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EDITORIAL

Genetic factors and cancer: diagnosis, prognosis and future perspectives

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Genetics is specifically responsible for several pathologies or, at the least, it is associated with a wide range of them, either as a primary causal agent (congenital genetic diseases) or secondary, being a factor within several possible for a given disease.

One of the most critical genetic concepts is developed from the phenotype, equivalent to the genotype associated with the environment. In other words, for a condition to manifest itself, cancer, for example, we need a genetic alteration within the environment, which somehow influences carcinogenesis from stochastic or induced interactions.

Cancer cases are approximately 80% and 90% associated with external causes, and environmental changes are mainly motivated by human actions, habits, and behavior, leading to an increased risk of different types of cancer. These changes lead to the formation of a cycle since man promotes environmental changes, leading to genetic modifications responsible for 10-20% of cancer formation. Although the percentage seems not to be significant, we have, in fact, several genetic mechanisms that will lead to the emergence of the most diverse types of cancer, including polymorphisms, mutations, oxidative stress, oncogenes, and genes that regulate the cell cycle, including apoptosis[1,2].

Polymorphisms can be directly associated with environmental factors, which modify nucleotide sequences by single base polymorphisms (SNP), leading to protein formation and structural changes. Mutations, in turn, can be spontaneous (it will take hundreds of years to express a specific condition) or induced, resulting from changes in the environment, whether food, ultraviolet radiation, or hormones. Alternatively, several mechanisms have shown the role of oxidative stress in tumorigenesis, from the release of free radicals, which enzymes of the antioxidant system will not neutralize. Once again, we think that these enzymes may have reduced activity due to the environment. When verifying the existence of oncogenes, we ask ourselves: do we have a way to control them? The answer is challenging to say the least.

The E6/E7 oncogenes present in human papillomavirus (HPV) are fundamental in carcinogenesis and possible targets for therapy[3]. However, when thinking about HPV, a virus that has, within the Unified Health System, a vaccination coverage offered free of charge to girls from 9 to 14 years old and boys between 11 and 14 years old, in addition to patients between 9 and 45 years old with some conditions that affect the immune system, more recently, we must continue vaccinating the population and reducing the risk of developing cervical cancer from HPV infection.

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Oncogenes or the suppression of regulatory genes are a common pathway to many types of cancer. For example, the p53 gene (tumor suppressor) encodes a protein of the same name, whose function is to identify within a mitotic cell cycle checkpoint whether the conditions for replication are correct. If this event is not suitable and the cell cannot repair the error, it causes apoptosis to occur from the signaling of other pathways and genes. Mutations in the p53 gene modify the apoptosis signaling mechanism since the protein can be altered or become nonfunctional in the cell-checking mechanism and consequently favor skin carcinogenesis, leukemia, adenocarcinoma, among others[4].

Also, concerning genes that promote and help in the apoptosis mechanism, the BRCA family genes (1 and 2) play a vital role in the diagnosis and prognosis of breast and ovarian cancer, and mutations found in these genes may indicate a greater aggressiveness of the tumor, which is related to hereditary characters. In addition to breast cancer, mutations in these genes have been associated with greater aggressiveness in pancreatic and prostate cancer[5].

Furthermore, when thinking about the future of genetics, understanding the role of different micro RNAs (miRNAs) is of paramount importance, as they can regulate transcription, preventing and leading to changes in gene function. Think about whether this occurs in a cell cycle regulatory gene. Along with miRNAs, the roles of epigenetic alterations, whose regulatory mechanisms are linked to DNA methylation, histone modification, chromatin remodeling, and non-coding RNA, are commonly unregulated in cancer, with tumor suppressor genes silenced by hypermethylation of the DNA in its promoters[6].

The genetic-hereditary component is inevitable. We can try controlling the environment, but the result of the equation in cancer formation is still unknown. After all, each individual has unique genetic characteristics and interactions with the environment.

We must increasingly think about genetic mechanisms as a propensity factor for tumorigenesis and not forget that technological tools to improve the diagnosis and prognosis of our patients will be increasingly linked to molecular biology tools.

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