). Some phthalates may therefore be potential CMR substances due to reprotoxic and carcinogenic properties. Toxicological studies in rodents indicate that DEHP exposure can cause liver cancer (Rao et al., 1990) and pancreatic tumors (David et al., 1997). Three studies in humans revealed an elevated pancreatic cancer risk in workers in flexible PVC processing (Chiazze and Ference, 1981; Dell and Teta, 1995; Selenskas et al., 1995). IARC initially classified DEHP as a 2B possible carcinogen to humans, but subsequently downgraded it to a non-classifiable grade 3 molecule (IARC, 2000a). However, none of the studies indicating elevated risk of pancreatic cancers in humans exposed to DEHP were considered (Melnick et al., 2000). Thus the IARC evaluation on DEHP, because it omitted critical and available information from relevant animal and human studies, was criticized by many scientists to such an extent that they asked IARC to schedule a new thorough and unbiased DEHP evaluation. In addition, vinylchloride monomer, but not PVC is mutagenic and thus can cause liver angiosarcoma and hepato-cellulars-carcinoma (HCC) (Kielhorn et al., 2000). Other occupational risks are related to many industrial substances and particularly to agrochemicals, more precisely to the use of pesticides by selected population of workers in agriculture and agrochemical industry (see further).

A major concern is the risk of childhood cancers following either parental or child exposure to occupational pollutants. Several studies, evaluating the effect of parental

exposure to solvents, paints, gasoline, gasoline exhaust confirmed an increased risk of leukemia and brain tumors in children (Wilkins and Koutras, 1988; Savitz and Chen, 1990; Smulevich et al., 1999; O'Leary et al., 1991; Feingold et al., 1992) to such an extent that in 1998, Colt and Blair (1998) wrote that there is "sufficient evidence that certain parental exposures may be harmful for children". However, how these different types of mutagenic chemicals or substances relate to the general population is still under investigation and needs further research efforts. Indeed, many mutagenic synthetic products, organochlorines or not, used to manufacture adhesives, resins, coatings, paints, dyes have been marketed over the post-war period without sufficient toxicological investigations and regulatory control.

3.2. Outdoor air pollution

Polycyclic Aromatic Hydrocarbons (PAH) resulting from the combustion of organic substances are mutagens (IARC, 1989). They are found in tobacco smoke as well as in factory smoke, waste incinerator emission and vehicle exhaust. They can adhere on fine carbon particles (PM 2.5) suspended in the air and thus penetrate the organism more extensively because particles accumulate near the ground at the level we breathe. In adults, long-term exposure to particles and so to PAH, i.e. in air of polluted cities increases the risk of death due to lung cancer by 8%, after controlling for tobacco smoking (Dockery et al., 1993; Pope et al., 2002; Cohen, 2003). In 2002, the American Cancer Society published the results of the widest survey carried out to date on 500,000 people residing in American cities: the higher the concentration of fine particles in the air, the higher the number of deaths due to lung cancer. This risk is significantly higher for the more polluted cities than for the less polluted ones, and smoking potentiates that effect (Pope et al., 2002). Moreover, in a recent European study, the proportion of lung cancers attributable to traffic-related air pollution and environmental tobacco smoke (ETS) in never- and ex-smokers was precisely estimated to be 5–7% and 16–24%, respectively (Vineis et al., 2007). In addition to PAH and fine and ultrafine carbon particles the air pollutant nitrogen dioxide (NO_2) may also be involved. NO_2 is the expression of a mixture of particles and gases related to traffic, power plants and/or waste incinerator emission. Experimental studies have shown that NO_2 can promote lung cancer induction (Ichinose et al., 1991; Ohyama et al., 1999) and facilitate metastatic dissemination (Richters and Kuraitis, 1983) and that after NO_2 combine with benzo[a]pyrene, the resulting product i.e. nitrobenzo[a]pyrene, is characterized by an increased mutagenicity (Tokiwa et al., 1994). In the previous epidemiological study, it was observed that higher exposure to NO_2 increases the risk of lung cancer in non-smokers (Vineis et al., 2007). Because they have more rapid rate of respiration and so inhale more pollutants per kg of body weight than adults, children are more susceptible to air pollution. Traffic exhaust is a major cause of children's exposure to air pollutants (IARC, 1989) and several authors in US as well as in Europe found a positive correlation between local traffic density at time of diagnosis and childhood leukemias with an estimated relative risk between 1.6 and 4.7 (Wertheimer and Leeper, 1979; Savitz and Feingold, 1989; Feychting et al., 1998; Harrison et al., 1999).

3.3. Indoor air pollution

Indoor air pollution is a growing scientific concern because indoor air can accumulate many carcinogenic pollutants. In addition to carbon particles and PAH, indoor air can accumulate ETS as well as chemicals such as biocides and also formaldehyde in addition to volatile organic compounds (VOC) such as benzene and 1,3 butadiene, which have been rated as carcinogens by IARC (1995b). The risk of lung cancer due to ETS exposure is much higher at work than at home and higher for ex-smokers than for never smokers (Vineis et al., 2007). Since children are most affected by chronic household exposure, they may be at higher risk of cancer. Particularly of concern is the association between parental and/or childhood exposure and the risk of childhood cancers and cancers in adulthood. In addition to an increased risk of adulthood lung cancer in children exposed to ETS (Vineis et al., 2007). several epidemiological studies have shown that indoor air pollution can be an important contributing risk factor for childhood cancers (Zhang and Smith, 2003). There is an increased relative risk of leukemia and lymphoma caused by indoor VOC (Smith, 1987; Weaver et al., 1998; Viegi et al., 2004) as well as by the indoor use of insecticides (Infante-Rivard et al., 1999; Meinert et al., 2000; Menegaux et al., 2006).

3.4. Biocides and pesticides

Over the last decades, several hundreds of pesticides have been marketed for intensive farming or domestic use (biocides). Many of them especially those bearing to the organochlorines, carbamates and carbinols groups are rated as *probable* or *possible* carcinogens, according to the US EPA and the IARC classification (IARC, 1991) while several are recognized as carcinogens in humans. Most of them having a molecular structure close to estrogens are endocrine disruptors. They may thus act as promoters, while others can in addition be mutagenic. Moreover many of them are also strong immunosuppressors, meaning that they can act also as promoters through immunosuppression (Repetto and Baliga, 1997; Hayes et al., 2006). In children, several epidemiological studies revealed an increased relative risk of cancers associated with parental exposure to pesticides be it occupational or non-occupational (Daniels et al., 1997; Zahm and Ward, 1998). The exposure observed occurs prior to and during pregnancy as well as post-natally. Paternal exposure to

pesticides is associated with an excess relative risk of leukemia (Ma et al., 2002), and of central nervous system (CNS) tumors (Cordier et al., 2001; Feychting et al., 2001) as well as of Wilm's tumors (Fear et al., 1998). Moreover a positive association has been found in several studies testing the direct exposure of children to pesticides (Daniels et al., 1997; Zahm and Ward, 1998; US-EPA, 2003; Menegaux et al., 2006). Collectively, these studies revealed an overall increase in relative risk of leukemia, NHL, brain tumors, Wilm's tumors, Ewing's sarcoma and germ cell tumors associated with parental or child exposures to pesticides. By contrast, in adults, epidemiological studies have provided conflicting results. A positive link between pesticides and breast or prostate cancers has been put forward (Charlier et al., 2003; Muir et al., 2004; Ibarluzea et al., 2004; Mills and Yang, 2006), whereas in other studies it cannot be confirmed (Post et al., 1999; Stellman et al., 2000; Gammon et al., 2002; Van Maele-Fabry and Willems, 2003). However, a strong association between pesticides and the relative risk of sarcoma (Hardell et al., 1995; Dich et al., 1997) and of Hodgkin and NHL (Hardell and Eriksson, 1999; Zheng et al., 2001; McDuffie et al., 2001; Hardell et al., 2003) has been found for 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane (DDT), chlorophenols and phenoxyherbicides. Likewise an increased risk of adult leukemia has been shown for carbon disulfide, phosphine (such as PH_3) and methylbromide (Dich et al., 1997). Indeed, due to their CMR effects, organochlorine pesticides are today almost universally banned in most industrialized countries. The danger of pesticides lies in the fact that as Persistent Organic Pollutants (POPs), they can persist over long periods in the environment and contaminate drinking water and food (Dougherty et al., 2000). Food component and ingredient analysis remains one of the most pertinent application of chemistry. However, policy is now insufficient to control pesticide use in many countries while the relevance of pesticides in carcinogenesis is steadily increasing. Indeed, pesticides can contaminate the body not only through ingestion, but also through air inhalation and skin contact, accumulate in adipose tissue (Lassiter and Hallam, 1990; Geyer et al., 1990, 1997), more specifically in fatty breast tissue (Muscat et al., 2003; Zou and Matsumura, 2003), pass through the placenta (Simonich and Hites, 1995) and accumulate in the milk of nursing mothers (Sharpe and Irvine, 2004). This led to the conclusion that children are at risk during three periods: *in utero* during pregnancy, during breast feeding and in the course of childhood, by inhaling biocides at home or by ingesting contaminated food (Guvenius et al., 2003; Menegaux et al., 2006). During recent years there has been an increasing incidence of male developmental reproductive disorders. These diseases have been grouped together in the testicular dysgenesis syndrome and include testicular cancer, cryptorchidism, hypospadias and low sperm count (Skakkebæk et al., 2001). Exposure to endocrine disrupting chemicals during foetal period has been postulated to be a risk factor. In a case-control study concentrations of certain persistent organic pollutants such as polychlorinated biphenyls were higher in mothers of young men with testicular cancer compared with mothers of controls (Hardell et al., 2006b).

