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Editor Note

Editorial Note on Organ Genomics

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EDITORIAL

Genomics is a general term that refers to the study of organisms' genomes. However, depending on the molecular levels or aspects, it can take several different forms: variomics focuses on sequence or structural changes, epigenomics focuses on DNA or histone modifications, cistromics focuses on cis-elements for transcriptional control, transcriptomics focuses on transcribed genomic regions, and interactomics focuses on three-dimensional chromosomal interactions. Cancer has become the "model disease" in medical science, attracting all of the attention of genomicists, particularly with the recent development of high-throughput sequencing technologies.

The International Cancer Genome Consortium (ICGC) was established in 2007 to bring together a number of collaborative efforts to research over 25,000 cancer genomes at the genomic, epigenomic, and transcriptomic levels . The Encyclopaedia of DNA Elements (ENCODE) is a public research consortium that seeks to classify all functional elements in the human genome using a range of cell types, including cancer cell lines.

Methods for analysing chromosomal interactions, such as ChIA-PET and Hi-C , have recently been combined with high throughput sequencing to analyse the interactomes of cancer cells. Despite the fact that cancer is the leading cause of death worldwide and therefore in the glare of medicine, it is an exceptional case of “gain-of-function” disease, as opposed to the majority of major organ-related diseases, which are “loss-of-function.” Mutations in the genomes of cells occur over time as a result of exposure to mutagens or replication errors.

Some of these mutations can up-regulate growth genes or down-regulate growth suppressor genes, giving cancerous cells an advantage to expand and migrate . Nonetheless, some of these mutations can trigger apoptosis or cause loss-of-function in functional genes. Mice with a CuZn superoxide dismutase deficiency are one example (CuZnSOD). Widespread oxidative damage and mutation accumulation were observed in the liver of mice with a high oxygen metabolism and vulnerability to oxidative damage when CuZnSOD was not present to superoxide radicals produced in the cytoplasm and nucleus. As a result, these mice's liver cells exhibit increase shepatocarcinogenesis and apoptosis, as well as a shorter lifespan.

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