SHORT COMMUNICATION

Transmitted Landscape of Oncogenesis, and DNA Repair

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DESCRIPTION

Genes called proto-oncogenes typically support cell growth and division to create new cells or to support cell survival. When a proto-oncogene mutates (changes) or has too many copies, it can be accidentally turned on (activated), at which time it is now referred to as an oncogene. The cell may then begin to expand uncontrollably, which might result in cancer. Normal proto-oncogene activity is comparable to how a car's gas pedal works. It aids in the cell's division and growth. An oncogene is comparable to a gas pedal that is jammed down, causing the cell to divide uncontrollably [1]. Oncogenes can be triggered (turned on) in cells in a variety of ways. For instance:

Genetic variations

Some individuals have variations in the 'code' of their genes, which might result in an oncogene that is always activated. These kinds of gene alterations may be inherited from a parent or may develop spontaneously over a person's lifetime as a result of an error in the gene copying process during cell division.

Epigenetic alterations

Genes may often be turned on or off by cells without causing changes to the genes themselves. Instead, genetic material (DNA or RNA) can have several chemical groups attached that influence whether a gene is switched on. These kinds of epigenetic alterations might occasionally cause the activation of an oncogene [2].

Chromosomal rearrangements

Each cell has long strands of DNA called chromosomes that encode its genes. A chromosome's DNA sequence can occasionally alter when a cell is dividing. By placing a gene that acts as a kind of "on" switch next to a proto-oncogene, this may keep the gene active even when it shouldn't. The cell's uncontrolled growth may be caused by this new oncogene.

Gene duplication

When a gene is duplicated in some cells, this might cause those cells to produce an excessive amount of a particular protein.

A hereditary alteration in an oncogene is connected to a limited subset of familial cancer disorders. These kinds of alterations can occasionally be the first stage in a cell's transformation into a cancer cell. However, rather than being inherited, the majority of oncogene-related alterations happen within a person's lifespan. A cell must create a fresh copy of its whole DNA when it divides to create new cells. This is a difficult procedure, and occasionally it leads to DNA errors [3].

The DNA repair genes behave like a mechanic fixing a car. They assist in correcting DNA errors, or if they are unable to do so, they induce the cell to die so that the errors cannot continue to create issues. These DNA repair genes can malfunction, which can lead to the accumulation of further errors inside the cell. Some of these might influence other genes, which would cause the cell to proliferate uncontrollably. Modifications in DNA repair genes can either be inherited from a parent or acquired over a person's lifespan, similar to other forms of gene changes.

The *BRCA1* and *BRCA2* genes are two examples of DNA repair genes. A pathogenic variation (mutation) in one of these genes increases the chance of developing certain cancers, especially breast and ovarian cancer in females. However, even in tumour cells from individuals who did not inherit one of these mutations, alterations in these genes can occasionally be observed [4].

CONCLUSION

A tumour suppressor gene generally aids in preventing the cell from proliferating too quickly. Cell division can spiral out of control when something goes wrong with a tumour suppressor gene, such a pathogenic version (mutation) that prevents it from functioning. Some familial cancer syndromes have been identified to have inherited alterations in tumour suppressor genes. They are the reason why some cancers run in families. However, rather of being inherited, the majority of tumour suppressor gene mutations occur throughout a person's lifetime.

REFERENCES

1. Zaĭchik V, Matveenko EG, Vtiurin BM, et al. Intrathyroid iodine in the diagnosis of thyroid cancer. Vopr Onkol 1982;28(3):18-24.
2. Zaichick V, Tsyb AF, Vtyurin BM. Trace elements and thyroid cancer. Analyst.1995;120(3):817-821.
3. Neĭmark II, Timoshnikov VM. Development of thyroid cancer in persons living in the endemic goiter area. Probl Endocrinol.1978;24(3):28-32.
4. Al-Sayer H, Mathew TC, Asfar S, et al. Serum changes in trace elements during thyroid cancers. Mol. Cell. Biochem. 2004;260(1):1-5.

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