



# **Development of Pediatric Cancer and Agrochemicals**

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### Introduction

Cancer is the result of the combination between two dey variables, endogenous and exogenous genetic or environmental or. About 85% of childhood cancers are probably risk factors associated with the environment, with most of them unknown. The significant progress in childhood cancer survival, in contrast to the ignorance or little information of the risk factors involved in the pathogenesis and development of this disease. Cancer is a disease of multifactorial origin that develops after long latency periods. At all ages, neoplasms are the end result of the variable interaction of two determinants, genetic and environmental. In turn, each of these determinants is made of a lot of different carcinogenic risk factors, being presently most of them unknown. In addition, each risk factor can group various carcinogens, as with snuff smoke [1], which contains 55 different carcinogenic chemicals. Overall, environmental factors are responsible for 80% of all cancers and for some authors, 90% also developed during pediatric age.

Pediatric cancer, but only represents 0.4% of all cancer population between 0 and 19 years old, leads a strong personal, family and social impact. It is primarily due to three factors: a) For the general population cancer is considered one of the most reliable prototypes very serious and potentially fatal disease; b) the increased vulnerability and anatomical and physiological immaturity c) The alteration of the biological course set, skipping the generation (parents and grandparents) [2].

Children are especially vulnerable to carcinogenic agents for the following reasons. Often, they are exposed to higher tissue concentrations for their energy-metabolic characteristics inherent to its rapid growth and cell development, breathing more air, ingesting more food and drink more fluids, compared to adults. The physiological immaturity difficult cautions, detoxification and removal of most chemical and physical carcinogens, significantly increasing the amount of exposure times. And in certain places, located in rural areas with low income, children with nutritional deficiencies, exposure is increased by the limitations and dietary needs that increase their contact with environmental chemicals. The behavior patterns of children eating from the ground and hand-mouth activities also promote their greater exposure and enters their bodies. For all these reasons, the adoption of numerous measures of protection against pediatric environmental carcinogens is necessary, that every day is more common in the environment. Raise public awareness of these risks for children is vital. A very important issue that should be seriously addressed at future due to the increasing numbers of pediatric cancers [3].

The special pediatric vulnerability affects a greater willingness to initiate the process of oncogenesis, which mainly end and, therefore, will be diagnosed in adulthood. Latency most oncogenic agents is several decades to develop different types of cancers that develop in adults, whereas there is a pediatric cancers marked shortening, since 40% of neoplasms They occurs before 4 years of age, which in actuality has a growing increase in the incidence of different types of cancers and the development of cancers in earlier stages of life.

### Periods of Exposure to Oncogenesis

Most studies investigating cancer etiology in relation to a prenatal or perinatal exposure are often based on a small number of cases because pediatric cancer and many of the potential risk factors have a low prevalence population, unlike the adult whose statistic is much higher.

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#### **Development of Pediatric Cancer and Agrochemicals**

The appearance of different types of childhood cancer is related to different environmental as constitutional determinants [4]. It is conditioned by evolutionary vulnerable periods of development. The different stages of exposure or critical developmental periods correspond to different stages of oncogenesis, preconception (germ cells), conception (fertilization of the egg), transplacental (fetus) and postnatal (newborn and other pediatric period). The actions of carcinogens in any of the four evolutionary stages may develop a cancer, not only in children but also in adults, depending on the latency that occurs in cancer to develop in the future.

Most chemical carcinogens enter the body through the skin and the respiratory and digestive mucous. By the bloodstream they reach all tissues, bio transformed and eventually eliminated, mainly by intestinal and urinary tract. Carcinogens pervade and potentially affect all body cells, including germ. They can cause genetic alterations, which would act as predisposing factors favouring the appearance of tumors in their offspring. Preconceptions in oncogenesis, environmental carcinogens affect the germ cells, causing alterations in the fetus and increase the risk of tumors in their offspring. In the male, spermatogenesis begins at puberty and ends elderly and there throughout life, a long period of exposure to the various carcinogens. In contrast, the mother all oocytes are formed during fetal life without new stem cells after birth to form, determining a short period in which to act carcinogens, only during the intrauterine period. Therefore, when epidemiological studies try to link parental occupational exposure and pediatric tumors it is mainly taking the impact on parents and mothers where no positive data are obtained to develop statistical studies. Also when evaluating different exposures preconception, maternal grandmothers and mothers should be included. To make a more precise statistical cases of pediatric cancers [5].

