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Review article

Exposure to Chemicals and Radiation During Childhood and Risk for Cancer Later in Life

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 A B S T R A C T

Purpose: Many chemical carcinogens are in food, water, air, household products, and personal care products. Although genetic susceptibility is an important factor in how an individual responds to exposure to a carcinogen, heritable genetic factors alone account for only a minor portion of cancer rates.

Methods: We review the evidence that early life exposure to carcinogenic chemicals and ionizing radiation results in elevations in cancer later in life.

Results: Because cells are rapidly dividing and organ systems are developing during childhood and adolescence, exposure to carcinogens during these early life stages is a major risk factor for cancer later in life. Because young people have many expected years of life, the clinical manifestations of cancers caused by carcinogens have more time in which to develop during characteristically long latency periods. Many chemical carcinogens persist in the body for decades and increase risk for all types of cancers. Carcinogens may act via mutagenic, nonmutagenic, or epigenetic mechanisms and may also result from disruption of endocrine systems. The problem is magnified by the fact that many chemical carcinogens have become an integral part of our food and water supply and are in air and the general environment.

Conclusions: The early life onset of a lifelong exposure to mixtures of multiple environmental chemical carcinogens and radiation contributes significantly to the etiology of cancer in later life.

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 IMPLICATIONS AND
 CONTRIBUTION

Most cancers are not solely a result of genetic factors. Exposure to radiation and cancer-causing chemicals in food, water, air, and consumer products early in life can lead to cancer later in life. Prevention of early life exposure to carcinogens is essential.

Cancer is the second leading cause of death in developed countries and is primarily a disease of older age. Although genetic susceptibility to cancer is important in individual responses to exposure to carcinogens, twin studies clearly show that only a small minority of cancers are due solely to heritable

genetic factors [1]. If most cancer is not from heritable genetic factors, it must be from environmental exposures. As stated in the recent report of the President's Cancer Panel, "It is more effective to prevent disease than to treat it, but cancer prevention efforts have focused narrowly on smoking, other lifestyle behaviors and chemopreventive interventions. Scientific evidence of individual and multiple environmental exposure effects on disease initiation and outcomes, and consequent health system and societal costs, are not being adequately integrated into national policy decisions and strategies for disease prevention" [2].

Most cancers appear in older adults, but inevitable exposures to radiation and chemicals, and lifestyles that expose people to carcinogens, begin early in life. The latency period between

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exposure to a carcinogen and clinical onset of cancer is usually long, often decades. As a consequence, exposures during gestation, childhood, and adolescence have been demonstrated to lead to cancer later in life [3,4]. Patterns of exposure to environmental chemical mixtures through diet and use of tobacco, alcohol, and personal care products are usually established during early life and are sources of exposure for the rest of life. Clearly the risk of development of cancer later in life depends on the inherent carcinogenicity of the chemical or radiation as well as the exposure magnitude and duration and to individual genetic susceptibility.

A variety of factors make exposure during early life important [5]. Age is one of the most important. The most vulnerable ages are the fetal and perinatal periods and the first years of life. Exposure to carcinogenic chemicals during fetal development poses a particularly significant risk because organ systems are developing [4]. During this phase, cells are replicating rapidly and if DNA damage occurs, permanent defects may result and lead to cancer later in life [6]. The fetus is completely dependent on the intrauterine environment, which is influenced by maternal health, diet, and chemical and radiation exposures. Many chemicals cross the placenta. Studies conducted between 2005 and 2011 reported the presence of at least 100 to more than 200 chemicals in the umbilical cord blood of 20 newborns [7,8]. Of these chemicals, 101 were present in every umbilical cord blood sample in the 2005 study. Known or suspected carcinogens have been found not only in human cord blood [9], but also in amniotic fluid [10], breast milk [11], and meconium [12].