3.5. Dioxins and other organochlorines

Organochlorines are CMR molecules, which result either from the combustion of organic substances in contact with chlorine or from industrial chemical synthesis. Examples are dioxins, which may be produced from the incineration of halogenated plastics (such as PVC), pesticides such as hexachlorobenzene (HCB) and Polychlorinatedbiphenyls (PCB). On the basis of a myriad of in vitro and animal studies, it has been clearly shown that these molecules are endocrine disruptors, may interfere with the developmental process *in utero* and can cause cancer through a promoting or cocarcinogenic effect (Stohs, 1990; Schiestl et al., 1997; Amaral-Mendes, 2002; Mandal, 2005). Moreover, some of them, such as PCB and dioxins have been shown in addition to be mutagenic (Giri, 1986; Schiestl et al., 1997). In 1979, following the Seveso accident, IARC rated 2-3-7-8 tetrachlorodibenzo-*p*-dioxin (TCDD) as a 2B possible human carcinogen (IARC, 1979), but this categorisation was upgraded to a recognized class 1 carcinogen in humans in 1997 (IARC, 1997b). Since then, some cluster studies have shown an increased relative risk of sarcoma and lymphoma in the vicinity of incinerators and related this increased risk to dioxin emission (Mukerjee, 1998; Viel et al., 2000; Floret et al., 2004). Although the carcinogenic mechanism of dioxins is not clear, a strong association between chronic exposure and an increased incidence of all cancer types has been put forward (IARC, 1997b) indicating the important and extensive spectrum of their carcinogenic effect. Other suspected carcinogenic organochlorines deal with the problem of long term exposure to chlorinated water which has been shown to be associated with an increased risk of cancer including bladder cancer (Cantor et al., 1987; Zierler et al., 1988; King and Marrett, 1996; Cantor et al., 1998; Villanueva et al., 2003, 2004), colorectal cancers (Hildesheim et al., 1998; King et al., 2000) and adult leukemia (Kasim et al., 2006). Chlorine is not itself carcinogenic but can combine with organic matter in drinking water and form chlorination disinfection by-products including trihalomethanes and haloacetic acids which have been demonstrated to be mutagenic *in vitro* and *in vivo* (Geter et al., 2004) and carcinogenic in animal models (Melnick et al., 2007). Finally, the danger of organochlorines lies in the fact that, as pesticides, they are POP and accumulate in the body's adipose tissue from which they are permanently released.

3.6. Food contaminants and food additives

Nitrates, pesticides and dioxins can contaminate drinking water and food. As pesticides, nitrates are used in intensive farming. Nitrates are not intrinsically carcinogenic, but can

be endogenously transformed into nitrites by the digestive bacterial microflora, while nitrites can be further transformed into *N*-nitroso compounds (NOC), i.e. into alkyl-nitrosamines and nitrosamides through nitrosation (Tannenbaum et al., 1980; Ward et al., 2005). Nitrites, alkyl-nitrosamines and nitrosamides are highly mutagenic molecules. Secondary or tertiary amines and amides are found as common dietary contaminants (Ward et al., 2005). Long-term exposure to food additives, including nitrite preservatives and artificial azo-dyes may be also involved in chemically- induced carcinogenesis, due to their mutagenic properties (Palmer and Mathews, 1986; Weisburger, 1986; Sasaki et al., 2002). In addition, Bisphenol A, a xenoestrogen used in plastic food containers, because it can migrate in food and be repetitively ingested, has been recently suspected to be carcinogenic in humans on the basis of results obtained from animal studies aiming at reproducing breast (Durando et al., 2007) and prostate cancer genesis (Ho et al., 2006; Prins et al., 2007). This led to the hypothesis that in association with individual diet and nutrition lifestyle-related factors, chronic exposure to food contaminants and food additives might be involved in carcinogenesis more frequently than it is usually appreciated. Since NOC are powerful transplacental neurocarcinogens (Rice et al., 1989), they may be involved in the recent increase of childhood brain tumors (Dietrich et al., 2005). In adults, a few epidemiologic studies have found an excess consumption of nitrates to be associated with an elevated risk for gastric and nasopharyngeal cancers (Cantor et al., 2006). Moreover, an increased risk of NHL (Ward et al., 1996) and of colon cancer (De Roos et al., 2003) has been put forward in association with drinking water with nitrates' concentration over the regulatory level. However, because the pollution by nitrates impacts the overall population and because the transformation of nitrates into mutagenic NOC depends on many factors, including diet and microflora modifications, their carcinogenic role is difficult to clearly assess through epidemiological studies (Ward et al., 2005). Before changes to the regulatory level for nitrates in drinking water can be considered, further comparative studies analyzing the precise role of nitrates in selected populations thus be performed.

3.7. Metals and metalloids

Several metals and metalloids have been rated as *certain* or *probable* carcinogens by the (IARC, 1980). Inhalation of arsenic oxides can cause lung cancer, but if they are swallowed, cancer can develop in bladder, kidney, liver and lung (Szymanska-Chabowska et al., 2002). Thus exposure to arsenic oxides has been reported to be associated with a very large spectrum of common cancer types. Aside from arsenic oxides exposure, lung cancer has been also reported to be associated with exposure to many metals, including lead, hexavalent chromium and nickel (IARC, 1990). Furthermore, exposure to hexavalent chromium or nickel has been found to be associated with nasopharyngeal carcinoma, exposure to lead or mercury to brain tumors, exposure to lead or cadmium to kidney cancer and exposure to cadmium to prostate cancer (Hayes, 1997; Cocco et al., 1999; Landrigan et al., 2000; Navas-Acien et al., 2002; Wesseling et al., 2002; Waalkes, 2003). The mechanisms of action of metals and metalloids are not clear yet. They could act as cocarcinogens by activating procarcinogens in the liver (Hayes, 1997; Cantor et al., 2006) or by increasing the promoting effect of estrogens (Gurpide et al., 1984). They could also act by replacing the natural enzyme-associated metal, thus inactivating the metabolic pathway of key enzymes. Carcinogenic metals and metalloids, such as arsenic, cadmium and nickel, and putative carcinogens including cobalt and lead can inhibit zinc finger containing DNA repair proteins. Damage of zinc finger in DNA repair proteins can therefore be regarded as a novel mechanism in carcinogenesis (Witkiewicz-Kucharczyk and Bal, 2006). Moreover, some metals and metalloids may also be mutagenic through other mechanisms. Indeed many of them can interact with DNA. Metal compounds such as Chromium(VI) are taken up by cells as chromate anion and are reduced intracellularly via reactive intermediates to stable Cr(III), which can directly interact with DNA. These Cr(III) intermediates may affect DNA by terminating replication or reducing replication fidelity thus leading to mutations (Snow, 1991, 1994). Cr(III) can also form DNA-proteins and DNA-aminoacids and glutathione crosslinks (Zhitkovich, et al., 1995, 1996). Platinum compounds (i.e. *cis*-diaminedichloroplatinum) are well-known strand breakers. They can form DNA crosslinks and DNA-protein crosslinks leading to mutations (Zwelling et al., 1979; Donahue et al., 1990). There is evidence that nickel may act via an epigenetic mechanism involving heterochromatic regions of the genome (Costa, 1991; Salnikow et al., 1994). However, how contamination of the environment by metals and metalloids may relate to the actual growing incidence of cancer is still under investigation.

3.8. Medicines and cosmetics

The best acknowledged class of pharmaceutical drugs classified as carcinogens corresponds to hormonal products, which include oral contraceptives and hormone replacement therapy (IARC, 1996b; Beral, 2003), as well as selected anti-estrogens such as tamoxifen (IARC, 1996b). Other carcinogenic medicines include many anticancer chemotherapeutic agents which can cause the late occurrence of secondary cancers in apparently cured cancer patients due to their mutagenic properties (Belpomme et al., 1974; Etiemble et al., 1979; Zahm and Devesa, 1995). However, the ratio between benefit and toxicity is so high that the use of chemotherapeutic agents in oncology is not questioned although efforts are made to adapt the dose or look for replacement products. By contrast the question is more difficult for drugs given to healthy persons. A serious

risk-benefit analysis has to be carried out for women, be they in search of contraception, management of menopausal symptoms by using substitutive treatments or prevention of breast cancer by tamoxifen. Several years ago, some anticancer agents were found to contaminate industrial workers involved in drug making and manufacturing, and nurses working in oncology departments, so that legal protection has followed, prohibiting their use by pregnant women (Sorsa and Anderson, 1996; Sorsa et al., 1985). A pending question remains, however, release of these mutagenic drugs in the environment through urine. Cosmetics which include some recognized carcinogenic molecules such as formaldehyde or hormonal products or some putative ones such as phthalates or parabens have been presently the object of many concerns. Parabens in underarm deodorants has been suspected to be an etiological factor of breast cancer (Darbre, 2001, 2003). The hypothesis was based on observations showing a disproportionately high incidence of breast cancer in the upper outer quadrant of the breast and that the left breast is more prone to the development of cancer than the right one in women as well as in men. However, this hypothesis is not validated yet and to our knowledge, no formal study has been designed and carried out to clarify the role of parabens. By contrast, it has been found that permanent hair-dyes, containing aromatic amines may increase the relative risk of bladder cancer by 3.3-fold among regular users relative to non-users (Gago-Dominguez et al., 2001), but by 1.22–1.50 in a more recent meta-analysis, indicating that personal use of hair-dyes increases bladder cancer risk only by 22–50% (Huncharek and Kupelnick, 2005). In addition, it has been shown that permanent hair-dyes increase the relative risk of adult acute leukaemia by 2.4 in women for a use of up to six times per year for more than 15 years (Rauscher et al., 2004). Although these data have been recently challenged (Takkouche et al., 2007), they have been confirmed in a systematic review of the scientific literature published since 1992: positive associations between personal hair-dye use and bladder cancer, acute leukemia, lymphoma and myeloma have been clearly observed in well-designed studies which have suggested that negative results may in fact be explained by differences in susceptibility to exposures (Rollison et al., 2006).

