

An Overview of Cancer Biology

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Editorial

Cancer is a disease of uncontrolled growth and proliferation in which cells have developed the ability to divide endlessly after escaping the body's regular growth control mechanisms. It's a multi-step procedure that necessitates the accumulation of several genetic alterations over a period of time. The activation of proto-oncogenes to oncogenes, dysregulation of tumour suppressor and DNA repair genes, and 'immortalization,' which will be covered in this chapter, are examples of genetic modifications.

The relevance of apoptosis and cell cycle regulation

Proliferation and advancement through the cell cycle are tightly controlled in normal cells by groups of proteins that interact with one another in a certain order [1]. Checkpoints ensure that each stage of the cell cycle is appropriately completed and that incompletely duplicated DNA is not passed on to daughter cells. Cyclin-dependent kinases are at the heart of this regulatory system (CDKs). CDKs are master protein kinases that phosphorylate and activate other downstream kinases to drive cell cycle progression through distinct phases. The presence of activating subunits called cyclins, which are synthesised and destroyed in a cell cycle-dependent manner, is required for CDK function. CDK inhibitors regulate cyclin-CDK complexes even more tightly [2].

Tumorigenesis and cell immortalization

The gaining of an infinite lifespan is known as immortality. Normal mammalian somatic cells replicate only a few times before senescence sets in. Even if they have stopped proliferating, senescent cells can still be metabolically active. The presence of telomerase, the enzyme responsible for preserving telomeres at the ends of chromosomes, is thought to play a role in immortalisation, which is an important step in the malignant transformation of normal cells [3]. Telomerase works to prevent telomere shortening, which would otherwise result in cell death, by lengthening telomeric DNA. Unlike normal cells, which do not have measurable levels of telomerase activity, roughly 90% of human tumour cells have an active telomerase enzyme.

Growth factors and receptors are two types of growth factors

Growth factors (GFs) serve a crucial physiological role in the normal process of tissue homeostasis and growth control. They send and receive growth signals from one cell to the next. Specific growth factor receptors detect these signals on the cell surface (GFRs) [4-6]. GFRs transmit the growth signal to target molecules that stimulate proliferation via signalling pathways.

Cell signalling's other components

Consistent stimulation of internal signalling components is an alternate way for cancer cells to become GF independent [7-9]. In normal cells, for example, the Ras protein is turned off and does not signal unless a GFR is activated, which then activates the Ras protein, changing it from a dormant state to an active, signal-emitting state through a series of intermediaries.

Following that, the Ras protein is able to release additional downstream signals that can induce proliferation. Because structurally altered Ras proteins can constantly deliver growth stimulatory signals into the inside of the cell in the absence of GFs, this signalling pathway is unregulated in cancer cells.

Oncogenes in cells

Oncogenes are genes that encourage autonomous cell proliferation in cancer cells, while proto-oncogenes are their normal cellular counterparts. Oncogenes have the ability to increase cell growth in the absence of normal mitogenic signals, whereas proto-oncogenes are physiologic regulators of cell proliferation and differentiation [10].

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