Cancer: Cells out of Control

AMI #4 ANATOMY

Normal cells do not live forever. Under certain circumstances, cells are programed to die, particularly during development. Cells that become damaged beyond repair will normally undergo this programed cell death (called **apoptosis** or **cell suicide**). Cancer cells evade this control and become immortal, continuing to divide regardless of any damage incurred. **Carcinogens** are agents capable of causing cancer. Roughly 90% of carcinogens are also mutagens, i.e. they damage DNA. Chronic exposure to carcinogens accelerates the rate at which dividing cells make errors. Susceptibility to cancer is also influenced by genetic make-up. Any one or a number of cancer-causing factors (including defective genes) may interact to induce cancer.

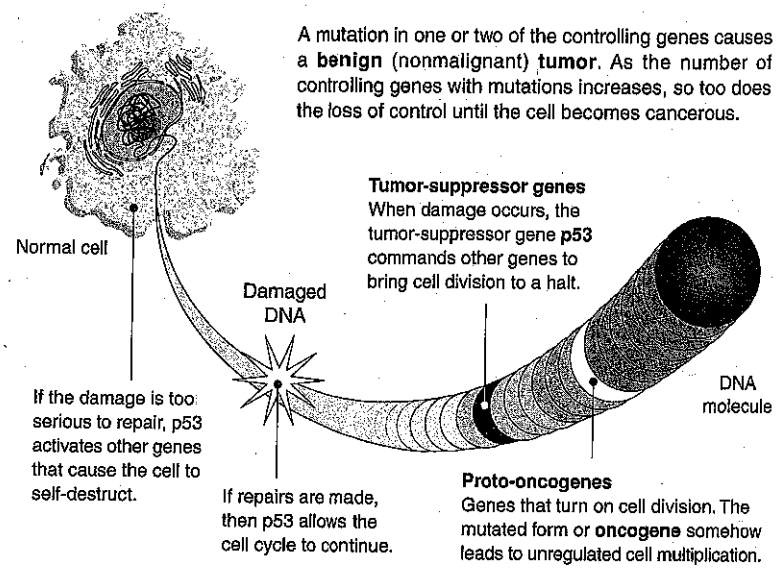
Cancer: Cells out of Control

Cancerous transformation results from changes in the genes controlling normal cell growth and division. The resulting cells become immortal and no longer carry out their functional role. Two types of gene are normally involved in controlling the cell cycle: **proto-oncogenes**, which start the cell division process and are essential for normal cell development, and **tumor-suppressor** genes, which switch off cell division. In their normal form, both kinds of genes work as a team, enabling the body to perform vital tasks such as repairing defective cells and replacing dead ones. However mutations in these genes can disrupt these checks and balances. Proto-oncogenes, through mutation, can give rise to **oncogenes**; genes that lead to uncontrollable cell division. Mutations to tumor-suppressor genes initiate most human cancers. The best studied tumor-suppressor gene is **p53**, which encodes a protein that halts the cell cycle so that DNA can be repaired before division.

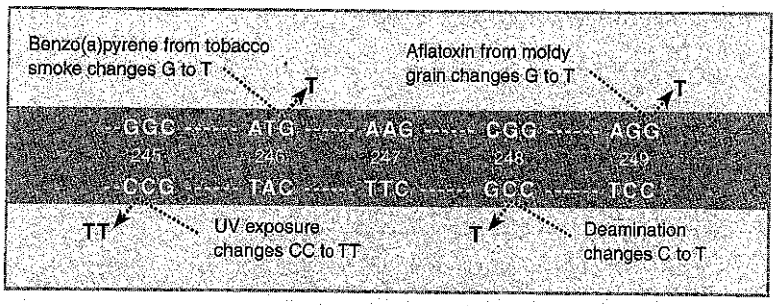
The panel, right, shows the mutagenic action of some selected carcinogens on four of five codons of the **p53 gene**.

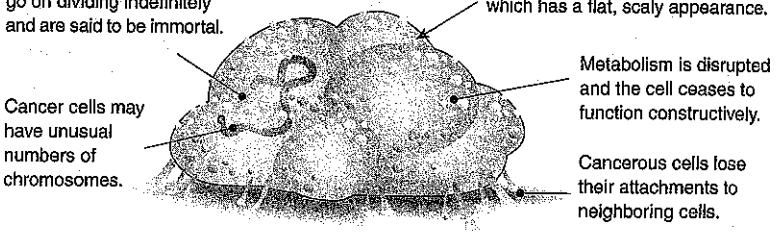
Features of Cancer Cells

The diagram right shows a single **lung cell** that has become cancerous. It no longer carries out the role of a lung cell, and instead takes on a parasitic lifestyle, taking from the body what it needs in the way of nutrients and contributing nothing in return. The rate of cell division is greater than in normal cells in the same tissue because there is no *resting phase* between divisions.



A mutation in one or two of the controlling genes causes a **benign** (nonmalignant) **tumor**. As the number of controlling genes with mutations increases, so too does the loss of control until the cell becomes cancerous.





1. Explain how cancerous cells differ from normal cells: _____

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2. Explain how the cell cycle is normally controlled, including reference to the role of **tumor-suppressor genes**:

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3. With reference to the role of **oncogenes**, explain how the normal controls over the cell cycle can be lost:

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