Table 2
Classification of some exogenous environmental factors according to their carcinogenic and cocarcinogenic properties

	Mutagen	Promoter	*Cocarcinogen*
Microorganisms			
EBV	M		
HBV/HCV	M	P	
HHMMTV	M		
HHV-8			C (?)
HIV		P	
HTLV-1	M		
HPV	M		
Helicobacter Pylori			C
Radiations			
Radioactivity	M	P	
UV	M	P	
EMF	M	P (?)	C
Particles and Xenochemicals			
Air fine particles[a]			C
Asbestos	M		C
Azoïc dyes	M		
Bisphenol A	M (?)	P	
β Naphylamine	M		
Benzene and derived molecules	M		C
DEHP, BBP	M	P	
Dioxins		P	
Formaldehyde and derived	M		
Hormonal residues		P	
Metals, metalloids	M		C
N-nitroso compounds[b]	M		
NO_2		P	C
Organochlorines	M (some ?)	P	
PAH[c]	M ($\geqslant$5 rings)	P ($<$5 rings)	C
PCB	M (some)	P	
Pesticides[d]	M (some)	P	
Vinyl chlorides (monomers)	M		

[a]Air carbon particles, especially PM$<$2.5 are vectors of chemicals, including PAH and organochlorines (pesticides).
[b]Nitrates, Nitrites, Nitrosamines, Nitrosamides.
[c]PAH of high molecular weight (5–7 rings) induce DNA adduction processes and so are mutagenic, while PAH with low molecular weight (3–4 rings) are non genotoxic promoters (Trosko and Upham, 2005).
[d]Act usually as endocrine disruptors or immunosuppressors (promoters), but some of them can be also mutagenic (see text).

4. Conclusion

Finally, from this overview, although there are still some persisting areas of uncertainty, it clearly appears that due to their mutagenic and/or promoting properties or to their cocarcinogenic effects, many exogenous environmental factors, including viruses, radiations and xenochemicals can contribute to cause a variety of cancers. In Table 2, we summed up most of the recognized environmental factors that are considered to be involved in carcinogenesis and tried to establish the weight of evidence according to which they can express their carcinogenic (i.e. mutagenic and/or promoting) or cocarcinogenic effects. This analysis could open a new way in considering the actual causes of cancers: it remains indeed to estimate the overall risk fraction for cancer attributable to these environmental factors.

References

Ahlbom, A., Day, N., Feychting, M., Roman, E., Skinner, J., Dockerty, J., Linet, M., McBride, M., Michaelis, J., Olsen, J.H., Tynes, T., Verkasalo, P.K., 2000. A pooled analysis of magnetic fields and childhood leukaemia. Br. J. Cancer. 83, 692–698.

Ahlbom, A., Cardis, E., Green, A., Linet, M., Savitz, D., Swerdlow, A., 2001. Review of epidemiological literature on EMF and health. Environ. Health Perspect. 109, 911–933.

Akerblom, G., 1999. Radon legislation and national guidelines. Swedish Radiation Protection Institute SSI Rapport: 99, 18.

Alexander, F.E., Chan, L.C., Lam, T.H., Yuen, P., Leung, N.K., Ha, S.Y., Yuen, H.L., Li, C.K., Lau, Y.L., Greaves, M.F., 1997. Clustering of childhood leukaemia in Hong Kong: association with the childhood

peak and common acute lymphoblastic leukaemia and with population mixing. Br. J. Cancer. 75, 457–463.
Amaral-Mendes, J.J., 2002. The endocrine disrupters: a major medical challenge. Food Chem. Toxicol. 40, 781–788.
Ando, T., Goto, Y., Maeda, O., Watanabe, O., Ishiguro, K., Goto, H., 2006. Causal role of *Helicobacter pylori* infection in gastric cancer. World J. Gastroenterol. 12, 181–186.
ASTDR (Agency for Toxic Substances and Disease RegistryToxicological Profile for Trichloroethylene), 1997. US Department of Health and Human Services. US Public Health Service, Atlanta, Georgia.
Beall, C., Delzell, E., Cole, P., Brill, I., 1996. Brain tumors among electronics industry workers. Epidemiology 7, 125–130.
Belpomme, D., Le Borgne de Kaouel, C., Ajuria, E., Jasmin, C., Dore, J.F., 1969a. Increase in antigenicity of permanent tissue culture lines ICI 101, ICI 104 and ICI 202 established from human leukaemic blood. Nature 222, 890.
Belpomme, D., Seman, G., Dore, J.F., Venuat, A.M., Berumen, L., Le Borgne de Kaouel, C., Mathe, G., 1969b. Established cell line (ICI-101) obtained from frozen human lymphoblastic leukemia cells: morphological and immunological comparison with the patient's fresh cells. Eur. J. Cancer. 5, 55–59.
Belpomme, D., Carde, P., Oldham, R.K., Mathe, G., Jacquillat, C., Chelloul, N., 1974. Malignancies possibly secondary to anticancer therapy. In: Mathe, G., Oldham, R.K. (Eds.), Recent Results in Cancer Research, Vol. 49. Springer, Berlin, Heidelberg, New York.
Belpomme, D., Irigaray, P., Sasco, A.J., Newby, J.A., Howard, V., Clapp, R., Hardell, L., 2007. The growing incidence of cancer: role of lifestyle and screening detection (Review). Int J Oncol 30, 1037–1049.
Belson, M., Kingsley, B., Holmes, A., 2007. Risk factors for acute leukemia in children: a review. Environ. Health Perspect. 115, 138–145.
Beral, V., 2003. Million women study collaborators: breast cancer and hormone replacement in the million women study. Lancet 362, 419–427.
Blot, W.J., Chow, W.H., McLaughlin, J.K., 1997. Wood dust and nasal cancer risk. A review of the evidence from North America. J. Occup. Environ. Med. 39, 148–156.
Blumberg, B.S., Larouze, B., London, W.T., Werner, B., Hesser, J.E., Millman, I., Saimot, G., Payet, M., 1975. The relation of infection with the hepatitis B agent to primary hepatic carcinoma. Am. J. Pathol. 81, 669–682.
Bonnet, M., Guinebretiere, J.M., Kremmer, E., Grunewald, V., Benhamou, E., Contesso, G., Joab, I., 1999. Detection of Epstein-Barr virus in invasive breast cancers. J. Natl. Cancer Inst. 91, 1376–1381.
Boutou, O., Guizard, A.V., Slama, R., Pottier, D., Spira, A., 2002. Population mixing and leukaemia in young people around the La Hague nuclear waste reprocessing plant. Br. J. Cancer. 87, 740–745.
Brooks, L.A., Crook, T., Crawford, D.H., 1999. Epstein-Barr virus and lymphomas. In: Newton, R., Beral, V., Weiss, R.A. (Eds.), Infections and Human Cancer. Cancer Surveys, Vol. 33. Cold Spring Harbor Laboratory Press, New York, pp. 99–123.
Callahan, R., Drohan, W., Tronick, S., Schlom, J., 1982. Detection and cloning of human DNA sequences related to the mouse mammary tumor virus genome. Proc. Natl. Acad. Sci. USA 79, 5503–5507.
Cantor, K.P., Hoover, R., Hartge, P., Mason, T.J., Silverman, D.T., Altman, R., Austin, D.F., Child, M.A., Key, C.R., Marrett, L.D., 1987. Bladder cancer, drinking water source, and tap water consumption: a case-control study. J. Natl. Cancer Inst. 79, 1269–1279.
Cantor, K.P., Lynch, C.F., Hildesheim, M.E., Dosemeci, M., Lubin, J., Alavanja, M., Craun, G., 1998. Drinking water source and chlorination byproducts. I. Risk of bladder cancer. Epidemiology 9, 21–28.
Cantor, K.P., Ward, M.H., Moore, L., Lubin, J., 2006. Water contaminants. In: Schottenfeld, D., Fraumeni, Jr., F. (Eds.), Cancer Epidemiology and Prevention, third ed. Kluwer Academic Publisher, Dordrecht, pp. 149–169.
Charlier, C., Albert, A., Herman, P., Hamoir, E., Gaspard, U., Meurisse, M., Plomteux, G., 2003. Breast cancer and serum organochlorine residues. Occup. Environ. Med. 60, 348–351.
Chiazze, L., Ference, L.D., 1981. Mortality among PVC-fabricating employees. Environ. Health Perspect. 41, 137–143.
Clapp, R., Howe, G., LeFevre, M., 2005. Environmental and occupational causes of cancer: review of recent scientific literature. The Lowell Center for Sustainable Production, University of Massachusetts Lowell. Available at: 〈http://www.sustainableproduction.org/publ.shtml〉.
Cocco, P., Heineman, E.F., Dosemeci, M., 1999. Occupational risk factors for cancer of the central nervous system (CNS) among US women. Am. J. Ind. Medv. 36, 70–74.
Cohen, A.J., 2003. Air pollution and lung cancer: what more do we need to know? Thorax 58, 1010–1012.
Colt, J.S., Blair, A., 1998. Parental occupational exposures and risk of childhood cancer. Environ. Health Perspect. 106, 909–925.
Cordier, S., Mandereau, L., Preston-Martin, S., Little, J., Lubin, F., Mueller, B., Holly, E., Filippini, G., Peris-Bonet, R., McCredie, M., Choi, N.W., Arsla, A., 2001. Parental occupations and childhood brain tumors: results of an international case-control study. Cancer Causes Control 12, 865–874.
Costa, M., 1991. Molecular mechanisms of nickel carcinogenesis. Annu. Rev. Pharmacol. Toxicol. 31, 321–337.
Cutchis, P., 1974. Stratospheric ozone depletion and solar ultraviolet radiation on earth. Science 184, 13–19.
Daniels, J.L., Olshan, A.F., Savitz, D.A., 1997. Pesticides and childhood cancers. Environ. Health Perspect. 105, 1068–1077.
Darbre, P.D., 2001. Under arm cosmetics and breast cancer: hypothesis. Eur. J. Cancer Prev. 10, 389–393.
Darbre, P.D., 2003. Underarm cosmetics are a cause of breast cancer. J. Applied Toxicol. 23, 89–95.
Darby, S., Hill, D., Auvinen, A., Barros-Dios, J.M., Baysson, H., Bochicchio, F., Deo, H., Falk, R., Forastiere, F., Hakama, M., Heid, I., Kreienbrock, L., Kreuzer, M., Lagarde, F., Makelainen, I., Muirhead, C., Oberaigner, W., Pershagen, G., Ruano-Ravina, A., Ruosteenoja, E., Rosario, A.S., Tirmarche, M., Tomasek, L., Whitley, E., Wichmann, H.E., Doll, R., 2005. Radon in homes and risk of lung cancer: collaborative analysis of individual data from 13 European case-control studies. BMJ 330, 223–228.
David, R.M., Moore, M.R., Finney, D.C., 1997. Chronic toxicity of di(ethylhexyl)phthalate in rats. Toxicol. Sci. 55, 433–443.
De Fabo, E.C., 2005. Arctic stratospheric ozone depletion and increased UVB radiation: potential impacts to human health. Int. J. Circumpolar Health. 64, 509–522.
De Maria, N., Colantoni, A., Fagiuoli, S., Liu, G.J., Rogers, B.K., Farinati, F., Van Thiel, D.H., Floyd, R.A., 1996. Association between reactive oxygen species and disease activity in chronic hepatitis C. Free Radical. Biol. Med. 21, 291–295.
De Roos, A.J., Ward, M.H., Lynch, C.F., Cantor, K.P., 2003. Nitrate in public water supplies and the risk of colon and rectum cancers. Epidemiology 14, 640–649.
De The, G., 1995. Viruses and human cancers: challenges for preventive strategies. Environ. Health Perspect. 103, 269–273.
Dell, L., Teta, M.J., 1995. Mortality among workers at a plastics manufacturing and research and development facility. 1946–1988. Am. J. Ind. Med. 28, 373–384.
Dich, J., Zahm, S.H., Hanberg, A., Adami, H.O., 1997. Pesticides and cancer. Cancer Causes Control 8, 420–443.
Dietrich, M., Block, G., Pogoda, J.M., Buffler, P., Hecht, S., Preston-Martin, S., 2005. A review: dietary and endogenously formed N-nitroso compounds and risk of childhood brain tumors. Cancer Causes Control 16, 619–635.
Dockery, D.W., Pope, C.A., Xu, X., Spengler, J.D., Ware, J.H., Fay, M.E., Ferris Jr., B.G., Speizer, F.E., 1993. An association between air pollution and mortality in six US cities. N. Engl. J. Med. 329, 1753–1759.
Doll, R., Peto, R., 1981. The causes of cancer: quantitative estimates of avoidable risks of cancer in the United States today. J. Natl. Cancer Inst. 66, 1191–1308.
Donahue, B.A., Augot, M., Bellon, S.F., Treiber, D.K., Toney, J.H., Lippard, S.J., Essigmann, J.M., 1990. Characterization of a DNA

