Text of Animation Videos

Resources for Understanding Cancer

Resource One

The rate and timing of cell division in your body normally are very precisely regulated. Cells are formed, mature, and eventually die.

As this happens, new cells divide, creating replacement cells. Chemical messengers that pass between neighboring cells help keep the rate of cell division equal to the rate of cell death.

Sometimes, a cell breaks free from its normal restraints and begins to follow its own pattern of cell division. This precancerous cell divides more often than normal, eventually producing a mass of cells that also divide more often.

Further changes in these cells can increase the frequency of cell division even more, until eventually a cancerous tumor develops.

At this point, the tumor grows large, but is confined to the tissue where it originated. Late in the development of cancer, some cells may gain the ability to move into blood vessels and travel to other parts of the body.

Resource Two

For many years, it was a mystery to scientists how cells controlled their cell division. Scientists now know that the chemical messages that cells receive from neighboring cells affect a complicated group of molecules in the cell. These molecules are called the "cell cycle clock."

The cell cycle clock integrates the mixture of signals the cell receives from its neighbors and determines whether the cell should move through each stage of growth and division. If the answer is "yes," the cell grows and divides.

The cell cycle is composed of four phases. In the G_1, or Gap 1, phase, the cell increases in size and prepares to copy its DNA.

Once all the necessary molecules are made, the clock moves the cell to the S phase, called S for "synthesis." This is when the cell copies its DNA.

After the DNA is copied, a second gap period, called G_2, occurs, and then the cell divides. The phase in which the cell divides is called M, for mitosis.

The new daughter cells immediately enter G_1. Depending on the signals they receive from neighboring cells and the decisions their cell cycle clocks make, they may go through the cell cycle again or stop cycling temporarily or permanently. Thus, in normal tissues, cell growth and division is precisely controlled by internal clocks.

Resource Three

Two types of genes play a major role in regulating the cell cycle. Genes called proto-oncogenes encourage cell division. Proteins produced by these genes act like accelerators, stimulating the cell to grow and divide.

Anim. 2

In contrast, genes called tumor suppressor genes inhibit cell division. Proteins produced by these genes act like brakes to slow down or stop cell division.

The balance between the activities of proto-oncogenes and tumor suppressor genes keeps normal cells dividing at a rate that is appropriate for their position and role in the body.

Resource Four

An important milestone in scientists' efforts to understand cancer came in the 1970s when it was shown that many cancer-causing agents also are able to cause changes in DNA that we call mutations.

In fact, research showed that in many cases, chemicals that are powerful cancer-causing agents also are powerful mutagens. Mutagens are agents that produce mutations. This is shown here on a graph that compares the ability of several chemicals to cause cancer with their ability to cause mutations.

In contrast, chemicals that had only a weak ability to stimulate the development of cancer were only weak mutagens.

We now know that some cancer-causing agents do not fit this simple pattern. But the fact that many cancer-causing agents also cause mutations gave scientists an important clue about what might cause cells to become cancerous.

Resource Five

Normal cell division in the body depends on a precisely regulated set of events that determine when a cell will divide and when it will not divide. Two types of genes, called proto-oncogenes and tumor suppressor genes, are primarily responsible for this regulation.

When mutated, however, proto-oncogenes can become what scientists call "oncogenes," genes that stimulate excessive division. This situation is similar to getting a car's accelerator stuck in the downward position: A cell that experiences such mutations tends to divide more frequently than it normally would.

In contrast, mutated tumor suppressor genes can become inactive. A cell that experiences a mutation in a tumor suppressor gene loses some of its crucial braking power. Again, the result is a tendency for the cell to divide more frequently than it normally would.

For a cancerous tumor to develop, mutations must occur in several of a cell's division-controlling genes. These mutations disturb the balance that normally exists between signals that stimulate cell division and signals that inhibit cell division. The result is uncontrolled division.