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Opinion Article

Metastasis and the Mechanism Involved in Progression of Cancer

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DESCRIPTION

Metastasis is the leading cause of mortality in cancer patients. Remarkable progress has been made in the past decades to understand molecular and cellular basis of this deadly process in cancer. Metastasis is the spread of cancer cells from one part to other parts of the body. In metastasis, cancer cells move away from the original (primary) tumor and circulate through the blood or lymph system, and form new tumors in other organs or tissues of the body. A new metastatic tumor is the same type of cancer as the primary tumor. For example, if breast cancer spreads to the lungs, the cancer cells in the lungs are breast cancer cells, not lung cancer cells.

Metastasis most commonly occurs when cancer cells leave the main tumor and invade the other organ. As the tumors increases they may have spread to nearby tissues and/or lymph nodes. These systems transport fluids throughout the body. In metastasis, cancer cells leave their original site of origin, travel through the blood or lymph system, and form new tumors in other parts of the body. Cancer can metastasize almost anywhere in the body. However, it usually moves to the bones, liver, or lungs. Metastasis is a general term used to describe the spread of cancer cells from a primary tumor to surrounding tissues and distant organs, and is a major cause of cancer morbidity and mortality. It is estimated that approximately 90% of cancer deaths are due to metastases.

The important processes involved in metastasis are cell adhesion, epithelial-mesenchymal junctions, vascular and lymphangiogenesis, role of cell junctions, and organ-specific metastasis. Cancer metastasis is the spread of cancer cells to tissues and organs and the formation of new tumors (secondary and tertiary lesions) is the only event leading to death in most cancer patients. At the time of cancer diagnosis, at least half of the patients already have clinically detectable metastatic disease.

Cancer cells are driven by genetic and epigenetic alterations within the tumor cell itself and its microenvironment.

Determining the biological mechanisms of the metastatic process it is critical for finding an open therapeutic methods for successful intervention.

Advances in cancer biology research and the emergence of new paradigms in the study of metastasis have revealed some of the molecular biology of this dissemination process. Invading tumor cells interact with other proteins and cells on their way to their target site. Cancer cell proliferation precedes the first step of the invasion-metastasis. This results in the chromosomal instability caused by continuous errors due to various genes involved in the chromosomal segregation during mitosis. Errors in chromosomal segregation lead to micronucleus rupture and secretion of genomic DNA into the cytosol, followed by activation of the cytosolic DNA-sensing pathway (cyclic GMP-AMP synthase-stimulating factor of the Interferon (IFN) gene) and downstream nuclear factors). Kappa activates the light. There is no doubt that human carcinogenesis is a dynamic process dependent on numerous variables and regulated at multiple spatial and temporal levels. However, viewing cancer as a dynamically complex system may reveal more about the underlying behavioral properties. It is encouraging that mathematicians, biologists and clinicians are working together to continue contributing to a common quantitative understanding of the complexity of cancer. This idea further helps to clarify concepts, interpret experimental data from old and new, point to alternative experiments, and classify knowledge gained based on similarities of very different tumors. Cancer is now recognized as a highly heterogeneous disease. Over 100 different types of human cancer have been identified, with different tumor subtypes found in specific organs. Furthermore, these tumors exhibit somatic mutations in a person. Epigenetic alterations of tumors are specific to individual neo plasms. It is now recognized that this genetic and phenotypic diversity largely determines self-progressive growth, invasiveness, tumor formation and metastatic potential of neoplastic diseases as well as their response or resistance to therapy, the complexity of cancer can explained histologically by using neoplasms similar to clinical diversity.

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