damage-recognition protein from mammalian cells that binds specifically to intrastrand d(GpG) and d(ApG) DNA adducts of the anticancer drug cisplatin. Biochemistry 29, 5872–5880.

Dougherty, C.P., Henricks Holtz, S., Reinert, J.C., Panyacosit, L., Axelrad, D.A., Woodruff, T.J., 2000. Dietary exposures to food contaminants across the United States. Environ. Res. 84, 170–185.

Draper, G., Vincent, T., Kroll, M.E., Swanson, J., 2005. Childhood cancer in relation to distance from high voltage power lines in England and Wales: a case-control study. B.M.J. 330, 1290–1993.

Durando, M., Kass, L., Piva, J., Sonnenschein, C., Soto, A.M., Luque, E.H., Munoz-de-Toro, M., 2007. Prenatal bisphenol a exposure induces preneoplastic lesions in the mammary gland in Wistar rats. Environ. Health Perspect. 115, 80–86.

Eilber, F.R., Morton, D.L., 1970. Immunologic studies of human sarcomas: additional evidence suggesting an associated sarcoma virus. Cancer 26, 588–596.

Epstein, S.S., 1994. Environmental and occupational pollutants are avoidable causes of breast cancer. Int. J. Health Serv. 24, 145–150.

Epstein, S.S., 2004. Legislative proposals for reversing the cancer epidemic and controlling run-away industrial technologies. In: Nicolopoulou-Stomati, P., Hens, L., Howard, C.V., Van Larebeke, N. (Eds.), Cancer as an Environmental Disease. Kluwer Academic Publisher, Dordrecht, pp. 149–169.

Etiemble, J., Bernard, J.F., Picat, C., Belpomme, D., Boivin, P., 1979. Red blood cell enzyme abnormalities in patients treated with chemotherapy. Br. J. Haematol. 42, 391–398.

Fear, N.T., Roman, E., Reeves, G., Pannett, B., 1998. Childhood cancer and paternal employment in agriculture: the role of pesticides. Br. J. Cancer. 77, 825–829.

Feingold, L., Savitz, D.A., John, E.M., 1992. Use of a job-exposure matrix to evaluate parental occupation and childhood cancer. Cancer Causes Control 3, 161–169.

Fews, A.P., Henshaw, D.L., Keitch, P.A., Close, J.J., Wilding, R.J., 1999a. Increased exposure to pollutant aerosols under high voltage power lines. Int. J. Radiat. Biol. 75, 1505–1521.

Fews, A.P., Henshaw, D.L., Wilding, R.J., Keitch, P.A., 1999b. Corona ions from powerlines and increased exposure to pollutant aerosols. Int. J. Radiat. Biol. 75, 1523–1531.

Feychting, M., Schulgen, G., Olsen, J.H., Ahlbom, A., 1995. Magnetic fields and childhood cancer-a pooled analysis of two Scandinavian studies. Eur. J. Cancer. 31A, 2035–2039.

Feychting, M., Forssen, U., Floderus, B., 1997. Occupational and residential magnetic field exposure and leukemia and central nervous system tumors. Epidemiology 8, 384–389.

Feychting, M., Svensson, D., Ahlbom, A., 1998. Exposure to motor vehicle exhaust and childhood cancer. Scand. J. Work Environ. Health. 24, 8–11.

Feychting, M., Plato, N., Nise, G., Ahlbom, A., 2001. Paternal occupational exposures and childhood cancer. Environ. Health Perspect. 109, 193–196.

Floret, N., Mauny, F., Challier, B., Cahn, J.Y., Tourneux, F., Viel, J.F., 2004. Dioxin emissions and soft-tissue sarcoma: results of a population-based case-control study. Rev. Epidemiol. Sante Publique. 52, 213–220.

Foliart, D.E., Pollock, B.H., Mezei, G., Iriye, R., Silva, J.M., Ebi, K.L., Kheifets, L., Link, M.P., Kavet, R., 2006. Magnetic field exposure and long-term survival among children with leukaemia. Br. J. Cancer. 94, 161–164.

Frisch, M., Biggar, R.J., Engels, E.A., Goedert, J.J., AIDS-Cancer Match Registry Study Group., 2001. Association of cancer with AIDS-related immunosuppression in adults. J. Am. Med. Assoc 285, 1736–1745.

Furmanski, P., Longley, C., Fouchey, D., Rich, R., Rich, M.A., 1974. Normal human mammary cells in culture: evidence for oncornavirus-like particles. J. Natl. Cancer Inst. 52, 975–977.

Gago-Dominguez, M., Castelao, J.E., Yuan, J.M., Yu, M.C., Ross, R.K., 2001. Use of permanent hair dyes and bladder-cancer risk. Int. J. Cancer. 91, 575–579.

Gallagher, R.P., Spinelli, J.J., Lee, T.K., 2005. Tanning beds, sunlamps, and risk of cutaneous malignant melanoma. Cancer Epidemiol. Biomarkers Prev. 14, 562–566.

Gallo, R.C., Montagnier, L., 2003. The discovery of HIV as the cause of AIDS. N. Engl. J. Med. 349, 2283–2285.

Gammon, M.D., Wolff, M.S., Neugut, A.I., Eng, S.M., Teitelbaum, S.L., Britton, J.A., Terry, M.B., Levin, B., Stellman, S.D., Kabat, G.C., Hatch, M., Senie, R., Berkowitz, G., Bradlow, H.L., Garbowski, G., Maffeo, C., Montalvan, P., Kemeny, M., Citron, M., Schnabel, F., Schuss, A., Hajdu, S., Vinceguerra, V., Niguidula, N., Ireland, K., Santella, R.M., 2002. Environmental toxins and breast cancer on Long Island. II. Organochlorine compound levels in blood. Cancer Epidemiol. Biomarkers Prev. 11, 686–697.

Geyer, H.J., Scheunert, I., Karl, R., Kettrup, A., Korte, F., Greim, H., Rozman, K., 1990. Correlation between acute toxicity of 2,3,7,8 tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) and total body fat content in mammals. Toxicology 65, 97–107.

Geyer, H.J., Schramm, K.W., Scheunert, I., Shughart, K., Buters, J., Wurst, W., Greim, H., Kluge, R., Steinberg, C.E., Kettrup, A., Madhukar, B., Olson, J.R., Gallo, M.A., 1997. Considerations on genetic and environmental factors that contribute to resistance or sensivity of mammals including humans to toxicity of 2,3,7,8 tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) and related compounds. Ecotoxicol. Environ. Saf. 36, 213–230.

Giri, A.K., 1986. Mutagenic and genotoxic effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin, a review. Mutat. Res. 168, 241–248.

Goguel, A., Cavigneaux, A., Bernard, J., 1967. Benzene leukemias in the Paris region between 1950 and 1965 (study of 50 cases). Nouv. Rev. Fr. Hematol. 7, 465–480.

Greaves, M., 2002. Childhood leukaemia. Br. Med. J. 324, 283–287.

Greaves, M.F., Alexander, F.E., 1993. An infectious etiology for common acute lymphoblastic leukemia in childhood? Leukemia 7, 349–360.

Greenland, S., Sheppard, A.R., Kaune, W.T., Poole, C., Kelsh, M.A., Childhood Leukemia-EMF Study Group, 2000. A pooled analysis of magnetic fields, wire codes, and childhood leukemia. Epidemiology 11, 624–634.