Exposures during this sensitive time carry carcinogenic potential for tumors that present clinically later in life. Epigenetic changes also result from exposures during this period. Some chemical carcinogens are persistent in the environment because they do not degrade rapidly. In the human body, they are not easily metabolized or excreted. Consequently they accumulate and may have half-lives of a decade or more. Highly chlorinated chemicals are lipophilic and poorly metabolized [13], whereas elements like lead are deposited in bones and teeth where they remain for long periods [14]. These reservoirs may, however, be mobilized under circumstances such as pregnancy and lactation. The combined effects of accumulating carcinogens, exposure to radiation, and epigenetic changes increase the child's risk for cancer as an adult. Additional factors that contribute to increased susceptibility throughout early life and childhood include small size and lack of developmental maturity. Compared with adults, children [4,15]:

- Have a higher respiratory rate, and consume more food and water per pound of body weight, exposing them to relatively greater quantities of pollutants from air, food, and water.
- Have less well-developed enzymatic systems for detoxifying contaminants.
- Have immune systems that are not fully developed.
- Are not as well able to recognize danger and escape independently.
- Are closer to the floor and are therefore closer to dust and spills.
- Have a greater likelihood of oral exposures because of hand-to-mouth behavior.

Puberty and adolescence are also periods of increased risk from exposure to chemicals. Although adolescents have greater independence than children regarding choice of activities, foods, and beverages, they still lack full neurological maturity that

limits their capacity for good judgment and may actually increase exposure to toxic substances such as tobacco, substances of abuse, and chemicals in some personal care products [16]. This may expose them to greater risks. During adolescence, the endocrine, reproductive, neurological, and other systems undergo remarkable development and growth. The developing tissues and functions of these organ systems are particularly sensitive to the effects of carcinogenic and endocrine-disrupting chemicals [17,18]. Endocrine disruptors interfere with the maintenance of normal hormone homeostasis by acting either as hormone agonists or antagonists or by altering metabolic processes [19]. These effects may predispose an individual to the eventual onset of cancer in hormone-sensitive organ systems such as the breast, ovaries, prostate, and testes [3,20].

Another factor that increases the risk of children and teenagers is that they have many more years to live. More years allows for greater acquisition and bioaccumulation of environmental chemicals, including persistent organic pollutants (POPs). An example is dioxin, a known human carcinogen, which has a half-life of about 7 years [21]. Because humans are unable to detoxify and excrete dioxin-like chemicals efficiently, the daily intake exceeds elimination under most circumstances. Therefore, levels in humans at background exposures increase with age [22]. In addition, concentrations of other POPs, including polychlorinated biphenyls (PCBs), chlorinated pesticides, and brominated flame retardants also accumulate in the adipose tissue. These additional toxins can potentiate the effect of earlier exposures, contributing to the onset of malignant disease many years, even decades, after the initial exposure. Therefore exposures that occur during any or all of the developmental phases of early life through adolescence may contribute to an elevated risk for cancer in later life.

Routes of Exposure

Carcinogens can be taken into the body by ingestion, inhalation, or dermal contact. Carcinogenic chemicals can be ingested through contaminated food, water, and even breast milk [23]. Ingestion is the most common pathway for exposure to POPs and heavy metals. Polycyclic aromatic hydrocarbons (PAHs), known to be carcinogenic, are in charred foods. Smoked and preserved meats contain nitrosamines, which are known to be carcinogenic. Although many carcinogens are synthetic, others are natural substances or metals. Arsenic, for example, tops the list of chemicals that concern the Agency for Toxic Substances and Disease Registry [24]. The major route of exposure to arsenic is drinking water. This exposure occurs because arsenic is in subsurface rock and soil and equilibrates with ground water. Dietary exposure to carcinogens depends on the type and quantity of foods consumed. In young children, hand-to-mouth behavior may result in significant ingestion of soil and dust containing carcinogenic substances [25]. Children and adolescents characteristically have different preferences for foods compared with adults [26] with children consuming more dairy products and adolescents consuming more fast foods [27]. Furthermore, teenagers generally eat greater quantities of food than adults. These factors often expose young people to greater intakes of chemical contaminants than is common with adults.

Inhalation is a major route of exposure to carcinogens in the air, including PAHs found in particulate air pollutants and tobacco smoke. Tobacco smoke also contains many other known

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