



Implications of genomic studies in highly precise cancer pharmacy drug

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DESCRIPTION

Cancer is a series of diseases caused by mutations in DNA that induce unregulated cell proliferation and malignancy. DNA mutations, rearrangements, deletions, amplification, and the addition or removal of chemical makers are all examples of abnormalities. These modifications can lead cells to create excessive amounts of certain proteins or produce malformed proteins that do not function properly. A combination of many genetic changes frequently works together to induce cancer. Gene changes can be passed down from one's parents, influenced by environmental circumstances, or occur naturally during cell division. Acquired or somatic alterations are the changes that occur during a lifetime and account for 90–95 percent of all cancer cases. Scientists can learn more about the molecular foundation of cancer growth, metastasis, and medication resistance once cancer-causing alterations have been identified. Clinical data on how patients responded to cancer treatment, laboratory trials using cell lines and model organisms, and big data analytic techniques are all used to do this. Bringing together massive genomic datasets and sharing them with researchers all around the world is becoming a more significant technique for cancer research, as it increases the data's potential and offers up new avenues for discovery. Scientists at the National Institutes of Health (NIH) and around the world are working hard to uncover the genetic abnormalities that underpin cancer, understand their roles in tumor development and spread, and use their findings to combat the disease.

Precision medicine is a strategy of using genomic information to improve cancer diagnosis and treatment options that are personalised to the tumors of individuals. As a result of research into the genomic changes linked to cancer, drugs have been developed to combat the disease in a variety of ways, including inhibiting enzymes that cause cancer cells to grow and survive abnormally,

blocking aberrant gene expression in cancer cells, and halting overactive molecular signalling pathways in cancer cells. These targeted medicines are formulated to overcome the differences between cancer cells and normal body cells. This makes them less harmful to patients than other treatments like chemotherapy and radiation, which can damage healthy cells. One example of precision medicine in clinical use is Imatinib (Gleevec), which inhibits overactivity of a protein (named Bcr-Abl tyrosine kinase) in individuals whose leukaemia is caused by a specific chromosomal translocation. Trastuzumab (Herceptin), on the other hand, inhibits a hyperactive signalling pathway (HER2 tyrosine kinase) in a subtype of breast cancer caused by numerous copies of the HER2 gene. Finally, the drugs erlotinib (Tarceva) and gefitinib (Iressa) inhibit the activation of a protein called EGFR, which is abnormally active in a subset of lung malignancies due to mutations in the protein.

Cancer genomics study further aids precision medicine by identifying cancer types and subtypes based on genetic characteristics. This cancer molecular taxonomy can help patients get a more exact diagnosis and, as a result, a more tailored treatment plan. Some researchers discovered various ways in which the molecular definition of cancer currently benefits patients. Breast cancer is divided into four categories based on molecular characteristics: Luminal A, Luminal B, Triple-negative/basal-like, and HER2 type. These subgroups differ in their aggressiveness and treatment response. Patients with breast cancer may benefit from a diagnosis and treatment plan based on their tumor's molecular subtype. Genomic screening can separate diffuse large B cell lymphoma into the ABC and GCB subtypes, finding patients who respond differently to existing chemotherapy regimens and molecularly targeted treatments. The Cancer Genome Atlas research discovered four subtypes of endometrial cancer that associated with patient survival in 2013 which included POLE ultramutated, microsatellite instability

(MSI) hypermutated, copy-number (CN) low, and CN high. This finding has already sparked additional clinical trials to see how these subtypes can improve endometrial cancer treatment in the future. Patients with lung cancer

who have a gene fusion including the *ROS1* gene often respond effectively to crizotinib, a targeted therapy. The condition is best defined and treated in these patients because of the specific genetic mutation.