Geter, D.R., Chang, L.W., Hanley, N.M., Ross, M.K., Pegram, R.A., DeAngelo, A.B., 2004. Analysis of in vivo and in vitro DNA strand breaks from trihalomethane exposure. J. Carcinog. 3, 2.

Griffin, B.E., 2000. Epstein–Barr virus (EBV) and human disease: facts, opinions and problems. Mutat. Res. 462, 395–405.

Guenel, P., Nicolau, J., Imbernon, E., Chevalier, A., Goldberg, M., 1996. Exposure to 50 Hz electric field and incidence of leukemia, brain tumors, and other cancers among French electric utility workers. Am. J. Epidemiol. 144, 1107–1121.

Guibout, C., Dore, J.F., Marholev, L., Rosenfeld, C., Belpomme, D., 1977. Serological identification of an antigen associated with acute lymphoid leukaemia. C. R. Acad. Sci. Hebd. Seances Acad. Sci. D. 285, 615–618.

Gurpide, E., Blumenthal, R., Fleming, H., 1984. Regulation of estrogen receptor levels in endometrial cancer cells. Prog. Clin. Biol. Res. 142, 145–165.

Guvenius, D.M., Aronsson, A., Ekman-Ordeberg, G., Bergman, A., Noren, K., 2003. Human prenatal and postnatal exposure to polybrominated diphenyl ethers, polychlorinated biphenyls, polychlorobiphenylols, and pentachlorophenol. Environ. Health Perspect. 111, 1235–1241.

Hagen, T.M., Huang, S., Curnutte, J., Fowler, P., Martinez, V., Wehr, C.M., Ames, B.N., Chisari, F.V., 1994. Extensive oxidative DNA damage in hepatocytes of transgenic mice with chronic active hepatitis destined to develop hepatocellular carcinoma. Proc. Natl. Acad. Sci. USA 91, 12808–12812.

Haider, T., Knasmueller, S., Kundi, M., Haider, M., 1994. Clastogenic effects of radiofrequency radiations on chromosomes of Tradescantia. Mutat. Res. 324, 65–68.

Hallberg, O., Johansson, O., 2002. Melanoma incidence and frequency modulation (FM) broadcasting. Arch. Environ. Health. 57, 32–40.

Hansen, J., Raaschou-Nielsen, O., Christensen, J.M., Johansen, I., McLaughlin, J.K., Lipworth, L., Blot, W.J., Olsen, J.H., 2001. Cancer incidence among Danish workers exposed to trichloroethylene. J. Occup. Environ. Med. 43, 133–139.

Hardell, L., Eriksson, M., 1999. A case-control study of non-Hodgkin lymphoma and exposure to pesticides. Cancer 85, 1353–1360.

Hardell, L., Hansson Mild, K., 2006. Mobile phone use and risk of acoustic neuroma: results of the interphone case-control study in five North European countries. Br. J. Cancer. 93, 1348–1349.

Hardell, L., Eriksson, M., Degerman, A., 1995. Meta-analysis of four Sweedish case-control studies on exposure to pesticides as risk-factor for soft-tissue sarcoma including the relation to tumour localization and histopathological type. Int. J. Oncol. 6, 847–851.

Hardell, L., Eriksson, M., Axelson, O., Flesch-Janys, D., 2003. Epidemiological studies on cancer and exposure to dioxins and related compounds. In: Schecter, A., Gasiewicz, T.A. (Eds.), Dioxins and Health, Second Ed. Willey, Hoboken, NJ, USA, pp. 729–764.

Hardell, L., Carlberg, M., Hansson Mild, K., 2006a. Pooled analysis of two case-control studies on use of cellular and cordless telephones and the risk for malignant brain tumours diagnosed in 1997–2003. Int. Arch. Occup. Environ. Health. 79, 630–639.

Hardell, L., van Bavel, B., Lindström, G., Eriksson, M., Carlberg, M., 2006b. In utero exposure to persistent organic pollutants in relation to testicular cancer risk. Int. J. Androl. 29, 228–234.

Hardell, L., Carlberg, M., Soderqvist, F., Hansson Mild, K., Morgan, L.L., 2007. Long-term use of cellular phones and brain tumours-increased risk associated with use for >10 years. Occup. Environ. Med. doi:10.1136/oem.2006.029751.

Harris, R.J., 2004. Viruses and tumours. 1965. Eur. J. Cancer. 40, 1993–1997.

Harrison, R.M., Leung, P.L., Somervaille, L., Smith, R., Gilman, E., 1999. Analysis of incidence of childhood cancer in the West Midlands of the United Kingdom in relation to proximity to main roads and petrol stations. Occup. Environ. Med. 56, 774–780.

Hayes, R.B., 1997. The carcinogenicity of metals in humans. Cancer Causes Control 8, 371–385.

Hayes, R.B., Gerin, M., Raatgever, J.W., de Bruyn, A., 1986. Wood-related occupations, wood dust exposure, and sinonasal cancer. Am. J. Epidemiol. 124, 569–577.

Hayes, T.B., Case, P., Chui, S., Chung, D., Haeffele, C., Haston, K., Lee, M., Mai, V.P., Marjuoa, Y., Parker, J., Tsui, M., 2006. Pesticide mixtures, endocrine disruption, and amphibian declines: are we underestimating the impact? Environ. Health Perspect. 114, 40–50.

HCCP (Harvard Center for Cancer Prevention) 1996. Human causes of cancer: Harvard School of Public Health, 〈www.hsph.harvard.edu/cancer/resources_materials/reports/index.htm〉.

Hennig, E.M., Suo, Z., Thoresen, S., Holm, R., Kvinnsland, S., Nesland, J.M., 1999. Human papillomavirus 16 in breast cancer of women treated for high grade cervical intraepithelial neoplasia (CIN III). Breast Cancer Res. Treat. 53, 121–135.

Henriksen, T., Dahlback, A., Larsen, S.H., Moan, J., 1990. Ultraviolet-radiation and skin cancer. Effect of an ozone layer depletion. Photochem. Photobiol. 51, 579–582.

Hewstone, R.K., 1994. Health, safety and environmental aspects of used crankcase lubricating oils. Sci. Total Environ. 156, 255–268.

Hildesheim, M.E., Cantor, K.P., Lynch, C.F., Dosemeci, M., Lubin, J., Alavanja, M., Craun, G., 1998. Drinking water source and chlorination byproducts. II. Risk of colon and rectal cancers. Epidemiology 9, 29–35.

Ho, S.M., Tang, W.Y., Belmonte de Frausto, J., Prins, G.S., 2006. Developmental exposure to estradiol and bisphenol A increases susceptibility to prostate carcinogenesis and epigenetically regulates phosphodiesterase type 4 variant 4. Cancer Res 66, 5624–5632.

Huncharek, M., Kupelnick, B., 2005. Personal use of hair dyes and the risk of bladder cancer: results of a meta-analysis. Public Health Rep 120, 31–38.

IARC (International Agency for Research on Cancer), 1977. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans, Vol. 14. Asbestos, IarcPress, Lycon.

IARC (International Agency for Research on Cancer), 1979. Chemicals and industrial processes associated with cancer in humans. IARC Monographs, Vols. 1–20 (Supplement 1).

IARC (International Agency for Research on Cancer), 1980. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Some Metals and Metallic Compounds, Vol. 23. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1984. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Polynuclear Aromatic Compounds, Part 2: Carbon Blacks, Mineral Oils (Lubricant Base Oils and Derived Products) and Some Nitroarenes, Vol. 33. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1989. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Occupational Exposures in Petroleum Refining; Crude Oil and Major Petroleum Fuels, Vol. 45. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1990. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Chromium, Nickel and Welding, Vol. 49. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1991. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Occupational Exposures in Insecticide Application, and Some Pesticides, Vol. 53. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1992. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Solar and ultraviolet radiation, Vol. 55. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1994a. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Schistosomes, Liver Flukes and Helicobacter pylori, Vol. 61. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1994b. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Hepatitis Viruses, Vol. 59. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1995a. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Human Papillomaviruses, Vol. 64. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1995b. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Wood dust and Formaldehyde, Vol. 62. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1995c. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Dry cleaning, Some Chlorinated Solvents and Other Industrial Chemicals, Vol. 63. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1996a. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Human Immunodeficiency Viruses and Human T-Cell Lymphotropic Viruses, Vol. 67. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1996b. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Some Pharmaceutical Drugs, Vol. 66. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1997a. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans 1997. Epstein-Barr Virus and Kaposi's Sarcoma Herpesvirus/Human Herpesvirus 8, Vol. 70. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 1997b. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Polychlorinated Dibenzo-para-Dioxins and Polychlorinated Dibenzofurans, Vol. 69. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 2000a. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Di(2-ethylhexyl)phthalate, Vol. 77. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 2000b. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Ionizing Radiation, Part 1: X- and Gamma (g)-Radiation, and Neutrons, Vol. 75. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 2002a. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Non-Ionizing Radiation, Part 1: Static and Extremely Low-Frequency (ELF) Electric and Magnetic Fields, Vol. 80. IarcPress, Lyon.

IARC (International Agency for Research on Cancer), 2002b. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Man-made vitreous Fibres, Vol. 81. IarcPress, Lyon.

Ibarluzea, J.M.J., Fernandez, M.F., Santa-Marina, L., Olea-Serrano, M.F., Rivas, A.M., Aurrekoetxea, J.J., Exposito, J., Lorenzo, M., Torne, P., Villalobos, M., Pedraza, V., Sasco, A.J., Olea, N., 2004. Breast cancer risk and the combined effect of environmental estrogens. Cancer Causes Control 15, 591–600.

Ichinose, T., Fujii, K., Sagai, M., 1991. Experimental studies on tumor promotion by nitrogen dioxide. Toxicology 67, 211–225.