The integration of one or two oncogenes in DNA, in the early stages of embryogenesis, affects the chromosomes of somatic and germ cells without altering their development, but this would produce increasing exponentially in tumor development in the future. In humans, for obvious reasons, the data for evidence of carcinogenicity prior to conception have had to be based on epidemiological studies based on the habits of parents and different exposures to carcinogenic environmental factors. There is much scientific evidence abalada in different articles, which are an increase in cancers in offspring of parents exposed to smoking, electromagnetic radiation, paints, hydrocarbons, nitrogen compounds derived from aromatic amines, inorganic combustion fumes, insecticides, pesticides and other today toxins found in the environment and ever increasing, due to the huge and growing environmental pollution.

#### **Development and Prevalence**

Under the vast amount of pesticides in the environment and the vast number of possible tissue "white" and end destinations that often differ depending on the stage of life when exposure occurs, leaving the need it becomes evident protective conditioning of all the scientific evidence of the safety of these substances based on the criteria of dangerousness as recommended by OMS. Functional deficits are not able to type "on" and "off" but cover a spectrum that goes from the inconsequential, it spends at very mild and goes to the very severe or totally debilitating. Consequently, it is difficult to quantify the degree of negative impact on neuro-development. Therefore, we face not only limitations on research techniques, but also to the intrinsic incompleteness of any scientific evidence to establish criteria for determining safety not include these findings. Because, if so, our regulatory approach should be much more rigorous to protect human and environmental health in the absence of full scientific certainty. Neither the current nor the proposed strategies protect public health or the environment. To locate pesticides in different ranges of danger WHO is based on the toxicity of the pesticide, as measured by the lethal dose 50 (LD50). This parameter is defined as a statistical value of the number of milligrams of the toxicant per kilo of weight required to kill 50 % of a large population exposed laboratory animals. Usually it expressed as a number, but in some cases may be a range. The LD50 in the case of pesticides, must be determined for the different routes of exposure (oral, dermal and respiratory) Meanwhile, several serious studies found that exposure to chemicals has been associated with increased risk of certain cancers among farmers and other agrochemical applicators. This has also been observed among families of rural workers and the general population living in agricultural areas, although specific exposures were not evaluated in most studies.

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Pesticide	Cáncer
Phenoxyacetic ACID (herbicides)	Non- Hodgkin lymphoma, soft tissue sarcoma, prostate carcinoma.
Organochlorine Insecticides	Leukemia, non- Hodgkin lymphoma, soft tissue sarcoma, pancreas, lung, breast Cancers.
Organophosphate Insecticides	Non- Hodgkin lymphoma,leukemia.
Insecticides Arsenicosos	Lung cancer and skin.
Triazine Herbicides	Ovarian cancer

 Table 1: Associations between different chemicals and different types of cancer.

## **Bibliography**

- 1. Hecht SS. "Tobacco smoke carcinogens and lung cancer". *Journal of the National Cancer Institute* 91.14 (1999): 1194-2210.
- 2. Quesnel S and Malkin D. "Genetic predisposition to cancer and familial cancer syndromes". *Pediatric Clinics of North America* 44.4 (1997): 791-808.
- Oxford Textbook of Public Health. 3<sup>rd</sup> ed. Oxford Textbook of Public Health. 3<sup>rd</sup>. New York: Oxford University Press (1997): 1043-1063.
- 4. National Childhood Cancer Registry. "ACCIS-Automated Childhood Cancer Information System".
- 5. Pesticide Outlook. "Royal Society of Chemistry". 1997

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