Infante-Rivard, C., Labuda, D., Krajinovic, M., Sinnett, D., 1999. Risk of childhood leukemia associated with exposure to pesticides and with gene polymorphisms. Epidemiology 10, 481–487.

Jackson, A.L., Loeb, L.A., 2001. The contribution of endogenous sources of DNA damage to the multiple mutations in cancer. Mutat. Res. 477, 7–21.

Kane, M.L., Ladov, E.N., Holdsworth, C.E., Weaver, N.K., 1984. Toxicological characteristics of refinery streams used to manufacture lubricating oils. Am. J. Ind. Med. 5, 183–200.

Karimi, M., Yarmohammadi, H., 2003. Seasonal variations in the onset of childhood leukemia/lymphoma: April 1996–March 2000, Shiraz, Iran. Hematol. Oncol. 21, 51–55.

Kasim, K., Levallois, P., Johnson, K.C., Abdous, B., Auger, P., Canadian Cancer Registries Epidemiology Research Group, 2006. Chlorination disinfection by-products in drinking water and the risk of adult leukemia in Canada. Am. J. Epidemiol. 163, 116–126.

Kauppinen, T., Toikkanen, J., Pedersen, D., Young, R., Ahrens, W., Boffetta, P., Hansen, J., Kromhout, H., Maqueda Blasco, J., Mirabelli, D., de la Orden-Rivera, V., Pannett, B., Plato, N., Savela, A., Vincent, R., Kogevinas, M., 2000. Occupational exposure to carcinogens in the European Union. Occup. Environ. Med. 57, 10–18.

Kelfkens, G., de Gruijl, F.R., van der Leun, J.C., 1990. Ozone depletion and increase in annual carcinogenic ultraviolet dose. Photochem. Photobiol. 52, 819–823.

Keydar, I., Ohno, T., Nayak, R., Sweet, R., Simoni, F., Weiss, F., Karby, S., Mesa-Tejada, R., Spiegelman, S., 1984. Properties of retrovirus-like particles produced by a human breast carcinoma cell line: immunological relationship with mouse mammary tumor virus proteins. Proc. Natl. Acad. Sci. USA 81, 4188–4192.

Kielhorn, J., Melber, C., Wahnschaffe, U., Aitio, A., Mangelsdorf, I., 2000. Vinyl chloride: still a cause for concern. Environ. Health Perspect. 108, 579–588.

King, W.D., Marrett, L.D., 1996. Case-control study of bladder cancer and chlorination by-products in treated water (Ontario, Canada). Cancer Causes Control 7, 596–604.

King, W.D., Marrett, L.D., Woolcott, C.G., 2000. Case-control study of colon and rectal cancers and chlorination by-products in treated water. Cancer Epidemiol. Biomarkers Prev. 9, 813–818.

Kinlen, L., 1988. Evidence for an infective cause of childhood leukaemia: comparison of a Scottish new town with nuclear reprocessing sites in Britain. Lancet 2, 1323–1327.

Kinlen, L.J., Hudson, C.M., Stiller, C.A., 1991. Contacts between adults as evidence for an infective origin of childhood leukaemia: an explanation for the excess near nuclear establishments in west Berkshire? Br. J. Cancer. 64, 549–554.

Koike, K., Tsutsumi, T., Fujie, H., Shintani, Y., Kyoji, M., 2002. Molecular mechanism of viral hepatocarcinogenesis. Oncology 62 (1), 29–37.

Kopf, A.W., Kripke, M.L., Stern, R.S., 1984. Sun and malignant melanoma. J. Am. Acad. Dermatol. 11, 674–684.

Krynska, B., Del Valle, L., Croul, S., Gordon, J., Katsetos, C.D., Carbone, M., Giordano, A., Khalili, K., 1999. Detection of human neurotropic JC virus DNA sequence and expression of the viral oncogenic protein in pediatric medulloblastomas. Proc. Natl. Acad. Sci. USA 96, 11519–11524.

Landrigan, P.J., Boffetta, P., Apostoli, P., 2000. The reproductive toxicity and carcinogenicity of lead: a critical review. Am. J. Ind. Med. 38, 231–243.

Lassiter, R.R., Hallam, T.G., 1990. Survival of the fattest: implication for acute effects of lipophilic chemicals on aquatic populations. Environ. Toxicol. Chem. 9, 585–595.

Latini, G., Del Vecchio, A., Massaro, M., Verrotti, A., De Felice, C., 2006. Phthalate exposure and male infertility. Toxicology 226, 90–98.

Lawson, J.S., Tran, D., Rawlinson, W.D., 2001. From Bittner to Barr: a viral, diet and hormone breast cancer aetiology hypothesis. Breast Cancer Res 3, 81–85.

Levi, F., Moeckli, R., Randimbison, L., Te, V.C., Maspoli, M., La Vecchia, C., 2006. Skin cancer in survivors of childhood and adolescent cancer. Eur. J. Cancer. 42, 656–659.

Lubin, J.H., Boice, J.D., 1997. Lung cancer risk from residential radon: a meta analysis of eight epidemiologic studies. Natl. Cancer Ins. 89, 49–57.

Lubin, J.H., Boice Jr., D., Edling, C., Hornung, R.W., Howe, G.R., Kunz, E., Kusiak, R.A., Morrison, H.I., Radford, E.P., Samet, J.M., Tirmarche, M., Woodward, A., Xiang, Y.S., Pierce, D.A., 1994. Radon and Lund Cancer Risk: A Joint Analysis of 11 underground miners studies. Vol. NIH, Publication No. 94-3644, US Department of Health and Human Services, Washington, DC.

Ma, X., Buffler, P.A., Gunier, R.B., Dahl, G., Smith, M.T., Reinier, K., Reynolds, P., 2002. Critical windows of exposure to household pesticides and risk of childhood leukemia. Environ. Health Perspect. 110, 955–960.

Mack, W., Preston-Martin, S., Peters, J.M., 1991. Astrocytoma risk related to job exposure to electric and magnetic fields. Bioelectromagnetics 12, 57–66.

Mackerer, C.R., Griffis, L.C., Grabowski Jr., J.S., Reitman, F.A., 2003. Petroleum mineral oil refining and evaluation of cancer hazard. Appl. Occup. Environ. Hyg. 18, 890–901.

Mandal, P.K., 2005. Dioxin: a review of its environmental effects and its aryl hydrocarbon receptor biology. J. Compt. Physiol. 175, 221–230.

McDuffie, H.H., Pahwa, P., McLaughlin, J.R., Spinelli, J.J., Fincham, S., Dosman, J.A., Robson, D., Skinnider, L.F., Choi, N.W., 2001. Non-Hodgkin's lymphoma and specific pesticide exposures in men: cross-Canada study of pesticides and health. Cancer Epidemiol. Biomarkers Prev. 10, 1155–1163.

McGrath, C.M., Jones, R.F., 1978. Hormonal induction of mammary tumor viruses and its implications for carcinogenesis. Cancer Res 38, 4112–4125.

McNally, R.J.Q., Alexander, F.E., Birch, J.M., 2002. Space–time clustering analyses of childhood and acute lymphoblastic leukaemia by immunophenotype. Br. J. Cancer. 87, 513–515.

Meinert, R., Schuz, J., Kaletsch, U., Kaatsch, P., Michaelis, J., 2000. Leukemia and non-Hodgkin's lymphoma in childhood and exposure to pesticides: results of a register-based case-control study in Germany. Am. J. Epidemiol. 151, 639–646.

Melnick, R.L., Brody, C., DiGangi, J., Huff, J., 2000. The IARC evaluation of DEHP excludes key papers demonstrating carcinogenic effects. Int. J. Occup. Environ. Health. 9, 400–401.

Melnick, R.L., Nyska, A., Foster, P.M., Roycroft, J.H., Kissling, G.E., 2007. Toxicity and carcinogenicity of the water disinfection byproduct, dibromoacetic acid, in rats and mice. Toxicology 230, 126–136.

Menegaux, F., Baruchel, A., Bertrand, Y., Lescoeur, B., Leverger, G., Nelken, B., Sommelet, D., Hemon, D., Clavel, J., 2006. Household exposure to pesticides and risk of childhood acute leukaemia. Occup. Environ. Med. 63, 131–134.

Mills, P.K., Yang, R., 2006. Regression analysis of pesticide use and breast cancer incidence in California Latinas. J. Environ. Health. 68, 15–22.

Miyazaki, S., Imatani, A., Ballard, L., Marchetti, A., Buttitta, F., Albertsen, H., Nevanlinna, H.A., Gallahan, D., Callahan, R., 1997. The chromosome location of the human homolog of the mouse mammary tumor-associated gene INT6 and its status in human breast carcinomas. Genomics 46, 155–158.

Moore, D.H., Charney, J., Kramarsky, B., Lasfargues, E.Y., Sarkar, N.H., Brennan, M.J., Burrows, J.H., Sirsat, S.M., Paymaster, J.C., Vaidya, A.B., 1971. Search for a human breast cancer virus. Nature 229, 611–614.

Muir, K., Rattanamongkolgul, S., Smallman-Raynor, M., Thomas, M., Downer, S., Jenkinson, C., 2004. Breast cancer incidence and its

possible spatial association with pesticide application in two counties of England. Public Health 118, 513–520.
Mukerjee, D., 1998. Health impact of polychlorinated dibenzo-p-dioxins: a critical review. J. Air Waste Manag. Assoc. 48, 157–165.
Muscat, J.E., Britton, J.A., Djordjevic, M.V., Citron, M.L., Kemeny, M., Busch-Devereaux, E., Pittman, B., Stellman, S.D., 2003. Adipose concentrations of organochlorine compounds and breast cancer recurrence in Long Island, New York. Cancer Epidemiol. Biomark. Prev. 12, 1474–1478.
Navas-Acien, A., Pollan, M., Gustavsson, P., Plato, N., 2002. Occupation, exposure to chemicals and risk of gliomas and meningiomas in Sweden. Am. J. Ind. Med. 42, 214–227.
Nitta, Y., Hoshi, M., Kamiya, K., 2002. Effects of radioactive iodine (131I) on the thyroid of newborn, pubertal and adult rats. Int. Cong. Ser. 1236, 127–131.
Ohyama, K., Ito, T., Kanisawa, M., 1999. The roles of diesel exhaust particle extracts and the promotive effects of NO_2 and/or SO_2 exposure on rat lung tumorigenesis. Cancer Lett 139, 189–197.
O'Leary, L.M., Hicks, A.M., Peters, J.M., London, S., 1991. Parental occupational exposures and risk of childhood cancer: a review. Am. J. Ind. Med. 20, 17–35.
Pagano, J.S., Blaser, M., Buendia, M.A., Damania, B., Khalili, K., Raab-Traub, N., Roizman, B., 2004. Infectious agents and cancer: criteria for a causal relation. Semin. Cancer Biol. 14, 453–471.
Palmer, S., Mathews, R.A., 1986. The role of non-nutritive dietary constituents in carcinogenesis. Surg. Clin. North Am. 66, 891–915.
Parkin, D.M., Bray, F., Ferlay, J., Pisani, P., 2001. Estimating the world cancer burden: Globocan 2000. Int. J. Cancer. 94, 153–156.
Pisani, P., Parkin, D.M., Munoz, N., Ferlay, J., 1997. Cancer and infection: estimates of the attributable fraction in 1990. Cancer Epidemiol. Biomarkers Prev. 6, 387–400.
Pollock, B.H., Jenson, H.B., Leach, C.T., McClain, K.L., Hutchison, R.E., Garzarella, L., Joshi, V.V., Parmley, R.T., Murphy, S.B., 2003. Risk factors for pediatric human immunodeficiency virus-related malignancy. J. Am. Med. Assoc. 289, 2393–2399.
Pope, C.A., Burnett, R.T., Thun, M.J., Calle, E.E., Krewski, D., Ito, K., Thurston, G.D., 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. J. Am. Med. Assoc. 287, 1132–1141.
Post, P.N., Stockton, D., Davies, T.W., Coebergh, J.W., 1999. Striking increase in incidence of prostate cancer in men aged <60 years without improvement in prognosis. Br. J. Cancer. 79, 13–17.
Prasad, K.N., Cole, W.C., Hasse, G.M., 2004. Health risks of low dose ionizing radiation in humans: a review. Exp. Biol. Med. (Maywood). 229, 378–382.
Preston, D.L., Pierce, D.A., Shimizu, Y., Cullings, H.M., Fujita, S., Funamoto, S., Kodama, K., 2004. Effect of recent changes in atomic bomb survivor dosimitry on cancer mortality risk estimates. Radiat. Res. 162, 377–389.
Prins, G.S., Birch, L., Tang, W.Y., Ho, S.M., 2007. Developmental estrogen exposures predispose to prostate carcinogenesis with aging. Reprod. Toxicol. 23, 374–382.
Raaschou-Nielsen, O., Hansen, J., McLaughlin, J.K., Kolstad, H., Christensen, J.M., Tarone, R.E., Olsen, J.H., 2003. Cancer risk among workers at Danish companies using trichloroethylene: a cohort study. Am. J. Epidemiol. 158, 1182–1192.
Rao, M.S., Yeldandi, A.V., Subbarao, V., 1990. Quantitative analysis of hepatocellular lesions induced by di(2 ethylhexyl)phthalate in F-344 rats. J. Toxicol. Environ. Health 30, 85–89.
Rauscher, G.H., Shore, D., Sandler, D.P., 2004. Hair dye use and risk of adult acute leukemia. Am. J. Epidemiol. 160, 19–25.
Repetto, R., Baliga, S.S., 1997. Pesticides and immunosuppression: the risks to public health. Health Policy Plan 12, 97–106.
Rice, J.M., Rehm, S., Donovan, P.J., Perantoni, A.O., 1989. Comparative transplacental carcinogenesis by directly acting and metabolism-dependent alkylating agents in rodents and nonhuman primates. In: Napalkov, N.P., Rice, J.M., Tomatis, L., Yamasaki, H. (Eds.), Perinatal and Multigeneration Carcinogenesis. International Agency for Research on Cancer, Lyon, pp. 17–34.
Richters, A., Kuraitis, K., 1983. Air pollutants and the facilitation of cancer metastasis. Environ. Health Perspect. 52, 165–168.
Rollison, D.E., Helzlsouer, K.J., Pinney, S.M., 2006. Personal hair dye use and cancer: a systematic literature review and evaluation of exposure assessment in studies published since 1992. J. Toxicol. Environ. Health B. Crit. Rev. 9, 413–439.
Ruder, A.M., Ward, E.M., Brown, D.P., 1994. Cancer mortality in female and male dry-cleaning workers. J. Occup. Med. 36, 867–874.
Saillenfait, A.M., Sabate, J.P., Gallissot, F., 2003. Comparative embryotoxicities of butyl benzyl phthalate, mono-n-butyl phthalate and mono-benzyl phthalate in mice and rats: in vivo and in vitro observations. Reprod. Toxicol. 17, 575–583.
Saillenfait, A.M., Sabate, J.P., Gallissot, F., 2006. Developmental toxic effects of diisobutyl phthalate, the methyl-branched analogue of di-n-butyl phthalate, administered by gavage to rats. Toxicol. Lett. 165, 39–46.
Salnikow, K., Cosentino, S., Klein, C., Costa, M., 1994. Loss of thrombospondin transcriptional activity in nickel-transformed cells. Mol. Cell. Biol. 14, 851–858.
Sasaki, Y.F., Kawaguchi, S., Kamaya, A., Ohshita, M., Kabasawa, K., Iwama, K., Taniguchi, K., Tsuda, S., 2002. The comet assay with 8 mouse organs: results with 39 currently used food additives. Mutat. Res. 519, 103–119.
Savitz, D.A., Chen, J.H., 1990. Parental occupation and childhood cancer: review of epidemiologic studies. Environ. Health Perspect. 88, 325–337.
Savitz, D.A., Feingold, L., 1989. Association of childhood cancer with residential traffic density. Scand. J. Work Environ. Health. 15, 360–363.
Schiestl, R.H., Aubrecht, J., Yap, W.Y., Kandikonda, S., Sidhom, S., 1997. Polychlorinated biphenyls and 2,3,7,8-tetrachlorodibenzo-p-dioxin induce intrachromosomal recombination in vitro and in vivo. Cancer Res 57, 4378–4383.
Schlom, J., Spiegelman, S., Moore, D., 1971. RNA-dependent DNA polymerase activity in virus-like particles isolated from human milk. Nature 231, 97–100.
Schuz, J., Jacobsen, R., Olsen, J.H., Boice Jr., J.D., McLaughlin, J.K., Johansen, C., 2006. Cellular telephone use and cancer risk: update of a nationwide Danish cohort. J. Natl. Cancer Inst. 98, 1707–1713.
Selenskas, S., Teta, M.J., Vitale, J., 1995. Pancreatic cancer among workers processing synthetic resins. Am. J. Ind. Med. 28, 385–398.
Sharpe, R.M., Irvine, D.S., 2004. How strong is the evidence of a link between environmental chemicals and adverse effects on human reproductive health? BMJ 328, 447–451.
Shea, K.M., 2003. American Academy of Pediatrics Committee on Environmental Health: pediatric exposure and potential toxicity of phthalate plasticizers. Pediatrics 111, 1467–1474.
Siemiatycki, J., Richardson, L., Straif, K., Latreille, B., Lakhani, R., Campbell, S., Rousseau, M.C., Boffetta, P., 2004. Listing occupational carcinogens. Environ. Health Perspect. 112, 1447–1459.
Simonich, S.L., Hites, R.A., 1995. Global distribution of persistent organochlorine compounds. Science 269, 1851–1854.
Skakkebæk, N.E., Rajpert-De Meyts, E., Main, K.M., 2001. Testicular dysgenesis syndrome: an increasingly common developmental disorder with environmental aspects. Human Reproduction 16, 972–978.
Smith, K., 1987. Biofuels, Air Pollution, and Health: A Global Review. Plenum Press, New York.
Smulevich, V.B., Solionova, L.G., Belyakova, S.V., 1999. Parental occupation and other factors and cancer risk in children: II. Occupational factors. Int. J. Cancer. 83, 718–722.
Snow, E.T., 1991. A possible role for chromium(III) in genotoxicity. Environ. Health Perspect. 92, 75–81.
Snow, E.T., 1994. Effects of chromium on DNA replication in vitro. Environ. Health Perspect. 102, 41–44.
Soble, S., Haislip, A., Hill, S., Garry, R., 1999. Human sequences related to mouse mammary tumor virus. In: Proceedings of the 11th

International Microbiology Congress. International Association of Microbiologists, Sydney.
Song, S., Pitot, H.C., Lambert, P.F., 1999. The human papillomavirus type 16 E6 gene alone is sufficient to induce carcinomas in transgenic animals. J. Virol. 73, 5887–5893.
Sorsa, M., Anderson, D., 1996. Monitoring of occupational exposure to cytostatic anticancer agents. Mutat. Res. 355, 253–261.
Sorsa, M., Hemminki, K., Vainio, H., 1985. Occupational exposure to anticancer drug-potential and real hazards. Mutat. Res. 154, 135–149.
Stellman, S.D., Djordjevic, M.V., Britton, J.A., Muscat, J.E., Citron, M.L., Kemeny, M., Busch, E., Gong, L., 2000. Breast cancer risk in relation to adipose concentrations of organochlorine pesticides and polychlorinated biphenyls in Long Island, New York. Cancer Epidemiol Biomarkers Prev 9, 1241–1249.
Stohs, S.J., 1990. Oxidative stress induced by 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Free Radic. Biol. Med. 9, 79–90.
Surralles, J., Autio, K., Nylund, L., Jarventaus, H., Norppa, H., Veidebaum, T., Sorsa, M., Peltonen, K., 1997. Molecular cytogenetic analysis of buccal cells and lymphocytes from benzene-exposed workers. Carcinogenesis 18, 817–823.
Szymanska-Chabowska, A., Antonowicz-Juchniewicz, J., Andrzejak, R., 2002. Some aspects of arsenic toxicity and carcinogenicity in living organism with special regard to its influence on cardiovascular system, blood and bone marrow. Int. J. Occup. Med. Environ. Health. 15, 101–116.
Takkouche, B., Etminan, M., Montes-Martinez, A., 2007. Personal use of hair dyes and risk of cancer: a meta-analysis. J. Am. Acad. Dermatol. 293, 2516–2525.
Talbot, S.J., Crawford, D.H., 2004. Viruses and tumours—an update. Eur. J. Cancer. 40, 1998–2005.
Tannenbaum, S.R., Young, V., Green, L., Ruiz de Luzuriaga, K., 1980. Intestinal formation of nitrite and *N*-nitroso compounds. IARC Sci. Publ. 31, 2819.
Tokiwa, H., Sera, N., Nakashima, A., Nakashima, K., Nakanishi, Y., Shigematu, N., 1994. Mutagenic and carcinogenic significance and the possible induction of lung cancer by nitro aromatic hydrocarbons in particulate pollutants. Environ. Health Perspect. 102, 107–110.
Tolbert, P.E., 1997. Oils and cancer. Cancer Causes Control 8, 386–405.
Tondel, M., Lindgren, L., Hjalmarsson, P., Hardell, L., Persson, B., 2006. Increased incidence of malignancies in Sweden after the Chernobyl accident—a promoting effect? Am. J. Ind. Med. 49, 159–168.
Trosko, J.E., Upham, B.L., 2005. The emperor wears no clothes in the field of carcinogen risk assessment: ignored concepts in cancer risk assessment. Mutagenesis 20, 81–92.
UNSCEAR report, 1988. Ionizing radiation sources and biological effects. United Nations Scientific Committee on the Effects of Atomic Radiation. United Nation, New York.
UNSCEAR report, 2000. Sources and Effects of Ionizing Radiation. United Nations Scientific Committee on the Effects of Atomic Radiation. United Nation, New York.
Urbach, F., 1991. Potential health effects of climatic change: effects of increased ultraviolet radiation on man. Environ. Health Perspect. 96, 175–176.
USEPA, 2003. Childhood Cancer. America's Children and the Environment. Measures of Contaminants, Body Burdens and Illness. United States Environmental Protection Agency. EPA 240-R-03-001, pp. 76-81.
Vaidya, A.B., Black, M.M., Dion, A.S., Moore, D.H., 1974. Homology between human breast tumour RNA and mouse mammary tumour virus genome. Nature 249, 565–567.
Van Den Bosch, C.A., 2004. Is endemic Burkitt's lymphoma an alliance between three infections and a tumour promoter? Lancet Oncol 5, 738–746.
Van Maele-Fabry, G., Willems, J.L., 2003. Occupation related pesticide exposure and cancer of the prostate: a meta-analysis. Occup. Environ. Med. 60, 634–642.
Viegi, G., Simoni, M., Scognamiglio, A., Baldacci, S., Pistelli, F., Carrozzi, L., Annesi-Maesano, I., 2004. Indoor air pollution and airway disease. Int. J. Tuberc. Lung Dis. 8, 1401–1415.
Viel, J.F., Baverel, J., Cohn, J.Y., 2000. Soft-tissue sarcoma and non-Hodgkin's lymphoma clusters around municipal solid waste incinerator with high dioxin emission levels. Am. J. Epidemiol. 152, 13–19.
Villanueva, C.M., Fernandez, F., Malats, N., Grimalt, J.O., Kogevinas, M., 2003. Meta-analysis of studies on individual consumption of chlorinated drinking water and bladder cancer. J. Epidemiol. Community Health. 57, 166–173.
Villanueva, C.M., Cantor, K.P., Cordier, S., Jaakkola, J.J., King, W.D., Lynch, C.F., Porru, S., Kogevinas, M., 2004. Disinfection byproducts and bladder cancer: a pooled analysis. Epidemiology 15, 357–367.
Vineis, P., Hoek, G., Krzyzanowski, M., Vigna-Taglianti, F., Veglia, F., Airoldi, L., Overvad, K., Raaschou-Nielsen, O., Clavel-Chapelon, F., Linseisen, J., Boeing, H., Trichopoulou, A., Palli, D., Krogh, V., Tumino, R., Panico, S., Bueno-De-Mesquita, H.B., Peeters, P.H., Lund, E.E., Agudo, A., Martinez, C., Dorronsoro, M., Barricarte, A., Cirera, L., Quiros, J.R., Berglund, G., Manjer, J., Forsberg, B., Day, N.E., Key, T.J., Kaaks, R., Saracci, R., Riboli, E., 2007. Lung cancers attributable to environmental tobacco smoke and air pollution in non-smokers in different European countries: a prospective study. Environ. Health. 6, 7–13.
Waalkes, M.P., 2003. Cadmium carcinogenesis. Mutat. Res. 533, 107–120.
Wakeford, R., 2004. The cancer epidemiology of radiation. Oncogene 23, 6404–6428.
Wang, Y., Go, V., Holland, J.F., Melana, S.M., Pogo, B.G., 1998. Expression of mouse mammary tumor virus-like env gene sequences in human breast cancer. Clin. Cancer Res. 4, 2565–2568.
Ward, M.H., Mark, S.D., Cantor, K.P., Weisenburger, D.D., Correa-Villasenor, A., Zahm, S.H., 1996. Drinking water nitrate and the risk of non-Hodgkin's lymphoma. Epidemiology 7, 465–471.
Ward, M.H., deKok, T.M., Levallois, P., Brender, J., Gulis, G., Nolan, B.T., VanDerslice, J., 2005. Workgroup report: Drinking-water nitrate and health–recent findings and research needs. Environ. Health Perspect. 113, 1607–1614.
Wartenberg, D., Siegel Scott, C., 2002. Carcinogenicity of trichloroethylene. Environ. Health Perspect. 110, A13–A14.
Wartenberg, D., Reyner, D., Scott, C.S., 2000. Trichloroethylene and cancer: epidemiologic evidence. Environ. Health Perspect. 108, 161–176.
Weaver, V.M., Buckley, T.J., Groopman, J.D., 1998. Approaches to environmental exposure assessment in children. Environ. Health Perspect. 106, 827–832.
Weggen, S., Bayer, T.A., von Deimling, A., Reifenberger, G., von Schweinitz, D., Wiestler, O.D., Pietsch, T., 2000. Low frequency of SV40, JC and BK polyomavirus sequences in human medulloblastomas, meningiomas and ependymomas. Brain Pathol 10, 85–92.
Weisburger, J.H., 1986. Role of fat, fiber, nitrate, and food additives in carcinogenesis: a critical evaluation and recommendations. Nutr. Cancer. 8, 47–62.
Weiss, N.S., 1995. Cancer in relation to occupational exposure to perchloroethylene. Cancer Causes Control 6, 257–266.
Wertheimer, N., Leeper, E., 1979. Electrical wiring configurations and childhood cancer. Am. J. Epidemiol. 109, 273–284.
Wesseling, C., Pukkala, E., Neuvonen, K., Kauppinen, T., Boffetta, P., Partanen, T., 2002. Cancer of the brain and nervous system and occupational exposures in Finnish women. J. Occup. Environ. Med. 44, 663–668.
Wilkins 3rd Jr., Koutras, R.A., 1998. Paternal occupation and brain cancer in offspring: a mortality-based case-control study. Am. J. Ind. Med. 14, 299–318.
Witkiewicz-Kucharczyk, A., Bal, W., 2006. Damage of zinc fingers in DNA repair proteins, a novel molecular mechanism in carcinogenesis. Toxicol. Lett. 162, 29–42.
Yu, Y., Morimoto, T., Sasa, M., Okazaki, K., Harada, Y., Fujiwara, T., Irie, Y., Takahashi, E., Tanigami, A., Izumi, K., 1999. HPV33 DNA in premalignant and malignant breast lesions in Chinese and Japanese populations. Anticancer Res 19, 5057–5061.
Zahm, S.H., Devesa, S.S., 1995. Childhood cancer: overview of incidence trends and environmental carcinogens. Environ. Health Perspect. 103, 177–184.

Zahm, S.H., Ward, M.H., 1998. Pesticides and childhood cancer. Environ. Health Perspect. 106, 893–908.

Zhang, J., Smith, K.R., 2003. Indoor air pollution: a global health concern. Br. Med. Bull. 68, 209–225.

Zheng, T., Zahm, S.H., Cantor, K.P., Weisenburger, D.D., Zhang, Y., Blair, A., 2001. Agricultural exposure to carbamate pesticides and risk of non-Hodgkin lymphoma. J. Occup. Environ. Med. 43, 641–649.

Zhitkovich, A., Voitkun, V., Costa, M., 1995. Glutathione and free amino acids form stable complexes with DNA following exposure of intact mammalian cells to chromate. Carcinogenesis 16, 907–913.

Zhitkovich, A., Voitkun, V., Costa, M., 1996. Formation of the amino acid-DNA complexes by hexavalent and trivalent chromium in vitro: importance of trivalent chromium and the phosphate group. Biochemistry 35, 7275–7282.

Zierler, S., Feingold, L., Danley, R.A., Craun, G., 1988. Bladder cancer in Massachusetts related to chlorinated and chloraminated drinking water: a case-control study. Arch. Environ. Health. 43, 195–200.

Zou, E., Matsumura, F., 2003. Long-term exposure to b-hexachlorocyclohexane (b-HCH) promotes transformation and invasiveness of MCF-7 human breast cancer cells. Biochem. Pharmacol. 66, 831–840.

Zwelling, L.A., Anderson, T., Kohn, K.W., 1979. DNA-protein and DNA interstrand cross-linking by cis- and trans-platinum(II) diammine-dichloride in L1210 mouse leukemia cells and relation to cytotoxicity. Cancer Res 39, 365–369.